

Attachment C – 11/13/2013 APC Public Comments Letters

The following public comment letters were received at the November 13, 2103 Advisory Planning Commission Meeting.



Lake Tahoe
Visitors Authority

November 1, 2013

Tahoe Regional Planning Agency
Governing Board
P.O. Box 5310
Stateline, NV 89449

Dear TRPA Governing Board:

At its October 10th meeting, the Lake Tahoe Visitors Authority (LTVA) Board of Directors discussed the merits of the South Lake Tahoe Tourist Core Area Plan. The unanimous decision of the board was in support of the plan and this letter is submitted to the Tahoe Regional Planning Agency Governing Board to encourage its approval.

The Tourist Core Area Plan will help the LTVA to better promote Lake Tahoe's South Shore and increase visitation. The plan outlines strategies to help connect visitors to recreation opportunities, a tactic that would dramatically improve the overall visitor experience. Additionally, the plan encourages redevelopment and improved transportation options, both essential elements to turning Tahoe South into a world class destination.

The LTVA Board of Directors commends the TRPA Governing Board for its recent approval of the Douglas County Area Plan. The approval of the Tourist Core Area Plan is the necessary next step to implement the Regional Area Plan. Thank you for your consideration in this matter.

Sincerely,

Patrick Ronan
Lakeshore Lodge & Spa

Jerry Bindel
Aston Lakeland Village

Tom Davis
City of South Lake Tahoe

Tamara Hollingsworth
Tahoe Beach & Ski Club

John Koster
Harrah's/Harveys

Nancy McDermid
Douglas County

Pete Sonntag
Heavenly Mountain Resort

California Location: 3066 Lake Tahoe Boulevard South Lake Tahoe, CA 96150 530-544-5050 phone 530-541-7121 fax
Nevada Location: 169 Highway 50 / P.O. Box 5878 Stateline, NV 89449-5878 775-588-5900 phone 775-588-1941 fax

TahoeSouth.com



Tahoe Douglas Visitors Authority

November 1, 2013

Tahoe Regional Planning Agency
Governing Board
P.O. Box 5310
Stateline, NV 89449

Dear TRPA Governing Board:

The Tahoe Douglas Visitors Authority (TDVA) Board of Directors adds its support of the South Lake Tahoe Tourist Core Area Plan. The approval of this plan is the necessary complement to the recently approved Douglas County Area Plan. Together, the two plans will help ensure a successful implementation of the Regional Plan.

The Tourist Core Area Plan offers significant benefits to locals and visitors alike. The plan encourages sustainable redevelopment opportunities, allowing for environmental threshold gains and improved transportation options. The plan encourages pedestrian focused activities thereby reducing vehicular miles travelled and improved visitor enjoyment of the area. Additionally, the plan will preserve neighborhood character while allowing for much needed improvements including the retirement of a significant amount of existing coverage.

Our community's future prosperity is dependent upon the approval of this plan and we strongly believe that it is in the best interest of visitors and locals alike. Environmentally and economically, it is the progressive step necessary to guarantee the integrated efforts to build the South Shore into a remarkable outdoor recreational destination are successful. The TDVA Board of Directors strongly urges the TRPA Governing Board to approve the South Lake Tahoe Tourist Core Area Plan.

Sincerely,

John Packer
Harrah's/Harveys

Mike Bradford
Lakeside Inn & Casino

Bryan Davis
Edgewood Tahoe

Nancy McDermid
Douglas County

Xenia Wunderlich
Harrah's/Harveys



TahoeChamber.org

Tahoe Regional Planning Agency
Board of Governors
P.O. Box 5310
Stateline, NV 89449

November 11, 2013

Re: Tourist Core Area Plan

The Board of Directors for the Lake Tahoe South Shore Chamber of Commerce (TahoeChamber) supports the approval of the South Lake Tahoe Tourist Core Area Plan. It is the necessary subsequent step to see through the implementation of the Regional Plan.

The TahoeChamber Board supported the concept of Area Plans during the TRPA's regional plan update process in the belief that the Area Plan with its reduction of duplicative permitting processes, will assist in stimulating some reinvestment of the built environment. Without this reinvestment our community will continue to lag and decline in appearance, profitability and environmental stewardship. In other words – doing nothing is not an option. The tourist core area is one of concentrated development and therefore has the greatest potential for environmental and scenic improvements through the enhanced design standards.

With smart planning as that envisioned under the plan, the tourist core area will better connect visitors to the world-class recreation opportunities that our community boasts. Enriching visitor experiences through improvements in both the built and natural environments is critical to the future prosperity of the region as whole. We encourage the TRPA Board of Governors to consider the overwhelming merits of the Tourist Core Area Plan which warrant its approval.

Sincerely,

Betty "B" Gorman, A.C.E.
President & CEO

Tamara Hollingsworth
Chair of the Board



Tahoe Regional Planning Agency
PO Box 5310
Stateline, NV 89449

Date: November 12, 2013
To: Tahoe Regional Planning Agency Advisory Planning Commission
From: The League to Save Lake Tahoe

Re: Findings for the Tourist Core Area Plan

Dear Members of the Advisory Planning Commission,

The League to Save Lake Tahoe (the League) appreciates the opportunities we have had throughout the planning stages of the Tourist Core Area Plan (TCAP). The League commends the work of both the City of South Lake Tahoe and Tahoe Regional Planning Agency (TRPA) staff. The League supports the Area Plan and all of its policies, but has comments regarding the associated TRPA Findings. The following comments address the Chapter 13 Findings within Attachment I – Motions, Findings, & Ordinance of the TRPA packet for the Advisory Planning Commission Meeting November 13, 2013.

TRPA is required to make findings for any amendment to the Regional Plan Update (RPU) to demonstrate how thresholds will be achieved and maintained. These are outlined in the Chapter 4 Findings for the TCAP and specify the policies NCR-2.1 and NCR-4.1.¹ These two policies were vetted throughout the planning stages of the TCAP and discussed at length at the Regional Plan Implementation Committee (RPIC) on October 24, 2013. They were ultimately updated to include more specific language on how the TCAP will achieve Sensitive Environment Zone (SEZ) restoration and reduction in coverage.² These specific policies should be called out in the Chapter 13 Findings for the TCAP. Chapter 13 of TRPA Code of Ordinances requires that Area Plans demonstrate how they are in conformance to the RPU. The RPU is based on incentives to encourage SEZ restoration and coverage reduction. It includes many goals and policies on how this will be achieved. Policies NCR-2.1 and NCR-4.1 in the TCAP help demonstrate how the TCAP is in conformance to the RPU and will be critical tools during the TCAP annual review. There are other SEZ and coverage policy cited in the Chapter 13 Findings. For consistency and adequacy all SEZ and coverage policy should be cited in Chapter 13 Findings. Policy NCR-2.1 should be included under the Chapter 13 Finding to “Protect and direct development away from the Stream Environment Zones and other sensitive areas, while seeking opportunities for environmental improvements within sensitive areas. Development may be allowed in Disturbed Stream Environmental Zones within Centers only if allowed development reduces coverage and enhances natural systems within the Stream Environment Zone.”³ Policy NCR-4.1 should be included under the Chapter 13 Finding to “Identify an integrated community

¹ APC 11/13/13 Packet, Attachment I, Chapter 4 Findings, 4.C (Soil Conservation), p 14 of Attachment I.

² APC 11/13/13 Packet, Attachment F- TCAP Modifications, p. 2 of Attachment F.

³ APC 11/13/13 Packet, Attachment I, Chapter 13 Findings, 1.A.7. p. 21 of Attachment I.

strategy for coverage reduction and enhanced stormwater management.⁴ Again, the policies not only demonstrate how the TCAP is in conformance to the RPU, but will be used in identifying how the TCAP is achieving RPU goals during the annual review.

Sincerely,

Shannon Eckmeyer

Policy Analyst

League to Save Lake Tahoe

⁴ APC 11/13/13 Packet, Attachment I, Chapter 13 Findings 1.B.5. p.23 of Attachment I



TRPA Advisory Planning Commission
128 Market St.
Stateline, NV 89449

November 12, 2013

Subject: Comments on City of South Lake Tahoe proposed Tourist Core Area Plan

Dear Members of the Advisory Planning Commission and TRPA staff:

The Friends of the West Shore (FOWS) and the Tahoe Area Sierra Club (TASC) appreciate the opportunity to provide additional comments on the proposed City of South Lake Tahoe (City) Tourist Core Area Plan (TCAP), and all related documents. As our collection of previous comments on the TCAP show, we have been extremely diligent providing comments, technical references, and recommendations to TRPA and City staff, the RPIC, TRPA APC, TRPA GB, SLT City Council, and SLT Planning Commission. Most comments and attachments have been included in GB packets.¹ The responses provided by staff in the “Attachment E, Response to Comments,” unfortunately still fail to address many of our concerns and questions, and with great disappointment, fail to respond to most of the technical information we provided from several of Tahoe’s most reputable scientific institutions (e.g. TERC, TSC, DRI) regarding matters which affect not only water quality, but public health and safety.

In order to achieve and maintain TRPA’s thresholds, and to protect public health and property, **we request the APC recommends TRPA staff to take the following actions, discussed in greater detail below, and recommends these same revisions be made in the relevant sections of the City’s TCAP.² Additional recommendations are included in the attached comments, including the incorporation of new nearshore information.** Our recommendations for TRPA and the City’s TCAP include:

- 1) Update Chapter 35: Natural Hazard Standards to address current flood hazards
- 2) Revise Code to remove variances that will allow degradation of the natural scenic quality
- 3) Evaluate the impacts of the off-road mobile sources of ozone precursors and adopt measures to reduce emissions to obtain the public health ozone standard *in the immediate future* (ozone standards are not long term standards such as mid-lake clarity);

The information available from the RPU/RTP EIR/S and EIS, and the TCAP’s environmental documentation is inadequate and as a result, TRPA’s threshold-related findings can not be made. Further, conforming to the RPU, which itself lacks adequate environmental review, does not ensure that public health and safety or environmental standards, will be achieved and maintained as required by the Compact. Please feel free to contact Jennifer Quashnick at jqtahoe@sbcglobal.net or Laurel Ames at laurel@watershednetwork.org if you have any questions.

Sincerely,

Laurel Ames,
Conservation Co-Chair,
Tahoe Area Sierra Club

Susan Gearhart,
President,
Friends of the West Shore

Jennifer Quashnick
Conservation Consultant

¹ See 10/21/2013 Comments, included in Attachment D: Public comments.

11/12/13 Attachments (links provided for easy reference and due to large file size):

- 2012 TRPA BMP Handbook (available at: <http://tahoebmp.org/BMPHandbook.aspx>)
 - 2NDNATURE, 2006, *Tahoe Basin BMP Monitoring Evaluation Process: Synthesis of Existing Research* prepared for USFS Tahoe Basin Management Unit.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/09/FinalReport_BMPSynthesis.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants, 2010, *Focused Stormwater Monitoring to Validate Water Quality Source Control and Treatment Assumptions* prepared for US Army Corps of Engineers, Sacramento District.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/09/FinalPhaseI_PLRM-Refinement_TechnicalReport.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Tahoe Stormwater and BMP Performance Database Monitoring and Reporting Guidance Document – Final and Appendices*. 2010
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/07/TahoeBMPDB_FinalGuidance_2010.jpg
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/09/TahoeBMPDB_FinalGuidance_Appendices.pdf
 - 2NDNATURE and Environmental Incentives, LLC. *Lake Tahoe Climate Change Science Synthesis, Aquatic Resources*. 2010.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/09/Global-Climate-Change_Science-Synthesis_2010.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Synthesis of Existing Information: Infiltration BMP Design & Maintenance Study* (for) the Tahoe Regional Planning Agency. 2011.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2011/09/BMPSynthesisFinal_reduced.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Pilot Catchment Validation Study*. 2012.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2012/06/PilotCatchmentValidationStudy_2N2012.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Focused Stormwater Quality Monitoring to Inform Assumptions and Evaluate Predictive Capabilities of Existing Tools*. 2012.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2012/06/FocusedStormwaterQualityResearch_2N2012.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Infiltration BMP Design and Maintenance Study: Final Report*. 2013.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2010/09/BMPAssessment_FINALREPORT.pdf
 - 2NDNATURE and Northwest Hydraulic Consultants. *Quantification + Characterization of Trout Creek Restoration Effectiveness and Stream Load Reduction Tool (SLRTv1) Methodology User Guidance and Beta Spreadsheet Tool*. 2013.
 - o http://www.2ndnaturellc.com/wp-content/uploads/2013/07/SLRTFinalReport_July2013_web.pdf
- Lake Tahoe Nearshore Evaluation and Monitoring Framework: <http://www.dri.edu/cwes>
- 10/23/13 Presentation to TRPA (attached and available online)
 - 10/23/13 DRI Press Release (attached and available online)
 - Executive Summary (attached and available online)
 - Full Report (not attached due to file size; available online)

Comments regarding the 11/13/2013 APC Staff Summary and Packet:

Attachment C: CEQA-IS-NEG-DEC TRPA-IEC-FONSE: IEC

Our previous comments to the TRPA, APC, RPIC, City Planning Commission, City Council, and TRPA and City staff include many comments and concerns regarding the failure of the IEC, and the RPU it tiers from, to adequately analyze the environmental impacts of the TCAP. We refer to our previous comments on TRPA's RPU EIS (provided exhaustively to TRPA and attached to our 10/24/13 Comments to the RPIC and previous letters as well), and to our comments regarding the TCAP's environmental analysis, which begin with our 8/28/13 comments to TRPA and the City of SLT regarding the IS/ND/IEC for the TCAP, and as addressed throughout all subsequent comments regarding the TCAP up to and including our 10/21/13 comments to the TRPA RPIC (the 10/21/13 Attachments include an extensive outline of our previous comments as well as the location of each comment letter).

In summary, there is a significant failure to analyze the impacts of the TCAP on TRPA's environmental thresholds, as well as applicable federal and state standards (including air quality), plus to consider the most recent science regarding significant threats to public health from numerous natural hazards (e.g. flooding, tsunamis, earthquakes).

FOWS and TASC request these impacts be adequately analyzed.

Attachments D and E: Public Comments and Responses

Staff's response to our 10/3/2013 comments to the RPIC overlooked numerous significant comments and questions we'd provided in our letter relating to public health and safety and human health, to note a few. We reiterated these issues in the 10/21/13 comments to the RPIC, which is included in "Attachment D: Public Comments" (pages 233-257) in the APC packet. We note the following additional technical documents attached to our 10/21/13 letter are not included with the comment letter in Attachment D:

New attachments (10/21/13):

- USGS 5.23.12 LiDAR LT faults
- USGS 5.23.12 LiDAR LT faults Shaded Relief Map
- USGS 5.23.12 LiDAR LT faults Figure 2
- US Report West Tahoe Fault 7-2013
- Three Faults under LT.Tahoetopia
- Livescience WTDPF earthquake
- 75-09 Ozone Trends CARB
- CARB staff report rev ozone 2005
- 2012 CARB Designations ozone
- CARB 2008 EI

We request TRPA staff provide the APC, and the public, with the full copy of our comments, as they also apply herein.

Further, the "Attachment E: Response to comments" included with the APC packet does not include responses to the questions that were originally skipped by staff, nor does the packet contain additional responses to those comments again raised for the 10/24/13 RPIC hearing.

We request TRPA staff provide written responses to these comments.

Attachment F: TCAP Modifications:

We appreciate the inclusion of a numerical standard for SEZ restoration as noted below in the packet.

Modifications to the Oct 1, 2013 City Council adopted Tourist Core Area Plan

SEZ Restoration and Coverage Reduction Strategy

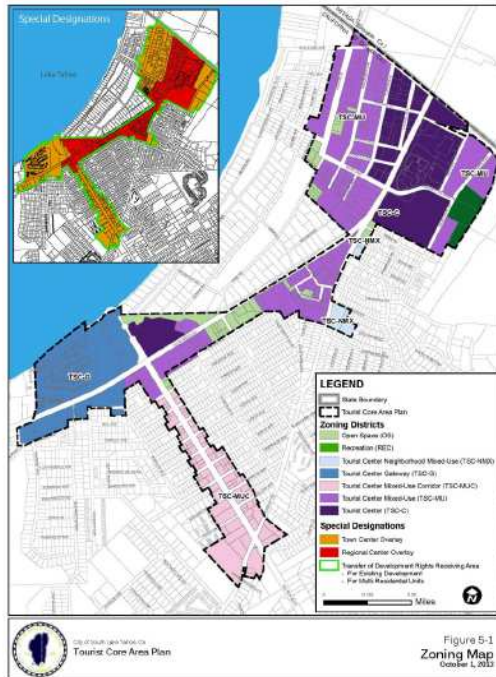
The following revisions intended to add more specificity to the TCAP SEZ Restoration and Coverage Reduction Strategy:

- **Policy NCR-2.1**
Increase the area of naturally functioning SEZs by preserving existing SEZs and restoring/rehabilitating 7 acres of disturbed SEZs. ~~where feasible.~~
- **Policy NCR-4.1**
~~Encourage onsite land~~ coverage reduction will occur primarily through environmental redevelopment by providing development incentives in centers that promotes the relocation and transfer of land coverage. The City will endeavor, where feasible, to reduce and avoid creating new coverage in order to benefit the objectives of the TCAP and other areas of South Tahoe.

However, with regards to the change requested by CTC (below), it is unclear where the priority restoration is or was, what change was made, nor can we locate the CTC comment in the record.

We request TRPA clarify this change and include CTC's comment in the record.

- **Figure 5-1 Zoning Map, p. 5-7**
"Priority Restoration Area" label deleted in response to CTC comment.



Attachment I – Motions, Findings & Ordinance

1) Findings are not supported by adequate environmental analysis:

As no new information has been presented to address the previous comments we have raised regarding the threshold-related findings, we refer again to our previous comments. Impacts which have not been adequately analyzed include, but are not limited to:

- Increases in coverage, including concentrated coverage closer to the Lake;
- Increases in air pollution that will impact human and forest health;
- Increased emissions per person per mile from use of waterborne transit;
- Lack of sufficient measures to guarantee SEZ restoration;
- Cumulative impacts of the TCAP in addition to impacts from adjacent Area Plans, CPs, and PAS's, regional increases allowed by the TRPA RPU, and the impacts of increased populations in areas surrounding the Basin (e.g. within a few hours' drive); and
- Impacts of natural hazards, including flooding, earthquakes,³ and tsunamis.

2) Finding that Area Plans will simply “not interfere” with threshold gain is inappropriate:

We have previously commented on the disagreement with the new Code, Section 13.5.C.6, stating Area Plans must “**6. Demonstrate that all development activity within Town Centers and the Regional Centers will provide for or not interfere with Threshold gain, including but not limited to measurable improvements in water quality.**” [Emphasis added]. It is not enough to “not interfere with threshold gain,” or “not hinder or impede”⁴ threshold gain. TRPA is responsible for maintaining a Regional Plan which helps **achieve and maintain the thresholds**. The Area Plans will become part of the RP, and therefore must also help achieve and maintain the thresholds.

This was clearly explained in federal Judge Carlton's ruling (Case 2:08-cv-02828-LKK-GGH Document 118, filed 9/16/2010) in favor of the TASC and LTLST challenge of TRPA's 2008 Shorezone Ordinances:

“More fundamentally, however, TRPA misunderstands the nature of the obligation to achieve and maintain the thresholds. It is not enough to show that the Amendments do not make the problem worse. TRPA must ensure that the ordinances, as amended, implement the regional plan in a way that will actually achieve the thresholds. With regard to thresholds not presently in attainment, TRPA's finding that the Amendments will not aggravate the problem is inadequate.” [Emphasis added].

We request the TRPA perform a proper analysis which examines the full impacts of all amendments to the Regional Plan, including the Area Plans that will also be amendments to the RP, and how the Plans will help TRPA achieve and maintain thresholds.

³ Since our 10/21/13 comments were submitted, numerous residents have observed California Geological Survey personnel performing digs to study fault lines in the Basin. Although adequate scientific data already exists regarding the presence and possible impacts of these fault lines, that CA GS and other experts are focusing on better identification of these hazards should be yet another reminder to TRPA that these hazards can not be ignored by TRPA or the local agencies.

⁴ TRPA's response to question number 99 (in July 2013 responses to our comments on the DC SSAP) regarding the definition of “not interfere with threshold gain” states “the term ‘not interfere with’ means that the Area Plan will not hinder or impede.”

3) *Information presented does not equate to substantial evidence in the record:*

We first reiterate that for the reasons noted throughout our comment letters, including this one, there is not evidence to support the findings TRPA is required to make. Where the findings are based on the ‘analysis’ in the RPU EIS, as noted, the RPU EIS is not technically adequate. Where the findings are based on the claim that future projects will have to identify and mitigate their project impacts, this ignores the cumulative nature of projects, plus it further exemplifies the deficiencies that began with the inadequate RPU EIS and which were carried through to the TCAP. Findings based on the City’s GPU EIR from 2011 are irrelevant for several reasons: 1) the TRPA documents state the baseline for the ND/IEC for the TCAP is 2013; 2) the GPU EIR also certified a plan that acknowledged it violated TRPA’s Regional Plan at that time; and 3) the GPU could not have contemplated, or analyzed, the cumulative impacts from the increases in development that were allowed by the December 2012 TRPA RPU Amendments.

For example, p. 5 notes:

The IS/ND/IEC is a program-level environmental document. No specific development projects are proposed at this time and no specific development projects were analyzed by the IS/ND/IEC. All future projects within the IS/ND/IEC are subject to the appropriate project-level environmental review and permitting by TRPA and/or CSLT based on the size, nature and location of the project (Section 13.7.3 of the TRPA Code of Ordinances). Project-level environmental documents will require identification of, and mitigation for, any potentially significant environmental impacts.

The IS/ND/IEC assessed potential impacts to the affected physical environment from implementation of the TCAP. Based on the review of the evidence, the analysis and conclusion in the IS/ND/IEC determined the implementation of the TCAP will not have a significant impact on the environment not otherwise evaluated in the RPU, RTP, and GP EISs and/or EIRs and potential significant impacts will be mitigated or addressed through adoption and implementation of the RPU, RTP, and GP. [Emphasis added].

Further, pages 6-7 note:

Chapter 4 Findings: The following findings must be made prior to adopting the TCAP:

1. Finding: The proposed area plan is consistent with, and will not adversely affect implementation of the Regional Plan, including all applicable Goals and Policies, Plan Area Statements and maps, the Code, and other TRPA plans and programs. Rationale: Land Use Policy 4.6 of TRPA’s Goals & Policies encourages the development of area plans that improve upon existing plan area statements and community plans or other TRPA regulations in order to be responsive to the unique needs and opportunities of the various communities in the Tahoe Region.

Rationale: Land Use Policy 4.6 of TRPA’s Goals and Policies encourages the development of area plans that improve upon existing plan area statements and community plans or other TRPA regulations in order to be responsive to the unique needs and opportunities of the various communities in the Tahoe Region. The TCAP includes all required elements identified in Land Use Policies 4.8, 4.9 and 4.10 as demonstrated in the Conformance Review Checklist.

The TCAP was prepared in conformance with the substantive and procedural requirements of the Goals and Policies, as implemented through TRPA Code of Ordinances, Chapter 13, Area Plans. The TCAP is consistent with the 2012 Regional Plan and Code, as shown in the Conformance Review Checklist and as demonstrated by the IS/ND/IEC. The TCAP contains the required contents of an Area Plan specified in the TRPA Code of Ordinances, Chapter 13, Area Plans, and

when implemented, will have a beneficial impact on the Regional Plan's ability to achieve and maintain the thresholds as demonstrated below in the Chapter 4 and 13 findings.

Pursuant to Code Section 4.4.2, TRPA considers, as background for making the Section 4.4.1.A through C findings, the proposed project's effects on compliance measures (those implementation actions necessary to achieve and maintain thresholds), supplemental compliance measures (actions TRPA could implement if the compliance measures prove inadequate to achieve and maintain thresholds), the threshold indicators (adopted measureable physical phenomena that relate to the status of threshold attainment or maintenance), additional factors (indirect measures of threshold status, such as funding levels for EIP projects), and interim and target dates for threshold achievement. TRPA identifies and reports on threshold compliance measures, indicators, factors and targets in the Threshold Evaluation Reports prepared pursuant to TRPA Code of Ordinances, Chapter 16, *Regional Plan and Environmental Threshold Review*.

TRPA relies upon the project's accompanying environmental documentation, Staff's professional analysis, and prior plan level documentation, including findings and EISs, to reach the fundamental conclusions regarding the project's consistency with the Regional Plan and thresholds. A project that is consistent with all aspects of the Regional Plan and that does not adversely affect any threshold is, by definition, consistent with compliance measures, indicators and targets. In order to increase its analytical transparency, TRPA has prepared worksheets related specifically to the 4.4.2 considerations, which set forth the 220 compliance and supplemental compliance measures, the 150 indicators and additional factors, and interim and final targets. Effects of the proposed project (here the TCAP) on these items, if any, are identified and to the extent possible described. TRPA cannot identify some target dates, status and trend for some threshold indicators because of a lack of available information. TRPA may still determine whether the project will affect the 4.4.2 considerations (and ultimately consistency with the Regional Plan and impact on thresholds) based on the project's specific environmental impacts related to those threshold indicators. [Emphasis added].

The last sentence underlined above appeared at the 13th hour before the DC SSAP was heard and approved by the TRPA GB on 9/24/2013.

We request that an adequate environmental analysis be performed and findings be based on substantial scientific evidence in the record. In addition, we previously questioned what the revision in the last sentence above meant,⁵ and reiterate that question herein for the TCAP.

In another example, page 7 notes:

Based on the IS/ND/IEC, the RPU, RTP, and GP EISs and/or EIRs, the RPU and RTP findings made by TRPA on December 12, 2012, and the Section 4.4.2 staff analysis, and using applicable measurement standards consistent with the available information, the TCAP will not adversely affect applicable compliance and supplemental compliance measures, indicators, additional factors, and attainment of targets by the dates identified in the 2011 Threshold Evaluation. The TCAP incorporates and/or implements relevant compliance measures, and with the implementation of the measures with respect to development within the TCAP, the effects are not adverse, and with respect to some measures, are positive. (See TCAP Threshold Indicators and Compliance Measures Worksheets) [Emphasis added].

The 2011 Threshold Report (TER) was not adopted, but rather 'issued.' TRPA also purposely excluded the TER from the Draft and Final EIS documents, claiming it was a

⁵ From our 9/24/13 comments: "This last sentence is new, and it is unclear what it means. It appears to suggest that TRPA is asking the GB to make threshold findings on 9/25/13 yet reserving the right to revise that conclusion later?"

separate document and did not provide a baseline for the RPU EIS (although as our RPU comments note, it clearly was relied on as baseline for the EIS). The TER also made recommendations, revisions, and other changes which were not aired through the same process as the RPU EIS, and the response to our detailed comments on the TER were not sufficiently addressed (examples were included with the attachments to our 12/11/12 comments to TRPA, included with a previous letter to the RPIC). Thus the reliance on the TER to make findings is suspect. Further, the threshold findings require that the RP, as amended, will help achieve and maintain thresholds. The TER does not guarantee threshold achievement and maintenance. This strange twist, from having to achieve and maintain thresholds, to now simply be 'consistent with' the TER, is also suspect. TRPA can not substitute the 2011 TER report that was 'issued' in December for making RP amendment findings.

TRPA anticipates that implementation of the TCAP will accelerate threshold gains as demonstrated below. Because the principal beneficial impacts of implementation of the TCAP depend upon the number and size of redevelopment projects, the specific extent and timing or rate of these beneficial effects of the TCAP cannot be determined at this time. However, pursuant to Chapter 13, TRPA will monitor all development projects within the TCAP through quarterly and annual compliance reports. These reports will be presented to the Governing Board annually for area plan recertification and used every four years to evaluate the status and trends related to thresholds. [Emphasis added].

This statement regarding cumulative *accounting* of unit does not replace the required (and missing) environmental analysis of the cumulative **impacts** of the TCAP, or the TRPA RPU.

Similarly, Section 4.4.2.C requires TRPA to confirm whether the proposed project is within the remaining capacity for development (e.g., water supply, sewage, etc.) identified in the environmental documentation for the Regional Plan. The TCAP does not affect the amount of the remaining capacities available, identified and discussed in the RPU EIS. The TCAP does not allocate capacity or authorize any particular development. To the extent the TCAP enables the use of redevelopment incentives, those incentives are within the scope of the incentives analyzed by the RPU and RTP EISs. [Emphasis added].

As the RPU EIS did not address all of the people that the RPU would draw, including countless visitors, the analysis failed to adequately assess the remaining capacities.

TRPA therefore finds that the TCAP is consistent with and will not adversely affect implementation of the Regional Plan, including all applicable Goals and Policies, Plan Area Statements and maps, the Code, and other TRPA plans and programs.

Where is the referenced 'demonstration?'

Although the staff summary includes more wording to 'explain' how findings can be made, including chapter 4 findings, the additional wording doesn't change the basic fact that the impacts of the TCAP have not been adequately analyzed.

Attachment J: Thresholds & Compliance Measures Checklist

It appears that the information provided in “Attachment J – Thresholds & Compliance Measures Checklist” was included as a means to “provide additional information to support the findings.” According to staff’s response to our comments that the Compact’s threshold findings can not be made for the DC SSAP (labeled comment number 141 in Appendix B of the September GB materials) “*Staff has revised the staff summary to provide additional information to support the findings.[PN]*”. At that time, the new information in this table was presented although its purpose was never clearly explained. The text references, which simply seem to explain and re-explain responses we’ve already heard in various iterations, do not provide any additional data or scientific evidence to support making the threshold findings.

Further, as noted in comments on the RPU and Area Plans, the RPU EIS did not analyze the carrying capacity of the thresholds, as required by the Compact. Page 8 states the following (included below), however the remaining capacity available associated with healthy air quality, healthy forests, healthy soils, adequate stormwater capture with climate change, noise, and other environmental resources impacted by development have not been analyzed. Therefore, there is no evidence to support the finding that the thresholds will not be exceeded.

The TCAP does not affect the amount of the remaining capacity available, as the remaining capacity for water supply, sewage collection and treatment, recreation and vehicle miles travelled have been identified and evaluated in the RPU EIS or RTP EIR/EIS. TRPA therefore finds that the TCAP will not cause the thresholds to be exceeded.

Finding 3 on pages 8-9 relates to air and water quality standards, stating:

3. Finding: Wherever federal, state or local air and water quality standards applicable for the Region, the strictest standards shall be attained, maintained, or exceeded pursuant to Article V(d) of the Tahoe Regional Planning Compact.

Rationale: Based on the following: (1) TCAP IS/ND/IEC; (2) RPU EIS; (3) RTP EIR/EIS; (4) GP EIR; (5) 2011 Threshold Evaluation Report; and (6) 2011 Indicator Summaries, adopted by the Governing Board or City Council, no applicable federal, state or local air and water quality standard will be exceeded by adoption of the TCAP. The proposed Area Plan does not affect or change the Federal, state or local air and water quality standards applicable for the Region. Projects developed under the TCAP will meet the strictest applicable air quality standards and implement water quality improvements consistent with TRPA Best Management Practices requirements and the Lake Tahoe TMDL and City’s Pollutant Load Reduction Plan (PLRP). Federal, state, and local air and water quality standards remain applicable for all parcels in the TCAP, thus ensuring environmental standards will be achieved or maintained pursuant to the Tahoe Regional Planning Compact.

TRPA’s rationale appears to neglect consideration of *increases* in air pollution, instead responding to a partial question about whether the TCAP will change the regulatory standards for air quality. This sidesteps the issue of whether the TCAP will result in the attainment of the strictest air and water quality standards in the jurisdiction for which it applies. As noted by our extensive comments related to ozone, as well as PM10, the Lake Tahoe Air Basin is not in attainment of California’s health-based standards, and the proposed RPU and TCAP do not provide evidence to support a finding that these standards will be attained by the plans, which will result in more air pollution. Also, in our previous comments to the RPIC and City entities, we explained why an estimate of

emission reductions from one source of air pollution (on-road motor vehicles) in 2035 – twenty-two years from now – does not support a finding of attainment of air quality standards in 2035, let alone *over the next 22 years when the public will continue to breath Tahoe’s air.*

Pages 12-21 outline additional discussion regarding the threshold findings. However, no additional information has been provided to support the outcomes that are claimed to happen from the TCAP. Examples include:

The TCAP accelerates threshold gain including water quality restoration and other ecological benefits, by supporting environmental redevelopment opportunities and Environmental Improvement Program (EIP) investments. The TCAP retains the Regional Plan established growth control system and provides incentives for property owners to hasten the transfer of development rights from sensitive or outlying areas to Town Centers and the Regional Center where redevelopment is better suited and will have beneficial or reduced adverse environmental impacts.

There is no evidence that the “redevelopment” proposed in the TCAP will result in environmental gain. As noted frequently in our comments, there is no evidence that the stormwater treatment facilities promoted by the TCAP, RPU, and TMDL, will remove the fine sediments as estimated or assumed. As the RPU and TCAP will increase coverage and do so in areas closer to the lake, there will be less opportunities for natural infiltration, thus the RPU and TCAP land use changes promote an outcome that will have to rely heavily on engineered facilities – including the use of stormwater filters. To date, laboratory studies of stormwater treatment filters generally reveal the finest particulates (those less than 20 microns) are not removed by the filters as assumed in models, and thus continue to Lake Tahoe. Only one very limited sample collection has been conducted in the Basin to test the effectiveness of media filter-based stormwater treatment.⁶ Further, scientists have noted that particles 5 microns and below are have the most impact on mid-lake clarity (noted in our RPU comments and originally stated in peer reviewer comments on the Technical TMDL, Lahontan Regional Water Quality Board). Evidence that filters will remove particles 5 microns and below in the Lake Tahoe Basin is also lacking. In addition, sufficient maintenance is required for the filters to perform (at whatever level they are designed to perform), yet maintenance has rarely been performed.⁷ There are also no data supporting any reductions in nitrogen from stormwater treatment facilities.⁸ Further, there has been no analysis of the true environmental impacts of tearing down the oft-referenced ‘old run-down motels’ and transferring them to the more urban centers (and then rewarded with up to 6x the number of units).⁹ All of these examples have been stated in our previous comments letters;

⁶ Two runoff events were sampled from the Ski Run Blvd. media filter, as outlined in referenced papers in TRPA’s 2012 BMP Handbook (handbook and papers included in attachments). The quality of the data are very poor, measurement tools were not working or not conducive to the setup, and the volume of water run through the media filter was very small compared to water volumes captured by Basins and other structures, and volumes anticipated in future flooding events (see 2NDNATURE and Northwest Hydraulic Consultants. *Tahoe Stormwater and BMP Performance Database Monitoring and Reporting Guidance Document – Final and Appendices*. 2010).

⁷ The lack of BMP maintenance is noted throughout the attached BMP studies.

⁸ As noted in TRPA’s 2012 BMP Handbook, Chapter 4: “The media filter demonstrated variable (typically poor) ability to remove dissolved nutrients from stormwater, including nitrate, nitrite, orthophosphate, and dissolved phosphorus.”

⁹ Detailed comments to illustrate this concern were included in the 10/3/2013 comments to the RPIC.

additional references related to stormwater treatments are noted in the 11/12/13 attachments.

We request TRPA and the CSLT to perform a sufficient environmental analysis of the impacts of the increased coverage, stormwater runoff, and related pollution sources.

The TCAP will help to promote environmental redevelopment within existing developed areas by allowing increased density and height provisions within Town Centers and the Regional Center that will serve as an incentive for private investment in redevelopment projects. Significant threshold gain will result from the application of existing Codes and requirements to individual projects. These redevelopment incentives are intended to increase the rate of redevelopment and will likewise increase the rate of threshold gain by accelerating the application of controls designed to enhance water quality, air quality, soil conservation, scenic quality and recreational improvements to projects that wouldn't otherwise be redeveloped absent TCAP provisions.

The 'application of existing Codes and requirements to individual projects' appears to mean that TRPA and the City will simply enforce existing requirements. How will adoption of the TCAP ensure more enforcement, especially with an extensive history of an ongoing lack of enforcement? Further, nothing in the TCAP provides the assurance that measures to control pollution, for example BMPs, will be adequately maintained - forever – yet the RPU and TCAP rely on these one-time installations to generate ongoing benefits.

We request TRPA and the City to show how full BMP compliance will be achieved throughout the entire Basin, and sufficient ongoing maintenance be assured.

The TCAP's proposed Development and Design Standards represent a significant step forward in enhancing the aesthetics of the built environment and will result in improvements to the scenic threshold as projects are approved and built. Redevelopment of existing Town Centers and the Regional Center is identified in the Regional Plan as a high priority, as many of the Region's environmental problems can be traced to existing "legacy" developments that were constructed without recognition of the sensitivity of the Region's natural resources and impact on Lake Tahoe. To correct this, environmentally beneficial redevelopment and rehabilitation of identified urban centers is a priority, and the Goals, Policies and Implementation Strategies identified in the TCAP along with application of existing City and TRPA Codes and regulations encourage environmentally beneficial redevelopment and rehabilitation.

These redevelopment incentives are intended to increase the rate of redevelopment and will likewise increase the rate of threshold gain by accelerating the application of controls designed to enhance water quality, air quality, soil conservation, scenic quality and recreational improvements to projects that wouldn't otherwise be redeveloped absent TCAP provisions.

What improvements to projects are guaranteed by the TCAP yet not guaranteed to occur without the TCAP? We have raised this question before but no clear answers have been provided.

We request TRPA to clarify what benefits are only guaranteed by adoption of the TCAP versus no TCAP.

The TCAP maintains or strengthens the noise standards and thresholds currently in effect. The TCAP maintains existing CNEL standards in the Stateline/Ski Run Community Plan or modifies them to be consistent with the Thresholds, including standards that apply to the highway corridors (i.e., the area within 300 feet from the roadway edge) influenced by traffic. In addition, the City recognizes and is adopting the project-specific noise reduction measures described in the RPU EIS with the TCAP.

The TCAP adopts the Mixed Use zoning approved in the RPU. The RPU EIS required mitigation be adopted for the noise impacts in these mixed use zones by the end of 2013. However, as noted in the TRPA APC packet for the 11/13/13 hearing, the TWG examining these mitigation measures failed to identify a policy to mitigate noise in mixed use areas, instead suggesting additional questions be included in the IEC:

Compliance with existing building codes would ensure that interior areas attain an acceptable noise level. However, depending on their placement and surrounding land uses, exterior activity areas, such as porches and balconies, could experience elevated noise levels. To address this impact, the EIS included Mitigation Measure 3.6-4, which requires that TRPA develop and implement an exterior noise policy for mixed-use development. [Emphasis added]

After considering several approaches and incorporating direction from the Governing Board, the TWG recommend that this mitigation measure be implemented through an addition to section II.6, Noise, in the IEC. (P. 8, Agenda Item V.A.)

This fails to provide any mitigation for the cumulative noise impacts in Mixed Use areas, and the TCAP would adopt and perpetuate this same failure to meet mitigation requirements.

We request the cumulative impacts of mixed use development on exterior noise levels (and interior noise levels during the warmer months when many people in SLT will have their windows open all day and night) be adequately analyzed.

Additional comments and information regarding Nearshore Impacts:

For years, we have raised concerns regarding Tahoe's declining nearshore conditions. Although scientists were still studying the nearshore, including cause and effect of the increase in algae and invasive species, several facts have been well-established for many years:

- 1) the nearshore is declining rapidly and in a manner very different from the mid-lake clarity;
- 2) algae growth results from the combination of nitrogen and phosphorous;
- 3) measures that may reduce fine particles and phosphorous do not reduce nitrogen in the same way;
- 4) fertilizer application adds nitrogen and phosphorous into Lake Tahoe's watershed; and
- 5) more pavement means more stormwater runoff with higher velocities.

These are all facts which should be undisputed, however, we have provided extensive comments and references in previous letters for this information. Readers may simply read the TERC's 2013 State of the Lake Report¹⁰ for much of this background information; other sources include public TMDL documents and the impervious coverage documents attached to our RPU comments and incorporated herein. On that note, we have repeatedly asked TRPA to address the nearshore, with specific emphasis on reducing both nitrogen and phosphorous.

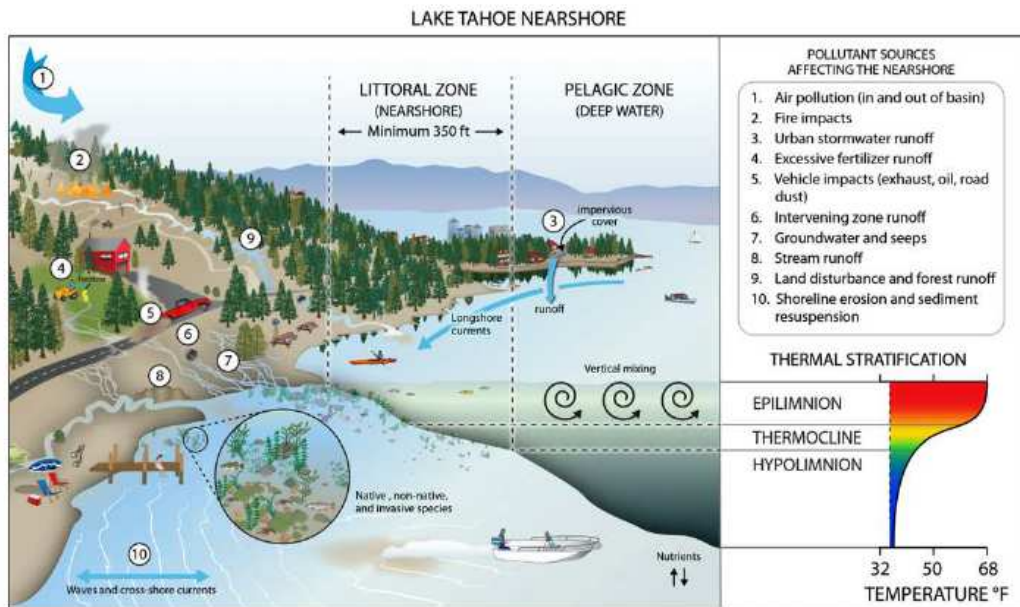
Reductions come from two ways: one, source control (e.g. less fertilizer); and 2) less coverage and stormwater treatment (e.g. wetlands, infiltration). The RPU, and the TCAP, do neither.

- The RPU's adopted 'alternative' specifically removed 'nitrogen' in the impact discussion, referring only to the future phase-out of phosphorous-containing fertilizers (DEIS Chapter 3.8, p. 3.8-23).
- The TCAP does not require reductions in nitrogen-containing fertilizers.
- The RPU also allows more coverage closer to the lake, creating more stormwater runoff. The TCAP carries forward this same impact.
- The RPU's EIS for this change, upon which the TCAP relies, claims the coverage can be treated; however, there is no actual evidence that existing technology can treat the amounts required, nor that BMPs will be properly maintained forever, let alone the 20-year span of the RPU.
- As noted in several previous comments, evidence shows that the 20-year BMP design is no longer sufficient to capture anticipated runoff in the Basin; nor is this one-size-fits-all approach appropriate for every part of the Basin since local topography and climate vary.

¹⁰ <http://terc.ucdavis.edu/stateofthelake/>

Thus, there remains no regulatory plan to improve the nearshore; instead, the RPU and associated Area Plans rely on unproven technologies, on perfect BMP maintenance (a highly unlikely situation), or simple neglect (e.g. failure to address nitrogen from fertilizers). The Desert Research Institute, working with several other research institutions, recently presented TRPA with the findings of their nearshore studies (we understand the first public presentation was at the 10/23/13 GB hearing). This information includes the following (full documents attached/links provided):¹¹

Nearshore Evaluation
 October 15, 2013
 Version 10.e



Illustration, L.J. Wible and A. Heysaert (Desert Research Institute), with additional clip art contributions courtesy of the Investigation and Application Network, University of Maryland Center for Environmental Science (ian.umces.edu/symbiol).

Figure 1-2. Illustration of important factors and processes affecting the lake nearshore environment.

¹¹ Images below taken from the DRI Executive Summary, p. 9-11.

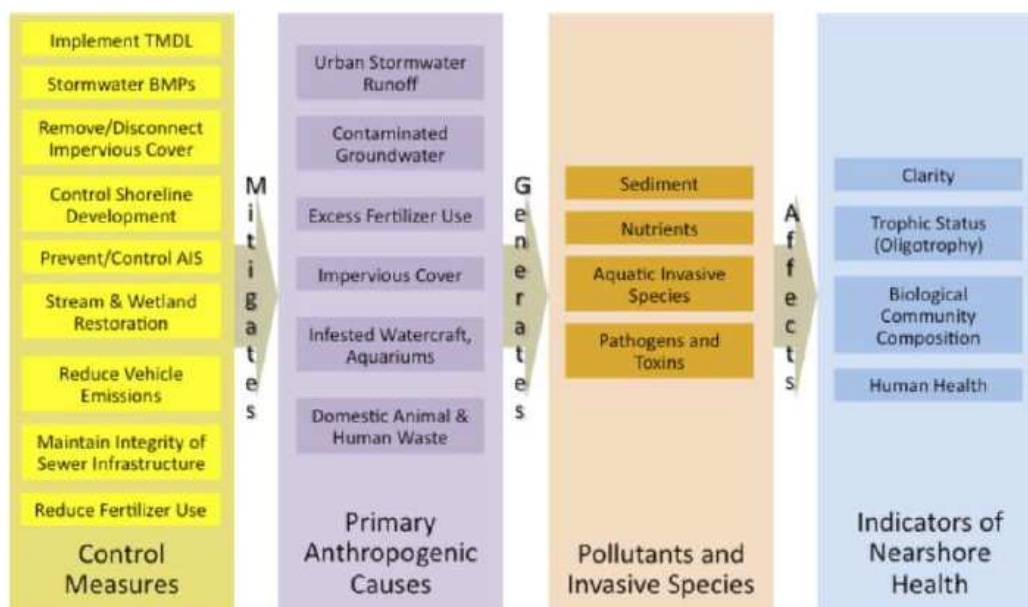


Figure 1-3. Examples from the nearshore conceptual model of progression from relevant control measures to indicators of nearshore health.

It must be acknowledged, however, that nearshore water quality is strongly influenced by localized pollutant input, so a load reduction that may improve the open-water may or may not have a directly comparable effect on all nearshore areas. For example, while load reductions along the south shore will contribute to an eventual improvement of open water clarity and a more immediate effect on that region's nearshore, its direct effect on the nearshore zone in the north lake may be delayed or attenuated. Water quality improvement projects should be selected to include those that (1) will have the most influence on both the nearshore and open water, and (2) are located in areas around the lake where measures of nearshore conditions indicate impairment.

While AIS may preferentially establish in some nearshore areas as a result of nearby watershed condition, this is not always the case, and once established they may not respond to watershed management activities. The establishment of invasive aquatic species in nearshore areas can precondition those areas for the introduction and establishment of subsequent undesired species by changing substrate and habitat conditions.

This report contains extensive information, however we'd like to point out some of the recurring themes:

- Pollutant sources affecting the nearshore include **urban stormwater runoff and excessive fertilizer runoff**;
- Anthropogenic causes include **urban stormwater runoff, excess fertilizer use, and impervious cover**.
- Example control measures include **Removing/Disconnecting Impervious Cover** (this is noted separate from the TMDL, thus reiterating that TMDL

implementation does not ‘negate’ removing impervious coverage), control shoreline development, and **reduce fertilizer use.**

The report also notes that nearshore conditions can not be examined from the larger, ‘lake-wide’ approach taken for mid-lake clarity. As noted in our comments to TRPA on the RPU (see 2012 collection), and various comments on the area plans in 2013, the RPU EIS analyses failed to examine the localized impacts of proposed land use changes, and looking merely at the Basin-wide level was not sufficient. TRPA’s response was to restate the TMDL would ‘likely help nearshore’ and that the local governments would perform more specific analysis at the local level. As illustrated by the DC SSAP or CSLT TCAP environmental documentation, no such analysis has been performed by the local governments. Instead, the Area Plan documents ‘tier’ from the RPU EIS, thus the additional analyses TRPA has stated would be done has not.

For years we have requested TRPA consider these nearshore issues and take actions that would reduce pollutants in the nearshore; we now reiterate that request to both TRPA and the CSLT with additional emphasis on and reference to the nearshore study noted herein.



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University of Nevada, Reno



FOR IMMEDIATE RELEASE: October 23, 2013

Scientists present integrated approach for evaluating and monitoring Lake Tahoe's nearshore ecology and aesthetics

(RENO): Scientists today presented research findings and recommendations to the Tahoe Regional Planning Agency Governing Board that address Lake Tahoe's aquatic nearshore environment and the heightened interest in understanding factors contributing to its apparent deterioration.

The Lake Tahoe Nearshore Evaluation and Monitoring Framework, a project funded during Round 10 of the Southern Nevada Public Land Management Act in 2010, was prepared for the USDA Forest Service by more than a dozen scientists and technical advisors from the Desert Research Institute, the University of Nevada, Reno and the University of California, Davis.

In addition, a Nearshore Agency Working Group compiled of key staff from the California Regional Water Quality Control Board, Lahontan Region (Lahontan Water Board), the Nevada Division of Environmental Protection, the Tahoe Regional Planning Agency, and the U.S. Environmental Protection Agency participated throughout the process, communicating agency needs and supporting the scientists with relevant information.

"This represents the initial collaborative step between the science community and the resource management agencies to develop a comprehensive approach for assessing and managing the nearshore ecology and aesthetics of Lake Tahoe," said Alan Heyvaert, Ph.D., principal investigator on the multi-year project and acting senior director of the Center for Watersheds and Environmental Sustainability at DRI.

The report does not recommend changes to existing state and TRPA legal or statutory definitions or standards affecting the Lake Tahoe nearshore. Rather, it explains, for the first time in one report, the unique aspects of this important zone; evaluates existing California, Nevada and TRPA standards and thresholds related to this region; presents a new conceptual model for evaluating nearshore environmental health; and proposes a monitoring strategy intended to help resource managers identify the most meaningful physical, chemical and biological indicators of healthy nearshore conditions.

"For monitoring and assessment purposes, the report defines "nearshore" as the zone from the low water elevation (6,223 feet), or the current shoreline, to the mid-summer thermocline, which has a depth of approximately 69 feet, and at minimum a distance of 350-feet from shore," Heyvaert explains.

"It is in the nearshore region that most people experience the lake," said report co-author Geoffrey Schladow,

[Download full report \(PDF\)](#)

The nearshore is an important zone of relatively shallow water around the lake's perimeter that is valued for its recreational and aesthetic qualities, as well as for the unique biological community that it supports.

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director of the UC Davis Tahoe Environmental Research Center. "This report is an important step as it establishes the scientific underpinnings of a successful nearshore restoration program."

Results from the report's widespread literature review and data summary indicate that conditions can differ widely around the lake's nearshore and create more localized effects as compared to the open-waters of Lake Tahoe, which tend to be more uniform.

The report also emphasizes that pollutants entering the lake from watershed or groundwater can be temporarily concentrated in the nearshore, before eventually being mixed and diluted in the open-water, resulting in biological responses not observed or recorded in Lake Tahoe's deep water.



Without a propeller and with its shallow draft, DRI's jet boat nearshore research vessel allows scientists to study changes in water clarity and other important nearshore characteristics in the shallow areas of the lake.

"The nearshore environment is inherently complex since it is immediately adjacent to stormwater flow and runoff from the developed and undeveloped portions of the surrounding watershed," said co-author John Reuter, research ecologist and associate director of the UC Davis Tahoe Environmental Research Center. "Therefore, it is recommended that a finer scale of evaluation and monitoring is necessary in this zone, especially for the nuisance blooms of attached algae found on rocks and other hard surfaces in the nearshore."

Reuter added that in addition to pollutant factors, there are numerous other aspects unique to the nearshore environment that can contribute to variation in conditions, such as greater vulnerability to increased temperature from climate change, effects of nearshore recreation, existing and future shorezone structures, and fluctuation in lake levels.

The report also summarizes the proposed and targeted research needs related to monitoring and management of the nearshore.

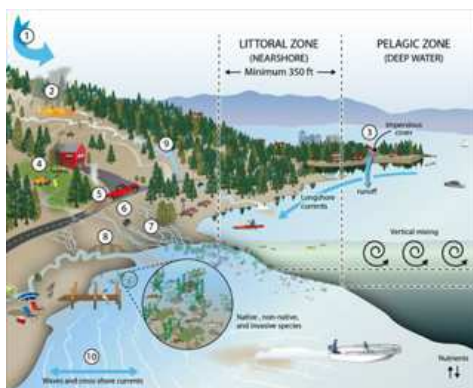
"The introduction of aquatic invasive species has already produced some profound changes in the nearshore," said co-author Sudeep Chandra, limnologist and director of University of Nevada Reno's Aquatic Ecosystems Analysis Laboratory. "Further establishment of aquatic invasive species in the nearshore has the potential to unravel the tremendous progress made toward protecting Lake Tahoe's clarity."

Chandra added that very little data exist on the nearshore community structure.

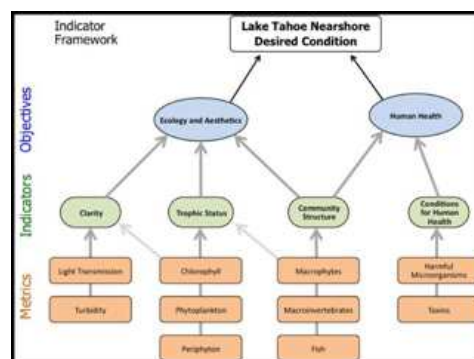
"We do not know the composition, distribution or abundance of most macro-organisms that inhabit the nearshore," he said. "Base data are urgently needed to describe these conditions before they change any further."

The findings and recommendations of the report are expected to support several agency statutory and programmatic needs by:

1. providing baseline information that could assist in developing data collection and analysis needed to inform any revisions or assessments of relevant state and TRPA standards;
2. supporting the development of products for the Tahoe Monitoring and Evaluation Program;
3. tracking the effectiveness of the Tahoe Total Maximum Daily Load Program and other Environmental Improvement Program efforts related to nearshore conditions and contributing to detection and management of aquatic invasive species in the nearshore.



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About the Desert Research Institute: DRI, the nonprofit research campus of the Nevada System of Higher Education, strives to be the world leader in environmental sciences through the application of knowledge and technologies to improve people's lives throughout Nevada and the world.

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Lake Tahoe Nearshore Evaluation and Monitoring Framework

EXECUTIVE SUMMARY

October 15, 2013



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Lake Tahoe Nearshore Evaluation and Monitoring Framework

October 15, 2013

The Nearshore Science Team (NeST) included water quality scientists and aquatic ecologists from the University of Nevada, Reno; the University of California, Davis Tahoe Environmental Research Center; and the Desert Research Institute Center for Watersheds and Environmental Sustainability.

A Nearshore Agency Work Group (NAWG) was created to communicate agency information needs and to contribute agency relevant information toward the effort. It was composed of representatives from the California Regional Water Quality Control Board, Lahontan Region (Lahontan Water Board), the Nevada Division of Environmental Protection (NDEP), the Tahoe Regional Planning Agency (TRPA), and the U.S. Environmental Protection Agency (USEPA).

Any questions regarding the Lake Tahoe Nearshore Evaluation and Monitoring Framework should be directed to:

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<http://www.fs.fed.us/psw/partnerships/tahoescience/>

The views in this report are those of the authors and do not necessary reflect those of the USDA Forest Service Pacific Southwest Research Station or the USDA Bureau of Land Management.



Lake Tahoe Nearshore Evaluation and Monitoring Framework

Version 10.e

Date: October 15, 2013

Nearshore Science Team (NeST) Contributors

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Abstract

Changes in nearshore conditions at Lake Tahoe have become evident to both visitors and residents of the Tahoe Basin, with increasing stakeholder interest in managing the factors that have contributed to apparent deterioration of the nearshore environment. This has led to joint implementation of a Nearshore Science Team (NeST) and the Nearshore Agency Working Group (NAWG), which together have contributed to a synthesis review of nearshore information and the development of a monitoring and evaluation plan that will track changes in nearshore conditions. A conceptual model is presented that conveys our contemporary understanding of the factors and activities that affect desired nearshore qualities. Results from review and analysis of historical data are provided, as well as an assessment on the adequacy of existing nearshore standards and associated indicators. The resulting nearshore monitoring framework will be used to guide development of an integrated effort that tracks the status and trends associated with nearshore conditions.

Recommended Citation: Heyvaert, A.C., Reuter, J.E., Chandra, S., Susfalk, R.B., Schladow, S.G. Hackley, S.H. 2013. Lake Tahoe Nearshore Evaluation and Monitoring Framework. Final Report prepared for the USDA Forest Service Pacific Southwest Research Station.

1.0 EXECUTIVE SUMMARY

1.1 Background

The nearshore of Lake Tahoe is an important zone of relatively shallow water around the lake perimeter that is much appreciated for the recreational and aesthetic qualities it provides, as well as for its vital biological habitat. Unfortunately, changes in nearshore conditions over time have become evident to both visitors and residents of the Tahoe Basin, along with increasing stakeholder interest in managing the factors that have contributed to apparent deterioration of the nearshore environment.

Heightened agency and public interest in understanding the nearshore environment has stimulated several independent research and monitoring efforts during this time, including nearshore studies on clarity and algae, as well as development of the Lake Tahoe TMDL (total maximum daily load) for managing pollutants that affect the pelagic (deep-water) clarity. This report is the result of a multi-year effort that for the first time summarizes available information on Lake Tahoe's nearshore condition, develops an integrated set of metrics and indicators to characterize nearshore condition, considers reference conditions and the relevance of existing thresholds and standards, and then provides recommendations for a monitoring and evaluation framework that can be used to guide the tracking of changes in nearshore condition and to support regional program planning needs.

Ultimately, the findings and recommendations of this project are expected to support several agency statutory and programmatic needs by: 1) providing baseline information to support assessment of relevant state and TRPA standards; 2) supporting the development of products for the Tahoe Monitoring and Evaluation Program; 3) tracking the effectiveness of the Tahoe TMDL Program and other EIP efforts related to nearshore condition; and 4) contributing to detection and management of aquatic invasive species in the nearshore.

1.2 Project Approach

This project represents an initial collaborative step between the science community and resource management agencies to develop a comprehensive approach for assessing and managing the nearshore ecology and aesthetics of Lake Tahoe. The Nearshore Science Team (NeST) included water quality scientists and aquatic ecologists from the University of Nevada, Reno (UNR), the University of California, Davis (UCD), and the Desert Research Institute (DRI). A Nearshore Agency Work Group (NAWG) was created to communicate agency information needs and to contribute agency relevant information toward the effort. It was composed of representatives from the California Regional Water Quality Control Board, Lahontan Region (Lahontan Water Board), the Nevada Division of Environmental Protection (NDEP), the Tahoe Regional Planning Agency (TRPA), and the U.S. Environmental Protection Agency (USEPA).

Completion of project components followed a logical sequence to inform successive steps in the process of assessing information and developing the final report, though several of these steps occurred iteratively (Figure 1-1). The initial task was to conduct a comprehensive literature review of available information relevant to the nearshore and to produce an annotated bibliography. This bibliography provided the basis for developing a conceptual model of the nearshore environment and the foundation for developing a desired condition statement and objectives, as well as a definition of the “nearshore” for monitoring and assessment purposes. It was also the source for much of the data summarized in the report for efficacy assessment of existing standards, and for developing an integrated set of metrics and indicators that were used to design the nearshore monitoring framework.

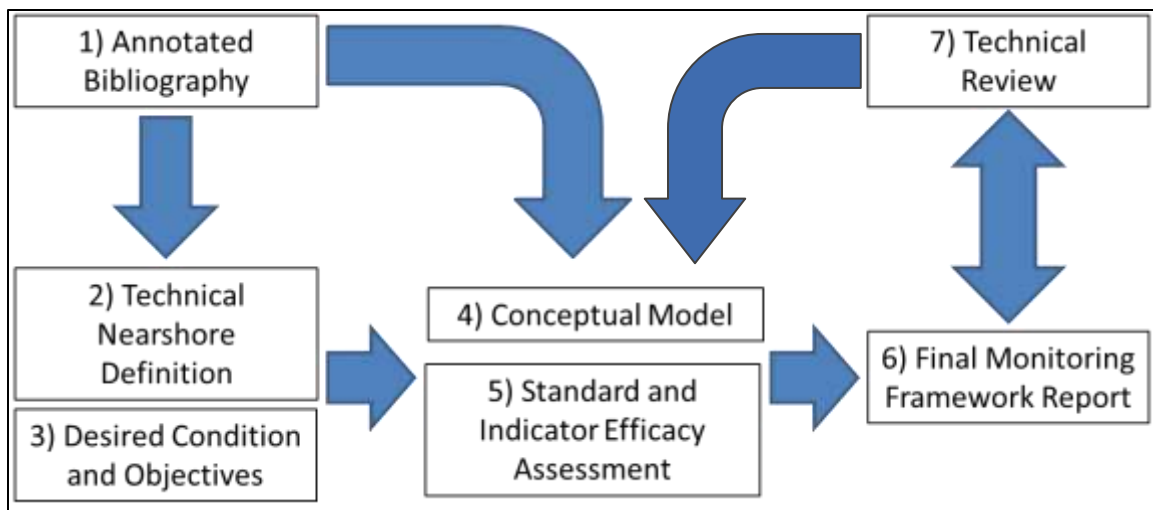


Figure 1-1. A schematic showing nearshore project tasks and sequence of workflow.

1.3 Summary of Project Components

- *Annotated bibliography* – Literature survey of data and information related to the nearshore of Lake Tahoe. Scientific journal articles as well as technical reports and academic theses/dissertations were included on topics such as water quality, ecology, algal species composition, periphyton growth and biomass, nutrients; fisheries, geology, etc.
- *Technical definition of nearshore* – Definition of the nearshore was developed for monitoring and evaluation purposes, based on existing definitions from Basin agencies, specific features of Lake Tahoe, and scientific literature.
- *Desired condition and objectives* – Developed narrative statements that summarize management objectives for a nearshore program that will guide actions taken to achieve the goal of its desired condition.

- *Conceptual model* – Summarized factors important to nearshore condition such as pollutant sources, watershed and in-lake processes, pollutants and affects, and controls within a qualitative, visual-based, format.
- *Current thresholds and standards* – Evaluated existing state and TRPA water quality-related standards and thresholds in terms of their relevance to nearshore assessment and management.
- *Indicators and metrics* – Developed a set of recommended indicators and associated metrics that would efficiently represent the complex interactions between various attributes (parameters) that constitute nearshore condition. Metrics are the measurable characteristics used in a monitoring design to evaluate the condition of specified indicators.
- *Existing nearshore data* – Available data were analyzed to provide summary assessments for each nearshore metric with regard to analysis of reference conditions, possible new or modified thresholds, and the creation of an integrated nearshore monitoring and evaluation program. Reference conditions were based on historical data, when available, otherwise on contemporary pristine, undisturbed or least disturbed conditions. Literature values were cited in the absence of Tahoe specific data. In some cases where sufficient data exist, options are discussed in consideration of different approaches.
- *Design of nearshore monitoring program* – Recommendations are provided for establishing a comprehensive monitoring program that allows nearshore condition to be evaluated for status and trends. Monitoring design is focused on the primary recommended metrics.

1.4 Nearshore Definition

This report does not recommend changes to existing state and TRPA legal or statutory definitions of the Lake Tahoe nearshore. Rather, it addresses unique aspects of the nearshore in context of framing the monitoring design through use of the following definition.

Lake Tahoe's nearshore for purposes of monitoring and assessment is considered to extend from the low water elevation of Lake Tahoe (6223.0 feet Lake Tahoe Datum) or the shoreline at existing lake surface elevation, whichever is less, to a depth contour where the thermocline intersects the lake bed in mid-summer; but in any case, with a minimum lateral distance of 350 feet lake ward from the existing shoreline.

The thermocline is a physical feature in lakes that represents a zone of rapid transition from warm surface water to underlying cold water. It is a seasonally dynamic stratification that strongly influences nearshore processes. The 31-year average August (maximum) thermocline depth in Lake Tahoe is 21 m (69 feet). This definition is more flexible than regulatory definitions, as is appropriate for guiding a monitoring approach that must adapt to natural variability in lake water levels and thermodynamic structure.

1.5 Desired Condition Statement and Objectives

A desired condition statement provides the focus for management and monitoring activities needed to achieve and maintain a preferred level of ecosystem quality. The desired condition statement for Lake Tahoe's nearshore was articulated as follows.

Lake Tahoe's nearshore environment is restored and/or maintained to reflect conditions consistent with an exceptionally clean and clear (ultra-oligotrophic) lake for the purposes of conserving its biological, physical and chemical integrity, protecting human health, and providing for current and future human appreciation and use.

Two overarching management objective statements were developed to support achieving the desired condition. The first is for preserving ecological and aesthetic characteristics of the nearshore:

Maintain and/or restore to the greatest extent practical the physical, biological and chemical integrity of the nearshore environment such that water transparency, benthic biomass and community structure are deemed acceptable at localized areas of significance.

Human experience at the lake is assumed to be equally or more strongly related to recreational interactions with the nearshore environment than it is to mid-lake clarity. Both the ability to see the bottom of the lake (transparency) and what is seen or felt on the bottom influence the nearshore aesthetic experience, which also reflects ecological conditions and processes. This report proposes that the nearshore ecology and aesthetic objective will be evaluated on the basis of three separate indicators (with associated metrics) that collectively provide assessment of:

- nearshore clarity,
- nearshore trophic status (nutrients and algal growth that indicate the degree of eutrophication), and
- nearshore community structure (biological composition).

The other objective is for sustaining conditions suitable for human health in the nearshore zone:

Maintain nearshore conditions to standards that are deemed acceptable to human health for purposes of contact recreation and exposure.

The focus for this objective is specifically on health risks associated with recreational exposure and not on attendant risks associated with water provided from the nearshore for municipal or domestic supply. Existing state and local programs enforce potable water supply standards. They also provide criteria for tracking the presence of pathogens and toxic compounds that may affect conditions for human health, which serves as the indicator for this objective.

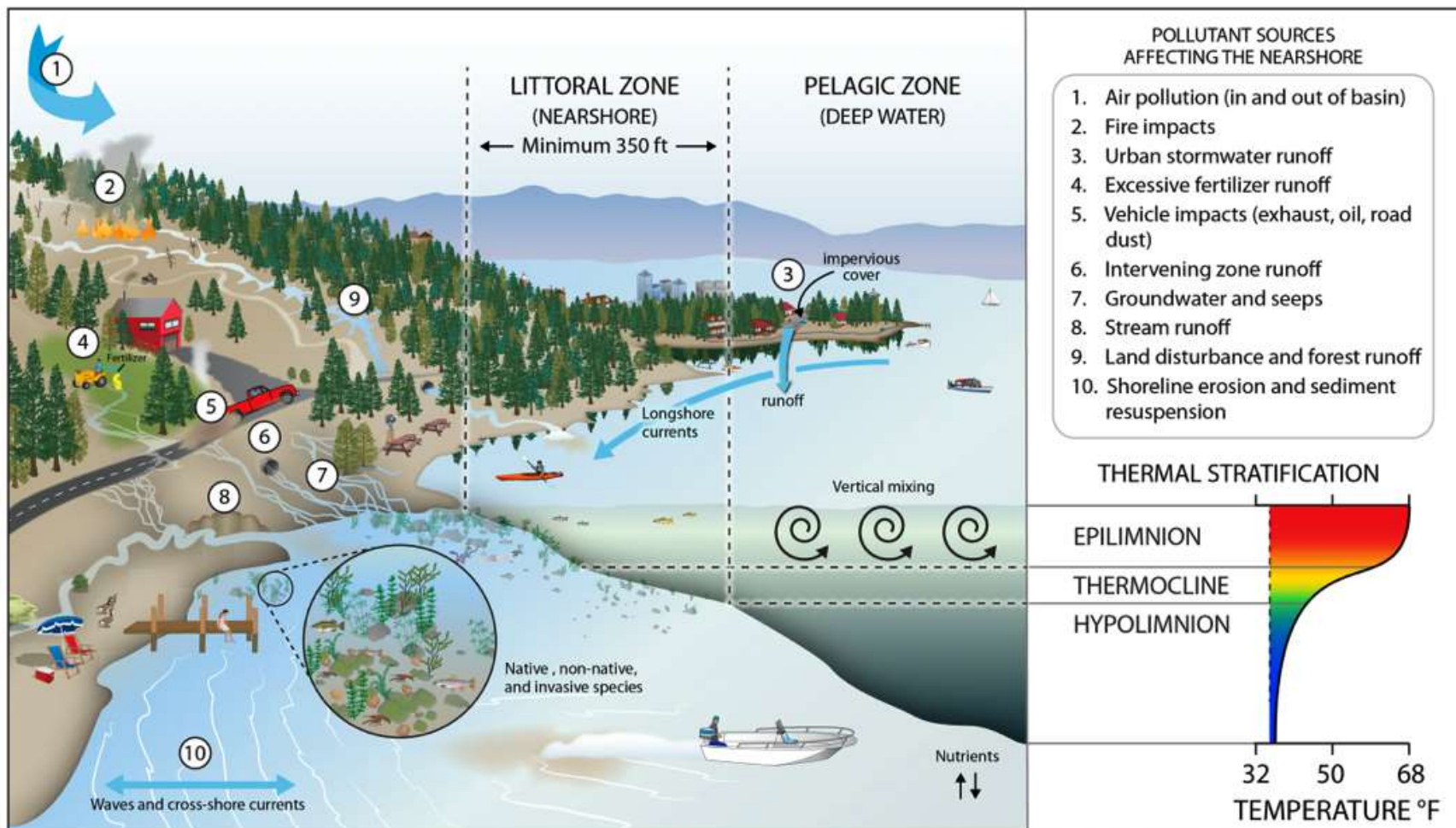
1.6 Conceptual Model

Results from review of available literature and data indicated that nearshore condition can differ widely around the lake based on factors such as adjacent land-use and urban development, non-point pollutant inputs, vicinity to stream inputs, water movement, water depth, substrate type, and other features of the lake bottom (Figure 1-2). Variations in these factors create more localized environmental conditions compared to the open-waters of Lake Tahoe that are more uniform. The nearshore environment is inherently more complex and active than the pelagic zone and it requires a different scale of evaluation and management. Some of these requirements for evaluation are addressed in this report.

A conceptual model of the nearshore was developed to illustrate relevant interactions between the natural and anthropogenic factors that affect important features and conditions of the nearshore. In many respects this nearshore conceptual model is quite similar to the mid-lake conceptual model, but with additional elements that emphasize how pollutants and other material that enter the lake from the watershed or groundwater will eventually be mixed and diluted to some extent in the open-water, these materials can be temporarily concentrated in the nearshore zone resulting in biological responses not typically observed in Lake Tahoe's deep water. In addition to the factors listed above, there are other aspects unique to the nearshore that can contribute to environmental condition, such as greater vulnerability to increased temperature from climate change, and impacts from nearshore recreation (e.g., higher levels of boat activity), domestic animals and wildlife activity, nearshore structures and habitat, and lake level changes.

Generally, the pollutant sources that affect nearshore conditions are the same as those identified in the Lake Tahoe TMDL, so the control measures to address those factors should be similar (Figure 1-3). We did not conduct a quantitative linkage analysis to determine the relative contributions from each potential nearshore pollutant source, as such analysis was beyond the scope of this project, but the science team consensus is largely consistent with previous expectations (TRPA, 1982) that "watershed activities which could alter the quality of the [mid-] lake will affect the littoral zone near the watershed earlier and to a greater extent than they will the open water." Therefore, it is anticipated that nutrient and fine sediment loading reductions that result from implementation of the Lake Tahoe TMDL will not only provide improved mid-lake clarity, but also will provide benefits for clarity and related characteristics in nearshore condition.

LAKE TAHOE NEARSHORE



Illustration, LJ Wable and A Heyvaert (Desert Research Institute), with additional clip art contributions courtesy of the Integration and Application Network, University of Maryland Center for Environmental Science (ian.umces.edu/symbols/).

Figure 1-2. Illustration of important factors and processes affecting the lake nearshore environment.

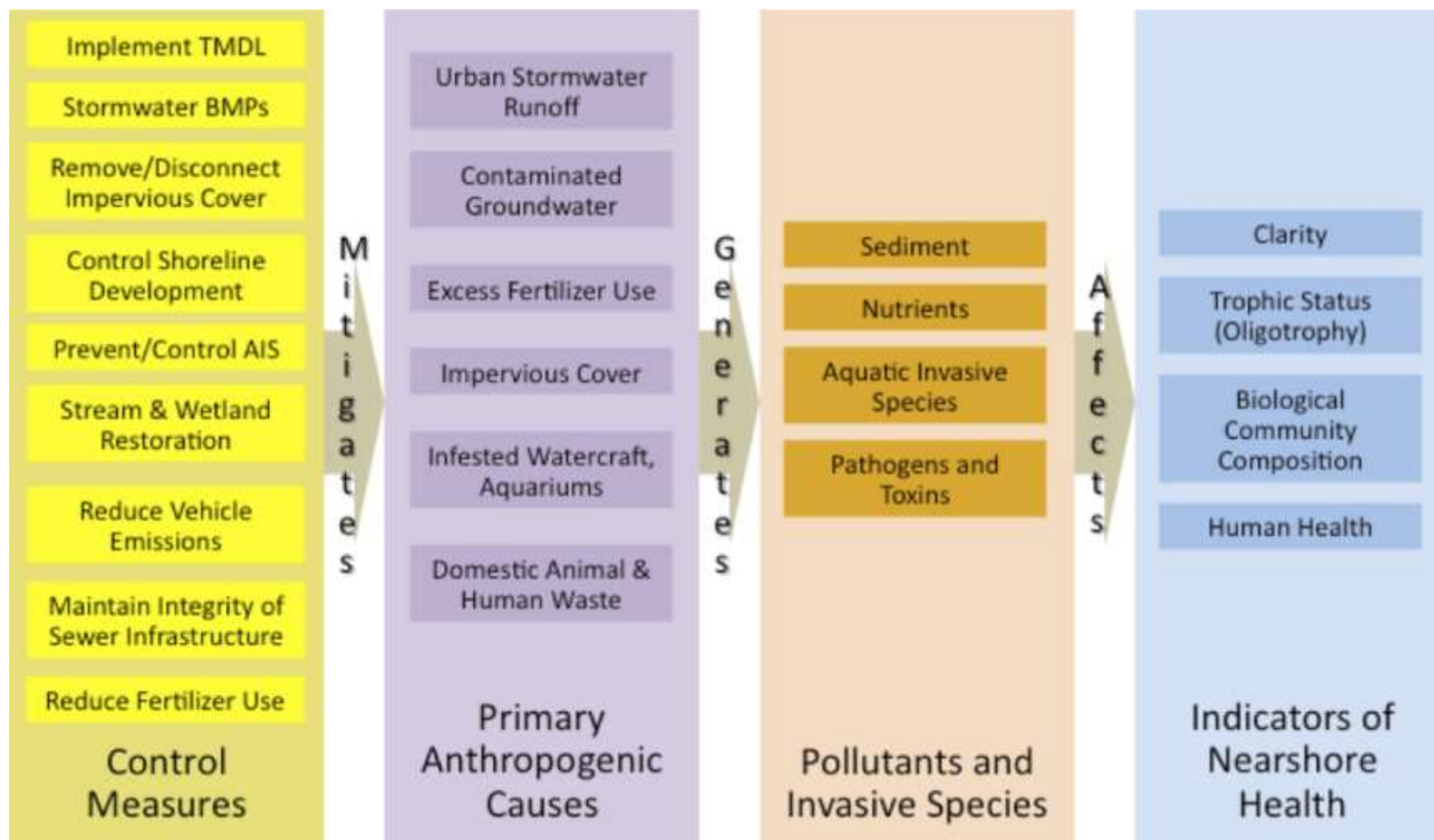


Figure 1-3. Examples from the nearshore conceptual model of progression from relevant control measures to indicators of nearshore health.

It must be acknowledged, however, that nearshore water quality is strongly influenced by localized pollutant input, so a load reduction that may improve the open-water may or may not have a directly comparable effect on all nearshore areas. For example, while load reductions along the south shore will contribute to an eventual improvement of open water clarity and a more immediate effect on that region's nearshore, its direct effect on the nearshore zone in the north lake may be delayed or attenuated. Water quality improvement projects should be selected to include those that (1) will have the most influence on both the nearshore and open water, and (2) are located in areas around the lake where measures of nearshore conditions indicate impairment.

While AIS may preferentially establish in some nearshore areas as a result of nearby watershed condition, this is not always the case, and once established they may not respond to watershed management activities. The establishment of invasive aquatic species in nearshore areas can precondition those areas for the introduction and establishment of subsequent undesired species by changing substrate and habitat conditions.

1.7 Evaluation of Existing Thresholds and Standards

An initial compilation of existing environmental standards and thresholds from California, Nevada and the TRPA consisted of 62 standards that were potentially applicable to Lake Tahoe's nearshore zone (see Report Appendix A). Some of these standards consisted of very specific numeric criteria while others were more general narrative statements. Several standards were consistent across agencies in terms of their specific characteristics and/or criteria, although some numerical criteria were not in alignment across all agencies.

The full set of 62 individual standards and thresholds was sorted into 38 categories based on related characteristics (see Report Appendix B). Then each of these categories was reviewed in terms of its relevance to monitoring and management of the nearshore at Lake Tahoe, with a brief narrative description and data assessment, as well as preliminary comments on reference conditions and whether the standard or threshold was sufficient to support desired conditions. These categories were then classified on the basis of (1) relevancy for nearshore assessment, and (2) relevancy to nearshore management for desired conditions. Nutrient loading standards, for example, are important for nearshore management since they fuel both phytoplankton and periphyton growth. Measurement of nutrient concentrations in the nearshore, however, is less relevant for assessing nearshore conditions because these concentrations can be quite ephemeral, with high input levels quickly reduced due to rapid algal uptake, sometimes yielding an apparent inverse relationship between nutrients and algal growth. The few available historic studies have not reported large and consistent differences in the spatial or temporal distribution of nutrient concentrations around the lake perimeter. Monitoring nutrient loading onshore is very important, however, and should be carried out as

part of a Tahoe regional stormwater monitoring program, in which the derived data from that program links to nearshore monitoring results.

Finally, a list of categories from nearshore standards was assembled that represented the attributes deemed as most “important” or “relevant” for assessing the achievement of nearshore desired condition. In turn, each of these categories of standards, as well as a few additional attributes, were linked to one or more of the four distinct nearshore indicators: clarity, trophic status, community structure (biological integrity), and conditions for human health. These formed the basis for design of the nearshore monitoring framework.

1.8 Design of the Nearshore Monitoring Framework

From the list of “important” or “relevant” categories for nearshore condition assessment, ten were selected to serve as primary metrics, with each metric representing a specific measurable response to anthropogenic impacts and to management actions taken to achieve objectives set forth for the nearshore desired condition. The benefit of this approach is that nearshore condition is not viewed as a series of individual standards subject to attainment determination, but rather as an interacting system of interdependent environmental factors evaluated on the basis of ecologically integrative response variables (Figure 1-4).

Consistent with the desired condition statement, four nearshore indicators were selected to provide a summary assessment on unique characteristics of the system. Obviously, the exceptional clarity for which Lake Tahoe has been long renowned is one of those unique characteristics extending to clear waters in the nearshore. Trophic status represents the amount of biological growth a system supports, generally reflected by very low algal biomass and low nutrient concentrations in Lake Tahoe. Community structure characterizes the aquatic species composition (richness), abundance and distribution. Nearshore conditions for human health are directly relevant to maintaining expected standards for safety and healthy recreational use of the lake.

Each metric associated with these indicators represents a key component of the nearshore ecosystem, as described below, and contributes to an integrated perspective on the health of the system. The traditional measure of Secchi disk clarity used in deep waters at Lake Tahoe does not function for the nearshore because water transparency can extend beyond the depth limits defined as nearshore. Instead, turbidity and transmissivity (light transmittance) are recommended as appropriate metrics for evaluating the nearshore clarity. Turbidity directly relates to existing nearshore standards (TRPA, CA and NV), but is not sufficiently sensitive to document visible changes in the nearshore at low range values typical of undisturbed areas. In these cases, transmissivity is a superior metric, but it has a shorter history of measurement in Lake Tahoe and does not currently link to existing standards.

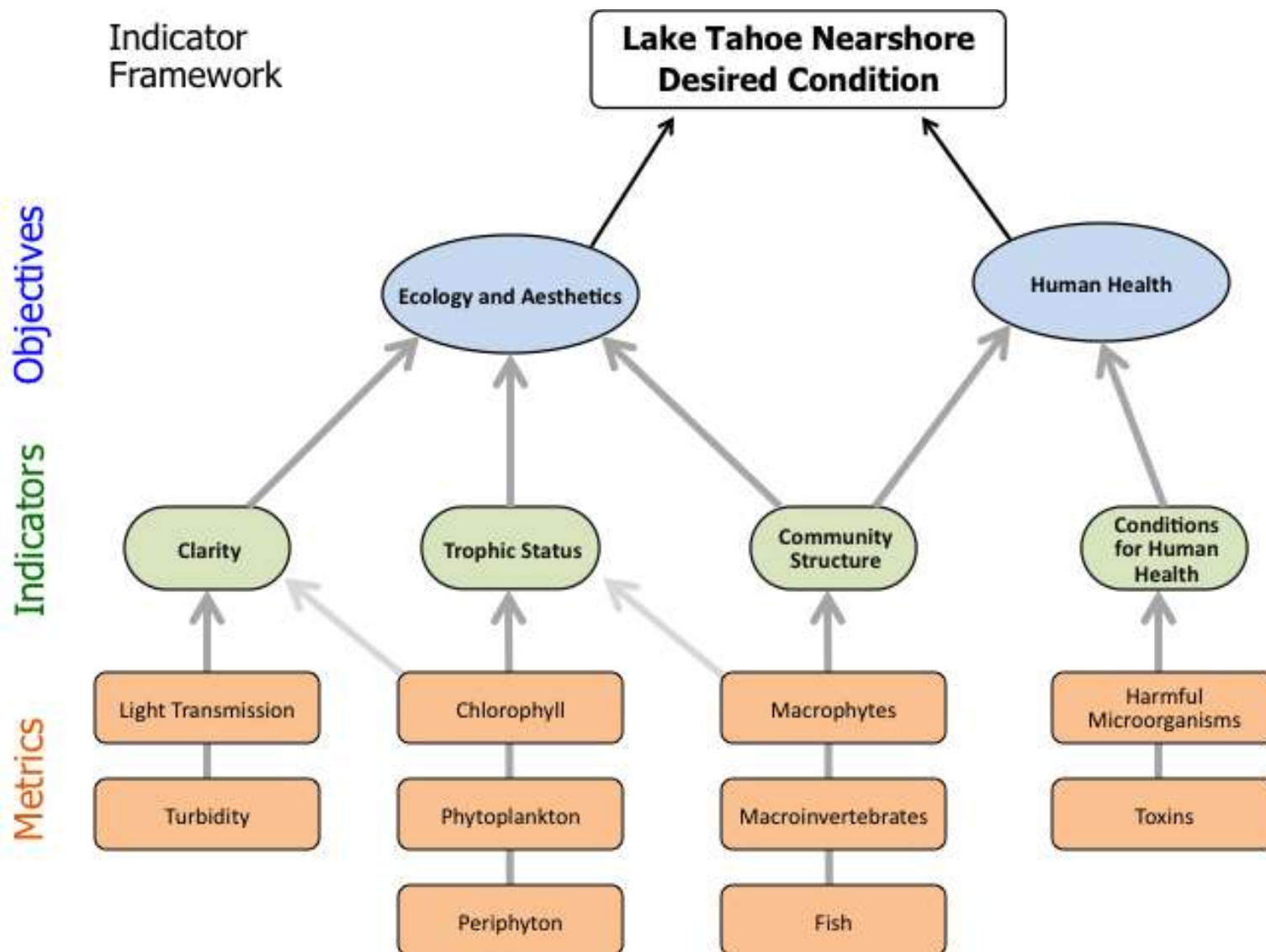


Figure 1-4. Simplified diagram of the Lake Tahoe nearshore monitoring framework, showing associations between metric data, aggregate indicators of condition, and nearshore objectives.

Chlorophyll concentration is a traditional measure of algal biomass (i.e. the concentration of algae in the water). Used in conjunction with an algal growth potential metric and phytoplankton (free-floating algae) identification, it provides a complete picture of trophic status (a measure of the biological productivity of a water body). The algal growth potential metric test uses chlorophyll measurements to determine how much algal growth can be supported by available nutrients in the water, and is more reliable than simply measuring nutrients at the very low concentrations typical in this lake. Phytoplankton counts, biomass, and algal growth potential each represent existing standards for the pelagic (deep) waters that are also consistent with evaluating nearshore conditions. Measurement of attached algae (periphyton), however, is unique to the nearshore. It is this tangible feature of the nearshore that individuals often perceive as evidence of undesirable conditions. The abundance and distribution of attached algae is variable in space and time and consequently difficult to measure in a representative manner. Fortunately, there is a long history of periphyton measurement at Lake Tahoe, which supports a robust analysis of spatiotemporal distributions and the potential development of appropriate targets or standards.

Macrophytes, macroinvertebrates, and fish are visible aquatic organisms that interact to create the habitats and diversity representative of Lake Tahoe's nearshore ecosystem. They also indirectly affect trophic status and in some cases with invasive species may contribute to diminished clarity of nearshore environments. This is one of the potential issues associated with changes in community structure resulting from the introduction of aquatic invasive species, as well as the inherent threat posed to native species and some endemic species by undesired nonnative species introductions. Nearshore surveys for each of the biological groups listed above will provide information needed for establishing suitable reference conditions and for detecting the spread or introduction of aquatic invasive species.

The proposed monitoring design includes full perimeter surveys conducted on a seasonal basis (four times per year) for turbidity, transmissivity, fluorescence (relative chlorophyll) and chlorophyll *a*, coordinated with location-based assessments of periphyton (attached algae), phytoplankton (free-floating algae), benthic macroinvertebrates, aquatic plants (macrophytes) and higher-level aquatic species that include fish and crayfish. For this initial monitoring effort, sampling four times per year should be considered a minimal effort; adjustments in sampling design may be considered as we improve our understanding of seasonal to annual variation in measurements and as funding allows over the long-term.

Measurements of turbidity, transmissivity, and relative chlorophyll are all done simultaneously, so there is minimal additional cost associated with each metric beyond the first parameter. During these perimeter circuits discrete samples will be collected for phytoplankton, absolute chlorophyll *a* concentration (and nutrients on occasion as secondary metrics) at

specified locations based in part on the longer-term range of responses observed in contiguous perimeter surveys. Initially, however, these discrete samples will be collected at ten locations in close proximity to established periphyton sampling sites or where some of the earliest studies were conducted from 1969–1974.

Attached algae abundance (periphyton biomass) is one of the more evident manifestations of changes in nearshore condition. It responds to lake conditions seasonally, so the sampling schedule is designed to track growth patterns that yield estimations of mean annual biomass. This sampling schedule follows existing routines and protocols, with site monitoring for periphyton biomass conducted 4-6 time per year at nine established locations and one additional spring synoptic conducted to assess biomass at forty locations around the nearshore.

Native and non-native aquatic plants would be monitored every other year on both a perimeter presence/absence and a relative abundance basis to detect changes and indicate potential effects of aquatic invasive plants on biological integrity. The macroinvertebrates would be monitored on a seasonal basis two times per year to detect shifts in community structure and impacts from environmental change. Detailed analysis of macroinvertebrate composition, distribution and abundance (CDA) obtained from samples collected at eleven sites will represent conditions over a range of substrates and including potential impacts from aquatic invasive species. This monitoring would be coordinated with efforts of the Lake Tahoe AIS Working Group.

Different fish species and crayfish migrate in and out of the nearshore seasonally, so these surveys should be conducted seasonally, four times each year, at eleven locations, and also during early summer at forty-nine spawning sites. The CDA analysis of fish and macroinvertebrate samples provides an assessment of changes in the aquatic community that will contribute to detection of AIS and evaluation of impacts on biological integrity. Again this monitoring would be integrated with efforts of the Lake Tahoe AIS Working Group.

Monitoring in the nearshore for harmful microorganisms or toxins that affect human health is proposed to be coordinated between the Lake Tahoe water quality agencies and local water purveyors. For example, samples for analysis of coliforms and *E. coli* are currently collected at beaches during recreational periods by regulatory agencies and some members of the Tahoe Water Suppliers Association. These programs are expected to continue in accordance with established state and federal requirements for the protection of drinking water, swimming, and other recreational activities. While chemical toxins are not generally considered an issue of concern at Lake Tahoe, any incident of localized chemical or sewage spills would require a rapid response monitoring assessment, which is outside the purview of routine monitoring.

1.9 Evaluation of Metrics for Reference Conditions and Standards Assessment

The primary metrics proposed for nearshore monitoring and condition assessment are presented and developed individually in this report. Each metric presentation begins with a brief review of its monitoring history at the lake, followed by an analysis of the available data, and then a discussion of potential standards and reference conditions (where applicable). It is important to distinguish between reference conditions and standards, because they are not necessarily synonymous.

Reference conditions represent a narrative or numeric description of a specific characteristic in the relative absence of human influence. They are used to inform a dialogue that establishes realistic targets or standards for effective management of an ecosystem to achieve desired conditions. In some cases of metric evaluation there were no available data on reference condition, or quite often the data available were too sparse to do more than provide a general sense of variation in reference condition. The following table summarizes our evaluation of data status for each of the proposed metrics (Table 1-1). The data quality itself is generally quite good, but the quantity is often insufficient to inform a detailed assessment. Given the general lack of nearshore data existing for most of these metrics, any discussion of standards and reference conditions is considered preliminary at this time. The exceptions are for periphyton and perhaps for turbidity, where longer-term nearshore monitoring has been conducted (although not as part of any regular program in the case of turbidity). The reference values presented in this report characterize conditions in the relative absence of human activities, and are considered representative of the unique attributes consistent with oligotrophic conditions in the nearshore of Lake Tahoe.

Future revision to existing standards or the development of new standards and thresholds should be linked directly to these recommended metrics and indicators. The data and the evaluations presented in this report will provide an essential scientific basis for these discussions and potential resulting actions.

Table 1-1. Summary of proposed nearshore metrics showing the relative availability of existing data for evaluation of existing state or TRPA standards, and to support linkage to specific numeric objectives.

Nearshore Metric	Associated Indicator	Data Basis	Link to Existing State or TRPA Standards
Turbidity	Clarity	Moderate	CA, NV, TRPA (Clarity)
Light Transmissivity	Clarity	Poor	CA, NV, TRPA (Clarity)
Chlorophyll	Clarity and Trophic Status	Moderate	CA (Biological Indicators)
Phytoplankton	Trophic Status	Poor	CA (Plankton Counts and AGP)
Periphyton	Trophic Status and Community Structure	Good	CA (Biological Indicators)
Macrophytes	Trophic Status and Community Structure	Poor	None
Macroinvertebrates	Community Structure	Poor	TRPA (Littoral Habitat)
Fish and crayfish	Community Structure	Poor	TRPA (Littoral Habitat)
Toxins	Human Health	Poor	CA, NV (CA Toxics Rule and Toxicity)
Pathogens	Human Health	Moderate	CA, NV (Bacteria)

1.10 Implementation of the Nearshore Monitoring Program

In designing the nearshore monitoring framework it was relevant to consider it in the context of other efforts in the Lake Tahoe Basin to reduce redundancy in monitoring efforts and to maximize monitoring investments. At Lake Tahoe, the central focus of water quality monitoring to date has been on characterizing conditions of Lake Tahoe’s deep-water clarity and the nearshore periphyton. The monitoring described in this report will aid in guiding the implementation of additional nearshore monitoring efforts, while also intersecting with other monitoring programs (e.g., tributary monitoring and urban stormwater monitoring). Although these other programs were not addressed as part of the nearshore monitoring design, it is expected they will provide much of the ancillary data needed to explain variation in nearshore conditions, assuming they are concurrently implemented (Figure. 1-5).

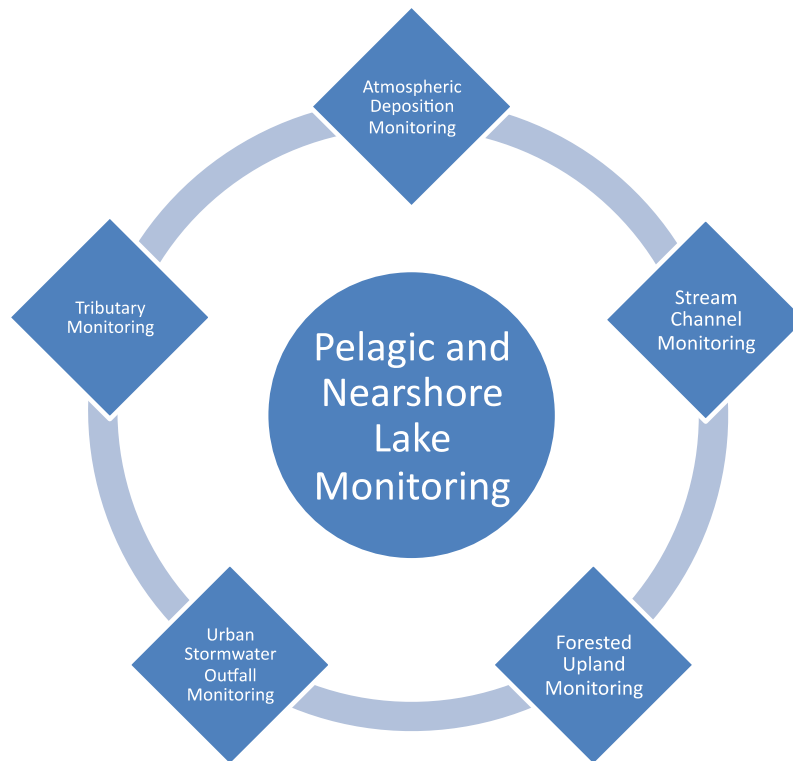


Figure 1-5. A generalized representation of other monitoring efforts anticipated in the Lake Tahoe Basin that would intersect with the nearshore monitoring program.

The nearshore monitoring framework is intended to answer key questions associated with both spatial and seasonal patterns of conditions in the lake's nearshore region. Its initial implementation will address the multiple dimensions of physical, chemical and biological characteristics in the nearshore to evaluate inherent variation within these parameters, especially in the cases of metrics and indicators for which little or no standardized monitoring data are currently available. For these indicators and metrics, subsequent data analysis and evaluation are expected to provide the basis for adjustments to initial monitoring design that will lead to improvements and a cost-efficient monitoring program (e.g., with optimal sampling frequency and locations). As a starting point, this initial monitoring framework is intended to provide the data needed to satisfy immediate management information needs for an evaluation of nearshore conditions, as well as to inform preliminary discussion on standards, and to inform progressive adjustments to the monitoring design and metric evaluation.

In most cases the metrics derive from or contain important elements of the standards reviewed in this report, although some additional attributes are to be measured as well (e.g., chlorophyll, macrophytes, and macroinvertebrates). Ultimately, it may be desirable to revise or replace existing standards with new standards that link directly to the primary nearshore

monitoring metrics. It was beyond the scope of this project, however, to provide the necessary level of analysis required by law to identify new standards, or to eliminate or modify existing standards. Rather, this report provides the scientific background that will help management agencies decide if and where they may want to address changes that would target specific features and metrics of nearshore condition.

A consistently implemented and standardized nearshore monitoring program will be essential to inform these efforts to update existing standards, including the validation of reference conditions, and for describing and confirming the spatial and temporal variation of metrics used to measure nearshore conditions. It will provide the quality and the quantity of data needed for evaluating progress in achieving management and restoration goals. It will also provide the basis for evaluating status and trends, and is designed to be flexible and scalable to accommodate available resources as well as changes in approach, information and techniques.

Taken in aggregate the ten primary metrics should provide a relatively comprehensive evaluation of status and trends for the most important and unique characteristics of the nearshore environment at Lake Tahoe. In some cases, any indication of change in status or trend would initiate an appropriate management or research initiative to address or investigate the specific causative factors and to develop suitable management or policy actions. The monitoring is focused on response variables, being the factors most sensitive and evident to changing biogeochemical conditions affecting the nearshore environment. It is not a research program, although specific questions that may arise in the context of evaluating these metrics could lead to important insights or to focused studies.

Conditions in the lake will continue to change over time as a consequence of changing patterns in land use, recreational activities, climate, species distributions, and other as yet potentially unidentified factors. A regular program of data collection allows the stakeholder community to detect and evaluate these changes in the context of natural variability and desired conditions.

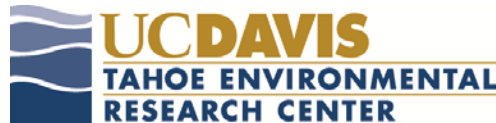
Ultimately, this nearshore monitoring program will be needed to help track anticipated benefits from environmental improvement projects and from loading reductions associated with implementation of the TMDL program. The nearshore areas of lakes are responsive to changing conditions in the watershed, since most external pollutant loading must pass through the nearshore before reaching pelagic open water areas. Therefore, it is expected that nutrient and fine sediment loading reductions will provide not only better mid-lake clarity, for which the TMDL was designed, but also will provide benefits to clarity and other characteristics of the nearshore.

Lake Tahoe Nearshore Evaluation and Monitoring Framework Report

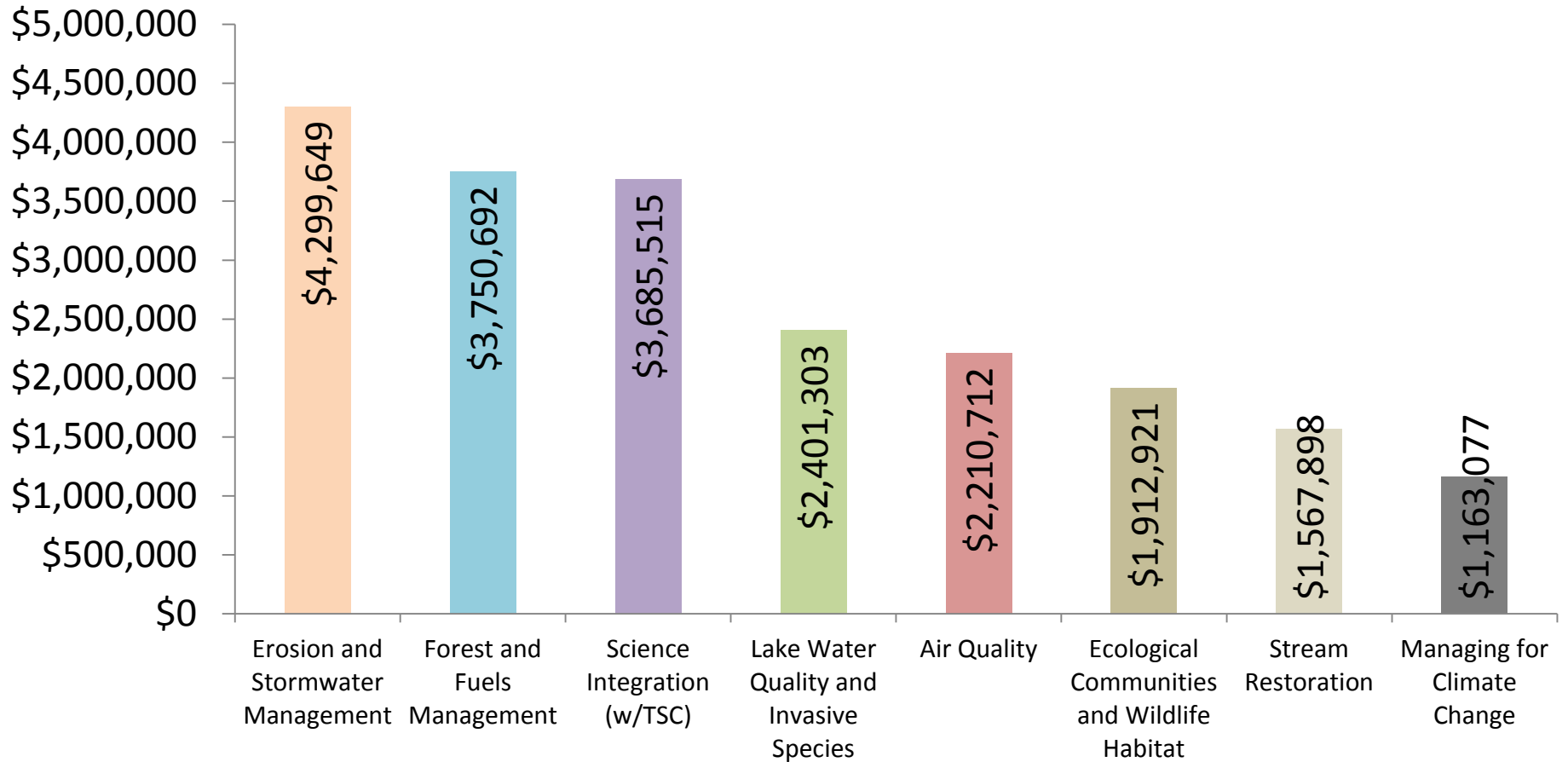
TRPA Governing Board
October 23, 2013

By Alan Heyvaert, Ph.D
Acting Senior Director
Center for Watersheds and Environmental Sustainability,
Desert Research Institute

Directed Action: SNPLMA - Research Round 10



SNPLMA Funded Research (Rounds 7 through 12)



Contemporary Research Efforts - Nearshore

- Inferring littoral substrates, fish habitats, and fish dynamics of Lake Tahoe using IKONOS data; TRPA
- Nearshore clarity at Lake Tahoe: status and causes of reduction; NV LTLP
- Predicting and managing changes in nearshore quality; SNPLMA-Round 7
- Monitoring past, present and future water quality using remote sensing; SNPLMA-Round 7
- Niches: nearshore indicators for clarity, habitat and ecological sustainability; SNPLMA-Round 8, NV-LTLP
- Development of a risk model to determine the expansion and potential environmental impacts of Asian clams; SNPLMA-Round 9, NV-LTLP
- Warm-water non-native fishes in Lake Tahoe; NV-LTLP
- Potential for pathogen growth, fecal indicator growth and phosphorus release under clam removal barriers; SNPLMA-Round 10
- Natural and human limitations to Asian clam distribution and recolonization-factors that impact the management and control in Lake Tahoe; SNPLMA-Round 10, NV LTLP
- Linking on-shore and nearshore processes: nearshore water quality monitoring buoy at Lake Tahoe – phases I and II; NV LTLP
- The ecology of curly leaf pondweed (*Potamogeton crispus*) and the potential for control using bottom barriers; SNPLMA-Round 11
- Testing the survival and growth of quagga mussel in Lake Tahoe; SNPLMA-Round 11
- Evaluation of nearshore ecology and aesthetics (this project); SNPLMA-Round 10

Existing and Current Monitoring

- Lake Tahoe water quality investigations: algal bioassay, phytoplankton, atmospheric nutrient deposition, and periphyton, 2000 to on-going; Lahontan, UC Davis
- Nearshore turbidity monitoring – various and intermittent efforts from 1965 to 2012; UC Davis, DRI, TRPA, NV LTLP
- Tributary pollutant concentration and loading monitoring; TRPA, USGS, USFS, UC Davis, NV LTLP
- Aquatic invasive species – prevention and control measure implementation and effectiveness monitoring; Various
- Shorezone ordinance effectiveness monitoring of gasoline constituents, fecal coliform, *e. coli*, and polycyclic aromatic hydrocarbons; USGS, TRPA, Lahontan
- Watercraft monitoring; TRPA
- Deep water Lake Tahoe Water Quality Monitoring, TRPA, UC Davis
- Urban stormwater pollutant monitoring (nearing implementation); Lahontan, USFS, Lahontan, TRCD

Project Overview

Nearshore Science Team (NeST) Contributors

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Nearshore Agency Working Group

Shane Romsos (TRPA), Dan Sussman (LRWQCB), Jason Kuchnicki (NDEP), Jacques Landy (USEPA).

Project Overview

➤ **Background:**

- Long-term monitoring monitoring and assessment in Lake Tahoe historically focused on mid-lake conditions
- Current nearshore monitoring insufficient to detect changing conditions
- Needed a comprehensive, integrated assessment of existing conditions

➤ **Project Objectives:**

- Review and synthesize existing research and monitoring data
- Document understanding of nearshore conditions
- Review standards and indicators for relevance to nearshore condition
- Develop an integrated nearshore monitoring design

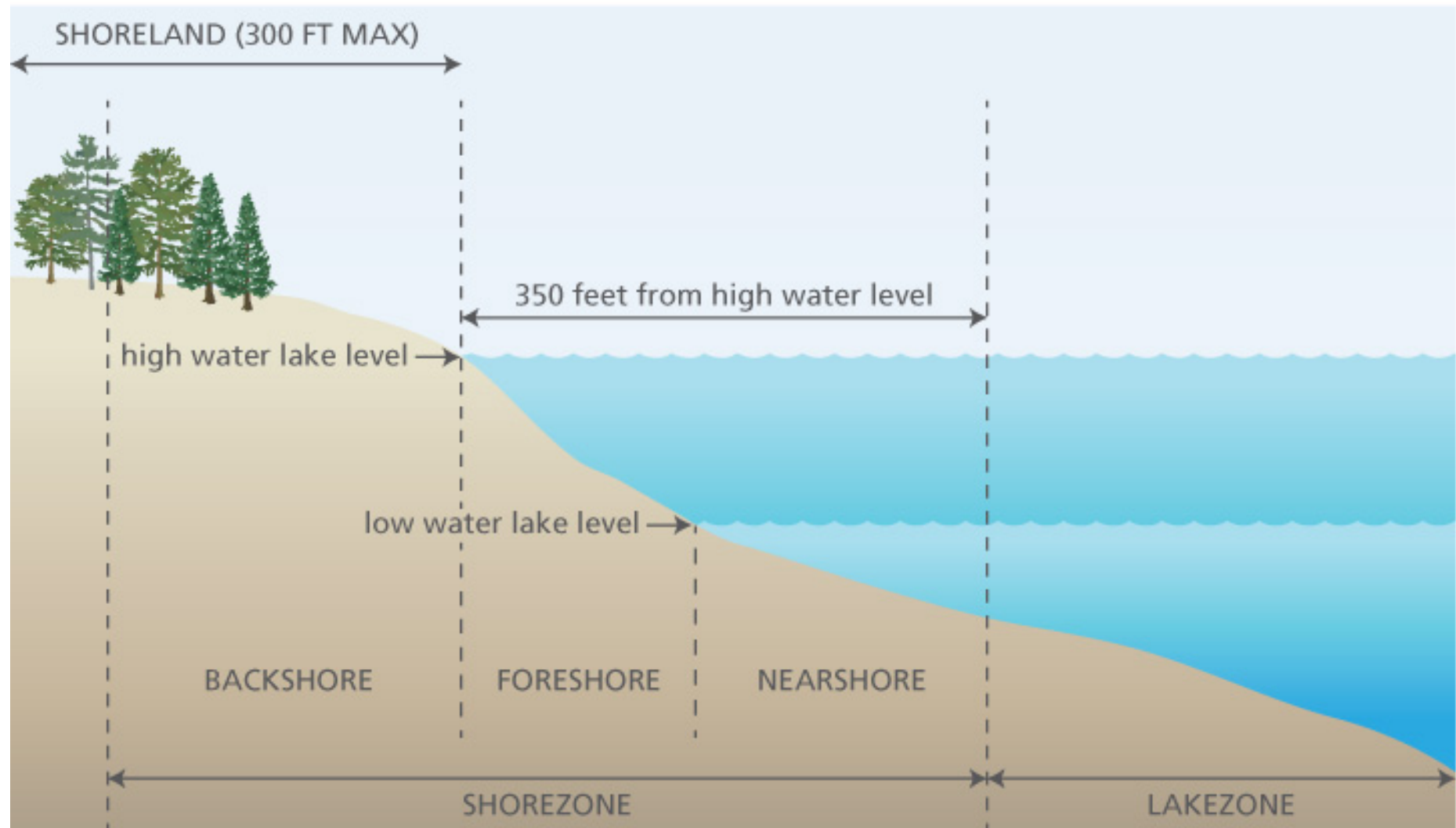
➤ **Major Products:**

- Conceptual model and indicator framework
- Assessment of exiting standards and indicators
- Nearshore monitoring and evaluation framework

Nearshore Desired Condition Statement

Lake Tahoe's nearshore environment is restored and/or maintained to reflect conditions consistent with an exceptionally clean and clear (ultra-oligotrophic) lake for the purposes of conserving its biological, physical and chemical integrity, protecting human health, and providing for current and future human appreciation and use.

Definition of shorezone areas (TRPA 2010)



Lake nearshore area as defined by TRPA (2010).

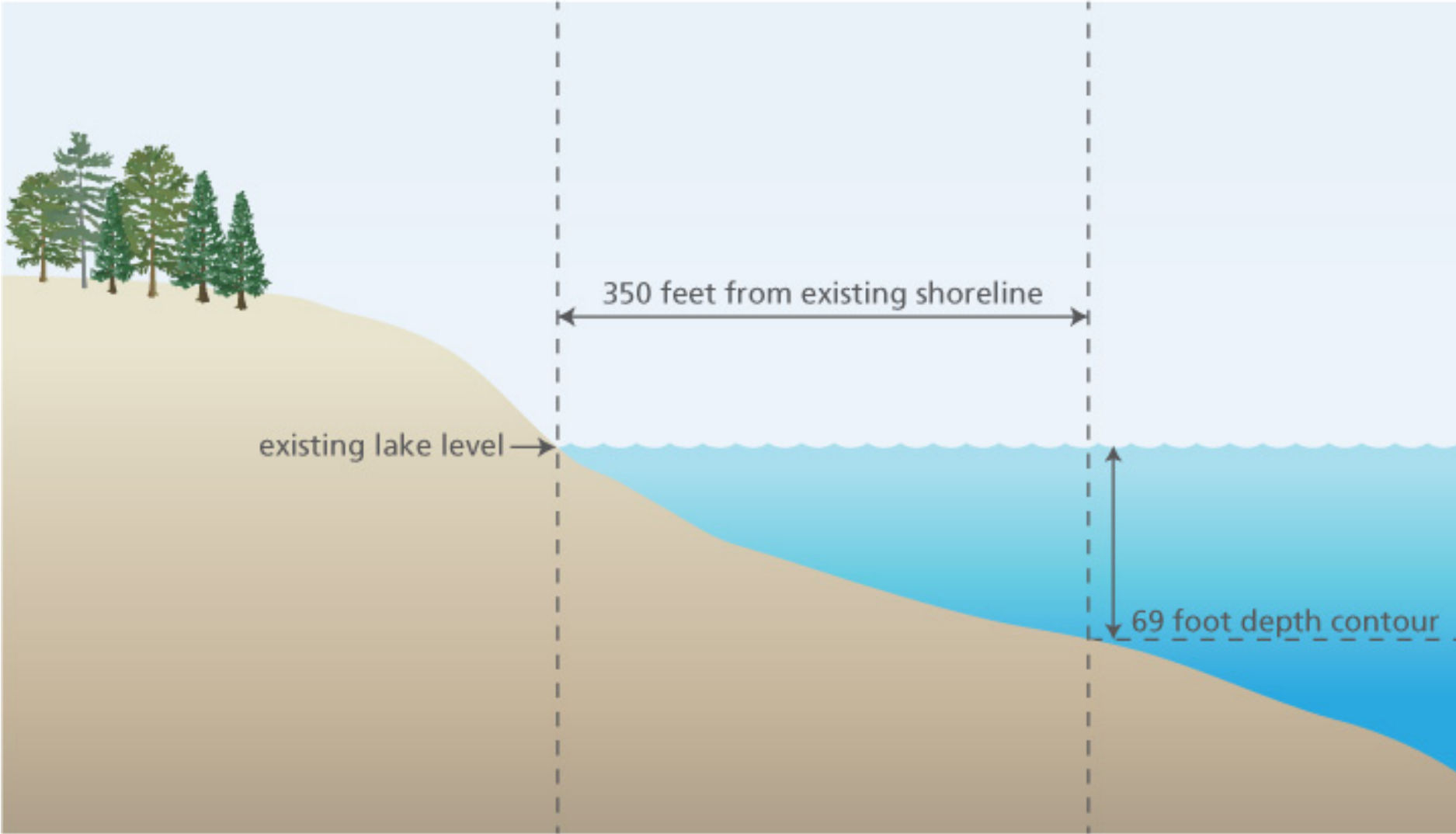
Nearshore Definition (Monitoring)

We do not recommend any changes to existing state and TRPA legal or statutory definitions of the Lake Tahoe nearshore. Rather, the following definition addresses unique aspects of the nearshore in the context of framing our monitoring design.

Lake Tahoe's nearshore for purposes of monitoring and assessment is considered to extend from the low water elevation of Lake Tahoe (6223.0 feet Lake Tahoe Datum) or the shoreline at existing lake surface elevation, whichever is less, to a depth contour where the thermocline intersects the lake bed in mid-summer; but in any case, with a minimum lateral distance of 350 feet lakeward from the existing shoreline.

- The 31-year average August (maximum) thermocline depth in Lake Tahoe is 21 m (69 feet).

Definition of nearshore zone for monitoring and assessment (NeST 2012)



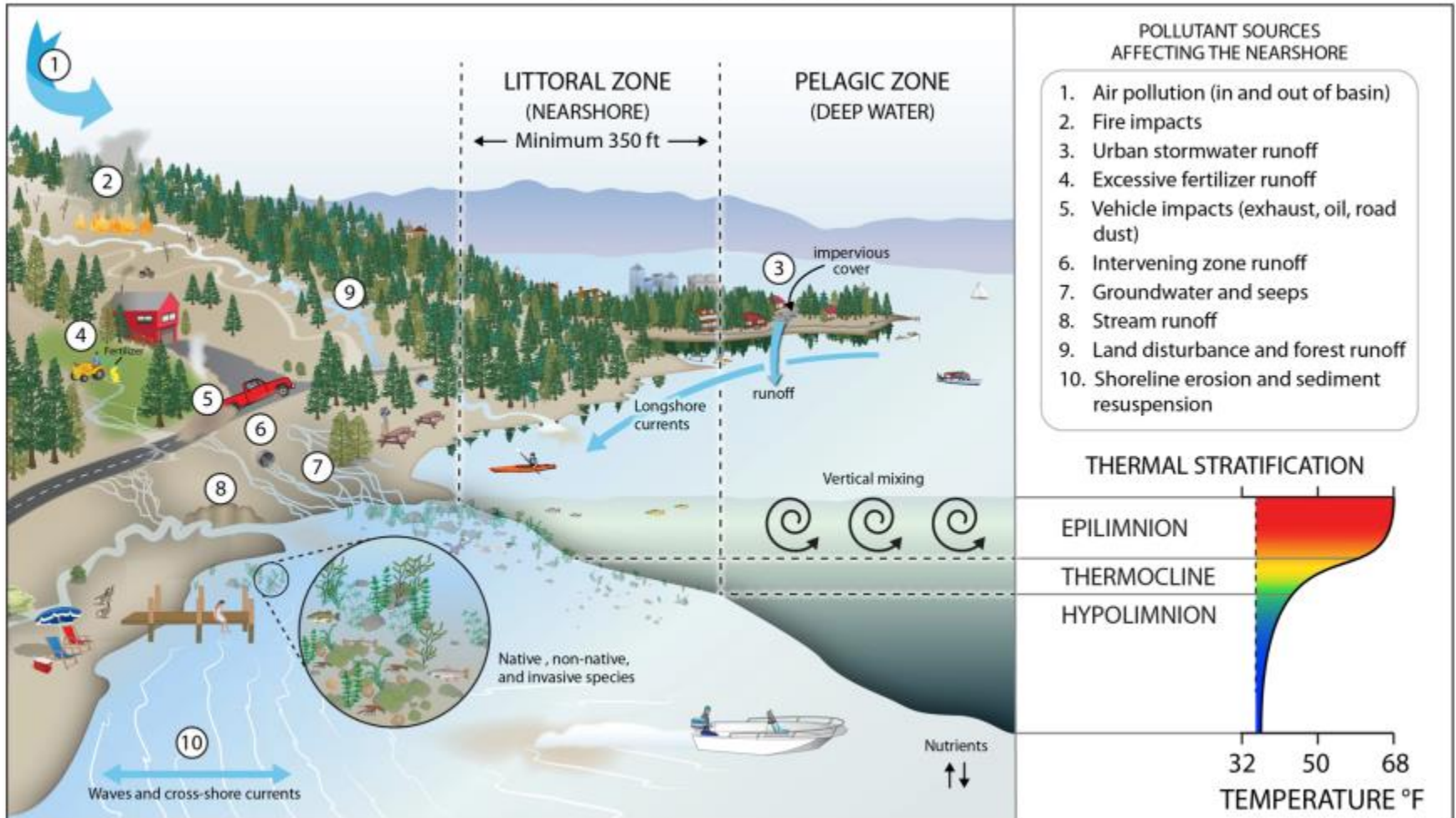
Lake nearshore area for monitoring and assessment (NeST, 2012), defined at the summer thermocline depth (typically 69 feet) or at 350 feet from the shoreline, whichever is greater. The depth and minimum lateral distance are taken from existing lake level rather than the high water level.

Natural rim (6223 feet)
350 foot from rim
30 foot depth contour
69 foot depth contour



Illustration of Factors Affecting Nearshore

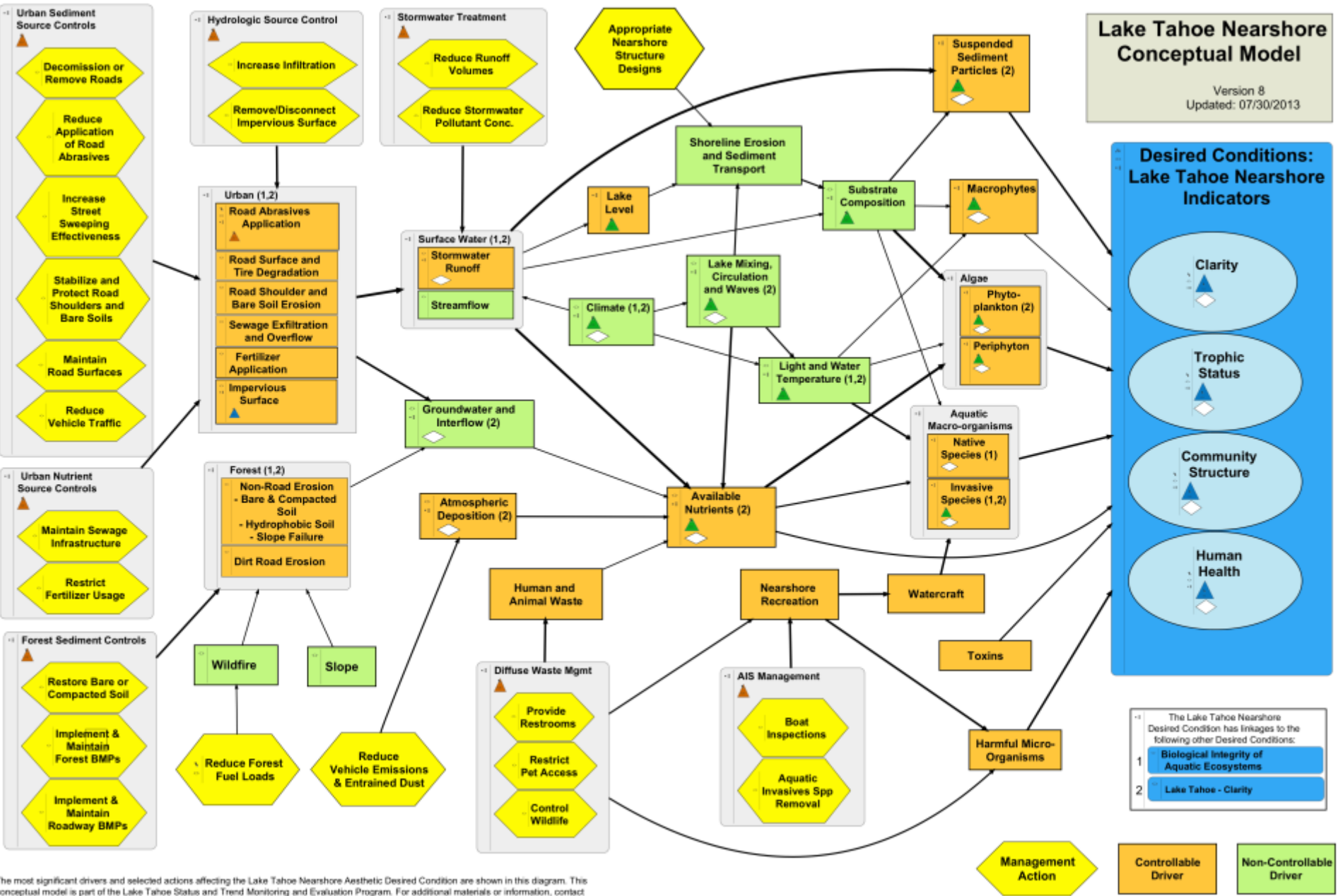
LAKE TAHOE NEARSHORE



Illustration, L.J. Wable and A. Heyvaert (Desert Research Institute), with additional clip art contributions courtesy of the Integration and Application Network, University of Maryland Center for Environmental Science (ian.umces.edu/symbols/).

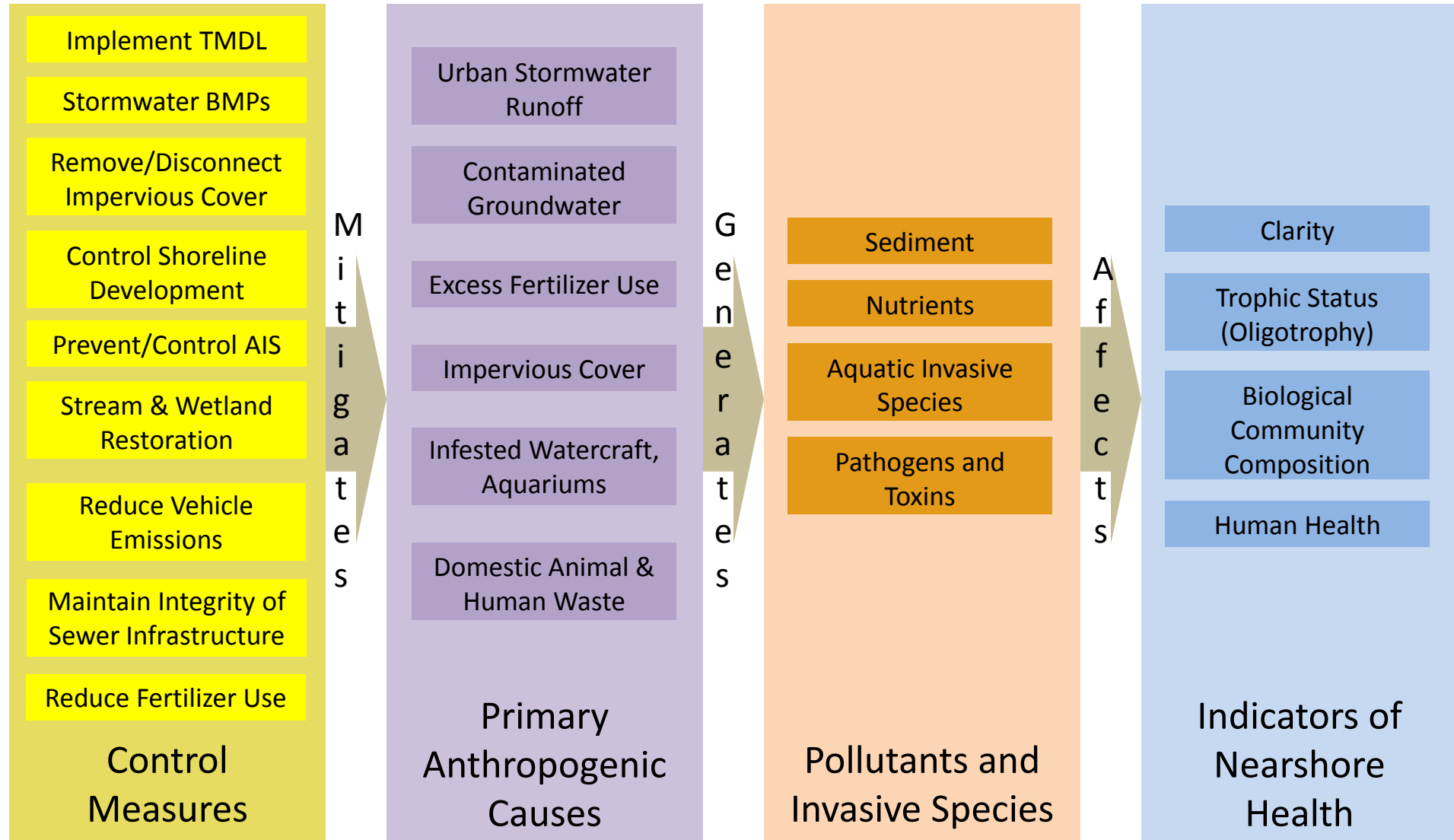
The nearshore conceptual model:

Lake Tahoe Nearshore Conceptual Model
Version 8
Updated: 07/30/2013



The most significant drivers and selected actions affecting the Lake Tahoe Nearshore Aesthetic Desired Condition are shown in this diagram. This conceptual model is part of the Lake Tahoe Status and Trend Monitoring and Evaluation Program. For additional materials or information, contact Shane Romsos, TRPA, email: sromsos@trpa.org, 775.589.5201.

Important Controllable Factors that Influence Nearshore Conditions



Assessment of Existing Standards

1. Agency representatives provided a list of existing standards (TRPA, NDEP, LRWQCB).
2. NeST sorted these standards into 38 categories with internally similar characteristics (Appendix A).
3. Brief narrative descriptions were developed for each category, along with preliminary data assessment (from bibliography) and a discussion of relevance to nearshore management or monitoring (Appendix B).
4. Categories of existing standards deemed “important” or “relevant” to both nearshore management and nearshore monitoring were carried forward and aggregated into a smaller list of internally related characteristics (Table 6-1).
5. A subset of this attribute list provided the basis for design of the integrated nearshore monitoring framework.

Table B-1. Existing Standards Potentially Relevant to the Nearshore of Lake Tahoe.

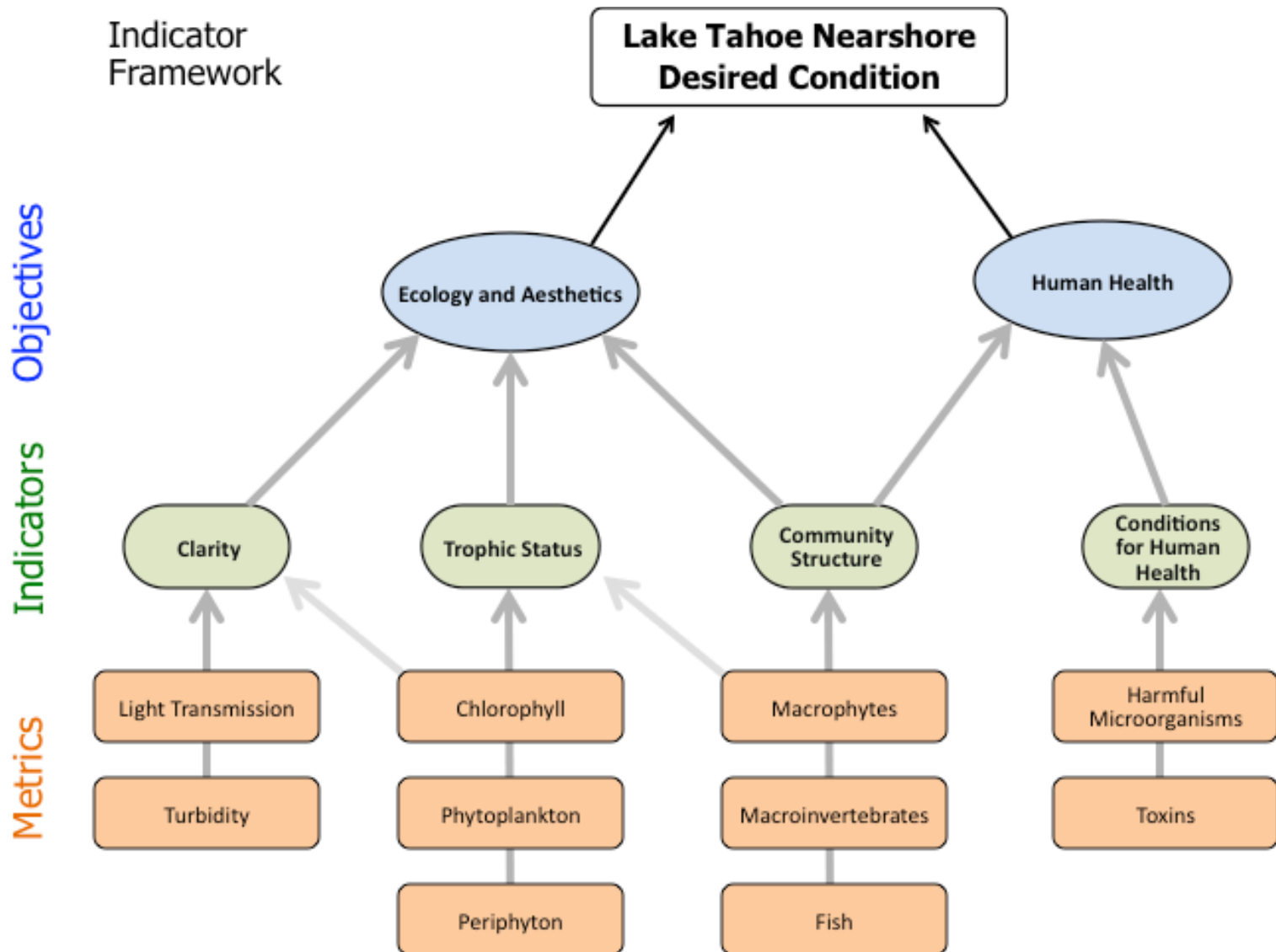
ID #	Parameter Category	Nearshore Management	Nearshore Monitoring
1	Total Nitrogen	Important	Relevant
2	Total Soluble Inorganic Nitrogen	Important	Relevant
3	Ammonia	Less relevant	Less relevant
4	Nitrite	Less relevant	Less relevant
5	Dissolved Inorganic Nitrogen Loading	(see #8)	(see #8)
6	Total Phosphorus	Important	Relevant
7	Soluble Phosphorus	Important	Relevant
8	Biostimulatory Substances	Important	Relevant
9	Clarity	Important	Important
10	Pytoplankton	Important	Important
11	Algal Growth Potential	Relevant	Relevant
12	Biological Indicators (with Periphyton)	Important	Important
13	Suspended Materials	Important	Relevant
14	Settleable Materials	Less relevant	Less relevant
15	Suspended Sediment Loading	(see #13)	(see #13)
16	Total Dissolved Solids	Relevant	Less relevant
17	Conductivity	Relevant	Less relevant
18	pH	Relevant	Less relevant
19	Sodium Absorption Ratio	Less relevant	Less relevant
20	Chloride	Less relevant	Less relevant
21	Sulfate	Less relevant	Less relevant
22	Boron	Less relevant	Less relevant
23	Chemical Constituents	Less relevant	Less relevant
24	<i>E. coli</i>	Important	Important
25	Coliform Bacteria	Relevant	Relevant
26	Fecal Coliform	Relevant	Relevant
27	Temperature	Relevant	Relevant
28	Temperature Change	Relevant	Relevant
29	Dissolved Oxygen	Relevant	Relevant
30	Aesthetic Condition	(see #9 and #12)	(see #9 and #12)
31	Color	Less relevant	Less relevant
32	Taste and Odor	Relevant	Less relevant
33	Floating Materials	Less relevant	Less relevant
34	Oil and Grease	Less relevant	Less relevant
35	Toxicity	Important	Important
36	Radioactivity	Less relevant	Less relevant
37	Aquatic Communities and Populations	Important	Important
38	Nondegradation	Important	Less relevant

Evaluation of Existing Standards

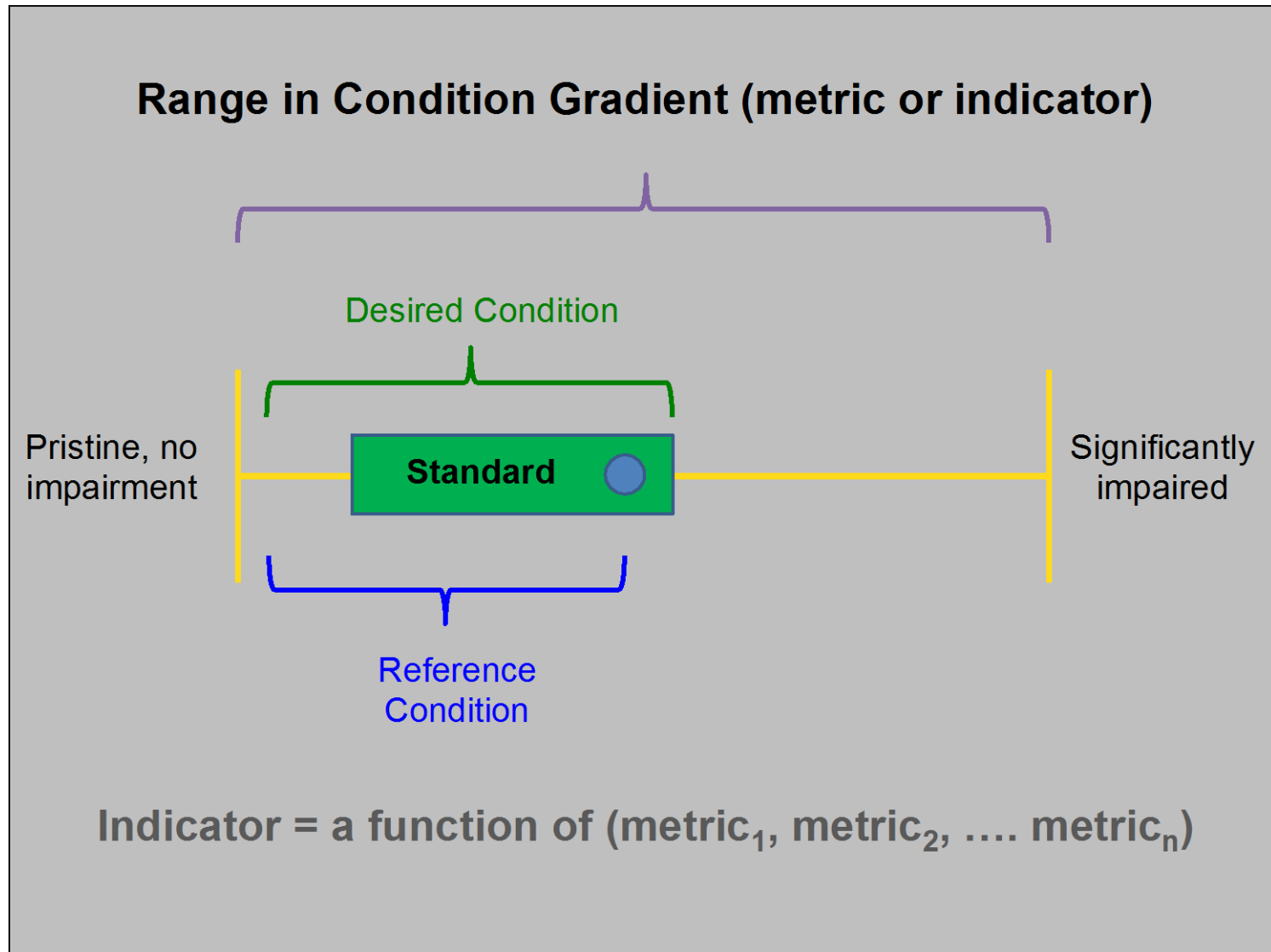
ID	Nearshore Attribute	Categories of Standards¹	Indicator Affiliation
A	Transmissivity	9, 30, 31	Nearshore Clarity
B	Turbidity	9, 30	Nearshore Clarity
C	Suspended Sediment	13, 15	Nearshore Clarity
D	Total Nitrogen	1	Nearshore Trophic Status
E	Total Phosphorus	6	Nearshore Trophic Status
F	Soluble Inorganic Nitrogen	2, 3, 4, 5, 8	Nearshore Trophic Status
G	Soluble Reactive Phosphorus	7, 8	Nearshore Trophic Status
H	Phytoplankton	10, 11	Nearshore Trophic Status
I	Periphyton	11, 12	Trophic Status and Community Structure
J	Toxicity	23, 34, 35	Conditions for Human Health
K	Pathogens	24, 25, 26	Conditions for Human Health
L	Temperature	27, 28, 35	Aquatic Community Structure
M	Community Composition	37, 38	Aquatic Community Structure
N	Chlorophyll	none	Clarity and Trophic Status
O	Macrophytes	none	Trophic Status and Community Structure
P	Macroinvertebrates	none	Trophic Status and Community Structure
Q	Fish and Crayfish	none	Trophic Status and Community Structure

¹ See Appendix B for discussion of referenced standards

Indicator Framework for Monitoring



Standards, Indicators and Metrics



Nearshore Monitoring Data Support for Existing Standards Evaluation or Revision

Nearshore Metric	Associated Indicator	Quality of Data Basis	Link to Existing State or TRPA Standards
Turbidity	Clarity	Moderate	CA, NV, TRPA (Clarity)
Light transmissivity	Clarity	Poor	CA, NV, TRPA (Clarity)
Chlorophyll	Clarity and Trophic Status	Moderate	CA (Biological Indicators)
Phytoplankton	Trophic Status	Poor	CA (Plankton Counts and AGP)
Periphyton	Trophic Status and Community Structure	Good	CA (Biological Indicators)
Macrophytes	Trophic Status and Community Structure	Poor	None
Macroinvertebrates	Community Structure	Poor	TRPA (Littoral Habitat)
Fish and crayfish	Community Structure	Poor	TRPA (Littoral Habitat)
Toxins	Human Health	Poor	CA, NV (CTR and Toxicity)
Pathogens	Human Health	Moderate	CA, NV (Bacteria)

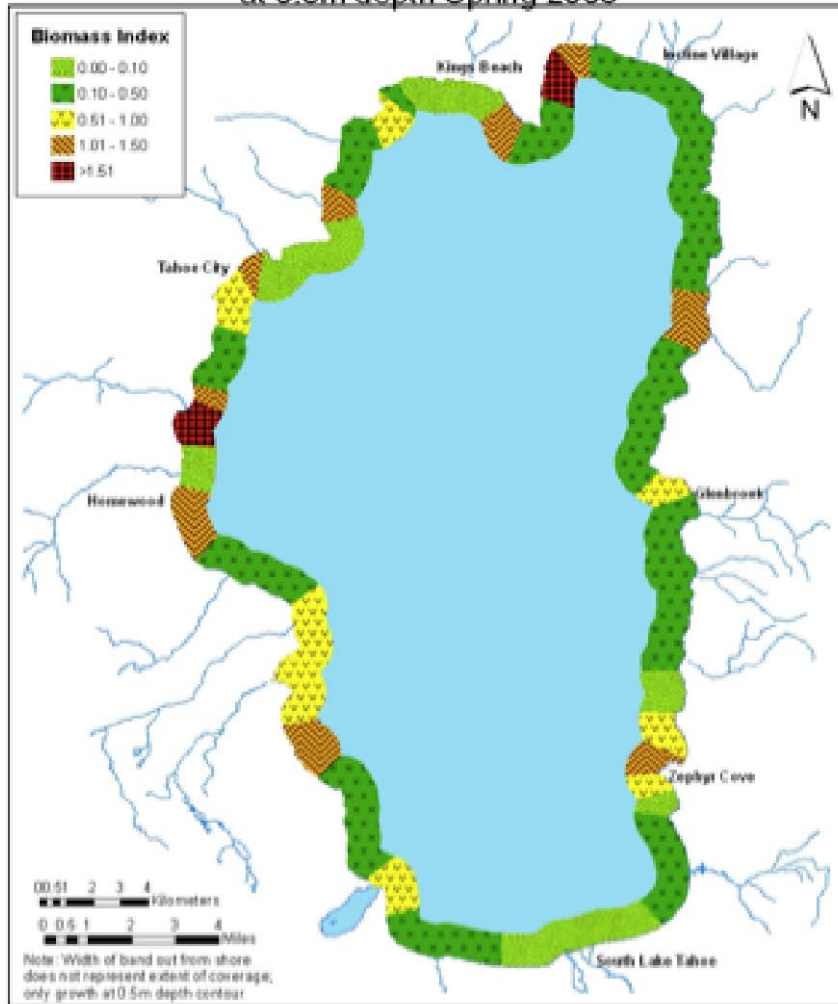
Periphyton

(remember N&P and not fine sediment controls growth)

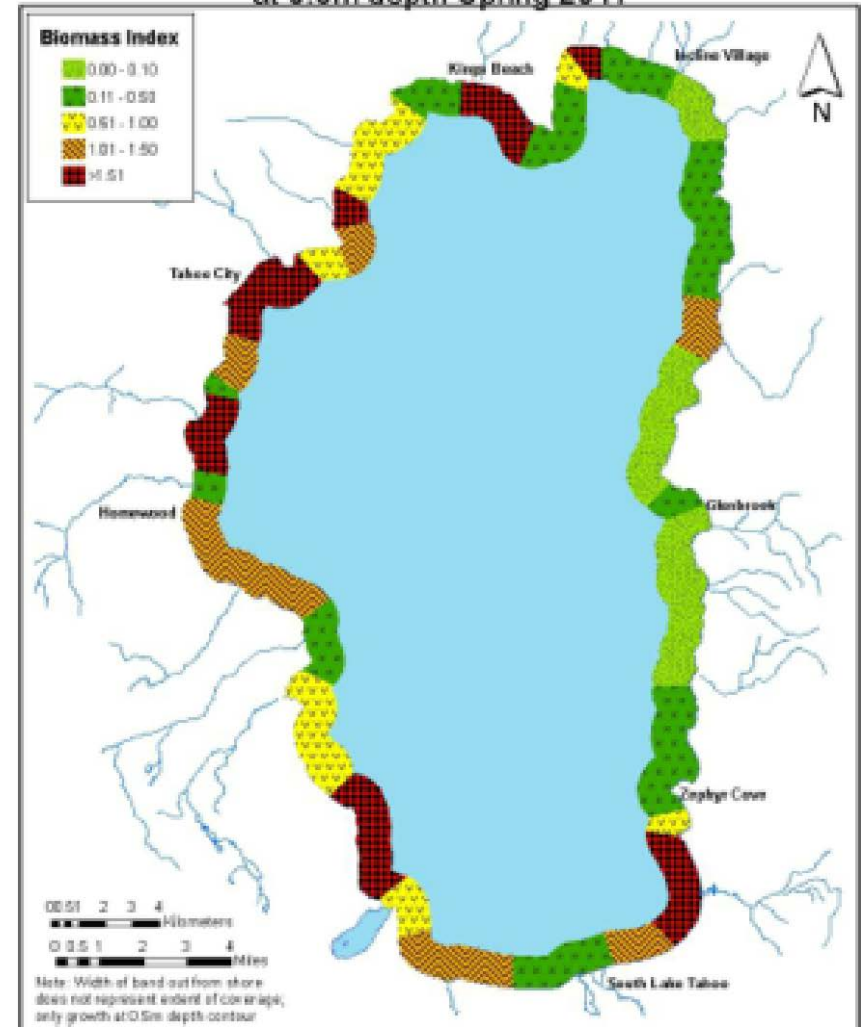


Nearshore Conditions are Spatially and Temporally Variable

Distribution of Periphyton Biomass at 0.5m depth Spring 2008



Distribution of Periphyton Biomass at 0.5m depth Spring 2011



Legend

— Edited Natural Rim

— 69ft Depth

Light Transmissivity
(percent)

0 - 75

75 - 85

85 - 88

88 - 91

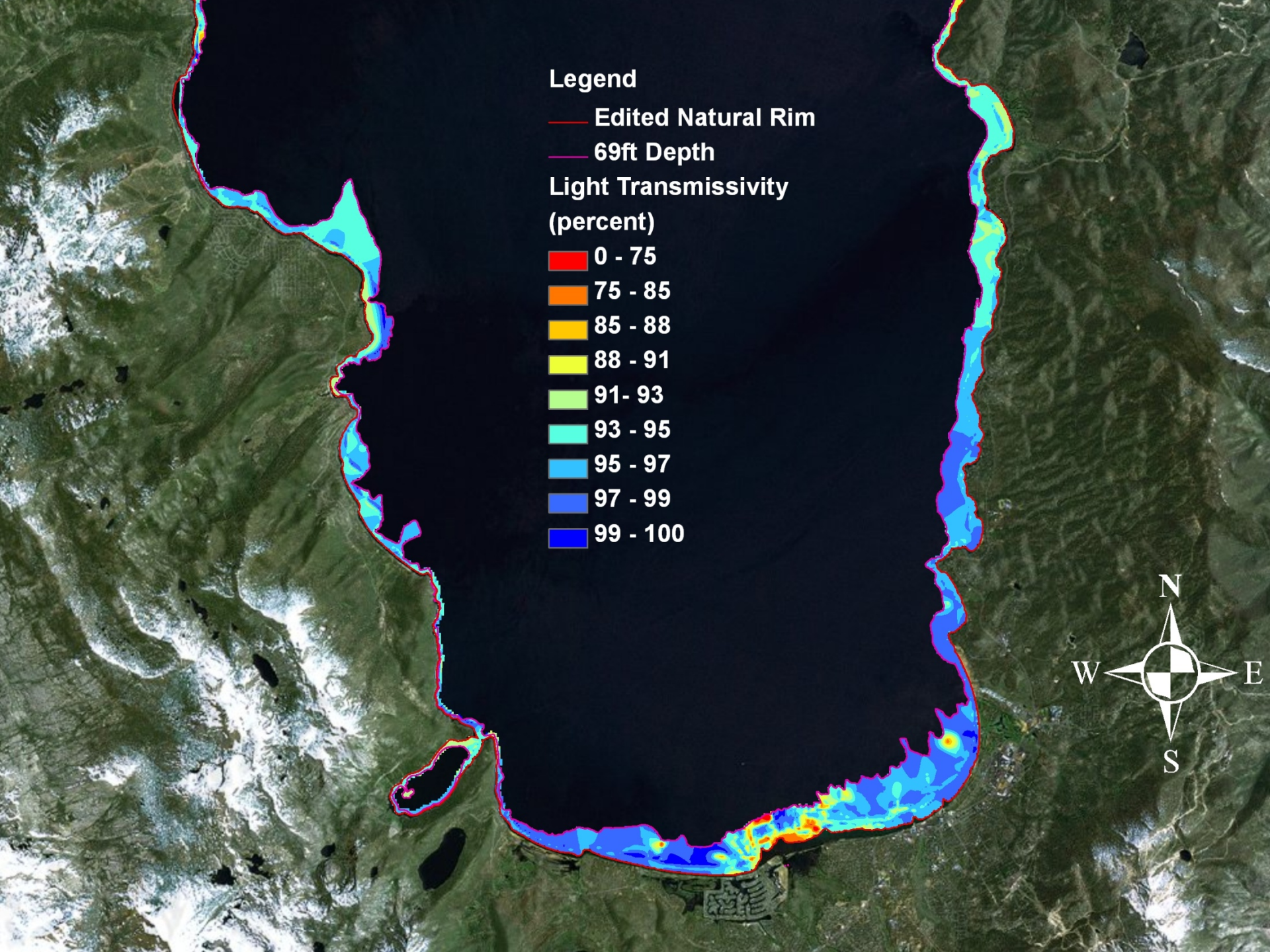
91 - 93

93 - 95

95 - 97

97 - 99

99 - 100



Nearshore Monitoring Design

Metric	When	Where	Note
Turbidity	4 times per year, seasonally (Jan-Mar, Apr-Jun, Jul-Sep, Oct-Dec)	Full-perimeter survey	Includes depth profiles at ten calibration sites for evaluation purposes and to inform other metrics.
Transmissivity	4 times per year, seasonally (Jan-Mar, Apr-Jun, Jul-Sep, Oct-Dec)	Full-perimeter survey	Includes depth profiles at ten calibration sites for evaluation purposes and to inform other metrics.
Chlorophyll	4 times per year, seasonally (Jan-Mar, Apr-Jun, Jul-Sep, Oct-Dec)	Full-perimeter survey, and discrete samples collected with phytoplankton	Ten calibration sites identified by metric response and as needed for depth profiles with collection of samples used in phytoplankton assessment.
Phytoplankton	4 times per year, seasonally (Jan-Mar, Apr-Jun, Jul-Sep, Oct-Dec)	Ten nearshore sites	Collected at ten calibration sites. Includes measurement of algal growth potential (AGP).
Periphyton	7 times per year, plus a spring synoptic	Nine fixed sites, and 40 sites during the spring synoptic	Approximately bimonthly sampling, plus spring synoptic between March to May.
Macrophytes	Biennial survey	Perimeter survey every other year	Visual presence/absence surveys.
Macro-invertebrates	2 times per year (spring and fall)	Eleven soft and hard substrate sites for CDA	Composition, distribution and abundance (CDA).
Fish and crayfish	4 times per year seasonally, plus biennial summer synoptic	Eleven locations for seasonal sampling, and forty-nine sites for summer survey	Composition, distribution and abundance in target areas, and for summer spawning survey.
Toxicity	Agency determination	Targeted by incident	In response to incidents or emerging concerns identified by LRWQCB or NDEP.
Harmful micro-organisms	Agency determination	As required for public safety, and where targeted by incident	Per state and federal requirements.

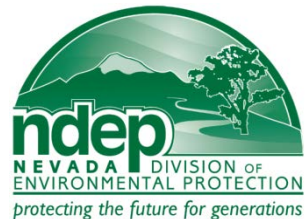
Next Steps

- Public Engagement
- Monitoring Program
- Targeted Research

Comments & Questions



Pacific Southwest
Research Station



Lahontan Regional Water Quality Control Board





TRPA Regional Plan Implementation Committee & TRPA Staff
Cc: City of South Lake Tahoe Staff
128 Market St.
Stateline, NV 89449

October 21, 2013

Subject: Comments on City of South Lake Tahoe proposed Tourist Core Area Plan

Dear Members of the Regional Plan Implementation Committee and TRPA staff:

The Friends of the West Shore (FOWS) and the Tahoe Area Sierra Club (TASC) appreciate the opportunity to provide additional comments on the proposed City of South Lake Tahoe (City) Tourist Core Area Plan (TCAP), and all related documents. We also thank members of the Regional Plan Implementation Committee (RPIC) for inviting additional comments and for the responses provided by staff. As our list of attachments on the TCAP will show, we have been extremely diligent providing comments, technical references, and recommendations to TRPA and City staff, the RPIC, SLT City Council, and SLT Planning Commission. Most comments and attachments have been included in GB packets.¹ The responses provided by staff in the “Attachment B, Response to Comments,” unfortunately fail to address many of our concerns and questions, and with great disappointment, fail to respond to most of the technical information we provided from several of Tahoe’s most reputable scientific institutions (e.g. TERC, TSC, DRI) regarding matters which affect not only water quality, but public health and safety.

In order to achieve and maintain TRPA’s thresholds, and to protect public health and property, we request the RPIC recommends TRPA staff to take the following actions, discussed in greater detail below, and recommends these same revisions be made in the relevant sections of the City’s TCAP.² Our recommendations for TRPA and the City’s TCAP include:

- 1) Update Chapter 35: Natural Hazard Standards to address current flood hazards
- 2) Revise Code to remove variances that will allow degradation of the natural scenic quality
- 3) Evaluate the impacts of the off-road mobile sources of ozone precursors and adopt measures to reduce emissions to obtain the ozone standard *in the immediate future* (ozone standards are not long term standards such as mid-lake clarity);

The information available from the RPU/RTP EIR/S and EIS, and the TCAP’s environmental documentation is not adequate and as a result, TRPA’s threshold-related findings can not be made. Further, Conforming to the RPU, which itself lacks adequate environmental review, does not ensure that public health and safety or environmental standards, will be achieved and maintained as required by the Compact. Please feel free to contact Jennifer Quashnick at jqtahoe@sbcglobal.net or Laurel Ames at laurel@watershednetwork.org if you have any questions.

Sincerely,

Laurel Ames,
Conservation Co-Chair,
Tahoe Area Sierra Club

Susan Gearhart,
President,
Friends of the West Shore

Jennifer Quashnick
Conservation Consultant,

¹ See next page: Attachments not included are attached again herein, or links are provided.

² We apologize this was apparently unclear in our previous comments (dated 10/3/13)² and we hope the RPIC will request staff to address all of our comments. “Staff” refers to TRPA and City staff, as appropriate.

Attachments (10/21/2013):

New attachments:

USGS 5.23.12 LiDAR LT faults
USGS 5.23.12 LiDAR LT faults Shaded Relief Map
USGS 5.23.12 LiDAR LT faults Figure 2
US Report West Tahoe Fault 7-2013
Three Faults under LT.Tahoetopia
Livescience WTDPF earthquake
75-09 Ozone Trends CARB
CARB staff report rev ozone 2005
2012 CARB Designations ozone
CARB 2008 EI

Previous/incorporated attachments:

Where documents are already available in staff reports, we note the report and page numbers in lieu of re-attaching

- **October packet** includes the GB and RPIC packets prepared for the 10/23 and 10/24 meetings.
 - o <http://www.trpa.org/wp-content/uploads/October-25-2013-Governing-Board-Packet-a.pdf>
- **September packet** includes the GB and RPIC packets prepared for the 9/25 meetings.
 - o <http://www.trpa.org/wp-content/uploads/September-25-2013-Governing-Board-Packet.pdf>

10/3/2013 TASC&FOWS Comments to RPIC and TRPA Staff regarding TCAP; (in October packet, p. 177-179)

Attachments to 10/3/13 Letter included:

9/24/2013 TASC&FOWS comments to TRPA GB on DC SSAP (main)
(in October packet, p. 145-176)

9/12/2013 TASC&FOWS comments to City of SLT Planning Commission (and attachments):
(in October packet, p. 93-101)

A-1: 9.11.2013 Email, Notice of Availability of Staff Packet;
(in October packet, p. 102)

A-2a: 9.12 A-2a.TASC comments CSLT GPU NOP 10.16.09
(in October packet, p. 103-107)

A-2b: 9.12 A-2b.10.16.09 TASC comments Attchmt 1.Riley
(in October packet, p. 108-125)

A-3: 9.12 A-3. SLTGPU_FEIR_3.0-Responses-to-Comments_02-2011
http://sltgpu.com/pdf/FEIR/SLTGPU_FEIR_3.0-Responses-to-Comments_02-2011.pdf

A-4a **7/17/13 TASC & FOWS Comments to RPIC on DC SSAP and related documents;** **ATTACHED – did not find in packets**

A-4b: 7/17/13 Addendum to 7/17/2013 Comments in A-4a;
(in October packet, p. 136-130)

A-5: 8/13/13 comments to APC regarding DC SSAP and related documents;
(in October packet, p. 131-139)

A-6a: 8/27.2013 comments to TRPA GB regarding DC SSAP and related documents.
(in October packet, p. 140-143)

A-6b: Simulated Images of resort recreation hotel/building on proposed T-RR parcel
(in October packet, p. 144)

9/30/2013 TASC&FOWS Comments to SLT City Council regarding TCAP (in October packet, p. 182-189)

Attachments to 9/30/13 Letter included:

9/24/2013 TASC&FOWS comments to TRPA RPIC on TCAP (and attachments):
(in October packet, p. 177-179)

9/24/2013 TASC&FOWS comments to TRPA GB on DC SSAP (main)
(in October packet, p. 145-176)

2013 Tahoe Science Consortium, ARkStorm@Tahoe Fact Sheet; *(in October packet, p. 190-191)*

2013 “Are you Ready for ARkStorm?” Expert Panel Discussion, invitation to 10/11/13 panel
(in October packet, p. 192)

10.21.13 FOWS&TASC Comments on CSLT TCAP for 10/24/13 RPIC

- 9/24/13 TASC&FOWS Comments to GB regarding DC SSAP; *(in October packet, p. 145-176)*
- Included as attachment to 9/24/13 letter to RPIC regarding CSLT TCAP); attachments to those comments, which were therefore part of the comments submitted on the TCAP, were provided to the RPIC, and listed in the comment letter (list repeated below for easy reference;

Attachments to 9/24/13 Letters include: (10/21/13 note: those attachments below that are not accessible online or already addressed above can be provided upon request)

Attachments to RPIC comments on DC SSAP and attached to RPIC comments on CSLT TCAP:
All attached comments are from TASC and/or FOWS, unless otherwise noted.

6/20/2013 comments to DC BOCC re: SSAP and documents (includes two attachments);
6/26/2013 comments to the TRPA RPIC re: DC SSAP;
7/17/13 TASC & FOWS Comments to RPIC (and addendum);
8/13/13 comments to APC regarding DC SSAP and related documents;
8/27/2013 comments to TRPA GB regarding DC SSAP, including attachment.
11/2/2010 Lahontan response to TASC 9/13/2010 TMDL comments

2012: TRPA RPU

6/17/2012 Joy Dahlgren's comments on RTP DEIS (transportation expert)
6/26/2012 Joy Dahlgren's comments on RPU DEIS (transportation expert)
6/27/2012 Joy Dahlgren's verbal comments to TRPA re: RPU/RTP DEIS
6/28/2012 Comments on RPU DEIS, RTP DEIR/S, and 2011 TER*
6/28/2012 NTPA & MAPF Comments on RPU DEIS; including Exhibits
7/25/2012 Comments on 2011 TER*
8/21/2012 Comments on RPU Committee Hearings*
10/23/2012 Comments on Bi-State, RPU process, and TER*
11/15/2012 Comments regarding RPU Concerns*
12/5/2013 Additional Comments re: RPU*
12/11/2012 Comments on Final RPU Package (w/Attachments 1-6)*

* Throughout our letters regarding the Area Plans and the DC SSAP, we have frequently referenced our comments on the RPU EIS that were submitted to the TRPA in 2012. We are attaching them to this letter so the full comments will be part of the record. In the case of the 6/28, 7/25, and 10/23, 2012 comments, there were extensive attachments submitted to TRPA via USB thumb drive. Attachments from 6/28/2012 are available at:

http://www.trpa.org/documents/reisc/5_Comment%20References/LTSLT_FOWS_TASC_references/
(accessed 9/23/2013). The other attachments will gladly be provided upon request.

- 8/28/13 TASC&FOWS comments to CSLT and TRPA staff regarding IS/ND/IEC for TCAP
(in September packet, p. 233)

Attachments to 8/28/13 Letter included:

1. Distribution of Ozone and Ozone Precursors in the Lake Tahoe Basin, USA. DRI.
(in September packet, p. 290-293)
2. The Use of Meteorological Regimes to assess the sources of ozone in the Lake Tahoe Basin. (Poster)
(in September packet, p. 247)
3. A Legacy of Research, Education, and Outreach. DRI. Presented at August 2012 Tahoe Summit. <http://www.dri.edu/2012-tahoe-summit> *(in September packet, p. 248-283)*
4. Contact Filter Data (summaries); *(in September packet, p. 284-289)*
5. TRPA Compact *(in September packet, p. 294-315)*

- 7/2/13 TASC&FOWS comments to CSLT City Council for TCAP workshop
(in September packet, p. 187-194)

- 7/11/13 TASC&FOWS comments to CSLT Planning Commission for TCAP workshop
(in September packet, p. 202-212)

Additional comments (10/21/13):

The TASC and FOWS appreciate the time taken by TRPA and City staff to address some of our more recent comments. However, there appears to be confusion regarding several of our comments, and a great deal of technical information, in addition to specific requested actions, was overlooked. The following comments are in addition to the collection of comments we have already submitted on the RPU EIS, RPU package, and all Area Plans. Specific requests are provided in **bold blue text** for clarity.

General Comment regarding TCAP's threshold benefits:

In general, there appears to be a misunderstanding regarding our comments on the TCAP's outcomes compared to a "no action" (no TCAP adoption) situation. Questions are not repeated in detail here, but can be found in the attachments. However, the concern we have raised is how the TCAP will ensure/guarantee threshold benefits above and beyond what would occur without the TCAP (this also affects the threshold findings). For example, the TCAP encourages SEZ restoration, but does not require it. Thus it does not *ensure* any more restoration than would occur without the TCAP. In another example, the TCAP relies on water quality BMPs and areawide projects to claim the TCAP will help reduce water pollution, however what is unclear is which projects will occur regardless of TCAP adoption, as the TCAP can not find threshold benefits from actions that are already occurring or will occur without it.

In summary, we ask staff to clearly explain what guaranteed benefits will result from the TCAP compared to what will happen without the TCAP, and reiterate previous comments to TRPA and City staff that 'encouraging, promoting, etc.' are not requirements which guarantee an outcome.

Specific comments regarding October Staff Packet:

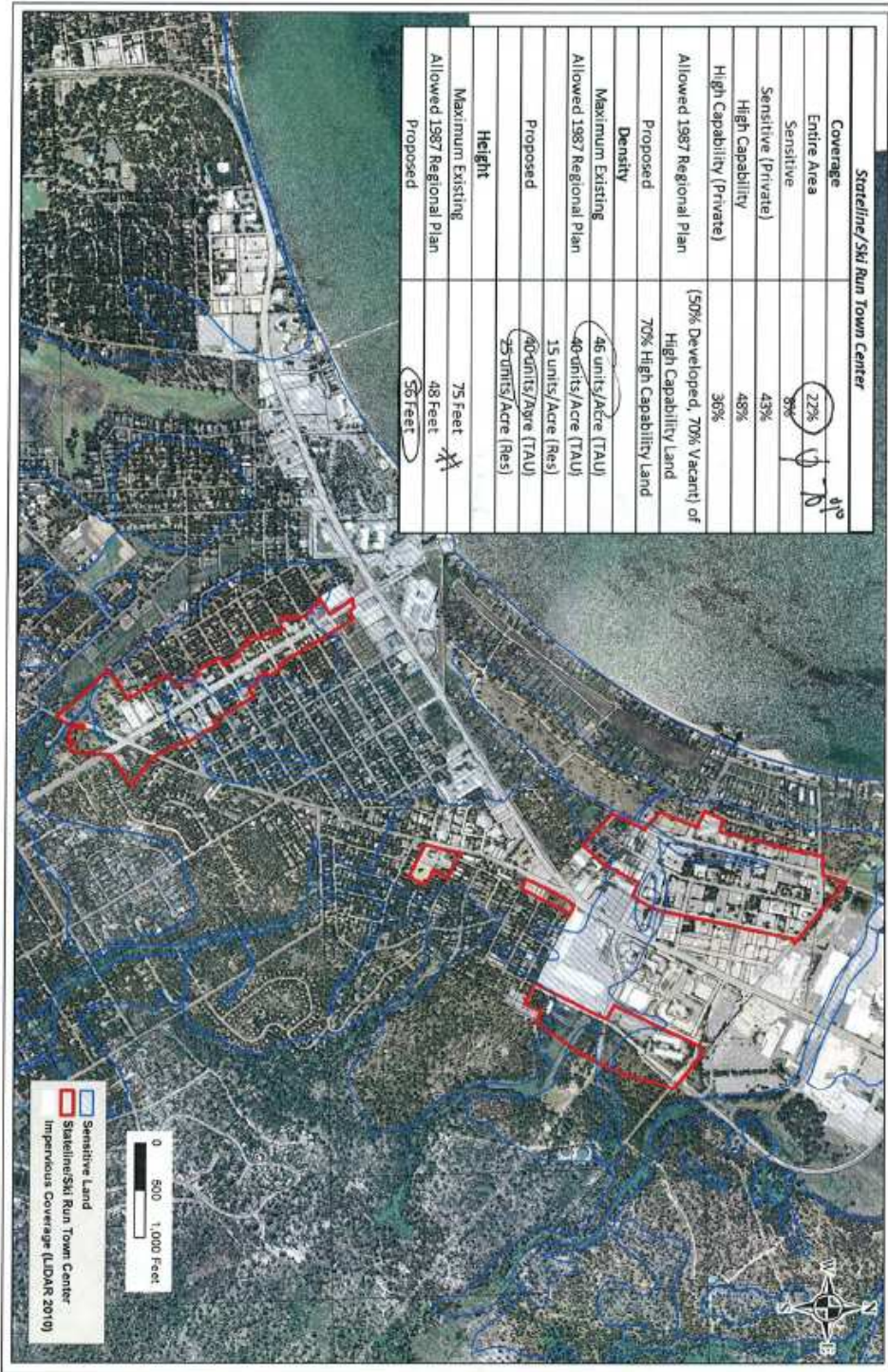
Page numbers note the **hard copy (and adobe reader) page numbers** in the October 2013 TRPA staff packet and are followed by additional comments.

Coverage: p. 213 (289)

5-1. An opportunity for hard coverage reduction is greater in the TCAP for development located in Land Capability Districts 4-7. Approximately, 68 percent of development is located on Land Capability District 4-7 and located on parcels with coverage that exceeds 70 percent. Much of this development is obsolete and in many cases does not meet current design standards

The original TCAP Town Center boundaries analyzed in the RPU included different statistics regarding existing coverage. Although the TCAP boundaries are greater than the Town Center boundaries analyzed by the RPU, it is unclear how much of the land (by land type) within the TCAP is currently covered.

We request TRPA provide an updated map to clarify. The image below was provided to the public in the latter half of 2012 related to the RPU's analysis and is on record with TRPA.



Waterborne Transit: p. 197-199 (273-275):

TASC, FOWS, and the LTSLT have asked about the environmental impacts of the waterborne transit that is promoted in the TCAP. The LTSLT's comment is noted as number 10 in the response:

10. One of the goals for transportation planning in the TCAP is Goal T-4, "to promote the use and expansion of multimodal transportation options including transit for visitors and residents.10" One of the specific policies related to this, is Policy T-4.2, "encourage the use of water borne transit along the south shore from Camp Richardson Resort to Zephyr Cove Resort and provides stops at Ski Run and Lakeside Marinas." Analysis of this transportation mode must show that promotion of water borne transit will lead to a reduction in air pollutants and will not have significant impact to Lake water clarity. An environmental analysis should be conducted to demonstrate the benefits and any environmental impacts associated with this type of transportation. The FONSI should demonstrate how this will be analyzed in the future or where it has been analyzed in the Regional Transportation Plan (RTP).

Appendix B includes the following response to number 10:

Policy T-4.2 is consistent with the City's General Plan and TRPA's Regional Plan and Regional Transportation Plan which all call for the consideration of waterborne transportation system in Lake Tahoe. Waterborne transit was analyzed programmatically in the RTP EIS/EIR as part of Transportation Package C, which includes the financially constrained list of projects, including new bicycle and pedestrian facilities, corridor revitalization projects, transit service and capital enhancements, and waterborne transit.

The RTP EIS/EIR concluded that the RPU would result in mobile-source CO emissions well within the emissions budgets allocated for transportation conformity. The transportation emissions budget is the basis for air quality planning efforts in the Lake Tahoe CO Maintenance Plan. If the transportation emissions budget is met, then the Basin is considered to be on track for maintaining attainment of the CO AAQS.

The RTP EIS/EIR also analyzed the effects of long-term operational emissions of ROG, NOX, PM10, and PM2.5 of Transportation Package C. As indicated in the transportation analysis prepared for the RTP/SCS, regional VMT in the Basin would increase by approximately 126,000 VMT/day by 2035. VMT per capita would decrease by approximately two percent by 2035 compared to 2010 conditions. Although VMT would increase, mobile-source operational emissions would decrease for all pollutants because of more stringent vehicle emission standards over the planning period. The emissions model used in this analysis (EMFAC 2011) accounts for vehicle emissions control measures contained in State Implementation Plans submitted to EPA, smog check programs, truck and bus emissions rules, and fuel economy standards. These regulatory programs are already in place or approved and will result in foreseeable mobile-source emission reductions in the study area.

Based on the results of the emissions modeling presented in the RTP EIS/EIR, mobile-source emissions of ozone precursors in the Lake Tahoe air basin would be expected to decrease substantially by 2035.

Based on the results of the emissions modeling, mobile-source emissions of ozone precursors in the Lake Tahoe air basin would be expected to decrease substantially by 2035. Emissions of PM10 and PM2.5 would decrease slightly by 2035 because vehicle emissions would improve substantially over the next 20 years as a result of more stringent federal and state standards, and very limited development would be allocated beyond what was authorized in the 1987 Regional Plan. Any increase in emissions resulting from additional population growth in the Region would be more than compensated by reduced rates of vehicle emissions in response to more stringent standards. Because substantial long-term reduction in emissions of ozone precursors and slight reduction in particulate matter, the impact of implementing Transportation Package C impact would be less than significant (RTP Draft EIS/EIR 2015, pages 3.4- 22 through 3.4-31).

The RTP EIS analyzed potential impacts to water quality from the effects of waterborne ferry operations under Impact 3.8-4. The EIS analysis concluded that these impacts would be less than significant due to vessel discharge regulations, requirements for runoff control and treatment, best management practices for avoiding accidental spills, and normal vessel nearshore speed limits to retard wakes (RTP Draft EIS/EIR 2015, pages 3.8-30 through 3.8-32). The RTP EIS analyzed potential impacts to water quality from the effects of waterborne

ferry operations under Impact 3.8-4. The EIS analysis concluded that these impacts would be less than significant due to vessel discharge regulations, requirements for runoff control and treatment, best management practices for avoiding accidental spills, and normal vessel nearshore speed limits to retard wakes (RTP Draft EIS/EIR 2015, pages 3.8-30 through 3.8-32). Moreover, any subsequent project proposal to implement waterborne transit on Lake Tahoe would be still be subject to project-level environmental review and permitting, in compliance with local, state, TRPA and federal law. [Emphasis added]

We raised the same issue. See page 209 (285):

25. The TCAP promotes waterborne transit, which is far more polluting per person mile than driving, yet this is not discussed.

Appendix B includes the following response:

At this time it is not known what type of vehicle would be used and therefore it is premature to determine if waterborne transit will be more polluting than automobiles. Any subsequent waterborne transit project that is proposed must meet the requirements of the TRPA Code and would be subject to project-level environmental review and permitting, and at that time the project would be required to demonstrate no significant impact on air quality, water quality and other resource areas.

These responses are confusing. Either the air quality impacts of waterborne were analyzed, as stated in the response to comment number 10, or they were not, as stated in the response to comment 25. Further, as noted in our 10/3/13 comments letter, the reference to the ‘reduction in mobile source emissions’ from the EMFAC analysis is incorrect. The RPU EIS modeled the anticipated emissions from *on-road* mobile sources, but not from *off-road* mobile sources,³ which according to CARB’s 2008 Emissions Inventory (EI)⁴ comprise *more than half* of the emissions for ozone precursors in the Basin. In fact, summertime sources such as boats may have an even greater impact on ozone levels because CARB’s EI is based on ‘tons per day’ (thus the average daily emissions for 365 days are represented in the values), yet in the Basin, boat use is generally confined to roughly 100 days per year, thus the daily emissions would be greater than the annual average emissions⁵ during the same months that ozone is a problem (e.g. add up the emissions times 365 days for the annual tons emitted, then divide that by 100 for average **per day summertime** emissions). Further, several TRPA summaries have erroneously claimed the *on-road* mobile source future reductions apply to all mobile sources. This is why we requested the following in our previous letter (which also provided more information regarding this error):

3. Evaluate the impacts of the following sources of ozone precursors and adopt measures to reduce emissions to obtain the ozone standard in the immediate future (ozone standards are not long term standards such as mid-lake clarity);
 - motorized watercraft
 - off road vehicles including snowmobiles and OHVs

Unfortunately this was not addressed by the responses in Appendix B. It is also confusing that, based on the response to comment 25, the air quality impacts could not be analyzed because the type of vessel that will be used is ‘not known,’ yet according to the response to comment 10, the *water* quality impacts already were analyzed and found to have no significant impact.

³ E.g. motorized watercraft, snowmobiles, and OHVs.

⁴ This is the same EI that the RPU EIS relied upon for estimates and is attached for reference.

⁵ Daily emissions would be derived from dividing CARB’s estimated Tons Per Year by 100, not by 365.

Further, we also note that TRPA has at its disposal the tools necessary to estimate the possible emissions ‘per person, per mile’ for a person using the waterborne transit versus driving. Appendix F of the RPU EIS (p. 54) provided general estimates of the air emissions from waterborne transit based on assumptions regarding use, hours, etc., utilizing a CARB model. These estimates were then incorporated into TRPA’s “RPU Emissions Modeling Summary (Year 2035)” for each alternative (pages 7-11).

- Estimates of ridership can be obtained based on existing transit (for example, NLT), and can be evaluated and presented based on various levels of ridership compared to capacity (e.g. 25%, 50%, or other percent ridership associated with a vessel that can carry, for example, 120 passengers). Emissions and number of miles from the one-way ride can be estimated, then divided by the number of passengers to obtain waterborne-based emissions per person per mile (PPPM).
- The PPPM emissions from driving the same number of miles can be easily estimated, and divided by the average number of people per vehicle in the Basin (the TMPO should have the most recent information for this), to obtain the on-road vehicle-based average emissions PPPM. This comparison can be done with some basic math and tools and information already at TRPA’s/TMPO’s and the City’s disposal.

We request staff provide a thorough, clear, and consistent response to these questions.

Air Quality Impacts and Human Health: Page 210 (286):

We have repeatedly requested that TRPA, and more recently, Douglas County and the City, evaluate the air quality impacts of placing more people in residential and tourist units adjacent to Tahoe’s highways, as would be done with most of the Centers in the RPU. It is well-established that exposure to air pollution causes significant human health impacts, including respiratory damage, heart damage, and premature death. There are also numerous scientific studies showing that those living near and adjacent to high-traffic roadways are exposed to higher concentrations of air pollution (several have been published by CARB; although this is an uncontested scientific fact, we would be glad to provide example studies upon request). As noted in our comments on the RPU/RTP documents in 2012, the studies that TRPA relied upon to conclude it was unnecessary to examine the human health impacts of the proposed Land Use changes in the RPU did not address the unique circumstances in the Basin which have a direct impact on our air quality.

As the RPU EIS relied on non-local information to “conclude” there were no impacts, our previous comments on the RPU EIS noted that the references used did not incorporate or account for the impacts of Tahoe’s frequent thermal inversions, which trap the pollutants at the surface (nor were the impacts of elevation on emissions considered). Thus, people living in apartments or condos adjacent to highway 50, as proposed in the TCAP, will be exposed to undiluted concentrations of pollution from vehicles on the highway, including CO, NO_x, VOCs, PM₁₀, PM_{2.5}, and toxic diesel emissions. During the winter months, inversions can last for entire days. In the summer, they are more common overnight than during the daytime, however, it’s important to consider that people (residents and visitors) in Tahoe are likely to open their windows for cool air overnight in the summer months, thus breathing the air pollution in as they sleep at night.

The meteorological information supporting Tahoe's inversions is extensive and well-documented; further, we provided TRPA with a preliminary study (2004) along highway 50, performed by a reputable researcher from UC Davis (Dr. Thomas Cahill), and funded by TRPA, which indicated diesel emissions were basically 'hanging in the air' during inversions. TRPA's RPU/RTP EIR/S documents did not address this issue, nor the study provided with our comments on the draft EIS. Unfortunately, our comments regarding this issue, and the public health impacts associated with it, were not addressed in Appendix B:

27. If the City is going to place more residents closer to the highway, this will increase their exposure to air pollution, especially during Tahoe's frequent inversions. Local conditions which affect how air pollution disperses, or doesn't, must be assessed. This was not considered in TRPA's RPU EIS.

Two responses were presumably provided in the packet to our comments. The first, noted below, is from Attachment B. The second excerpt is from "Attachment C: Impact Analysis on Adjacent Residential Areas:"

Based on the results of the emissions modeling, mobile-source emissions of ozone precursors in the Lake Tahoe Basin would be expected to decrease substantially by 2035. Because substantial long-term reduction in emissions of ozone precursors and slight reduction in particulate matter, the impact of implementing the Regional plan is considered less than significant (RTP Draft EIS 2015, page3.4-31). Because the TCAP is required to be consistent with the Regional Plan, implementation of the TCAP would also be expected to result in a substantial long-term reduction in emission (TCAP IS/ND page 50).

Page 286 (359):

In response to this comment on air pollution, the RPU EIS, and TCAP Initial Study/Initial Environmental Checklist were carefully reviewed and the environmental documentation determined to be adequate as presented.

Thus, this question has remained unaddressed for almost two years, yet the impacts of the Mixed-Use pattern combined with the increases in residential and tourist populations (which the RPU and TCAP encourage to be placed in units essentially along highway 50 and Ski Run Blvd.) and total VMT, have not been considered. The TCAP unfortunately perpetuates the inadequate analysis of human health impacts in TRPA's RTP/RPU environmental documents by tiering from the RPU EIS (e.g. see response to comment 85, p. 237).

In addition, the RPU/RTP's assessments only consider future reductions from one source - the on-road mobile sources - and only in 2035⁶. We have asked for an assessment of the intervening years, but no responses have been provided. For example, what will air quality be like in just five years? Ten years? Next year? All residents and visitors that rely on air to breathe between now and 2034 should be interested in these answers.

We therefore request that a scientific analysis based on Tahoe-specific meteorological and air quality data be performed to evaluate the potential exposure to air pollution by

⁶ Estimates of CO are the exception, as intermediate years were evaluated to meet federal Conformity requirements. However, an assessment of CO emissions and the impacts of the Basin's inversions, coupled with the RPU's and TCAP's proposed increased placement of humans in the location of the Basin's historical CO "hotspots" and increased wintertime VMT, has not been performed.

those living, working, or visiting these units. Further, we have asked TRPA and local governments to examine the air pollution that will occur between now and 2035, as air pollution will not magically wait until 2035 to cause negative public health impacts.

SEZ Restoration: Page 238-239 (314-315):

As our comments have noted, the TCAP does not appear to ensure any more SEZ restoration with its adoption than without. For example, we asked:

91. What does SEZ restoration priority mean? How to do ensure these parcels will be restored?

We appreciate the City's inclusion of more wording regarding SEZ restoration (noted in previous comments), however, as noted in the response to this comment, the TCAP still fails to ensure more SEZ restoration will actually occur:

Similar to TRPA's Restoration Plan Area designation the TCAP identifies certain parcels as SEZ restoration priority areas. These areas have been substantially degraded by prior development and are targeted for environmental restoration. This designation is consistent with proposed TCAP Policy NCR-2.2 which states the following: "Protect and direct development from SEZs, and encourage the removal and transfer of existing development from disturbed SEZs that can be feasibly restored by creating incentives for their removal."

Because the parcels are privately owned, there is no guarantee that restoration will occur. However, the designation focuses the City's efforts in areas where restoration is feasible and desirable and allows the City to collaborate with private properties owners to achieve the TCAP restoration goals.

We request that staff examine the guaranteed SEZ restoration that *will occur* (not 'is likely to' or 'is incentivized', etc.) due to adoption of the TCAP versus without its adoption to assess the threshold benefits of the TCAP.

Environmental impacts of transferring existing development: Pages 242-243 (317-318)

There has been an ongoing assumption that transferring development from existing, "old motels" to Centers (which then increases the number of units, coverage, etc., as a result of various incentives), will provide a net environmental benefit. However, the actual impacts of these transfers have never been examined. We have raised the question verbally, and in writing, and clarified specifically what the question was (see the 'equation' laid out in our 9/30/13 comments – page 7 - for additional detail). Attachment B includes the following comment, noted by several paragraphs of responses which do not address the question of impacts (please see pages 242 and 243 for the response).

110. With transfer what are the impacts from transferring old motels on Hwy 89 and relocating them closer to the Lake? What are the impacts of allowing these transfers?

We request that staff examine the actual impacts of these transfers, following the equation laid out in our previous comments, to assess the true net benefits to water quality, soil conservation, air quality, noise, and natural scenic quality from the associated demolition, restoration activities (which may actually cause more pollution upfront, for years), construction, increases in number of units and coverage resulting from transfer incentives, threshold impacts in the 'new' location, changes in VMT, increases in population, etc.

Current increasing trend in ozone: Pages 246-247 (322-323)

We have noted throughout comments on the RPU/RTP environmental documents, and subsequent comments on the DC SSAP and the City's TCAP that contrary to the RPU's claim (through the 2011 Threshold Evaluation Report [TER]), ozone levels in the Basin are not decreasing, nor is there information to conclude the California standard is being attained within the TCAP boundaries since 2009 (CA's official designation for the Lake Tahoe Air Basin is still nonattainment-transitional), as implied in the 2011 TER:

"Interim Target: The Highest 8-hour Average Concentration of Ozone indicator is currently in attainment with the CA standard of 0.070 ppm and therefore, it is not necessary for an interim target to be established."

"Target Attainment Date: The Highest 8-hour Average Concentration of Ozone indicator is currently in attainment with the CA standard of 0.070 ppm and therefore, it is not necessary for an interim target date to be established." (2011 TER, Chapter 3, p. 3-31).

We have essentially been 'beating a dead horse' with our repeated comments on this issue. Not only is ozone not getting better, but it's getting worse, and as noted in the TER report excerpt, TRPA chose not to acknowledge this, nor require reductions. We have provided extensive detail on this issue, including ozone measurements published by DRI and TSC in 2011 (from 2010 data throughout the Basin), yet this has not been addressed. Rather, Attachment B fails to address all but one of the outreach materials we'd included (which we'd included for understanding by the general public and only in addition to the technical information), and mischaracterizes the document in a way which makes it appear irrelevant. The Attachment combines our comments into the following:

113. DRI study for Aug 2012 Summit shows ozone is increasing and California standard violated. This is inconsistent with threshold evaluation and the Regional Plan so findings cannot be made based on the Regional Plan.

The response in Attachment B states:

The document the commenter cites is a media publication piece prepared for the 2012 Summit that profiles Nevada's contribution to research in the Lake Tahoe Basin rather than a peer-reviewed scientific study. The publication mentions concerns with elevated ozone levels (circa 2006) and its impacts on human health and described ongoing studies being conducted by Dr. Gertler and DRI and their attempts at trying to get a better handle on the sources of ozone in the basin. The summary further mentions the need to find effective strategies to manage growth and development while minimizing the effects of the pollution. The statements made by Dr. Gertler are not inconsistent with the proposed policies or strategies of the RPU and the TCAP to implement a land use and transportation system that reduces the emissions of ozone precursors in the Basin. As described in the RPU DEIS, ozone precursors would be expected to decrease substantially by 2035 under the adopted RPU land scenario and implemented in the TCAP.

This response fails to address the other technical information we have provided regarding ozone, which we will not repeat herein, although ample details are provided in the attachments. However, to address the response regarding one of the documents we have submitted, we note that the inclusion of "(circa 2006)" by staff is misleading, is not supported by any materials from the DRI summary, and not supported by the facts. *If Dr. Gertler has provided a response to staff stating his article was referencing 2006 information, we request this be provided and clarified with the response.* However, an observation of the facts would suggest that the statement was certainly not based on data from "circa 2006":

- First, this was a media publication prepared by an extremely reputable scientific institution – the Desert Research Institute (DR) – prepared for an annual, large, well-attended public and political event - the 2012 Tahoe Summit. It would make little sense for a reputable scientist (e.g. Dr. Alan Gertler) to make such a statement about doing current research based on data at least six years old.
- Second, as staff can clearly examine in the October packet, the research summary includes information that CA standards for ozone have been violated, and are approaching federal standards. As shown by TRPA’s own final 2011 TER, the CA standard was first violated in 2006 and later (monitoring data for the CA side only exists through 2009). CARB adopted the more stringent 8-hour standard in 2006 based on research indicating the previous standard was not protective enough of sensitive populations.⁷ It would make little sense to assume Dr. Gertler was referring back to data running through the late 80’s, when the prior 1-hour CA standard had first been reported as violated. In addition, although CARB’s 1975-2009 ozone summary (attached) highlights violations of the 8 hour standard before 2004, the 0.070 8-hour ppm standard was not applicable until 2006, at which point, violations of the state ozone standard began and continued through the last year there was monitoring. It is far more likely that these are the violations the DRI report referred to.
- Third, as provided in previous letters, we have included technical papers showing the ozone measurements performed in 2010.
- Finally, no information has been provided to date regarding the air quality measurements of ozone on the CA side of the Lake since 2009, and violations of the 8-hour standard did occur that year.

We therefore request staff perform a technically-sound analysis of existing ozone conditions, trends, sources, and impacts, and based on this information, identify how the RPU and TCAP will ensure ozone standards are attained – and air quality in the Basin is healthy – well before 2035.

⁷ <http://www.arb.ca.gov/regact/ozone05/revstaff.pdf>: (also attached): “...The current California ambient air quality standard for ozone is 0.09 ppm averaged over one hour and was set by the Board in 1988. The data indicate that the current standard alone is not sufficiently protective of human health. Based on the review of the scientific literature and recommendations by OEHHA, the staff recommends that the following revisions be made to the California ambient air quality standard for ozone:

1. Ozone will continue to be the pollutant addressed by the standard.
2. Ozone 1-hour-average Standard – retain the current 1-hour-average standard for ozone at **0.09 ppm, not to be exceeded.**
3. Ozone 8-hour-average Standard – establish a new 8-hour-average standard for ozone at **0.070 ppm, not to be exceeded.**”

Cumulative impacts: Pages 274-275 (350-351)

The cumulative impacts of the TCAP have also not been adequately addressed. As noted in one of our comments on this matter:

23. As noted throughout, the RPU EIS did not sufficiently analyze the cumulative impacts of the new RPU. Further, the RPU made assumptions about local government planning in order to ‘evaluate’ the increased GHGs, Air pollutants, etc., but the IS/ND/IEC includes no comparison of whether those assumptions were comparable to what the TCAP proposes. Further, the impacts within the TCAP and to adjacent areas have not been assessed. The impacts of other Area Plans, which all thus far favor drawing more traffic along highway 50, have not been analyzed. Both the TRPA Compact and CEQA require the analysis of cumulative impacts (including reasonably foreseeable projects/plans, for which other Area Plans qualify). Finally, the combined scenic impacts of all Area Plans have not been addressed.

The response states:

The cumulative impacts analysis of the RPU EIS (TRPA 2012a, pages 4-2 through 4-10) includes environmental enhancement projects, land management plans, TTD/TMPO projects and programs, and other development projects. The TCAP IEC is tiered from this EIS, including its comprehensive cumulative analysis.

The scope and characteristics of cumulative actions have not substantially changed; no additional cumulative projects or programs are known at this time.

TRPA and City staff disagrees with the comment that the RPU EIS analysis is inadequate. The RPU EIS thoroughly examined the environmental consequences from the RPU proposals.

We disagree, as detailed in hundreds of pages of comments to the TRPA, Douglas County, and City of SLT, the RPU EIS was not technically adequate. Further, the RPU EIS was not certified as a CEQA document. Responses also state that the environmental analysis was also tiered from the City’s GPU EIR document (May 2011), which was found to meet CEQA. However, the GPU EIR could not have contemplated the cumulative impacts of the increases included in the RPU EIS (and RTP EIR/S), including but not limited to the new zones “Resort Recreation Districts” which allow for significant new development on currently undeveloped Edgewood and Heavenly parcels. These changes will result in more traffic, air pollution, water pollution, noise, and increased scenic impacts (associated with allowing all five casinos to build ‘up and out’) than the future development contemplated in the GPU EIR. As a result, CEQA requirements to examine the cumulative impacts from the TCAP, adjacent uses, and reasonably foreseeable development, have not been met for the TCAP.

We request TRPA and City staff evaluate the cumulative impacts of the TCAP, as well as the impacts within the TCAP from the adjacent DC SSAP, and the reasonably foreseeable Heavenly Resort Recreation Parcel (as specifically identified and rezoned by the RPU).

Natural Hazards:

Flooding: Pages 71 (27)

Another issue for which we have been a broken record includes the need to address flood plain protection, especially in light of the increased flooding that will occur from climate change. Previous comments have referenced – and included – climate change information from reputable scientists and institutions for which TRPA often relies on, including the UC Davis Tahoe Environmental Research Center (TERC) the Tahoe Science Consortium (TSC), DRI, U.S. Geological Survey (USGS), and others. *There is no question that the Basin will experience (and has to a degree already) more flooding events due to climate change.* For years we have raised this issue to TRPA, the heart of which is not only an environmental issue, but also a public health and safety issue. Scientists have raised it as well, given presentations to the TRPA Board, invited agency staff to attend scientific presentations, and most recently, invited agency representatives to the ARkStorm presentation noted in our attachments (10/11/2013).

We will have more flooding in the Basin; however, contrary to this information, the RPU adds more pavement, which takes away the ability for soils to help manage and reduce flooding. We have noted several other cities where removing pavement is being encouraged in previous comments. The RPU proclaims it can handle the increased runoff through stormwater management facilities, including basins, vaults, etc., all of which are governed by outdated regulations requiring a design based on the 20-year, 1-hour storm (1 inch/hour rainfall). The science is clear – with more flooding, the old design standards will not be good enough. Yet, responses continue to claim no responsibility for addressing the science that has been available for years. For example, the packet includes the following response in the minutes from the 9/25/13 RPIC hearing (p. 27):

Laurel Ames, Tahoe Area Sierra Club said many have seen how much of Ski Run is in a stream environment zone.

Mr. Marshall said we do have a flood plain regulation in our existing Regional Plan.

Laurel Ames, Tahoe Area Sierra Club asked if that is correct or is it just mapped flood plains.

Mr. Marshall said we have flood plain regulations that address flood plain issues. The designations are from FEMA flood maps.

Laurel Ames, Tahoe Area Sierra Club said Ski Run Blvd. is not in a FEMA flood map.

Mr. Marshall said there is not a lot we can do if the agency that is responsible for addressing flood plains hasn't mapped it. [Emphasis added]

We disagree, as did Congress when it approved the 1980 Compact, which required TRPA to adopt [ETCCs] to maintain public health and safety within the region (excerpts below):

Article I (a):

(6) Maintenance of the social and economic health of the region depends on maintaining the significant scenic, recreational, educational, scientific, natural public health values provided by the Lake Tahoe Basin.

Article II:

(i) “Environmental threshold carrying capacity” means an environmental standard necessary to maintain a significant scenic, recreational, educational, scientific or natural value of the region or to maintain public health and safety within the region. Such standards shall include but not be limited to standards for air quality, water quality, soil conservation, vegetation preservation and noise. [Emphasis added]

The Compact also required TRPA to revise the RP as needed to achieve the purposes of the Compact:

Article V:

(d)...The agency may, however, adopt air or water quality standards or control measures more stringent than the applicable State implementation plan or the applicable Federal, State, or local standards for the region, if it finds that such additional standards or control measures are necessary to achieve the purposes of this compact. Each element of the regional plan, where applicable shall, by ordinance, identify the means and time schedule by which air and water quality standards will be attained. [Emphasis added]

Whether the USACE or FEMA has officially mapped an area or not is not relevant when it comes to TRPA’s responsibility to address the flooding that science clearly shows will occur in the area. Although the extent of future flooding may not be certainly predicted, the meteorological changes that will result in more flooding have already started and are well-established. That said, Mother Nature does not recognize the boundaries of old USACE maps (which only mapped three areas in the Basin, which include the Upper Truckee River, Bijou Creek, and Trout Creek – also noted our previous comments).

Earthquakes and Tsunamis:

We have also raised the issue of earthquakes, tsunamis, and seiches, to TRPA in the past. However, to ensure this information is adequately considered and in the record, we have attached several documents, including a publication by the USGS, which identify the seismic hazards in the Lake Tahoe Basin (excerpts from selected articles are also included below):

US Report West Tahoe Fault 7-2013:

Experts are concerned about the potential for an earthquake and tsunami. But if the most active fault, West Tahoe, is the source, the consequences would be cataclysmic, producing a type of tsunami most never heard of.

Should a quake occur, there would be a phenomenon many of us have never considered. Engineer Gordon Seitz deemed the situation “urgent enough” that the study needed to be done:

“Unlike ocean tsunamis, whose massive waves break and then disappear, a tsunami in Lake Tahoe would go back and forth for hours, hitting one side of the lake and then the other, again and again, Seitz said.”

The California Geological Survey [explained the phenomenon](#):

“There are several faults thought capable of generating large earthquakes in the region that encompasses Lake Tahoe, Truckee and Carson City, Nev., including the Genoa, Antelope Valley, Incline Village and West Tahoe faults. The latter fault runs under part of Lake Tahoe and may be able to generate damaging seiche waves (essentially a tsunami in an enclosed body of water)...

USGS 5.23.12 LiDAR LT faults:

CARNELIAN BAY, Calif. — Results of a new U.S. Geological Survey study conclude that faults west of Lake Tahoe, Calif., referred to as the Tahoe-Sierra frontal fault zone, pose a substantial increase in the seismic hazard assessment for the Lake Tahoe region of California and Nevada, and could potentially generate earthquakes with magnitudes ranging from 6.3 to 6.9. A close association of landslide deposits and active faults also suggests that there is an earthquake-induced landslide hazard along the steep fault-formed range front west of Lake Tahoe.

The science is there, and it is TRPA’s responsibility to protect public health and safety in the Region. Hazards such as flooding, earthquakes, tsunamis, and seiches, are also expected to severely impact environmental thresholds and measures in place (including new and existing construction, areawide treatment systems, BMPs, etc.). This will be made worse if land use plans have failed to account for these changes, let alone proposed development that will make them worse (e.g. more coverage). Unfortunately the City has relied upon TRPA to perform its Compact-mandated job, and in this case, TRPA has failed to do so, leaving the City’s residents unprotected from floods and other natural hazards. Further, the City should note TRPA’s repeated responses regarding the “regional” 30,000 foot level view (“analysis”) taken in the RPU EIS – meaning TRPA is not looking at the local details necessary to protect the City’s citizens, thus the City needs to do so.

In view of the significant changes that are being expected with climate change, it’s time for the TRPA and the City to start seriously planning for these impacts. At a minimum, technically-sufficient, thorough evaluations of the hazards and threats to people are needed, and future Land Use should incorporate measures to reduce the potential hazards (and the exposure of people to such hazards). For example, the more land use planning accounts for flooding impacts, the better the chances of reducing the impacts when flooding occurs. Further, real estate information should include the details to direct people to information that is readily available about increase in flooding, flood height, mapped or not, earthquakes and seismic hazards, including tsunamis and seiches, as well as information regarding emergency escape plans. This information should also be made available to all existing residents and all guests to the Basin, as their health and safety are also at risk.

Findings (beginning on page 294 of packet):

The responses to previous comments regarding findings have generally included reiterations of the TCAP’s tiering from the RPU EIS, various ‘incentivized’ (but not often guaranteed) benefits that are expected, etc. We have reviewed the discussion in the packet related to TRPA’s findings and believe the evidence to support the findings has not been presented (or is not available). The following includes excerpts from the staff summary and additional comments.

1. Finding: The proposed ordinance and TCAP could not have a significant effect on the environment and a finding of no significant effect shall be prepared in accordance with TRPA’s Rules of Procedure.

Rationale: ...These program-level environmental documents include a regional and city-wide cumulative scale analysis and a framework of mitigation measures that provide a foundation for subsequent environmental review at an Area Plan level. Because the TCAP is consistent with the Regional Plan, Regional Transportation Plan (“RTP”), and CSLT General Plan, which all have approved program-level

EISs and/or EIRs, the policies and objectives of the TCAP are within the scope of these program-level EISs and/or EIRs.

As noted above, the GPU EIR could not have possibly analyzed the cumulative impacts of the RPU that was adopted almost two years after the EIR's analysis, and the RPU EIS only viewed these changes from a 'programmatic' level. The IS/ND essentially refers back to the GPU EIR and RPU EIR as the supporting basis for conclusions. Thus, the localized plan-level impacts of the TCAP have not been analyzed and **there is no evidence to support the finding that the TCAP could not have a significant effect on the environment.**

Chapter 4 Findings: The following findings must be made prior to adopting the TCAP:

1. Finding: The proposed Area Plan is consistent with, and will not adversely affect implementation of the Regional Plan, including all applicable Goals and Policies, Plan Area Statements and maps, the Code, and other TRPA plans and programs.

Rationale:...The TCAP is consistent with the 2012 Regional Plan and Code, as shown in the Conformance Review Checklist and as demonstrated by the IS/ND/IEC. The TCAP contains the required contents of an Area Plan specified in the TRPA Code of Ordinances, Chapter 13, *Area Plans*, and when implemented, will have a beneficial impact on the Regional Plan's ability to achieve and maintain the thresholds as demonstrated below in the Chapter 4 and 13 findings.

...The TCAP will not adversely affect applicable compliance measures, indicators, additional factors and supplemental compliance measures and target dates as identified in the 2011 Threshold Evaluation indicator summaries. TRPA anticipates that implementation of the TCAP will accelerate threshold gains as demonstrated below.

Finding: Wherever federal, state or local air and water quality standards applicable for the Region, the strictest standards shall be attained, maintained, or exceeded pursuant to Article V(d) of the Tahoe Regional Planning Compact.

...Based on the following: (1) TCAP IS/ND/IEC; (2) RPU EIS; (3) RTP EIR/EIS; (4) GP EIR; (5) 2011 Threshold Evaluation Report; and (6) 2011 Indicator Summaries, adopted by the Governing Board or City Council, no applicable federal, state or local air and water quality standard will be exceeded by adoption of the TCAP.

As noted above, air quality standards are already being exceeded. TRPA's analyses of these impacts in the noted environmental documents fail to adequately analyze the existing conditions and trends related to ozone, and the RPU and TCAP propose increases in uses and activities that will increase emissions of ozone precursors. There is also no evidence or analysis upon which to base the assumption that exceedances will not occur between 2013 and 2034. Further, as discussed in our comments, the existing 20-year storm design for water quality is inadequate to address actual present and future conditions. As a result, there is no evidence to suggest that even if BMPs based on this design were properly installed, operated, and adequately maintained – forever – that water quality standards will not be exceeded. In addition, TRPA's own statistics show a relatively poor compliance record for BMP installation, and there is no field data or other clear, technical measurements that previously-installed BMPs have been adequately maintained. **As a result, there is not evidence to support this finding.**

4. Finding: The Regional Plan and all of its elements, as amended, achieves and maintains the thresholds.

For the reasons exhaustively explained in our multiple comment letter - including but not limited to the failure of the EIS to adequately analyze multiple impacts and address current

scientific information (e.g. impacts of climate change), we do not believe there is substantial evidence in the record that the RPU adopted in 2012 achieves and maintains the thresholds. **That said, the TCAP, which promotes the same deficiencies, also fails to be based on evidence to support this finding.**

Detailed Comments (Originally stated in 10/3/12 comments but not addressed;⁸ repeated in 10/21 letter for reference):

We provide the following comments from our 10/3/13 comments to the RPIC for ease of reference:

1. Update Chapter 35: Natural Hazard Standards to address current flood hazards

The current RP Code, Chapter 35, begins with the following:

“This chapter sets forth regulations pertaining to recognition of natural hazards, prevention of damage to property, and protection of public health relating to such natural hazards. It implements provisions of the Goals and Policies and the Water Quality Management Plan for the Lake Tahoe Region pertaining to avalanche and mass instability, floodplains, and wildfire.” [Emphasis added]

Unfortunately, the Codes requirements are minimal, and limit regulations to merely assessing ‘additional development’ in floodplains - but only for those floodplains identified by the U.S. Army Core of Engineers (USACE), which are few, or the limits of the flood insurance program by the Federal Emergency Management Agency (FEMA).⁹ The most ‘current’ flood plain identification in the Tahoe Basin addresses just 3 of the 63 tributaries, and is based on documents by USACE, both dated 1969:

- Floodplain Information, Upper Truckee River, South Lake Tahoe, CA. Prepared for El Dorado County (USACE, October 1969).
- Floodplain Information, Trout and Bijou Creeks, South Lake Tahoe, CA. Prepared for El Dorado County (USACE, July 1969).

Section 35.4.1.D provides for the following:

In areas where the U.S. Army Corps of Engineers or Federal Emergency Management Agency has not prepared 100-year floodplain maps and where TRPA has reason to believe that a flood hazard may exist, the limits of the 100- year floodplain shall be determined by application of standard hydrologic data and methods (e.g., rational method, unit hydrograph, watershed cross-sections) applied by a competent professional and approved by TRPA. [Emphasis added]

We request the TRPA do the following in order to protect the environment, lives, property, and public services:

- 1) No new or additional development is allowed in flood-prone areas until completion of a flood protection plan.
- 2) Within one year,
 - a. determine the current 100-year flood plains in the areas where USACE and FEMA have not prepared maps, and
 - b. evaluate current conditions and information to update the flood plain maps for the areas addressed by the 1969 USACE maps based on current information.
- 3) Within 18 months, adopt Code which increases and ensures the flood plain protection that will be necessary to address the Basin’s future climate.

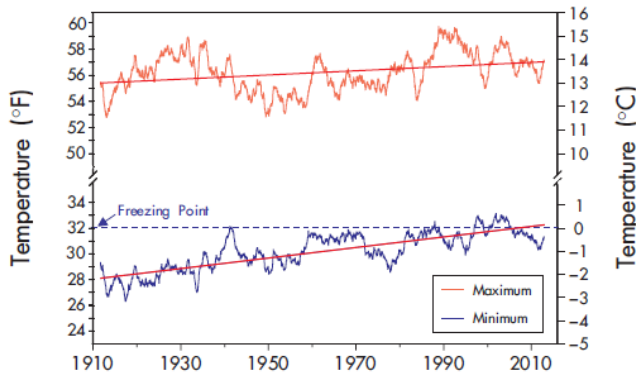
⁸ See comment 127 and response, p. 252 of packet.

⁹ Code 35.4.1, sections A-D.

Existing Conditions and State of Knowledge:

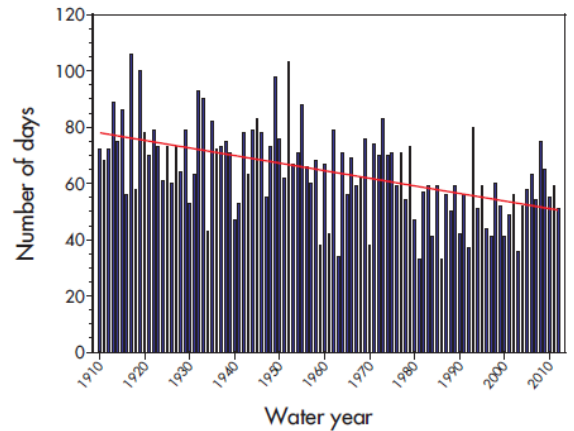
The change in the Basin’s hydrology and climate since 1969 have been dramatic. Changes in air temperature, percent of precipitation falling as rain versus snow, and other parameters all specific to the Lake Tahoe Basin, have been extensively evaluated by researchers for years. The Tahoe Environmental Research Center (TERC) published a “State of Climate Change 2013” Report¹⁰ which summarizes information from these studies. Below we have included some examples of information provided in that report, and specifically request TRPA observe the differences between conditions in 1969 compared to present conditions. Even this brief, visual comparison indicates the need to re-evaluate our flood plains in the Basin. We also request TRPA examine the report in full, and review the other information TERC has published regarding climate change impacts in the Basin, available at: http://terc.ucdavis.edu/publications/Climate_Change_Report_2010.pdf

AIR TEMPERATURE



Daily air temperatures have increased over the 100 years measured at Tahoe City. The long-term trend in daily minimum temperature has increased by more than 4 °F (2.2 °C), and the long-term trend in daily maximum temperature has risen by less than 2 °F (1.1 °C).

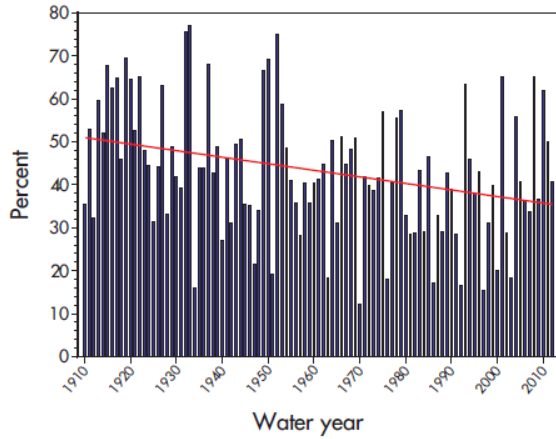
BELOW-FREEZING AIR TEMPERATURES



The number of days when air temperatures averaged below freezing has declined by about 25 days since 1911. The 2012 Water Year extended from October 1, 2011, through September 30, 2012.

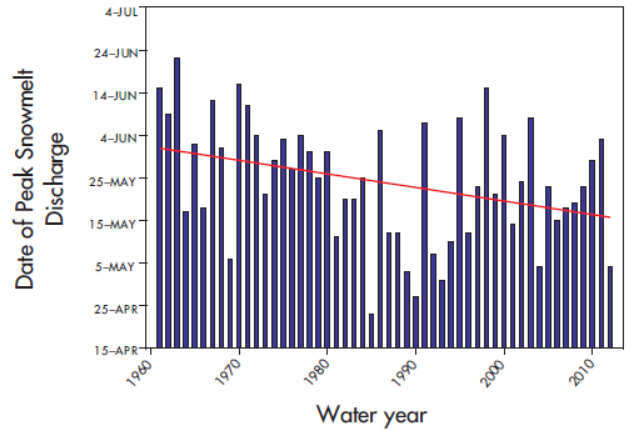
¹⁰ <http://terc.ucdavis.edu/stateofthelake/StateOfClimateChange2013.pdf>

SNOW AS A FRACTION OF ANNUAL PRECIPITATION



Snow has declined as a fraction of total precipitation, from an average of 51 percent in 1910 to 36 percent in present times

SHIFT IN SNOWMELT TIMING



The date on which peak snowmelt occurs varies from year to year, since 1961 it has shifted earlier an average of 2 weeks (16.3 days).

The report summarizes the following on page 8:

Key findings of the study till the end of the 21st Century included, for the range of scenarios tested:

1. Air temperature increases as high as 10 °F
2. The fraction of snow to rain could fall to 0.1-0.2, leading to reduced water storage in the spring snow pack and increases in drought severity
3. Changes in stream low-flow conditions could render the lower reaches of some streams completely dry more often
4. Dramatic increases in flood magnitude
5. Sediment and nutrient loading to Lake Tahoe from streams should not increase substantially
6. Overall fine sediment load reductions should still be achievable if storm water treatment facilities are properly sized
7. Lake Tahoe could cease to mix to the bottom for extended periods, resulting in complete oxygen depletion in the deep waters with loss of habitat and an increase in sediment nutrient release,
8. Lake surface level is more likely to drop below the natural rim for extended time periods. [Emphasis added]

With the new and extensive knowledge of the flood dangers we are all facing, now is not the time for TRPA to hide behind very limited Code language.

Future Conditions

Residents and business owners existing in or near the historic floodplain which runs through the proposed TCAP and surrounding areas, will find themselves dealing with **major flooding**, as scientifically-based forecasts for increased flooding events predict more flooding in the Basin, and the TRPA RPU, as implemented through the proposed TCAP, did not plan for accommodation of increased stormwater:

“...Landfalling ARs are storm events with the potential to deliver extreme amounts of precipitation to the West Coast, including California and Nevada, over a just a few days. The name “ArkStorm” was coined to describe large AR storm sequences, which, for instance, can produce precipitation in California that in places can exceed totals experienced only once every several hundred to 1,000 years...” ARKstorm@Tahoe Fact Sheet (attached).

The recent experiences of residents in Lyons and Boulder, CO, should be heeded as the Basin is facing a similar future, as current City Plans do not account for increased flooding.

2. Revise Code to remove variances that will allow degradation of the natural scenic quality

Although there were provisions under the 1987 Code which allowed exceptions to the general 26 foot height maximum in most areas, the new RPU doubled the heights allowed in many areas (in some cases more than doubled, e.g. the Regional Tourist District in the proposed TCAP), increased the densities, and increased the allowed coverage in many Centers where current scenic views exist. Thus, as more development may threaten scenic resources, the protection of those resources must be enhanced. We request TRPA make the following amendments to the Code:¹¹

a. 13.5.3.E.2. Building Height

- a. Area Plans may allow building heights up to the maximum limits in Table 13.5.3-1 above.
- b. Building height limits shall be established to ensure that buildings do not project above the forest canopy, ~~ridge lines existing buildings~~, or otherwise detract from the viewshed.
- c. Area Plans that allow buildings over two stories in height shall, ~~where feasible~~, include provisions for transitional height limits or other buffer areas adjacent to areas not allowing buildings over two stories in height.

13.5.2.E.3.c. Viewsheds ~~should~~ shall be ~~considered~~ protected in all new construction. Emphasis ~~should~~ shall be placed on lake views from major transportation corridors.

- b. **13.5.2.E.3.a.** Area Plans may include alternative sign standards. For Area Plans to be found in conformance with the Regional Plan, the Area Plan shall demonstrate that the sign standards will ~~not create negative~~ minimize and mitigate significant scenic impacts and will ensure movement ~~move-towards~~ attainment or achieve the adopted scenic thresholds for the Lake Tahoe region.

c. 37.7. FINDINGS FOR ADDITIONAL BUILDING HEIGHT

The findings required in this chapter are as follows:

37.7.1. Finding 1

When viewed from major arterials, scenic turnouts, public recreation areas, or the waters of Lake Tahoe, from a distance of 300 feet, the additional height will not cause a building to extend above the forest canopy, when present, or to block views of mountains beyond existing structures a ridgeline. For height greater than that set forth in Table 37.4.1-1 for a 5:12 roof pitch, the additional height shall not increase the visual magnitude beyond that permitted for structures in the shoreland as set forth in subsection 66.3.7, Additional Visual Magnitude, or Appendix H, Visual Assessment Tool, of the Design Review Guidelines.

37.7.2. Finding 2

When outside a community plan, the additional height is consistent with the surrounding uses. Within a Town Center, the additional height may extend up to 42 feet, if consistent with surrounding building height. Within the entire portion of the Regional Center, from the intersection of Pioneer Trail and highway 50 west, the additional height may extend up to 42 feet, if consistent with surrounding building height.

37.7.3. Finding 3

With respect to that portion of the building that is permitted the additional height, the building has been designed to minimize interference with existing views within the area ~~to the extent practicable~~.

37.7.4. Finding 4

¹¹ Proposed deletions have been ~~struck out~~ and additions underlined.

The function of the structure requires a greater maximum height than otherwise provided for in this chapter, [and there is no alternative location for the structure that would not impair scenic quality.](#)

37.7.5. Finding 5

The portion of the building that is permitted additional building height is adequately screened, as seen from major arterials, the waters of lakes, and other public areas from which the building is frequently viewed. In determining the adequacy of screening, consideration shall be given to the degree to which a combination of the following features causes the building to blend or merge with the background.

- A. The horizontal distance from which the building is viewed;
- B. The extent of screening; and
- C. Proposed exterior colors and building materials.

37.7.6. Finding 6

The building that is permitted additional building height is located within an approved community plan or Ski Area Master Plan that identifies the project area as being suitable for the additional height being proposed.

37.7.7. Finding 7

The additional building height is the minimum necessary to **feasibly** implement the project and there are no **feasible** alternatives requiring less additional height.

37.7.8. Finding 8

The maximum building height at any corner of two exterior walls of the building is not greater than 90 percent of the maximum building height. The maximum height at the corner of two exterior walls is the difference between the point of lowest natural ground elevation along an exterior wall of the building, and point at which the corner of the same exterior wall meets the roof. This standard shall not apply to an architectural feature described as a prow.

37.7.9. Finding 9

When viewed from a TRPA scenic threshold travel route, the additional building height granted a building or structure shall not result in the **net** loss of [any existing](#) views to a scenic resource identified in the 1982 Lake Tahoe Basin Scenic Resource Inventory. TRPA shall specify the method used to evaluate potential view loss.

37.7.10. Finding 10

The building is no more than two stories above grade (excluding basement) in height.

37.7.11. Finding 11 (Specification of Special Height Districts in Adopted Redevelopment Plans)

...

37.7.12. Finding 12 (Establishing Maximum Allowable Building Heights Within Special Height Districts)

...

37.7.13. Finding 13 (Additional Height for View Enhancement)

- A. The view enhancement is provided in the same threshold roadway travel route as the project in which the building using the additional height is located;
- B. For views of the natural landscape and views of major visual features, no building or structure greater than five feet in height is closer than 100 feet from the viewpoint to the resource;
- C. For view enhancements of views of Lake Tahoe, no building or structure exists between the viewpoint and Lake Tahoe;
- D. For the purposes of creating a view enhancement, TRPA shall find, in addition to the findings in subparagraphs A, B, and C above, that the created view is available for a continuous distance of at least ~~200~~ [500](#) feet as seen from the threshold roadway travel route; and

...

37.7.15. Finding 15 (Additional Height for Special Projects within North Stateline Community Plan)

Additional height may be specified within the North Stateline Community Plan subject to the following requirements:

...

- D. New structures along State Route 28 shall be set back from the travel route edge of pavement a minimum of 40 feet and stair-stepped upslope, providing a transition of height

across the site (See Figure 37.7.15-A). Additional height for new structures satisfying these requirements may be permitted as follows:

1. The maximum permissible height for structures with a minimum set back of 40 feet from the State Route 28 edge of pavement shall be ~~58~~ 26 feet.
2. The maximum permissible height for structures with a minimum set back of 60 feet from the State Route 28 edge of pavement shall be ~~67~~ 32 feet.
3. The maximum permissible height for structures with a minimum set back of 180 feet from the State Route 28 edge of pavement shall be ~~75~~ 42 feet.

[Figure 37.7.15-A: Setback Measurement on State Route 28]

...

37.7.16. Finding 16 (~~Three- or Four- Two~~ Story Buildings in Town Centers and Two Three- to Six-Story Buildings in the Regional Center)

In order to mitigate for potentially significant scenic impacts resulting from three- ~~or four-~~ story buildings in the Town Centers and from three- to six-story buildings in the Regional Center, TRPA shall make the following findings:

- A. The project shall meet findings 1, 3, 5, and 9 in Section 37.7.

37.7.17. Finding 17 (Redevelopment in High Density Tourist District within Existing Visual Prominence)

To mitigate for potentially significant scenic impacts resulting from buildings up to 197 feet in the High Density Tourist District, proposed development in the High Density Tourist District shall achieve the following performance standards:

- A. The height and visual mass of any redeveloped existing high-rise structures projecting above the forest canopy shall ~~not increase the visual prominence over-~~ negatively impact or block views from baseline scenic conditions as viewed and evaluated from key scenic viewpoints, including, but not limited to, views from the Van Sickle Bi-State Park, scenic roadway units, scenic shoreline units, and public recreation areas.
- B. When considering visual prominence, the following factors will be considered: building mass, contrast, location, articulation, color, materials and architectural style; and the quality of landscape features and views that are blocked or revealed.

...

d. TABLE 13.5.3-1: MINIMUM DEVELOPMENT STANDARDS FOR AREA PLANS

We request this table is revised to reflect a maximum of 42 feet in Town Center Overlays, and 95 feet in the Regional Center Overlay, with the exception of the portion of the Regional Center from the intersection of Pioneer Trail and highway 50 west, where the 42 foot height maximum will apply.

3. Evaluate the impacts of the following sources of ozone precursors and adopt measures to reduce emissions to obtain the ozone standard in the immediate future (ozone standards are not long term standards such as mid-lake clarity);
 - o motorized watercraft
 - o off road vehicles including snowmobiles and OHVs

The RPU EIS considered just one source of emissions of ozone precursors (NOx and VOCs)– on road motor vehicles, yet CARB’s Emissions Inventory (2008, as referenced by TRPA’s RPU EIS) shows that **off road** vehicles may contribute equal, if not higher amounts, of ozone precursors (approx. 47% of NOx emissions and 35% of VOC emissions¹²) – especially during the summer months when conditions are prime for ozone formation. However, the RPU EIS erroneously

¹² These estimates are based on annual averages, and therefore do not consider the increased use in off-road mobile sources in the Tahoe Basin during the summer months..

concluded positive trends in ozone (which is not the case in recent years, as shown by DRI data provided to TRPA in previous comments), that ozone standards were being met (as the California standard was and remains designated nonattainment-transitional), and then justified more traffic and development by estimating the future emissions from just one source group – on road motor vehicles, which comprise roughly 41% of the NOx emissions and 22% of VOC emissions. The technical analysis in the RPU EIS was even further flawed by the inadequacy of the air quality analysis to address what happens between 2013 and 2035, as the future on-road vehicle emissions were only assessed for 2035 (using EMFAC 2011; this statement applies to all pollutants but CO). Per this ‘logic’ as used in the EIS, public health (and forest health) standards could conceivably be violated every year between now and 2034, so long as they are attained in 2035.

We request the TRPA do the following in order to protect public health (from respiratory damage and other impacts of ozone) and lives (air pollution can be fatal), forest health and the environment, water resources, property, and public services:¹³

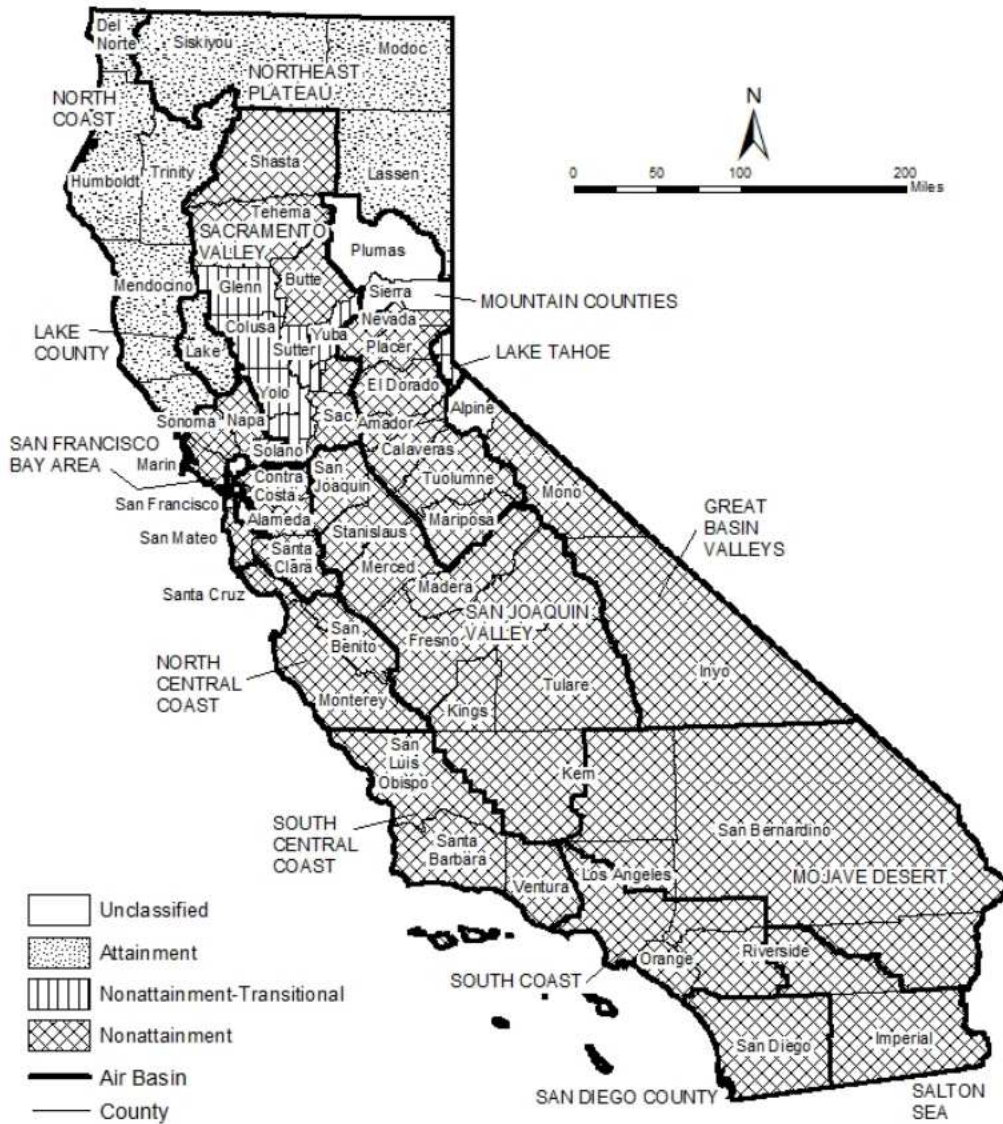
- a. No increase in the use of off-road motor vehicles (and/or associated increases in recreational capacity for these vehicles) will be allowed in the Lake Tahoe Basin until completion of thorough air quality analysis, and installation of an adequate, Basin-wide air quality monitoring network which can ensure air quality standards are not being violated.
- b. No new allocations, or expanded development projects that will result in more than 200 additional VMT per day, will be permitted until the following is completed:

Within one year, we request TRPA to:

- i. Evaluate the emissions of ozone precursors specific to the Lake Tahoe Basin, using local information wherever feasible (e.g. boat counts done by TRPA); update the impact analysis to analyze the affects of Tahoe’s inversions on air quality concentrations, movements, and dilution;
- ii. Perform a thorough scientific assessment of air quality conditions to identify the relative impacts of each source, and the reductions needed from each source to achieve standards (much like the Tahoe TMDL was done for water quality, only humans don’t have the luxury of breathing bad air for 65 years);
- iii. Install a Basin-wide network of air quality monitoring, with monitors located to address TRPA, federal, state, and local standards (e.g. local ozone monitors for human health where the greatest populations are, and locate ozone monitors for forest health in the forests).

¹³ It has been well established for decades that ozone damages pine trees and makes them more susceptible to other detrimental impacts (e.g. bark beetles); in fact, TRPA’s 1983 EIS for the thresholds was heavily focused on protecting forests from ozone damage. An unhealthy forest affects the entire ecosystem, water resources, and is more susceptible to fires like the Angora Fire, which can move from forests into communities and threaten lives, damage property and impact public services.

2012 Area Designations for State Ambient Air Quality Standards OZONE



Source Date:
February 2012
Air Quality Data Branch, PTSD



Tahoe Regional Planning Agency
Attn: Regional Plan Implementation Committee
128 Market Street
Stateline, NV 89449

July 17, 2013

Subject: Douglas County South Shore Area Plan and Related Documents

Dear Chair Clem Shute and Members of the Regional Plan Implementation Committee:

The Friends of the West Shore (FOWS) and the Tahoe Area Sierra Club (TASC) appreciate the opportunity to provide additional comments on the proposed Douglas County South Shore Area Plan (SSAP), the Delegation MOU for Area Plan implementation, the revised Initial Environmental Checklist (IEC), the responses to our previous questions, and related documents. We appreciate the efforts by TRPA and Douglas County staff to prepare the additional information in the staff report for the 7/18 RPIC hearing. We also thank members of the RPIC for requesting TRPA provide these answers.

We are, however, very concerned with the lack of clarity of the process for adoption of the DC SSAP by the County and by TRPA. In June, the Douglas County Board of County Commissioners (BOCC) approved the SSAP and delegation MOU, and “accepted” the related documents (e.g. IEC), however none of these documents were finalized. In fact, the MOU contained many areas where TRPA would later ‘insert language.’ The MOU also contained new changes which increase the environmental disturbance that would be allowed through exempt and qualified-exempt activities and projects, yet the environmental impacts of these changes had not been analyzed, nor were they contemplated or included in the RPU EIS. Some changes were not even proposed prior to adoption of the TRPA RPU in December 2012. Further, the Douglas County Board of County Commissioners (BOCC) set a tentative date in August for further review of the SSAP and MOU based on feedback from TRPA staff, RPIC, APC, and GB.

We have reviewed the 165-page staff packet prepared for the 7/18/2013 RPIC meeting, and provide additional comments below. Further, many of our most basic concerns still apply:

1. We do not believe the Compact allows the delegation of authority that is associated with this Area Plan and the MOU. We also believe the increases in exemptions and qualified-exemptions are improper.
2. The environmental impacts of the SSAP, and the cumulative impacts of the SSAP and reasonably foreseeable projects (e.g. the Heavenly Cal Base expansion), to areas within and adjacent to the SSAP (traffic being one of the greatest impacts of activities adjacent and in to the Basin), and the entire Basin, have not been adequately analyzed;
3. The SSAP does not show how it will achieve and maintain TRPA’s thresholds, and TRPA treats the Area Plans as if they must merely avoid ‘impeding’ threshold attainment and maintenance. Yet upon adoption, the Area Plan will become part of the RP, and TRPA must make findings for RP amendments that the amendment will

- help achieve and maintain the thresholds. In fact, as reiterated by TRPA staff on 6/26,¹ *“That leads into the Governing Board action when they will decide whether or not to amend the Regional Plan by including this area plan. The key finding is that the Regional Plan achieves and maintains Thresholds.”*
4. TRPA led the public to believe additional environmental review would be performed for the Area Plan² which would analyze the more localized impacts of Area Plans, as compared to the regional, ‘broad scale’ policy changes contemplated in the RPU EIS, yet the IEC includes no additional analysis. The IEC also fails to examine whether the ‘broad-scale’ assumptions in the RPU EIS comport to the proposed SSAP e.g. will transfers of development follow the general assumptions made for the RPU EIS regarding the location of future land use and associated traffic?
 5. The SSAP IEC “tiers” from the RPU EIS. As the RPU’s EIS is the subject of a pending lawsuit, it is premature to tier off the RPU EIS; and
 6. The SSAP, including TRPA’s MOU template and Conformity Checklists that will be used for all Area Plans, carry forward the problems and deficiencies of the RPU process and documents. We incorporate herein all comments by TASC and FOWS on the RPU, and all subsequent comments submitted related to Area Plans and RPU implementation (in 2013).

We are also very concerned with the ‘mix and match’ process that appears to be guiding the SSAP. It is unclear when a finalized SSAP, MOU, Conformity Checklist, and IEC will be heard for approval by Douglas County (details below). Finally, as noted in our previous comments (include our 3/27/2013 comments to TRPA), without sufficient analysis, the Compact’s mandated threshold-related findings³ can not be made.

“With the limited review scheduled for Area Plans combined with the noted environmental review documents, we are very concerned that there will not be adequate environmental evaluation and analysis of the local, regional, and cumulative environmental impacts of the Area Plans. Without adequate analysis, there would not be sufficient evidence to make the Compact’s threshold-related findings for Area Plans.”

The information available with the SSAP is not adequate and as a result, TRPA’s threshold-related findings can not, at this time, be made. Please feel free to contact Jennifer Quashnick at jqtahoe@sbcglobal.net or Laurel Ames at laurel@watershednetwork.org if you have any questions.

Sincerely,



Laurel Ames,
Conservation Co-Chair,
Tahoe Area Sierra Club



Susan Gearhart,
President,
Friends of the West Shore



Jennifer Quashnick
Conservation Consultant,
Friends of the West Shore

¹ This statement by John Hester is included in the minutes from the 6/26 meeting, page 8.

² Examples were provided in previous comments to Douglas County and TRPA.

³ Code of Ordinances, Chapter 4, Section 4.4, Threshold-Related Findings.

Throughout these comments, we refer to the minutes from the 6/26 RPIC meeting and ‘staff responses’ provided in the RPIC packet for the 7/18 meeting (titled: “ATTACHMENT 1: Questions and Comments, Responses to Questions and Comments, and Source of Question/Comment Process”). Unless otherwise noted, page numbers refer to the hard copy numbers in the RPIC packet.

I. Process Concerns

We have significant concerns regarding the process for the development and approval of the SSAP and associated documents. Not only has the process been rushed, and numerous changes not analyzed, but there has been little certainty regarding when the SSAP, MOU, and related documents will be finalized and brought for final approval by the BOCC. It is also unclear how this process – review/approval by local government, then review/recommendation by RPIC, then APC, then the GB, will work, if the RPIC will identify additional needs for area plans. This must be made clear and understandable, especially as future Area Plans are already scheduled for various reviews by their local governments, for public workshops, for review by the RPIC, and then up to a month or more later, review by the APC and then hearing by the GB. As noted in the minutes from the last RPIC meeting, the SSAP appears to be a mobile document, going back and forth between the County and TRPA:

“The Code specifies in 13.6.2.A that the area plan should be approved by the lead agency prior to TRPA’s review for conformance. That is not to say that we couldn’t come to the Regional Plan Implementation Committee prior to that for identification of issues. We advised Douglas County and the City of South Lake Tahoe to go through their planning commission and governing body to get the plan the way they believe it should be; we have worked with them at the staff level, it will then go through their process then it comes to TRPA. We have identified three steps; the Regional Plan Implementation Committee, the Advisory Planning Commission and the Governing Board. If there changes that the Board decides are needed for this plan to be found in conformance, it would go back to the local government. We did not want to go back to the local government after the RPIC, after APC and again after Governing Board. If overtime, you feel we need to adjust this process staff would be happy to do this.” (Page 8).

Obviously the BOCC did not adopt the final SSAP or MOU, because:

- The MOU was unfinished, as noted by several areas italicized where language would be added;
- Changes included in the MOU had not been adequately analyzed by TRPA (including the increased exempt and qualified exempt activities in the delegation MOU);
 - o TRPA did not perform any assessment of the impacts of these changes in the MOU;⁴
- The Conformity Checklist included items which do not conform to the RPU (e.g. the golf course use on the T-RR parcel, the lack of sufficient design guidelines for the casino towers, etc.);⁵

⁴ When members of the public raised the issue, include our groups, Douglas County stated TRPA said they would do an IEC for the MOU-related changes in July – after BOCC approval.

⁵ As noted below, although language has been added to the IEC regarding golf course uses, this does not change the fact that TRPA’s Code would not allow the new fertilizer use as it would affect several streams and areas of runoff.

- The BOCC ‘accepted’ an IEC that was, and remains, incomplete.

In fact, recognizing all of these gaps, during the 6/20 BOCC meeting, a tentative date for another hearing of the SSAP (based on review by TRPA) was set for 8/15/2013.

II. Area Plans must demonstrate threshold achievement:

We have previously commented on the disagreement with the new Code, Section 13.5.C.6, stating Area Plans must “**6. Demonstrate that all development activity within Town Centers and the Regional Centers will provide for or not interfere with Threshold gain, including but not limited to measurable improvements in water quality.**” [Emphasis added]. It is not enough to “not interfere with threshold gain,” or “not hinder or impede”⁶ threshold gain. TRPA is responsible for maintaining a Regional Plan which helps **achieve and maintain the thresholds**. The Area Plans will become part of the RP, and therefore must also help achieve and maintain the thresholds.

This was clearly explained in federal Judge Karlton’s ruling (Case 2:08-cv-02828-LKK-GGH Document 118, filed 9/16/2010) in favor of the TASC and LTLST challenge of TRPA’s 2008 Shorezone Ordinances:

“More fundamentally, however, TRPA misunderstands the nature of the obligation to achieve and maintain the thresholds. It is not enough to show that the Amendments do not make the problem worse. TRPA must ensure that the ordinances, as amended, implement the regional plan in a way that will actually achieve the thresholds. With regard to thresholds not presently in attainment, TRPA’s finding that the Amendments will not aggravate the problem is inadequate.” [Emphasis added].

A proper analysis must examine the full impacts of all amendments to the Regional Plan, including the Area Plans that will also be amendments to the RP, and how the Plans will help TRPA achieve and maintain thresholds. However, the IEC, TRPA’s responses, MOU, and other documents associated with the SSAP, have seemingly been viewed as plans that must simply ‘not interfere with’ threshold achievement and maintenance.

III. Presence of Regulations does not negate requirement to analyze environmental impacts:

The final RPU EIS, and the revised IEC, have made conclusions of ‘insignificance’ based upon the mere presence of regulations.

Final TRPA EIS, p. 3-53:

Once an Area Plan is in place, any proposed project would be subject to restrictions identified in the conforming Area Plan. Approval of the Area Plan would be subject to regulations specific to Resort Recreation areas, including requirements that any residential, commercial, or tourist uses are transferred from outside the area and result in the retirement of existing development (Final Draft Code Section 13.5.3.3). Any project proposed within the Resort Recreation District would be required to comply with all pertinent sections of the Final Draft Code of Ordinances, including ordinances related to site development (Chapters 30–39), growth management (Chapters 50–53),

⁶ TRPA’s response to question number 99 regarding the definition of “not interfere with threshold gain” states “the term ‘not interfere with’ means that the Area Plan will not hinder or impede.”

and resource management and protection (Chapters 60–68). Any project would also be required to comply with project-level environmental documentation requirements (Final Draft Code Chapter 3), which require identification and mitigation of any potentially significant environmental impacts. Prior to approving any project within the Resort Recreation District, TRPA must make the findings described in Final Draft Code Sections 4.4.1 and 4.4.2 demonstrating that the proposed project would not prohibit attainment or maintenance of any Threshold Standards. For these reasons, the impact of this land use designation change would be less than significant for the proposed Edgewood Mountain Resort Recreation area and mitigation would not be required... (p. 3-53). [Emphasis added].

Revised IEC, page 40:

“The Regional Plan EIS analyzed the changes in land use designation from Conservation to Resort Recreation, and the establishment of Mixed-Use zoning. This IEC assesses the potential effects of the proposed changes in land use resulting from the implementation of the SSAP. The existing requirements for site specific environmental review, combined with TRPA and Douglas County resource protection polices, provide the protections to ensure that all proposals are consistent with the Regional Plan and the potentially significant environmental impacts are identified, assessed, and mitigated. Therefore, although the SSAP would allow new uses that are currently prohibited under the existing community plans and PASs, the new uses are consistent with the types of uses envisioned in the Regional Plan and analyzed in the RPU EIS or are consistent with typical uses in similar areas throughout the Region. Further, because the regulations are in place today that would protect water supply, water quality, natural resources, recreation, scenic resources and TRPA environmental thresholds (as described above), the impact of the addition of golf course as a permissible use would be less than significant and mitigation would not be required.” (Page 40).

However, finding that an activity will comply the regulations does not assure that all impacts will be avoided or mitigated, and does not allow the agency to bypass the environmental analysis of the impacts. For example, compliance with the TMDL may account for certain impacts (fine sediment) but not others (nitrogen pollution). Case law supports this as well.⁷

⁷ “Compliance with the law is not enough to support a finding of no significant impact” under CEQA. *Californians for Alternatives to Toxics v. Dep’t of Food & Agric.* (“CATS”), 136 Cal. App. 4th 1, 17 (2005); *City of Antioch v. City Council*, 187 Cal. App. 3d 1325, 1331 (1986) (conformity with general plan does not “justify a finding that the project has no significant environmental effect”). An agency may not use compliance with a regulatory scheme to avoid meaningful consideration of “issues raised by the proposed project.” *CATS*, 136 Cal. App. 4th at 16. (EIR’s reliance on pesticide registration process did not account for project’s “specific uses of pesticides”); *Oro Fino Gold Mining Corp. v. County of El Dorado*, 225 Cal. App. 3d 872, 876 (1990) (reliance on compliance with local noise standards inadequate to find that significant impacts would be avoided, given testimony about noise from past projects); *Berkeley Jets*, 91 Cal. App. 4th at 1373, 1381 (“fixed” noise standard could not be “sole criterion” for determining significance of aircraft noise). In another example, *Cmties. for a Better Env’t v. Cal. Res. Agency*, 103 Cal.App.4th 98, 113-14 (2002) (confirming that regulatory standard may not be applied in way to foreclose substantial evidence of significant effect).

IV. Change from Community Plans and Plan Area Statements to Area Plans:

IV.A Previous Community Plans based on certified EIS:

We have consistently asked for environmental impacts of the RPU, and now, the Area Plans, to be analyzed – we have not dictated whether this be done through an EA or EIS. However, as impacts of the RPU must be fully analyzed, and the RPU EIS certified in December 2012 fails to do so, it is expected that an adequate analysis would require the comprehensive review of an EIS. What we can not speculate about is what level of environmental review Douglas County would need to perform if TRPA's RPU EIS were adequate. It is, however, worth noting that the existing Kingsbury and Stateline Community Plans were adopted after review in an EIS (see TRPA's November 1993 GB packet). Although the community plans were intended to further "implement" the 1987 Regional Plan (for which an EIS was certified), they were still subject to additional environmental impact analysis. As the Area Plans are also intended to implement the 2012 Regional Plan (for which an EIS was certified), should they not be subject to the same environmental review requirements? Why is such review not required now, yet the circumstances essentially mirror what was done in the past?

IV.B Confused Baseline and Existing conditions for Area Plan analysis:

There remains confusion regarding the 'baseline' that must be used for assessing the impacts of changes that will result from Area Plans. In addition, our comments regarding the RPU's 'baseline' still apply. In terms of the Area Plans, when we previously asked TRPA staff to provide the 'existing condition' regulations for the public to be able to compare what is allowed prior to adoption of the Area Plan compared to what was proposed (e.g. we asked this about height and density to Placer and El Dorado County as well as TRPA staff in previous meetings so the public would be aware of what had been allowed under the previous CP/PAS and could compare to what was proposed), TRPA responded that the RPU adopted on 12/12/12 equated to what was "currently allowed." However, TRPA has recognized that the existing Plan Area Statements and Community Plans still remain in effect prior to Area Plan adoption. Also, environmental review documents (e.g. an EIS) were certified prior to approval of the Community Plans. The RPU EIS did not update or replace the detailed impact analysis that was done at the community level to support the Community Plans, nor does the IEC provide a comparable impact analysis of the Area Plan either.

It appears TRPA has taken two conflicting approaches – in some cases, TRPA has stated the 'existing conditions' are the RPU's new limits. In other cases, as noted below, TRPA states that the existing CPs/PASs are the baseline. If a project applicant would have to consider the CP/PAS as the 'baseline' or "no action" alternative now (see below), then why wouldn't the Area Plan analysis have to use the same baseline? Further, given the original CP's were adopted after a full EIS was completed and certified, the Area Plans should have to clearly compare the proposed changes to the original Community Plans

and analyze those impacts. The only comparison presented appears to be the table of uses.

“What is currently in effect today is the 2012 TRPA Regional Plan and the updated Code of Ordinances. When the Regional Plan was adopted, the community plans and plan area statements remained in effect. These were the plans to implement the 1987 Regional Plan. Douglas County currently has three community plans; Kingsbury, Stateline and Roundhill and thirty plan area statements. We have design standards and guidelines in place for the three Douglas County community plans that were adopted in 1993. There is the Tahoe Basin regulations and Douglas County Code which is code regulations to implement the community plans and plan area statements. The overall objective is to replace the community plans and plan area statements with area plan to implement the Goals & Policies of the 2012 Regional Plan.” (RPIC meeting minutes from 6/26, page 9).

Mr. Marshall said because it is essentially is the existing regulatory regime that is in place. The baseline is existing conditions but if we are comparing a no project for example, it is the existing regulatory regime which is the plan area statements. Until an area plan is adopted you do not have these other uses available in that area. What we are trying to do is disclose that there is going to be “new” use because we are shifting to an area plan, but then where is that environmental analyzes for those new uses. That was conducted, so we are tiering to the RPU EIS. The best we can fully disclosing the change in use on the ground and pointing to where the environmental analyses of that change took place either in the checklist or tiering to the RPU EIS.

Mr. Yeates asked if the Regional Plan Update EIS would have looked at the addition of these uses being proposed in this area plan.

Mr. Marshall said yes that is correct.

Mr. Marshall said he does not necessarily agree that is the baseline. He feels the baseline is the existing regulatory regime in place which are these plan area statements until they are replaced by the area plan. The analysis for that change was done in the RPU EIS, but the actual change does not happen until you take the action of approving the area plan.” (p. 18). [Emphasis added].

IV.B.1 Comparison of Height and Density in PAS/CPs vs. SSAP:

Additionally, with regards to assessing the baseline/existing conditions versus proposed (or RPU-related) changes, we have requested that the public be provided with the height and density that was previously allowed (before the RPU adoption) so it can reasonably be compared to what the RPU (and proposed Area Plan) would allow. We do not feel this is an unreasonable request. In fact, according to Ascent Environmental, this information was provided for permitted land uses:

“Mr. Yeates said one of the attachments to the Douglas County report prepared was called TRPA Initial Environmental Checklist, Appendix B, Summary of proposed SSAP land use changes...It suggests to him that it is a new use that may have some environmental impact. For example, it lists employee housing, multiple family dwellings, multiple person dwellings, bed and breakfast facilities, hotel, motel and other transient dwellings, time sharing, time sharing with residential design, eating and drinking place, outdoor retail sales, retail personal service; all of these are listed as new. That suggest that this may have a potential environmental impact that was not analyzed in the prior Regional Plan because by name a new use.

Nanette Hansel, Ascent Environmental said they prepared these tables; and what they are intended to show is a comparison of the permitted uses that are allowed under the existing plan area statements and community plans compared with what would be allowed by the zoning in the South Shore Area Plan. Where there is a new use that is a use that would not be allowed if the plan

area statement was in place. The Initial Environmental Checklist looked at uses from a perspective of did the Regional Plan contemplate that type of use. For Plan Area Statement 80, Resort Recreation parcel; the Regional Plan contemplated residential uses as an allowed use, our IEC analyses tiered from that. The intent when we looked through these use changes was to give consideration to uses that could have localized effects that weren't contemplated, but if they fell within the broader use category that was contemplated in the Regional Plan we tiered from that and did not go any further since this is still a plan level document." (page 17). [Emphasis added]

Conversely, we would like to see a "comparison of the permitted heights and densities that are allowed under the existing [PASs and CPs] compared with what would be allowed by the zoning in the South Shore Area Plan."

V. Inadequate Environmental Analysis of Individual and Cumulative Impacts:

V.A Lack of analysis of localized impacts:

Further, the RPU EIS's 'broad' view did not analyze the localized impacts of the Area Plan, and the IEC does not provide any additional analysis of environmental impacts. Although some additional mapping was done regarding the golf course use on the T-RR parcel, this new information is simply a presentation of available data and regulations. Further, staff's response to question number 75 confirms the Area Plans will replace the existing Community Plans and Plan Area Statements. The CPs were originally approved after detailed, site-specific analyses were performed in an EIS (this was in addition to the regional EIS for the 1987 Regional Plan).

Currently, TRPA has proclaimed the RPU EIS provides the broad, regional review, yet unlike before, the local government and TRPA are not providing a localized impact analysis, instead referring to the broad, programmatic level RPU EIS as having evaluated the impacts. Thus, it appears the agencies are 'skipping' the localized impact analysis as if it doesn't count. The Compact's requirements for environmental analysis have not been changed since the Community Plan for this Area was adopted in 1993.⁸ What makes the Compact's standards any less relevant in 2013?

V.B Request for identification of referenced analyses in RPU EIS:

During public comment, a request was made that the IEC include specific references to where the RPU EIS analyzed an impact.⁹ The IEC has not been revised to reflect this information, although if the IEC is relying on an analysis done elsewhere, the specific location of that analysis should be identified for the public. For example, where the IEC

⁸ The courts continue to use NEPA and CEQA for guidance when reviewing TRPA's requirements as well, and those regulations have maintained some very basic review and disclosure requirements for decades.

⁹ "Ellie Waller, Tahoe Vista resident said she will submit her comments that she would have made today. Since Ascent has been used as the expert for the Regional Plan and helping Douglas County, she asked to see how they determined that tiering references were appropriate. Where in the Regional Plan Update is it identified that an environmental analysis is equal to or greater than what Douglas County may have to do. It should not be that difficult to align the sections in Chapter 13 from the Regional Plan." (p. 16 in RPIC minutes).

states “This potential effect is the same as the RPU, and therefore the analysis is tiered from and consistent with the RPU EIS,” we believe the IEC should clarify *where* the impact was analyzed in the EIS.

V.C Lack of Cumulative Impacts Analysis:

In TRPA’s Table of Responses to Comments, a question from the public is summarized as: “4. How is the cumulative impact of each Area Plan addressed?” The response to this question below is followed by another response to our question (labeled number 35 in the Table):

TRPA assesses the cumulative impacts arising from the “new” policies within Area Plans in both the RPU EIS and the environmental document accompanying each Area Plan. The RPU EIS itself represents a “cumulative” examination of the impacts of area planning policies, including, e.g., those impacts from redevelopment, height limitations, and traffic (including both VMT and LOS). The main “cumulative” effects analysis therefore has already occurred in the RPU EIS. However, TRPA must ensure that for each Area Plan considered, no unique circumstances exist that might cause insignificant impacts that when aggregated with other reasonably foreseeable actions might have a significant effect. For the SSAP, the IEC concludes that no such circumstances exist. [JM]

The cumulative impacts analysis of the RPU EIS (TRPA 2012a, pages 4-2 through 4-10) includes environmental enhancement projects, land management plans, TTD/TMPO projects and programs, and other development projects. The SSAP IEC is tiered from this EIS, including its comprehensive cumulative analysis. The scope and characteristics of cumulative actions have not substantially changed; no additional cumulative projects or programs are known at this time. [AE]

The responses refer back to the RPU EIS as supposedly performing the cumulative impact analysis. However, as noted in our extensive comments filed with TRPA the RPU EIS was wholly inadequate. For example, the RPU’s cumulative impacts analysis performs no actual review, rather, it refers back to the individual chapters for each subject area, claiming an estimate of buildout will suffice (below). However, the RPU EIS notes it only compared the proposed changes under each alternative.¹⁰ This did not account for the impacts of development already on the ground, nor does it include the future consequences from reasonably foreseeable projects, and impacts from increased growth in urban areas within a few hours’ drive to the Basin. Cumulative impacts include the impacts of actions not under the control of TRPA.

One of the most obvious examples of cumulative impacts in the Basin is reflected by our traffic. As development outside of the Basin increased, so did visitation to the Basin. With the expansion of Northstar came more visitors who drive to north shore, and many, around the Lake. Add these impacts to increases from TRPA’s Plan and the results are **cumulative**. But, if TRPA’s logic is applied to environmental disclosure laws, then there

¹⁰ “...the analysis in Chapter 3 is cumulative because it assesses the build-out condition of each alternative. Because the Regional Plan Update is a policy-level undertaking, and the precise locations, magnitudes, and character of most projects implementing the Plan cannot yet be known, descriptions of cumulative effects below are qualitative and do not include quantification of specific effects associated with cumulative impacts.” (RPU EIS, Chapter 4).

would be no reason for NEPA or CEQA, or TRPA's Compact, to require a cumulative impacts analysis, so long as the individual impacts were assessed.

In addition, although the RPU EIS claims all transportation impacts were analyzed, this is not correct. Not only were the impacts of visitor traffic inadequately evaluated, as explained in our comments on the RPU EIS, RTP EIR/S, and other Mobility 2035 documents, but the RPU EIS failed to include reasonably foreseeable projects in its cumulative impacts assessment, which at the time included expansions of projects like the Squaw Valley Village and Northstar. The traffic impacts from these two examples, and other projects, will no doubt generate traffic throughout the entire Lake Tahoe Basin (note that both projects had been proposed to the public prior to the release of the draft RPU EIS, and were certainly 'reasonably foreseeable').

In summary, TRPA explains that the IEC concluded no cumulative impacts. However, this is because the IEC refers back to the RPU EIS, claiming that TRPA already examined the cumulative impacts. Once again TRPA and the County appear to be tossing the ball back and forth at each other. Ultimately, TRPA must meet the Compact, which requires adequate environmental review. Yet as noted in our comments on the RPU EIS, the EIS itself also fails to perform an adequate cumulative impacts review.

V.D No "Regional" Planning:

TRPA's response to a question from the public regarding height and scenic standards¹¹ reveals a failure to analyze the regional impacts of development. For example, the height of one project may block views of the Lake and mountain from many areas outside of the Area Plan. Further, increases in buildings will have a negative impact on the view of areas from the Lake, mountains, and other areas of significance. Further, although a single project may protect 'some' portion of an existing view, when multiple projects are allowed, the cumulative impacts can be orders of magnitude greater. However, based on TRPA's logic, impacts need only be considered at the project-level review. Is the developer going to perform the regional analysis that TRPA has pushed off to the locals (who are now pushing it off to the individual project level)?

The RPU EIS and the SSAP IEC examine the impacts associated with the changes in policies, not individual projects (which will be analyzed if and when considered by TRPA for approval). In many instances, the RPU EIS made assumptions regarding future development in order to assess the impacts from policy changes, but that plan level, programmatic analysis cannot substitute for subsequent project level environmental documentation (although TRPA expects that some amount of tiering to the RPU EIS may occur in project level documents). [JM]

On the other hand, it appears that TRPA is anticipating that project-level reviews will also simply refer back to the RPU EIS for the analysis of impacts, as noted in the tiering comment above. Thus, we have TRPA putting analysis off to the locals, the locals saying TRPA already analyzed it and/or it will be analyzed at the project level, and now we can

¹¹ 7. How is the distinction made between Area Plan standards (e.g., height, scenic standards) and projects?

expect a developer to refer back to the RPU EIS and likely the Area Plan IEC and claim their impacts were already analyzed too – thus avoiding *any* consideration of the regional, cumulative, and possibly individual impacts of any development! **Where is the regional planning?**

V.E Revisions to Impacts in IEC:

V.E.1 Comments regarding the IEC's conclusions of "no" or "mitigated" impacts:

In response to our comments regarding the inexplicable claim in the IEC that there were often "no" impacts, staff provided some generalized responses in the Table. The information in the revised IEC still does not support the conclusion of no or mitigated impacts. Some of staff's responses are noted below, followed by additional comments:

[In addition to repetitive statements like "no change compared to RPU" ...]

• Introduction of new vegetation, excessive fertilizer, barrier to replenishment of existing species: Existing protections pertaining to fertilizer use and management remain in effect, and no projects are proposed that would increase fertilizer use.

Note that a former TRPA source control measure for nitrogen included restricting or banning fertilizer sales and use. The nearshore impacts we are seeing today refute the claim that 'all is well.' Whether a project specifically proposes to increase fertilizer use or not, if a project will add turf and lawns to the Basin, fertilizers will be required. The proposed golf course use on the Edgewood Mountain parcel – a reasonably foreseeable project - will clearly require new fertilizer use.

• Traffic generation and impacts on transportation: The SSAP would not alter or revise regulations pertaining to trip generation and is consistent with the Regional Plan in terms of traffic generation. While development and redevelopment within the SSAP as a whole is likely to generate 100 or more new daily vehicle trip ends (criterion used to determine the need for project specific traffic analyses), any project that would generate that volume would prepare the analyses and mitigate for any impacts in accordance with the TRPA Code.

This statement confirms the SSAP will create additional traffic beyond the limits considered significant by TRPA's Code. Therefore, the SSAP itself must show how it will mitigate these impacts to less than significant. It is inappropriate to put this off to multiple "project-level" reviews, nor can environmental analysis be avoided by stating regulations will be in place to mitigate impacts.

• Scenic and visibility: As described in the IEC, the SSAP would implement the provisions of the Regional Plan (such as increased density and height in community centers) intended to incentivize redevelopment, while protecting scenic resources through South Shore Design Standards and Guidelines and Chapters 4, 36, 37, and 66 of the TRPA Code (see page 48 -53 of the IEC). Because the SSAP does not propose revisions to the Regional Plan, nor include proposals for development projects, no additional analysis is required. [AE]

See comments regarding the cumulative impacts to scenic views and other resources. Further, we remind TRPA and DC the Compact's focus was to protect the scenic quality of the natural environment. The design standards and guidelines are based on the design of buildings, not the protection of view sheds. Resolution 82-11 requires

that the built environment “**be compatible with** the natural, scenic, and recreational values of the basin.”

V.E.2 Examples: Impact Revisions:

Several were changed from “No” impact to “yes” with an asterisk. However, nothing has been proposed to reduce the impacts; instead, the revised IEC appears to simply reiterate the claim that TRPA’s RPU already analyzed it - - not adequately, we note -- and/or already concluded it was an acceptable impact. Although many of our comments still stand, we add the following examples:

(i) Example Revision: Air Quality and Human Health impacts:

The question regarding diesel impacts was changed to “yes.” (p. 15). The IEC concludes: “*While the proposed SSAP could result in increased development, redevelopment, and construction activity resulting in increased use of diesel fuel, this increase in fuel use would not result in the exposure of sensitive receptors to toxic air contaminant emissions.*”

What information supports the claim that the increased use of diesel will not expose people to TACs? None is provided.

Therefore, because measures identified in the RPU EIS that would reduce construction-related TAC emissions to the extent feasible have been adopted by Douglas County and are part of the proposed SSAP, subsequent projects under the SSAP involving the use of heavy-duty diesel-fueled construction equipment would not result in the exposure of sensitive receptors to TACs.

Where will the cumulative, localized impacts of the diesel emissions from all of the construction at Edgewood, including Edgewood Mountain, and possibly the casinos, be analyzed? Also, when examined locally, thousands of people will be exposed to these emissions within the DC SSAP – a situation that may not exist regionally, and was not analyzed by TRPA “regional” programmatic EIS.

(ii) Example revision: Transportation:

The IEC’s Transportation section has been revised to include the following statement:

** While the proposed SSAP would permit increased development which could result in generation of 100 or more DVTE, this change is consistent with the increase in development envisioned in the Regional Plan and analyzed in the Regional Plan Update EIS, which is projected to result in a 10 percent reduction in VMT region wide. Further, the proposal under consideration is not a single project (to which the standard of 100 or more DVTE is applicable), but an area plan, the implementation of which would likely result in that level of traffic increase.*

First, this is not correct. The RPU claims a reduction in future ‘per capita’ VMT, although even those estimates are flawed because they exclude through trips and the increased VMT associated with increasing visitor attractions in the Basin. The RPU does, however, acknowledge that overall Basin-wide VMT, which is the threshold standard - - not “per capita” - - will increase. Second, as noted in this

language, the Area Plan will result in this level of traffic increase, and this must be analyzed.

V.F IEC Revisions ignore potential impacts in lieu of evaluating them:

The IEC went from failing to analyze the impacts of the SSAP to instead, trying to delete language that impacts may result. This is reflective of the overall nature of this process to skirt responsibility for any impacts. The following text with strikethroughs in the revised SSAP IEC on pages 29 and 32 provide a glaring example. Who will protect people and wildlife from the noise impacts of construction and increases in development? Pretending there is no impact in the document doesn't actually remove the impact.

~~“Consistent with the RPU EIS findings, the development of new residential and tourist accommodation uses in community centers within the SSAP could place new, more noise sensitive land uses in locations where ambient noise levels are incompatible. The noise environment in the two SSAP community centers (i.e., the T-MU/TC and T-T/HDT zoning districts) is influenced by multiple noise sources, including highways and roadways; transit vehicles; delivery trucks serving commercial establishments; heating, air conditioning, and ventilation equipment on buildings; light industrial uses; outdoor events with amplified sound; and landscape maintenance activities. All new residential units constructed in these areas would achieve an acceptable interior noise level of 45 dBA CNEL, as required by the 2006 International Residential Code (International Code Council 2006), which is also required in Section 20.690.030 of the Douglas County Code. However, depending on their design and location, the outdoor activity areas of new residential and tourist accommodation uses may be exposed to exterior noise levels that are incompatible with such uses.”~~

~~During construction, nearby residences and other noise sensitive receptors could be exposed to noise levels that exceed applicable Douglas County and TRPA standards outside of the exempt hours between 8:00 a.m. and 6:30 p.m., and/or expose nearby noise sensitive receptors to excessive or severe noise levels. Therefore, construction activities could expose people to severe and/or nuisance noise levels unless mitigation is incorporated on a project specific basis. Construction activities that occur between 8:00 a.m. and 6:30 p.m. are exempt from Douglas County and TRPA standards CNEL noise standards.~~

In apparent response to comments that the SSAP IEC failed to perform the additional environmental analysis that TRPA led the public to expect, additional language has been added to “explain” - yet again - why no such environmental analysis will occur. Page 36 states:

The uses that would be permissible within the T-RR zoning district (a portion of PAS 080) reflect the mix of uses envisioned for the two resort recreation areas in the Regional Plan (recreation, residential, tourist accommodation, and commercial uses [TRPA 2012b: page 3-53]); and would be subject to specific requirements in Chapter 13 of the TRPA Code, including a requirement that any new development be the result of transfers of development that result in the retirement of existing development. In general, the uses that would be allowed within the T-RR zoning district are consistent in character with currently allowed uses or those analyzed in the RPU EIS and most do not have unique characteristics that warrant additional analysis in this IEC. Continuation of uses that are currently permissible but that could have unique localized effects (such as equestrian stables and off-road vehicle courses) were not considered further in this IEC, because there is no change resulting from SSAP implementation. Some uses that are listed as permissible under the existing PASs, such as cemeteries and rural sports facilities (including shooting and archery ranges), would be eliminated. Although golf courses, which would be a new permissible use with

the SSAP, are consistent with the “Resort Recreation” land use classification and are listed recreation uses in Chapter 21 of the TRPA Code, this new use could have localized impacts and therefore, is discussed below. In considering effects of golf course uses within the T-RR zoning district, the *Edgewood Lodge Golf Course Improvement Project EIS*, certified by the TRPA Governing Board on August 22, 2012, was reviewed as it relates to fertilizer use and related effects on surface and groundwater quality.

The Edgewood project EIS was certified before the RPU and SSAP documents were approved, and well before the SSAP IEC came forward with the proposal to increase the extent of the golf course on to the T-RR parcel. The RPU, as approved on 12/12/12, still contains the prohibition to allow a new use that will generate fertilizer runoff into sensitive areas, streams, and eventually, the Lake.

V.G IEC’s additional information related to change in use for golf course:

The IEC includes “additional analysis” of the impacts of the proposed golf course use on the T-RR parcel, beginning on page 36:

“GOLF COURSES

Implementation of the SSAP would add golf courses as a permissible use throughout the Edgewood Mountain parcel in the T-RR zoning district (currently PAS 080). The Chapter 21 use definition for golf courses includes a wide range of golf facilities, including driving ranges, putting greens, and accessory uses (Section 21.4 of the TRPA Code). Golf facilities can range in size and character (such as 9 hole versus 18 hole, or putting greens only and no fairways). As no projects have been proposed nor applications submitted, it would be speculative to assume that a golf course project would be proposed, or to make assumptions as to the size or nature of any potential future golf facility within the T-RR zoning district.” [Emphasis added]

This is blatantly misleading to the public, as the SSAP clearly includes the desire to add a golf course facility to the T-RR parcel. There has been a great deal of discussion about this, and the County obviously performed ‘additional review’ of the golf course use for a reason. Throughout the revised IEC checklist, proposed changes include focusing on a golf course use, and removing other possible uses. It is inexplicable to claim this would be ‘speculative.’

“Generally, a golf course within the T-RR zoning district could require additional water to irrigate turf areas and landscaping; the use of fertilizers for landscaping establishment and maintenance; grading and tree removal; land coverage related to cart paths and accessory facilities; and recreation allocations. Fertilizer use, grading, and the creation of additional impervious surfaces would have the potential to affect water quality in Edgewood Creek, Eagle Rock Creek, and Lake Tahoe (located just over 0.5 mile west of US 50 and the western boundary of the T-RR zoning district). Tree removal and grading have the potential to affect scenic quality and habitat. However, any future golf course development within the T-RR zoning district would be subject to subsequent environmental review and permitting by TRPA and required to adhere to the regulations that would protect these resources as described below.”

“Furthermore, Section 60.1.8.A of the TRPA Code prohibits the use of fertilizers in SEZ areas, except for maintenance of pre-existing landscaping. Approximately 22 percent of the 250-acre Edgewood Mountain parcel (T-RR zoning district) is located within SEZ (Exhibit 1; Table 6). Golf course facilities would be prohibited in these areas. Additionally, fertilizer use would be subject to the SEZ setbacks described in Section 53.9.3 of the TRPA Code; a minimum separation

of 10 to 60 feet would be required between the edge of fertilized landscaping and the edge of SEZ.
“ [Emphasis added]

Further, at the last RPIC meeting, TRPA counsel explained:

“...[the] difference in a programmatic plan level environmental analysis and a project level environmental analysis. When you are making changes for example a potential that would allow a new use while you may not be able to analyze its specific impacts because you are uncertain of where the development may be proposed then that can be deferred until later. However, you have to undertake an analysis of the general consequences of allowing a particular zone change.”

With the change to Resort Recreation by TRPA, and subsequently to T-RR in the SSAP, the specific inclusion of a golf course use, and discussion regarding where it may be placed in terms of land capability, it is obviously clear where ‘the development may be proposed’ thus the specific impacts of this land use change can and must be analyzed.

Although we appreciate the inclusion of close-up map of the land capability where the “speculative” golf course use is desired, as the IEC now explains, the golf course would be limited to the forested areas in the eastern portion of the T-RR zoning district.¹² First, we reiterate the concern about nutrients entering SEZs and creeks and being carried to Lake Tahoe. Second, given that golf courses usually have open areas without trees, it appears that a significant number of trees would have to be cut to accommodate the ‘speculative’ golf course on these areas. This will change the entire character of this portion of forest, will negatively impact scenic views, wildlife habitat, and create numerous other significant environmental impacts.

Although the IEC states that such impacts will be analyzed at the “project-level” (see below), the cumulative impacts of cutting down the trees and reshaping the forest, planting grass and/or creating other disturbance to this area, impacts to views from throughout the Basin that will now include another disturbed area of forest, increases in runoff due to changes to currently healthy, infiltrating soils, etc., will all experience cumulative impacts from this proposed change. These changes only further compound the impacts associated with the approved Edgewood Lodge expansion and the plan’s allowed changes for the casino towers, all of which will create dominant buildings that will block existing views.

These must be analyzed and a project-level analysis will not be able to adequately consider the cumulative impacts to not only areas within the SSAP, but also to adjacent Area Plans (e.g. Tourist Core) and the entire Basin (e.g. Lake Tahoe). For example, the SSAP will draw more tourists to the area, a majority of which will come through South

¹² From the revised IEC: “Finally, while Section 30.5 of the TRPA Code prohibits new disturbance in low capability lands (LCDs 1a, 1b, 1c, 2, and 3), except under certain circumstances, Douglas County has restricted golf development to high capability lands (LCDs 4, 5, 6, and 7) by adding a footnote to the permitted use table in Section 20.703.090 of the Douglas County Code. Less than half of the T-RR zoning district is located in high capability lands. The 1.9 acres of land in LCD 7 at the corner of US 50 and SR 207 is too small to accommodate golf facilities. For these reasons, golf uses would be limited to the forested areas in the eastern portion of the T-RR zoning district, beyond the meadow area flanking US 50 and Lake Parkway (Exhibit 1).”

Lake Tahoe (e.g. visitors from the Bay Area and Sacramento Valley). This will increase traffic through Meyers and the City of SLT. TRPA claims the RPU analyzed these impacts, but the RPU EIS only examined the ‘assumed’ differences in the additional development under each alternative (see our 6/20 and 6/26 comments regarding the need to compare the assumptions in the RPU EIS to the SSAP’s land use scheme).

“Any future golf facility would be subject to subsequent project-level environmental documentation requirements (Chapter 3 of the TRPA Code), which require identification and mitigation of any potentially significant environmental impacts. Prior to approving any project within the T-RR zoning district, including any potential future golf facilities, TRPA must make the findings described in Sections 4.4.1 and 4.4.2 of the TRPA Code demonstrating that the proposed project would not prohibit attainment or maintenance of any Threshold Standards.” [Emphasis added]

We remind TRPA that its purpose is not to ‘prevent projects from getting in the way of threshold achievement or maintenance.’ Although the reference above may be to a project, the RPU change that rezoned this parcel to Resort Recreation was based on an *amendment* to the Plan. Further, TRPA’s approval of an Area Plan will also be an *amendment* to the Regional Plan. As noted previously, this requires the Area Plan to demonstrate actual benefits towards achievement and maintenance of the thresholds.

VI. Concerns with MOU:

Although staff stated changes would be made based on some of our comments on the proposed delegation MOU,¹³ we still have many concerns (in addition to the delegation itself). Comments include, but are not limited to, the following:

VI.A MOU – Tracking vs. Monitoring:

We asked that the MOU clearly identify the difference between science-based monitoring and tracking.¹⁴ For example, the MOU requires tracking of the County’s permitting activities. However, the MOU language refers to this as monitoring, yet monitoring the status of the thresholds in Lake Tahoe typically involves the *physical measurements* of environmental conditions. The response in the Table does not actually address our comment. Strangely, even as TRPA appears to explain why the word ‘monitoring’ will still be used, a definition of the activity is provided which clearly states “tracking.” Why not just revise the language to be clear?

36. Tracking vs. monitoring

Monitoring is used here to describe the process of TRPA staff receiving information on all building permits and checking a portion of those for compliance with the relevant Code requirements. The way it is used is consistent with the Merriam-Webster definition: “to watch, keep track of, or check usually for a special purpose”. [JH]

¹³ A revised MOU was emailed by TRPA on Tuesday, July 16; our comments are based on the packet made available July 11.

¹⁴ In our email to John Hester on 7/1/2013, we requested: “The documents also need to clearly separate and distinguish science-based monitoring from tracking (e.g. quarterly reports required by the MOU represent tracking, not monitoring as stated in the documents).”

VI.B MOU-related increases in exempt and qualified exempt activities:

In response to our concerns regarding the increases in exempt and qualified exempt activities included in the proposed delegation MOU (labeled comment no. 55 in the RPIC Table), TRPA staff are preparing an IEC for these changes.¹⁵ Because the changes allow more coverage and disturbance, more polluting activities, etc., to occur without any review, the cumulative impacts could be substantial. We remain concerned the IEC will fail to analyze the cumulative impacts of these changes. In addition, TRPA intends to use this MOU format for all Area Plan delegations, thus the impacts of these changes will occur basin-wide. Where will these cumulative impacts be analyzed? Finally, we note that we do not believe the Compact allows for such exemptions.

VI.C MOU failure to address thresholds:

As noted in our previous comments, the MOU language does not even mention TRPA's thresholds, yet the Area Plans are supposed to be adopted as part of the Regional Plan, and thus responsible for helping to achieve and maintain thresholds. According to TRPA's response in the Final EIS, page 3-50:

To be found in conformance with the Regional Plan, the Area Plan must demonstrate that it will achieve and maintain TRPA Threshold Standards. (p. 3-49 – 50). [Emphasis added].

If the Area Plan must demonstrate it will achieve and maintain thresholds, the MOU should require this (we note staff's response that the MOU will be amended to include this reference). However, this does not resolve the disconnect between TRPA monitoring the thresholds and local government's making planning approvals in a separate, disconnected process.¹⁶ How can the Area Plans ensure development is commensurate with achieving and maintaining thresholds if TRPA will only 'report' on the status of thresholds every four years? How much environmental damage could be approved in that four year time frame? There needs to be a direct connection between all development approvals and the measured status of the thresholds. (This also reiterates one of the reasons we do not believe the Compact allows for this level of delegation – one regional body should be responsible for approving development that is consistent with the thresholds). Additionally, TRPA was tasked with doing the same thing – to review the threshold standards and adjust the RP as needed to ensure standards are met – yet even TRPA, as the only agency involved, failed to do so.¹⁷ What previously required only TRPA review and amendments will now require both TRPA and local governments' review and amendments.

¹⁵ "A separate Initial Environmental Checklist for the MOU was prepared and will be made available for public review prior to the Advisory Planning Commission and Governing Board meetings. [PN]" (p. 25).

¹⁶ "The proposed MOU requires the local jurisdiction to impose project conditions of approval and inspect permitted projects for conformance with Regional Plan which will ensure conformity with soil conservation, scenic quality and other applicable thresholds. TRPA will continue to be responsible for on-the-ground threshold monitoring. [PN]" (p. 19).

¹⁷ This is detailed in our comments to TRPA on the RPU throughout 2012.

VI.D Public Notification for Activities Reduced:

TRPA's responses explain that in the shift from the RPU to the Area Plan, property owners will be provided less notice of possible activities on adjacent properties than TRPA has provided.¹⁸ As the Area Plan is a document which implements the Regional Plan, we believe that property owners should be afforded the same level of notice as TRPA would provide. Further, there is no definition in the Code or in the Rules of Procedure of an "affected property owner". TRPA and the City have been known to not inform property owners that were immediately adjacent to the property, but more than 100 feet from the project, as if that made a difference to the affected adjacent property owners. The AP codes and guidance and the Rules of Procedure must be amended to provide guidance to the notice provisions, and to assure that adjacent property owners are informed. In fact, property owners who will be impacted by several months or years of construction emissions and noise should be notified, property owners several parcels away that will have their views cut off should be notified, and property owners who will be subject to new costs due to their planned future inclusion in an area-wide treatment scheme to solve a project's need for better BMPs should be notified months, and preferably years, before any approval actions are taken on the project.

VI.E Lag time from TRPA-approved amendments to Area Plan amendments:

Below is our question followed by the staff response in the RPIC packet:

"53. The Public Entity has one year to amend the Area Plan to demonstrate conformity with the TRPA amendment." Will TRPA enforce the RPU amendments at the local level in the meantime? Currently, amendments take effect in 60 days; this may allow a 10 month additional lag time.

Response:

"Regional Plan and Code amendments will be applied and enforced from the effective date of the amendment and are not dependent on Douglas County amending the area plan. [PN]"

How will this work if Douglas County is approving projects based on the Area Plan, and TRPA's amendments will directly impact projects?

VI.F Alternative/Substitute Standards:

In our previous comments on the MOU, we asked the following questions:

"The following standards shall apply to activities authorized under this MOU. The Parties shall consult with each other regarding any uncertainties about these standards. Alternative standards may be approved by the TRPA MOU Coordinator when the results are determined to be equal or superior to these standards."

- It is unclear if the parties' consultation will involve the public – this should be a public process, just as the MOU requires public hearing.
- What constitutes an "Alternative standard?"
- What criteria will be used to assess whether a standard is "equal or superior?"

¹⁸ "The TRPA Code does not specify when notices to affected property owners must be provided, however TRPA's practice is to provide notice 14 days prior to a hearing. Douglas County proposes 10 days which is consistent with Douglas County Code for the entire County. [PN]" (page 22).

- How will the public be included in this ‘equal or superior’ determination, as it affects the MOU, which requires public hearing?

We also asked the following with regards to the MOU Coordinator:

“Communication: The Parties shall each designate a liaison for direct communication of matters related to this MOU. The Public Entity liaison and the TRPA MOU Coordinator shall meet at least once per year to review this MOU and to establish policy directives, training needs, and renew communications.”

- What are the qualifications for the Public Entity liaison and the TRPA MOU Coordinator? How will these representatives be selected and overseen?
- How will the public be included in this process? Given the annual review promised by TRPA and the public nature of the MOU document, a clear public participation process must be included.

Staff’s response (below) does not address our questions. Further, “may require” an amendment does not guarantee the public will be involved in any determinations of ‘equal or superior’ standards:

Alternative standards that are equal or superior may be developed based on best professional judgment and may require amendment of the MOU depending on the scope and scale of the proposed alternative standards. Amendment of the MOU will require a public hearing and approval by the TRPA Governing Board. [PN] [Emphasis added]. (p. 31).

TRPA and Douglas County will each select their own public liaisons that will be familiar with permitting regulations and processes for resolving disputes. [PN]

The determination of whether substitute standards are equal or superior can not be done ‘in house’ by agency staff, without consideration for the public process. These responses do not state who’s professional judgment will be used, what their qualifications will be, how public input will be solicited, and what other parameters will be used to assess this.

VI.G Cumulative noise impacts:

The increased exempt and qualified-exempt activities allowed by the MOU will allow more noise-generating activities to occur. We are concerned that this will increase noise in the Basin, and the impacts of these changes have not been analyzed. We hope the IEC being prepared for these changes fully analyzes this impact.

VII. Environmental Impacts of Proposed T-RR/Golf Course:

TRPA’s response (below) to our comments about the failure to analyze the impacts of a new golf course on the T-RR parcel (by both the RPU EIS and IEC) implies that the RPU EIS did contemplate the golf course use –which we also note is not the same as analyzing the impacts of the new use. However, what we found was an indirect reference in the RPU EIS to this use, but no analysis of the impacts. RPIC members raised the same question:

Mr. Shute said he has reviewed part of the RPU EIS particularly dealing with the golf course. What is says is that it is for possible use and would have to be looked at more closely when it

comes to an area plan. Now we are talking about a zoning change or an area plan that would actually contemplate a golf course. Whereas, the RPU EIS did not have analysis of the impact on wildlife and the water quality of a golf course. He does not want an answer orally today; he wants a written explanation of how you feel the law is satisfied by the evaluation that was done for this golf course in particular.

Mr. Marshall said some background on how this environmental analysis is tiered. For the Initial Environmental Checklist, pages 3, 4, and 5 have a methodology and assumption section which goes through the tiering concept. In general, for each section it references the specific page numbers of the RPU EIS that had that tiered analysis. For golf courses, we may need to bolster some analysis in the IEC to say that although it is consistent with what was contemplated; if we are making effective this new use thorough the area plan then we need to make certain that we disclose potential impacts and determine whether or not it either requires mitigation.

Mr. Shute said it needs to be answered either by how it's already been evaluated or how you plan to evaluate it. He asked what is the master plan proposed policy with respect to realigning Highway 50. (p. 20).

VII.A Indirect reference does not provide substitute for environmental analysis:

The RPU EIS did not analyze this change. The revised IEC also fails to analyze the environmental impacts of this rezoning, although the SSAP clearly identifies golf course uses as a desired project on the T-RR parcel. Strangely, TRPA's response to the questions about the golf course impacts would suggest that this change was analyzed, somewhere – but the facts don't support this assertion.

“The RPU EIS recognized that the land use designation change from conservation to resort recreation that occurred with the approval of the RPU could include golf course uses on the Edgewood Mountain parcel. See RPU Final EIS at page 3-48. The SSAP IEC further analyzes the golf course use on the parcel at page 36. [JM]”

VII.B “Recognition of possible use” does not provide substitute for environmental analysis:

The Final EIS includes a Master Response related to the changes to Resort Recreation, which begins on page 3-47. Excerpts and comments from the Master Response are below:

...The Recreation designation proposed in Alternative 3 could have expanded permissible uses (subject to Area Plan approval) to potentially include additional recreation uses such as golf courses, downhill ski facilities, and outdoor recreation concessions. The additional uses are generally consistent with existing PAS management policies for the area and the corresponding range of permissible recreation uses...

The only reference to golf courses in this entire section is what is noted above. This was not an analysis of the impacts, but rather a statement that the draft RPU Alternative could have allowed additional uses such as golf courses, but then the EIS proceeds to explain how the final Alternative was revised to include the new Resort Recreation Land Use, and narrow it to two parcels. Although the reader may infer that this means golf courses could be allowed on the Resort Recreation parcels, this is never stated, nor analyzed, in

the EIS. All the public has is this indirect reference to something that “could have happened” but didn’t.

VII.C IEC information regarding T-RR parcel does not provide substitute for environmental analysis:

As noted during public comment on June 26th, the RPU EIS analyzed a language change, not the impacts of the regulatory change.¹⁹ The following excerpts from the Final EIS Master Response indicate additional, more site-specific planning will occur at the Area Plan development stage:

...These properties—the approximately 250-acre Edgewood Mountain area adjacent to the proposed High Density Tourist District (see Exhibit 3-1), and the approximately 65-acre Heavenly California Base area near the proposed Regional Center (see Exhibit 3-2)—would receive the new designation to distinguish them from the existing Recreation designation. These two areas were considered appropriate for additional uses and building subdivision allowances (i.e., to permit creation of condominiums, not new subdivision of land) because they are in close proximity to already developed centers—Edgewood to the most intensely developed casino core at South Shore, and Heavenly California Base area to the heavily developed South Shore (proposed Regional Center) and to an existing, heavily used ski area base facility and parking lot...

...New uses that are not currently allowed in the area could be proposed under an Area Plan that would be subject to conformance review, as described in Chapter 13 of the Final Draft Code. The Area Plan would be subject to separate environmental review, as required by Chapter 3 of the Final Draft Code. While no new uses are currently proposed for the area, it is expected that additional uses would be proposed during development of an Area Plan. Primary uses are listed in Section 21.4 of the Final Draft Code. New development within the area could include residential, tourist accommodation, or commercial uses as long as the development meets the new Resort Recreation designation requirements...

...The Area Plan development and approval process would provide the opportunity for more detailed, site-specific planning, which would be subject to a conformance review and environmental review. The Area Plan review requirements would ensure that, based on the additional site specific planning, all proposals are consistent with the Regional Plan and that potentially significant environmental impacts are identified, assessed, and mitigated. (p. 3-49)

...Area Plans will include more site-specific criteria than the Regional Plan, commensurate with their smaller-scale planning area. To be found in conformance with the Regional Plan, the Area Plan must demonstrate that it will achieve and maintain TRPA Threshold Standards. (p. 3-49 – 50). [Emphasis added].

Although TRPA considers the IEC an acceptable “environmental review,” the IEC does not actually perform any additional review. There are no calculations, no estimates, and no alternatives. The IEC does not compare the proposed SSAP to a “no action” alternative (perhaps with the exception of the “use tables”), to existing conditions on the ground (which are different than existing regulations approved by TRPA last December), nor does the IEC provide an evaluation of possible alternatives.

¹⁹ “Ellie Waller, Tahoe Vista resident said she still has issues with baseline definition and identification. When we changed Heavenly and Edgewood to resort recreation we did a language change. There was no environmental impact analyzed for what conservation land is today which is what she is calling a baseline and starting with resort recreation as a baseline. What was analyzed was a language change, not an impact.” (RPIC minutes, p. 21).

VII.D Impacts of increased recreational capacity:

A review of our comment includes:

80. (Other IEC issues.) The commenter expresses that the SSAP will create additional recreation demand and capacity that needs to be analyzed.

Staff's response states:

“As described in the IEC, development associated with the SSAP could generate additional recreation demand by increasing the concentration of residents and visitors in the area, but existing recreation opportunities are numerous and can meet that potential increase in demand within and in the immediate vicinity of the SSAP, and more than half of the SSAP includes land designated for recreation purposes. It is anticipated that development within the SSAP could expand public recreation opportunities within the SSAP limits. Therefore, any new demand that is created by development within the SSAP would be easily met. The *physical* effects of any proposed recreation facilities would be evaluated in accordance with TRPA Code and other regulations. [AE]”

There is a lack of confidence that the physical impacts to the environment of increased recreation uses will be analyzed. First, as we see for the change to Resort Recreation, impacts from this change have not been adequately analyzed by TRPA, nor by Douglas County, and the revised IEC puts off further analysis to the project-level, yet sets the stage for a developer to ‘tier’ off the RPU EIS in the future. Further, a project-specific review will not account for the cumulative impacts to the Basin and larger South Shore area. Second, TRPA recently approved a change from a horse-drawn sleigh ride to a 20 unit snowmobile track on the T-RR property, without any environmental analysis of the impacts. Yet snowmobiles create significant impacts to air quality, water quality, noise, wildlife, SEZs and soils, etc. Third, as these comments repeatedly note, TRPA has done its best to avoid analyzing the impacts of this change – a change which allows new coverage and uses on land once zoned conservation!

“81. As noted throughout this letter, there are several proposals in the SSAP that do not ‘conform’ to TRPA’s RPU, yet the Conformity Checklist states that they do. For example, TRPA’s RPU would prohibit the development of a new golf course, as a new source of fertilizer use, on the TRR parcel because according to DC’s own maps, this parcel is comprised of sensitive and SEZ lands. Exemptions for fertilizer use only apply to existing approved uses.

The TRPA land capability maps indicate the presence of both high and low capability land within the TRR Edgewood parcel. The exact location of high capability and low capability land would be verified as a part of a project application. The TRPA Code prohibits the development of a golf course on low capability land. [PN] “

VII.E Increased Fertilizer Use will Impact Thresholds:

The addition of a new golf course will result in increased fertilizer use, which will increase the phosphorous and nitrogen entering Lake Tahoe. Staff's response to our concerns about this did not address the RP's prohibition on new sources of major fertilizer use in areas where the fertilizer will *runoff into* sensitive/SEZ areas. Further, allowing more fertilizer use will contribute to the declining nearshore conditions in Lake

Tahoe – yet TRPA just adopted a management standard aimed to help protect nearshore conditions.

The RPU adopted the following standard (included in Resolution 82-11):

Attached Algae

MANAGEMENT STANDARD

Implement policy and management actions to reduce the areal extent and density of periphyton (attached) algae from Lake Tahoe’s nearshore.

It is a well-known fact that nutrients are used to increase the growth of plants in gardens, landscaping, and farms - - and therefore also perform the same function for algae in the Lake, including the nearshore.²⁰ It is also scientific fact that stormwater treatment facilities – including natural infiltration and engineered facilities (e.g. stormwater filters) do not remove nitrogen.²¹ Therefore, introducing more nitrogen into the Basin, which would be a result of the increased fertilizer use needed to support the golf course, will negatively impact Tahoe’s nearshore (and summer-time mid-lake) clarity, both of which continue to degrade. As a result, this change would have a negative impact on two of TRPA’s thresholds. Findings that this change in use would not harm, let alone would help achieve and maintain the thresholds, can not be made.

VIII. Additional Comments:

VIII.A Visual Prominence remains undefined:

The RPU does not include criteria to define “visual prominence.” Considering the parameters in the response²² noted in the RPIC packet still provides no way to assess whether the (undefined) ‘visual prominence’ will be changed by a plan or project. The SSAP carries forward this same failure, thus there remains no definition or criteria that actually provides for an assessment of visual prominence. Given the SSAP includes what would be the largest increase in buildings in the entire Basin (through the expansion of the casino towers), it is even more imperative that this term be defined and clear measures to evaluate it be developed.

²⁰ Interested readers are referred to the 2012 State of the Lake Report, and other publications, by the Tahoe Environmental Research Center (TERC).

²¹ We have provided specific information in previous comments to TRPA and all agencies involved in the TMDL and Lake Clarity Crediting Program regarding this fact.

²² “Because the SSAP is in conformance with the Regional Plan and proposes no modifications or revisions, any projects proposed in the SSAP would be subject to the mitigation measures adopted as part of the RPU EIS, including no increase in visual prominence, described in TRPA Code Section 37.7.17.B: “When considering visual prominence, the following factors will be considered: Building mass, contrast, location, articulation, color, materials and architectural style; and the quality of landscape features and views that are blocked or revealed. [AE]” (RPIC packet, p. 42).



Ozone Trends Summary: Lake Tahoe Air Basin

Year	Days > Standard		1-Hour Observations			8-Hour Averages				Year Coverage			
	State	National	State	Nat'l	State	National	State	National	Year	Min.	Max.		
	1-Hr	8-Hr	1-Hr	'08 8-Hr	Max.	D.V. ¹	D.V. ²	Max.	D.V. ¹	Max.	'08 D.V. ²	Min.	Max.
2009	0	1	0	0	0.077	0.09	*	0.071	0.077	0.070	*	100	100
2008	0	5	0	1	0.091	0.09	*	0.077	0.077	0.077	*	100	100
2007	0	5	0	0	0.090	0.08	*	0.073	0.075	0.073	*	94	94
2006	0	2	0	0	0.086	0.09	*	0.075	0.075	0.075	*	100	100
2005	0	0	0	0	0.073	0.07	*	0.068	0.068	0.067	*	98	98
2004	0	0	0	0	0.066	0.09	*	0.062	0.079	0.061	*	0	6
2003	0	2	0	0	0.086	0.09	0.083	0.071	0.071	0.070	0.066	95	96
2002	0	1	0	1	0.083	0.08	0.083	0.079	0.072	0.079	0.066	98	98
2001	0	2	0	1	0.088	0.08	0.083	0.077	0.074	0.077	0.067	98	98
2000	0	2	0	0	0.083	0.08	0.081	0.072	0.077	0.072	0.069	98	98
1999	1	3	0	1	0.095	0.08	0.081	0.079	0.077	0.079	0.069	99	99
1998	0	7	0	1	0.081	0.08	0.081	0.077	0.077	0.077	0.069	97	97
1997	1	1	0	0	0.095	0.08	0.083	0.071	0.074	0.071	0.068	97	97
1996	0	2	0	0	0.083	0.08	0.083	0.074	0.079	0.073	0.071	98	98
1995	0	5	0	2	0.092	0.08	0.086	0.089	0.077	0.089	0.070	95	95
1994	0	6	0	2	0.086	0.09	0.083	0.079	0.079	0.079	*	94	94
1993	0	2	0	0	0.090	0.09	0.080	0.071	0.071	0.071	*	98	98
1992	1	20	0	5	0.100	0.09	0.090	0.083	0.083	0.082	0.075	0	91
1991	0	10	0	2	0.090	0.09	0.090	0.081	0.085	0.081	0.076	93	93
1990	0	8	0	1	0.090	0.09	0.090	0.080	0.085	0.080	0.075	94	94
1989	2	20	0	11	0.100	0.09	0.090	0.085	0.085	0.085	0.076	7	96
1988	0	20	0	8	0.090	0.09	0.090	0.085	0.083	0.085	0.074	98	99
1987	0	17	0	5	0.090	0.09	0.090	0.083	0.079	0.082	0.071	96	99
1986	0	3	0	2	0.090	0.08	0.080	0.080	0.080	0.080	0.069	86	97
1985	1	3	0	2	0.100	0.08	0.080	0.086	0.079	0.086	0.068	92	96
1984	0	1	0	0	0.080	0.08	0.080	0.073	0.071	0.072	0.067	89	97
1983	0	1	0	0	0.080	0.09	0.080	0.071	0.076	0.071	0.069	88	95
1982	0	1	0	1	0.090	0.10	0.090	0.080	0.083	0.080	*	100	100
1981	2	11	0	2	0.100	0.10	0.100	0.083	0.083	0.082	*	99	99
1980	0	5	0	2	0.090	0.09	*	0.080	0.083	0.080	*	69	89
1979	0	4	0	0	0.080	0.09	0.090	0.075	0.083	0.075	*	95	98
1978	1	11	0	7	0.100	0.10	0.090	0.086	0.088	0.086	*	0	94
1977	1	31	0	20	0.100	0.10	0.090	0.088	0.088	0.087	*	79	92
1976	2	5	0	2	0.100	0.10	0.100	0.095	0.095	0.095	*	56	99
1975	0	6	0	5	0.090	0.09	*	0.083	0.083	0.082	*	32	88



Info: Click on a column header for more information about the statistic in that column.

Years: Annual Ozone statistics are available for this basin from 1975 through 2009.

Notes: All concentrations expressed in parts per million.

The national 1-hour ozone standard was revoked in June 2005 and is no longer in effect. Statistics related to the revoked standard are shown in *italics* or *italics*.

State exceedances shown in **yellow**. National exceedances shown in **orange**.

An exceedance is not necessarily a violation.

¹ D.V. = State Designation Value

² D.V. = National Design Value

* There was insufficient (or no) data available to determine the value.

Go to: [Data Statistics Home Page](#) [Trends Summaries Start Page](#) [PM2.5/PM10 Trends for this Basin](#)

ALMANAC EMISSION PROJECTION DATA (PUBLISHED IN 2009)

2008 Estimated Annual Average Emissions

LAKE TAHOE AIR BASIN

All emissions are represented in Tons per Day and reflect the most current data provided to ARB.

[Download these results \(as a comma delimited file\).](#)

[Download more detailed data \(as a comma delimited file\).](#)

[Start a new query.](#)

STATIONARY SOURCES	TOG	ROG	CO	NOX	SOX	PM	PM10	PM2.5
FUEL COMBUSTION								
ELECTRIC UTILITIES	0.00	0.00	0.01	0.05	0.00	0.00	0.00	0.00
MANUFACTURING AND INDUSTRIAL	0.00	0.00	0.00	0.04	0.00	0.00	0.00	0.00
SERVICE AND COMMERCIAL	0.01	0.00	0.02	0.09	0.00	0.01	0.01	0.01
OTHER (FUEL COMBUSTION)	0.00	0.00	0.01	0.02	-	0.00	0.00	0.00
* TOTAL FUEL COMBUSTION	0.01	0.01	0.04	0.20	0.00	0.01	0.01	0.01
CLEANING AND SURFACE COATINGS								
LAUNDERING	0.00	0.00	-	-	-	-	-	-
DEGREASING	0.12	0.09	-	-	-	-	-	-
COATINGS AND RELATED PROCESS SOLVENTS	0.10	0.09	-	-	-	-	-	-
ADHESIVES AND SEALANTS	0.06	0.05	-	-	-	-	-	-
* TOTAL CLEANING AND SURFACE COATINGS	0.27	0.24	-	-	-	-	-	-
PETROLEUM PRODUCTION AND MARKETING								
PETROLEUM MARKETING	0.23	0.04	-	-	-	-	-	-
* TOTAL PETROLEUM PRODUCTION AND MARKETING	0.23	0.04	-	-	-	-	-	-
INDUSTRIAL PROCESSES								
MINERAL PROCESSES	-	-	0.01	0.00	0.00	0.01	0.00	0.00
* TOTAL INDUSTRIAL PROCESSES	-	-	0.01	0.00	0.00	0.01	0.00	0.00
** TOTAL STATIONARY SOURCES	0.52	0.29	0.05	0.21	0.00	0.02	0.02	0.02
AREAWIDE SOURCES	TOG	ROG	CO	NOX	SOX	PM	PM10	PM2.5
SOLVENT EVAPORATION								
CONSUMER PRODUCTS	0.42	0.36	-	-	-	-	-	-
ARCHITECTURAL COATINGS AND RELATED PROCESS SOLVENTS	0.19	0.18	-	-	-	-	-	-
PESTICIDES/FERTILIZERS	0.02	0.02	-	-	-	-	-	-
ASPHALT PAVING / ROOFING	0.35	0.35	-	-	-	-	-	-
* TOTAL SOLVENT EVAPORATION	0.97	0.91	-	-	-	-	-	-
MISCELLANEOUS PROCESSES								
RESIDENTIAL FUEL COMBUSTION	2.82	1.24	11.82	0.33	0.05	1.95	1.82	1.75
FARMING OPERATIONS	0.91	0.07	-	-	-	0.12	0.06	0.01
CONSTRUCTION AND DEMOLITION	-	-	-	-	-	0.89	0.43	0.04
PAVED ROAD DUST	-	-	-	-	-	2.43	1.11	0.17
UNPAVED ROAD DUST	-	-	-	-	-	2.40	1.42	0.14
FUGITIVE WINDBLOWN DUST	-	-	-	-	-	0.04	0.02	0.00
FIRES	0.00	0.00	0.01	-	-	0.00	0.00	0.00
MANAGED BURNING AND DISPOSAL	0.50	0.23	2.75	0.07	0.01	0.32	0.32	0.30
COOKING	0.01	0.01	-	-	-	0.04	0.03	0.02
* TOTAL MISCELLANEOUS PROCESSES	4.25	1.55	14.58	0.40	0.06	8.19	5.21	2.43
** TOTAL AREAWIDE SOURCES	5.22	2.45	14.58	0.40	0.06	8.19	5.21	2.43
MOBILE SOURCES	TOG	ROG	CO	NOX	SOX	PM	PM10	PM2.5
ON-ROAD MOTOR VEHICLES								
LIGHT DUTY PASSENGER (LDA)	0.33	0.31	2.83	0.18	0.00	0.01	0.01	0.01

LIGHT DUTY TRUCKS - 1 (LDT1)	0.45	0.42	4.65	0.31	0.00	0.02	0.02	0.01
LIGHT DUTY TRUCKS - 2 (LDT2)	0.30	0.27	2.93	0.33	0.00	0.02	0.02	0.01
MEDIUM DUTY TRUCKS (MDV)	0.15	0.14	1.61	0.18	0.00	0.01	0.01	0.01
LIGHT HEAVY DUTY GAS TRUCKS - 1 (LHDV1)	0.04	0.04	0.36	0.04	0.00	0.00	0.00	0.00
LIGHT HEAVY DUTY GAS TRUCKS - 2 (LHDV2)	0.04	0.04	0.33	0.02	-	0.00	0.00	0.00
MEDIUM HEAVY DUTY GAS TRUCKS (MHDV)	0.04	0.03	0.35	0.03	-	-	-	-
HEAVY HEAVY DUTY GAS TRUCKS (HHDV)	0.04	0.03	0.54	0.05	-	0.00	0.00	-
LIGHT HEAVY DUTY DIESEL TRUCKS - 1 (LHDV1)	0.00	0.00	0.01	0.07	-	0.00	0.00	0.00
LIGHT HEAVY DUTY DIESEL TRUCKS - 2 (LHDV2)	0.00	0.00	0.01	0.04	-	0.00	0.00	0.00
MEDIUM HEAVY DUTY DIESEL TRUCKS (MHDV)	0.01	0.00	0.04	0.18	0.00	0.01	0.01	0.01
HEAVY HEAVY DUTY DIESEL TRUCKS (HHDV)	0.04	0.04	0.15	0.54	0.00	0.02	0.02	0.02
MOTORCYCLES (MCY)	0.08	0.07	0.46	0.02	-	0.00	0.00	0.00
HEAVY DUTY DIESEL URBAN BUSES (UB)	0.00	0.00	0.00	0.03	-	0.00	0.00	0.00
HEAVY DUTY GAS URBAN BUSES (UB)	0.00	0.00	0.02	0.00	-	-	-	-
SCHOOL BUSES (SB)	0.00	0.00	0.02	0.02	-	0.00	0.00	0.00
OTHER BUSES (OB)	0.01	0.01	0.12	0.04	-	0.00	0.00	0.00
MOTOR HOMES (MH)	0.01	0.01	0.35	0.02	-	0.00	0.00	0.00
* TOTAL ON-ROAD MOTOR VEHICLES	1.54	1.42	14.79	2.11	0.01	0.09	0.09	0.07
OTHER MOBILE SOURCES								
AIRCRAFT	0.30	0.27	2.72	0.20	0.03	0.09	0.09	0.09
COMMERCIAL HARBOR CRAFT	0.05	0.04	0.18	0.56	0.00	0.02	0.02	0.02
RECREATIONAL BOATS	0.89	0.84	6.50	0.32	0.00	0.05	0.05	0.03
OFF-ROAD RECREATIONAL VEHICLES	0.61	0.57	1.64	0.02	0.00	0.01	0.01	0.01
OFF-ROAD EQUIPMENT	0.52	0.46	3.45	1.36	0.00	0.09	0.08	0.08
FARM EQUIPMENT	-	-	-	-	-	-	-	-
FUEL STORAGE AND HANDLING	0.04	0.04	-	-	-	-	-	-
* TOTAL OTHER MOBILE SOURCES	2.40	2.21	14.49	2.46	0.04	0.26	0.25	0.22
** TOTAL MOBILE SOURCES	3.94	3.64	29.28	4.57	0.05	0.35	0.34	0.29
GRAND TOTAL FOR LAKE TAHOE AIR BASIN	9.67	6.38	43.91	5.18	0.11	8.56	5.57	2.74

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Air Resources Board

State of California

Governor Arnold Schwarzenegger

**October 2005 Revisions* to
Chapters 1-2
Appendix B
Reference Lists: (Chapters 1-11, Appendix B, and Appendix G)
of the March 11, 2005 Staff Report
Review of the
California Ambient Air Quality Standard
for Ozone
October 27, 2005**

California Environmental Protection Agency

Air Resources Board

***Revisions noted in underline (new text) and strikeout (deleted text).**

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California Environmental Protection Agency

Alan C. Lloyd, Ph.D., Secretary
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Chapter 1
Executive Summary

Chapter 2
Overview and Staff Recommendations

1 Executive Summary

The California Health and Safety Code in section 39606, requires the Air Resources Board to adopt ambient air quality standards at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety. Ambient air quality standards are the legal definition of clean air. In December 2000, as a requirement of the Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Stats. 1999, Health and Safety Code 39606 (d)(1)), the Air Resources Board (ARB or Board), approved a report, "Adequacy of California Ambient Air Quality Standards" (ARB and OEHHA, 2000) that contained a brief review of all of the existing health-based California ambient air quality standards.

Following this review, the standard for ozone, currently set at 0.09 parts per million (ppm) for one hour, was prioritized to undergo full review after review of the standards for particulate matter and sulfates. Staff from ARB and the Office of Environmental Health Hazard Assessment (OEHHA) have reviewed the scientific literature on public exposure, atmospheric chemistry, health effects of exposure to ozone, and welfare effects. This Staff Report or Initial Statement of Reasons (Staff Report) presents the findings of the review and the staff recommendations to revise the ozone standard in order to adequately protect public health. The proposed amendments to the ambient air quality standard for ozone are based on the health effects review contained in Volume III of this Report and the recommendation of OEHHA, as required by Health and Safety Code section 39606(a)(2).

1.1 Summary of the Staff Report/Initial Statement of Reasons

1.1.1 Health Effects of Ozone

Controlled human exposure studies demonstrate that ~~Scientific studies show that exposure to ozone~~ exposure can result in reduced lung function, increased respiratory symptoms, increased airway hyperreactivity, and increased airway inflammation. Epidemiologic studies indicate that exposure to ozone is also associated with premature death, hospitalization for cardiopulmonary causes, emergency room visits for asthma, and restrictions in activity.

In controlled human exposure studies (see Chapter 9), exercising individuals exposed for 1 hour (hr) to an ozone concentration as low as 0.12 parts per million (ppm) or for 6.6 hours to a concentration as low as 0.08 ppm experienced lung function decrements and symptoms of respiratory irritation such as cough, wheeze, and pain upon deep inhalation. The lowest ozone concentrations at which airway hyperreactivity (an increase in the tendency of the airways to constrict in reaction to exposure to irritants) has been reported are 0.18 ppm ozone following 2-hour exposure in exercising subjects, 0.40 ppm following 2-hour exposure in resting subjects, and 0.08 ppm ozone in subjects exercising for 6.6 hr. Airway inflammation has been reported following 2-hour exposures to 0.20 ppm ozone and following 6.6-hour exposure to 0.08 ppm ozone.

Additional support for the exposure/response relationship for ozone health effects is derived from animal toxicological studies, which have shown that chronic ozone exposure can induce morphological (tissue) changes throughout the respiratory tract, particularly at the junction of the conducting airways and the gas exchange zone in the deep lung. In addition, the magnitude of ozone-induced effects is related to the inhaled dose (ozone concentration times breathing rate times exposure duration). Of these three factors ozone concentration is the most significant in predicting the magnitude of observed effects, followed by ventilation rate. Exposure duration has the least influence of the three factors.

Epidemiological studies (see Chapter 10) have shown positive associations between ozone levels and several health effects, including decreased lung function, respiratory symptoms, hospitalizations for cardiopulmonary causes, emergency room visits for asthma, and premature death. Children may be more affected by ozone than the general population due to effects on the developing lung and to relatively higher exposure than adults. There is little information available on the effects of ozone exposure on infants. Also, asthmatics may represent a sensitive sub-population for ozone. Since most California residents are exposed to levels at or above the current State ozone standard during some parts of the year, the statewide potential for significant health impacts associated with ozone exposure is large and wide-ranging.

1.1.2 Summary of Non-health Issues

The Staff Report contains reviews and discussions of non-health topics to provide a context for the health review and the staff recommendations for the State ozone standard. Almost all of the ozone in California's atmosphere results from reactions between substances emitted from sources including motor vehicles and other mobile sources, power plants, industrial plants, and consumer products. These reactions involve volatile organic compounds (VOC) and oxides of nitrogen (NO_x) in the presence of sunlight (Chapter 3). Ozone is a regional pollutant, as the reactions forming it take place over time, and downwind from the sources of the emissions. As a photochemical pollutant, ozone is formed only during daylight hours under appropriate conditions, but is destroyed throughout the day and night. Thus, ozone concentrations vary depending upon both the time of day and the location. Even in pristine areas there is some ambient ozone that forms from natural emissions that are not controllable (Chapter 4). This is termed "background" ozone. The average "background" ozone concentrations near sea level are in the range of 0.015 to 0.035 ppm, with a maximum of about 0.04 ppm.

The Staff Report includes an overview of statewide ozone precursor emissions that are involved in the formation of ozone (Chapter 5). The Staff Report also includes a discussion of the current ultraviolet photometry monitoring method, and a listing of approved samplers (Chapter 6). Although there are two measurement methods for ozone approved for use in the U.S. by the U.S. Environmental Protection Agency (USEPA), the method based on ultraviolet photometry is almost universally used in practice and is approved for use in California for state air quality standards.

The Staff Report includes a summary of current air quality in California, as well as long-term trends in statewide ozone concentrations (Chapter 7). Ozone is monitored continuously at approximately 175 sites in California. The highest number of exceedance days for both the State and federal 1-hour standards occurred in the San Joaquin Valley Air Basin and the South Coast Air Basin. Both areas had more than 115 State standard exceedance days and 31 or more federal standard exceedance days during each of the three years from 2001 through 2003. The Sacramento Metro Area, Mojave Desert Air Basin, and Salton Sea Air Basin all averaged more than 50 State standard exceedance days and averaged 6 or more federal standard exceedance days during 2001 through 2003. The remaining five areas (Mountain Counties Air Basin, San Diego Air Basin, San Francisco Bay Area Air Basin, South Central Coast Air Basin, and the Upper Sacramento Valley) averaged from 12 to 45 State standard exceedance days. The Upper Sacramento Valley area had no exceedances of the federal standard while the Mountain Counties Air Basin, San Diego Air Basin, San Francisco Bay Area Air Basin, and South Central Coast Air Basin each averaged 1 to 2 federal standard exceedance days for the three-year period.

The range of the measured maximum 1-hour concentrations tends to follow a similar pattern. The South Coast Air Basin showed the highest values, with measured concentrations of 0.169 ppm or higher during 2001 through 2003. The next highest 1-hour ozone concentrations occurred in the Salton Sea Air Basin and San Joaquin Valley Air Basin, which had concentrations of 0.149 ppm or higher during all three years. During 2001 through 2003, neither the State nor federal 1-hour standard was exceeded in the Lake County Air Basin, North Coast Air Basin, or Northeast Plateau Air Basin. Data for four additional areas, Great Basin Valleys Air Basin, Lake Tahoe Air Basin, North Central Coast Air Basin, and the Upper Sacramento Valley show exceedances of the State standard, but not the federal 1-hour standard (as described earlier, representative data for the Northeast Plateau Air Basin and Great Basin Valleys Air Basin are available for 2002 and 2003 only). Both the State and federal 1-hour standards were exceeded during at least two of the three years in all other areas.

Californians' indoor and personal exposures to ozone are largely determined by the outdoor ozone concentrations in their community. Nonetheless, some Californians experience a substantial exposure to ozone indoors, due to the increasing use of certain types of appliances and equipment that emit ozone. Children and those who are employed in outdoor occupations or exercise heavily outdoors, experience substantially greater exposures to ozone than the rest of the population, because they spend time outdoors during peak ozone periods.

A review of welfare effects, including effects of ozone on forest trees, agricultural crops, and materials is also discussed in this report (Chapter 8). Elevated concentrations of ozone can cause adverse effects on agricultural crops, forest trees and materials at current ambient levels, and the proposed health-based ozone standards should also provide protection to crops, forests and materials. In broad terms, impacts to crops are generally more severe than for forest trees owing to their inherently more vigorous rates of growth. Discussed in the

subsection on crops and the methods used to expose plants to ozone. This is followed by an examination of the physiological basis of ozone damage to plants, with special emphasis on carbon metabolism and the resulting impacts on crop growth and yield. Data collected since the 1950s on mixed conifer forests in the San Bernardino Mountains and the Sierra Nevada indicate that increasing numbers of ponderosa and Jeffrey pines exhibit ozone-specific needle damage due to the pollutant's cumulative effects. Also discussed are the impacts of ozone on materials, including building materials, rubber, paint, and fabrics. Although the proposed ozone standards are based on human health effects, progress toward attaining the proposed standards will provide welfare benefits.

1.2 Staff Recommendations for the Ozone Standard

California ambient air quality standards are defined in the Health and Safety Code section 39014, and 17 Cal. Code Regs. section 70101, and comprise four elements: (1) a definition of the air pollutant, (2) an averaging time, (3) a pollutant concentration, and (4) a monitoring method to determine attainment of the standard. The current California ambient air quality standard for ozone is 0.09 ppm averaged over one hour and was set by the Board in 1988. The data indicate that the current standard alone is not sufficiently protective of human health. Based on the review of the scientific literature and recommendations by OEHHA, the staff recommends that the following revisions be made to the California ambient air quality standard for ozone:

1. Ozone will continue to be the pollutant addressed by the standard.
2. Ozone 1-hour-average Standard – retain the current 1-hour-average standard for ozone at **0.09 ppm, not to be exceeded.**
3. Ozone 8-hour-average Standard – establish a new 8-hour-average standard for ozone at **0.070 ppm, not to be exceeded.**
4. Ozone Monitoring Method: retain the current monitoring method for ozone which uses the ultraviolet (UV) photometry method for determining compliance with the State ambient air quality standard for ozone. Incorporate by reference (17 Cal. Code Regs. section 70101) all federally approved UV methods (i.e., samplers) for ozone as "California Approved Samplers". This will result in no change in air monitoring equipment practices, but will align state monitoring requirements with federal requirements.

These recommendations are based on the following findings:

- a. Reduced lung function and increased respiratory or ventilatory symptoms following 1-hour exposure to 0.12 ppm ozone with moderate to heavy exercise.
- b. Increased airway hyperreactivity following 2-hour exposure to 0.18 ppm in exercising subjects.
- c. Airway inflammation following 2-hour exposure to 0.20 ppm ozone in exercising subjects

- d. Reduced lung function, increased respiratory and ventilatory symptoms, increased airway hyperreactivity, and increased airway inflammation following 6.6 to 8-hour exposure to 0.08 ppm ozone.
- e. Evidence from epidemiological studies of several health endpoints at current ambient concentrations of ozone including premature death, hospitalization, respiratory symptoms, and restrictions in activity and lung function.
- f. Evidence from epidemiological studies of emergency room visits for asthma suggesting a possible threshold concentration between 0.075 and 0.11 ppm from analyses based on a 1-hour averaging time, and a possible threshold concentration between 0.070 and 0.10 ppm from analyses based on an 8-hour averaging time.
- g. There is no evidence that children and infants respond to lower ozone concentrations than adults. Their risk is primarily related to their greater ventilation rate and greater exposure duration.
- h. The dose-rate of ozone inhalation influences the magnitude of observed effects.

The staff recommendations for revision of the California ambient air quality standard for ozone are primarily based on controlled human exposure studies. Epidemiologic data contributed to development of the margin of safety.

1.3 Other Recommendations

In light of the adverse health effects observed at current ambient concentrations and the lack of a demonstrated effect threshold for the population as a whole, staff makes the following comments:

1. Fund additional research investigating the responses of human subjects to multi-hour exposures to ozone concentrations between 0.04 and 0.08 ppm.
2. The standards should be revisited within five years, in order to re-evaluate the evidence regarding the health effects associated with ozone exposure.
3. In any air basin in California that currently attains the ambient air quality standards for ozone, air quality should not be degraded from present levels.

1.4 Estimated Health Benefits Impacts of Ozone Exposure

~~Staff estimates that attainment of the proposed ozone standards throughout California would avoid a significant number of adverse health effects each year, specifically:~~

Exposure to ozone at current ambient levels has substantial health impacts, including, but not limited to, death, hospitalization, emergency room visits, and symptoms of respiratory irritation. Staff estimates that the annual health impact

of exposure to ozone at current levels, compared to attainment of the proposed State 8-hour and 1-hour ozone standards throughout California includes:

- 630 (310 – 950 probable range) ~~580 (290 – 870, probable range)~~ premature deaths for all ages.
- 4,200 (2,400 - 5,800, 95% confidence interval (CI)) ~~3,800 (2,200 – 5,400, 95% confidence interval (CI))~~ hospitalizations due to respiratory diseases for all ages.
- 660 (400 – 920, 95% CI) ~~600 (360 – 850, 95% CI)~~ emergency room visits for asthma for children under 18 years of age.
- 4.7 million (1,200,000 – 8,600,000, 95% CI) ~~3.3 million (430,000 – 6,100,000, 95% CI)~~ school absences for children 5 to 17 years of age.
- 3.1 million (1.3 million – 5.0 million, 95% CI) ~~2.8 million (1.2 million – 4.6 million, 95% CI)~~ minor restricted activity days for adults above 18 years of age.

These health impact estimates are based on the results of epidemiologic studies on the health effects of ozone exposure and an exposure reduction methodology modified from analyses conducted by the U.S. EPA and other investigators (see Appendix B). The health impacts estimates were made for only a small number of the known health effects of ozone exposure and, consequently, underestimate the total public health impact. The health impacts assessment was not used to select the appropriate levels of the proposed ozone standards.

For comparison purposes, we also estimated the health impacts o current ozone levels compared to attainment of the federal 8-hour ozone standard of 0.08 ppm, and the health impacts of current ozone levels compared to attainment of the State 1-hour ozone standard alone. Specifically, we estimate that 360 (180 – 550, probable range) premature deaths annually are related to current ozone levels, compared to statewide attainment of the federal 8-hour standard, and about 540 (270 – 810, probable range) premature deaths annually are related to current ozone levels, compared to statewide attainment of the State 1-hour standard. Results for other health endpoints are in Appendix B.

The differences between the results are the “incremental” impacts of not attaining the State 1-hour and 8-hour standards, compared to the federal 8-hour ozone standard. However, it is more reasonable to consider attainment of the two State standards together, compared to current ozone levels, since it is unlikely that control strategies will be geared to fist attain one standard and then the other. Nonetheless, the current impact of not attaining the federal 8-hour standard is about 360 premature deaths annually, with an additional 270 deaths associated with not attaining the proposed State 8-hour standard, making the total estimated impact of not attaining both standards 630 deaths. Similarly, the current impact of not attaining the State 1-hour standard is about 540 premature deaths annually, with an additional 90 deaths associated with not attaining the proposed 8-hour standard, making the total estimated impact of not attaining both the State 1-hour and 8-hour ozone standards 630 premature deaths. More detailed discussion of this analysis is available in Appendix B.

As discussed in Appendix B, there are a several important assumptions and uncertainties in this analysis. Some have to do with study design, statistical methods, and choice of epidemiological studies used to develop the concentration-response (CR) functions used in the analysis. Few studies have investigated the shape of the CR function, or whether there is a population response threshold for health endpoints other than emergency room visits for asthma. Further uncertainty is added by assumptions in the statewide exposure assessment. It should also be noted that since several health effects related to acute exposure, and effects of chronic ozone exposure, are not included in the estimates, the health benefits associated with lowering ozone exposure are likely underestimated.

1.5 Public and Peer Review of the Staff Recommendations

The draft version of this Staff Report was released to the public on June 21, 2004 and presented for review and comment at public workshops during 2004 on July 14 in Sacramento, July 15 in El Monte, July 16 in Fresno, and August 25 in Sacramento.

The draft Staff Report was peer reviewed by the Air Quality Advisory Committee (AQAC). AQAC is a scientific peer review committee, appointed by the University of California, to independently evaluate the scientific basis of staff findings and recommendations in the draft Staff Report for revising the California ambient air quality standard for ozone. The AQAC held a public meeting to discuss its review of the draft Staff Report, comments submitted by the public, and staff responses to those comments. AQAC concluded that the report was well written and researched, and that the proposed revision to the State ozone standard was adequately supported. AQAC findings, public comments, and staff responses can be found in Appendices C-E. Following the meeting of the Air Quality Advisory Committee (AQAC), staff revised the draft Staff Report based on comments received from AQAC and the public.

1.6 Environmental and Economic Impacts

The proposed ambient air quality standards will in and of themselves have no environmental or economic impacts. Standards simply define clean air. Once adopted, local air pollution control or air quality management districts are responsible for the adoption of rules and regulations to control emissions from stationary sources to assure their achievement and maintenance. The ARB is responsible for adoption of emission standards for mobile sources and consumer products. A number of different implementation measures are possible, and each could have its own environmental or economic impact. These impacts must be evaluated when the control measure is proposed. Any environmental or economic impacts associated with the imposition of future measures will be considered if and when specific measures are proposed.

1.7 Environmental Justice Considerations

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption,

implementation, and enforcement of environmental laws, regulations, and policies. The available literature suggests there appears to be no special vulnerability related to race, ethnicity or income level, although there may be higher exposure. Ambient air quality standards define clean air; therefore, all of California's communities will benefit from the proposed health-based standards.

1.8 Comment Period and Board Hearing

Release of this Staff Report opens the official 45-day public comment period required by the Administrative Procedure Act prior to the public meeting of the Air Resources Board to consider the staff's recommendations. Please direct all comments to either the following postal or electronic mail address:

Clerk of the Board
Air Resources Board
1001 "I" Street, 23rd Floor
Sacramento, California 95814
ozone05@listserve.arb.ca.gov

To be considered by the Board, written submissions not physically submitted at the hearing must be received at the ARB no later than 12:00 noon, April 27, 2005. Public workshops will be scheduled for April 2005 to present the final staff recommendations and receive public input on the Staff Report. Information on these workshops, as well as summaries of the presentations from past workshops and meetings are available by calling 1-916-445-0753 or at the following ARB website:

<http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm>.

An oral report summarizing the staff recommendations for revising the ozone standard will be presented to the Board at a public hearing scheduled for April 28, 2005.

The staff recommends that the Board adopt the proposed amendments to the ambient air quality standards for ozone as stated above. The proposed amendments and their basis are described in detail in this Staff Report, which contains the findings of ARB and OEHHA staff's full review of the public health, scientific literature, and exposure pattern data for ozone in California. Due to the extensive nature of the literature review and the hundreds of studies reviewed, the Staff Report is divided into four volumes. Volume I contains the Executive Summary, Overview and Staff Recommendations, and Appendix A, the proposed amendments to the California Code of Regulations (amended regulatory text). Volumes II through IV present more detailed discussions of the material that is summarized in Volume I. Volume II includes background material on non-health topics, including chemistry of ozone formation and deposition, ozone precursor sources and emissions, ozone exposure and background levels, measurement methods, and welfare effects of ozone exposure. Volume III contains a summary of ozone health effects and an in-depth discussion of the basis for the staff recommendation. Volume IV includes several appendices, including an analysis of the estimated health benefits associated with attainment of the proposed

standards, summaries of Air Quality Advisory Committee and public comments and staff responses, and supplemental animal toxicologic data.

1.9 References

Air Resources Board and Office of Environmental Health Hazard Assessment (2000). Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. Sacramento, CA. Available at <http://www.arb.ca.gov/ch/programs/sb25/airstandards.htm>.

2 Overview and Staff Recommendations

Ozone (O₃) can damage human cells upon contact, and has been implicated in a variety of adverse health effects. Scientific studies show that exposure to ozone can result in reduced lung function, increased respiratory symptoms, increased airway hyperreactivity, and airway inflammation. Exposure to ozone is also associated with premature death, hospitalization for cardiopulmonary causes, emergency room visits for asthma, and restrictions in activity. Ozone forms in the atmosphere as the result of reactions involving sunlight and two classes of directly emitted precursors. One class of precursors includes nitric oxide (NO) and nitrogen dioxide (NO₂), collectively referred to as nitrogen oxides or NO_x. The other class of precursors includes volatile organic compounds (VOCs, also called reactive organic gases or ROG), such as hydrocarbons. Ozone forms in greater quantities on hot, sunny, calm days. In metropolitan areas of California and areas downwind, ozone concentrations frequently exceed existing health-protective standards in the summertime. The current California ambient air quality standard for ozone is 0.09 ppm for one hour.

The sources of ozone precursor emissions within California have been grouped into three major categories: point sources, which are distinct facilities such as power plants and factories; mobile sources, which includes cars, trucks, and off-road mobile equipment; and area-wide sources, which include agricultural and construction activities, and consumer products. VOCs are emitted from vehicles, factories, fossil fuels combustion, evaporation of paints, and many other sources. NO_x is emitted from high-temperature combustion processes, such as at power plants or in motor vehicle exhaust .

The concentrations of ozone measured in the air vary both regionally and seasonally throughout California. For example, the Los Angeles area and the San Joaquin Valley experience highest ozone levels in the state. Ozone concentrations are typically higher during the summer months than the winter months.

To help understand which sources contribute to high ozone levels, the ARB has developed and maintains detailed facility and source specific estimates of the overall estimated ozone precursor emissions. Only the precursor gases are estimated. As a complement to emission inventory and routinely collected air quality monitoring data, the ARB conducts atmospheric modeling, using these precursor emission inventories and other appropriate information, to estimate ozone levels

2.1 Setting California Ambient Air Quality Standards

Ambient air quality standards (AAQS) represent the legal definition of clean air. They specify concentrations and durations of exposure to air pollutants that reflect the relationships between the intensities and composition of air pollution and undesirable effects (Health and Safety Code section 39014). The objective of an AAQS is to provide a basis for preventing or abating adverse health or welfare effects of air pollution (17 Cal. Code Regs. section 70101).

Health and Safety Code section 39606(a)(2) authorizes the Air Resources Board (Board) to adopt standards for ambient air quality "in consideration of public health,

safety, and welfare, including, but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy." Standards represent the highest pollutant concentration for a given averaging time that is estimated to be without adverse effects for most people. Standards are set to ensure that sensitive population sub-groups are protected from exposure to levels of pollutants that may cause adverse health effects. A margin of safety is added to account for possible deficiencies in the data and measuring methodology. Health-based standards are based on the recommendation of the Office of Environmental Health Hazard Health Assessment (OEHHA).

Recent legislation requires that infants and children be given special consideration when ambient air quality standards are adopted. As part of its recommendation to the ARB, the statute requires OEHHA to use current principles, practices, and methods used by public health professionals to assess the following considerations for infants and children:

1. Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
2. Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
3. The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.
4. The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

The law also requires that the scientific basis or the scientific portion of the method used to assess these considerations be peer reviewed (Health and Safety Code section 39606(c)). The draft Staff recommendations and their bases, including OEHHA's assessment and recommendation, is peer reviewed by the Air Quality Advisory Committee (AQAC). AQAC is an external peer review committee established in accordance with section 57004 of the Health and Safety Code and appointed by the President of the University of California a University of California. The AQAC meets to independently evaluate the scientific basis of draft recommendations for revising the California ambient air quality standards.

Ambient air quality standards should not be interpreted as permitting, encouraging, or condoning degradation of present air quality that is superior to that stipulated in the standards. Rather, they represent the minimum acceptable air quality. An AAQS adopted by the Board is implemented, achieved, and maintained by numerous rules and regulations that limit pollution from specific sources of ozone precursors. These rules and regulations are primarily, though not exclusively, emission limitations established by the regional and local air pollution control and air quality management districts for stationary sources, and by the Board for vehicular sources and consumer products (see generally, Health and Safety Code sections 39002, 40000, and 40001).

2.2 Current California Ambient Air Quality Standard for Ozone

The current California ambient air quality standard for ozone, established in 1988, is 0.09 ppm (180 $\mu\text{g}/\text{m}^3$) for a one-hour average. This value is not to be exceeded. This standard was established based on the following most relevant effects, which are listed in the table of standards (17 Cal. Code Regs. section 70200):

a. Short-term exposures:

- (1) Pulmonary function decrements and localized lung edema in humans and animals.
- (2) Risk to public health implied by alterations in pulmonary morphology and host defence in animals.

b. Long-term exposures: Risk to public health implied by altered pulmonary morphology in animals after long-term exposures and pulmonary function decrements in chronically exposed humans.

c. Welfare effects:

- (1) Yield loss in important crops and predicted economic loss to growers and consumers.
- (2) Injury and damage to native plants and potential changes in species diversity and number.
- (3) Damage to rubber and elastomers and to paints, fabric, dyes, pigments, and plastics.

The US EPA has set national ambient air quality standards, as noted in the table below. The federal one-hour standard will be phased out beginning in June 2005. The Federal Clean Air Act gives California authority to set its own ambient air quality standards in consideration of statewide concerns. California has the largest number of exceedances of the Federal 8-hour ozone standard in the United States, supporting California's need to address a significant statewide public health issue.

Current Ambient Air Quality Standards for Ozone

Averaging Time	California Standard	Federal Standard
1 Hour	0.09 ppm (180 $\mu\text{g}/\text{m}^3$)	0.12 ppm (235 $\mu\text{g}/\text{m}^3$)
8 Hour	—	0.08 ppm (157 $\mu\text{g}/\text{m}^3$)

2.3 History of Ozone/Oxidant Standards

The first state oxidant standard was set in December 1959 by the state Department of Public Health (DPH), which had the responsibility for setting air pollution standards before the creation of the ARB. This standard was set at 0.15 ppm, averaged for one hour. The standard was for oxidant, rather than ozone, because the monitoring method available at that time, the potassium iodide (KI) method, measured all ambient oxidant

gases, including ozone and other oxidants such as peroxyacetyl nitrate (PAN) nitrogen dioxide, photochemical aerosols, and other unknown oxidants.

In 1969, the newly-created ARB reviewed the oxidant standard set by DPH and revised the standard to a concentration of 0.10 ppm, averaged over one hour, not to be equaled or exceeded. The information considered by the Board in 1969 included adverse effects upon: (1) the health of humans and animals; (2) vegetation; (3) materials; and (4) visibility. Eye irritation was listed as the most relevant effect of oxidant.

In 1974, the Board introduced ultraviolet photometry as the monitoring method for the standard. However, since ultraviolet photometry measures only ozone, the Board changed the designation of the standard from “oxidant” to “oxidant (as ozone).” Because only ozone was to be measured, the Board changed the most relevant effect from: “eye irritation” (which is caused primarily by peroxyacyl nitrates or PANs) to “aggravation of respiratory disease” (which is caused primarily by ozone).

In 1988, the Board changed the designation of the standard from “oxidant (as ozone)” to “ozone”, and revised the standard to a concentration of 0.09 ppm, averaged over one hour, to reflect that the listed relevant effects were related to ozone exposure, rather than to oxidants in general.

For comparison, in 2000, the World Health Organization established a guideline value for ozone in ambient air of 120 $\mu\text{g}/\text{m}^3$ (0.061 ppm) for a maximum period of 8 hours per day (WHO 2000).

2.4 Review of the California Ambient Air Quality Standards

The Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Stats. 1999, ch. 731) required the ARB, in consultation with the OEHHA, to evaluate all health-based standards by December 31, 2000, to determine whether the standards were adequately protective of the health of the public, including infants and children (Health and Safety Code section 39606 (d)). At its December 7, 2000 meeting, the Board approved a report, “Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act” (ARB, et al., 2000), prepared by ARB and OEHHA staffs. The Adequacy Report concluded that health effects may occur in infants and children and other potentially susceptible subgroups exposed to ozone at or near levels corresponding to the current standard. The report identified the standard for ozone as having the second highest priority for further detailed review and possible revision. The standard for PM10 (including sulfates) had the highest priority and was reviewed and revised in 2002, including establishment of a new standard for PM2.5.

2.5 Findings of the Standard Review

2.5.1 Chemistry and Physics

Most of the ozone in California's air results from reactions between substances emitted from sources including motor vehicles, power plants, industrial plants, consumer products, and vegetation. These reactions involve volatile organic compounds (VOCs, which the ARB also refers to as reactive organic gases or ROG) and oxides of nitrogen (NO_x) in the presence of sunlight. Ozone is a regional pollutant, as the reactions forming it take place over time, and downwind from the precursor sources. As a

photochemical pollutant, ozone is formed only during daylight hours under appropriate conditions, but is destroyed throughout the day and night. Thus, ozone concentrations vary depending upon both the time of day and the location. Ozone concentrations are higher on hot, sunny, calm days. In metropolitan and downwind areas of California, ozone concentrations frequently exceed regulatory standards during the summer.

2.5.2 Ozone Background

Even in pristine areas there is some ambient ozone that forms from natural emissions that are not controllable. This is termed “background” ozone. Overall, it appears that “background” ozone in California is dominated by natural tropospheric and stratospheric processes. The effects of occasional very large biomass fires and anthropogenic emissions are secondary factors. The foregoing discussion indicates that average “natural background” ozone near sea level is in the range of 0.015 to 0.035 ppm, with a maximum of about 0.04 ppm. Exogenous enhancements to “natural” levels generally are small (about 0.005 ppm), and are unlikely to alter peak concentrations.

At altitudes above 2 km stratospheric intrusions can push peak ambient concentrations to 0.045 to 0.050 ppm. The timing, spatial extent, and chemical characteristics of stratospheric air mass intrusions makes these events recognizable in air quality records, providing that the affected region has a fairly extensive monitoring network and that multiple air quality parameters (CO, VOC, PM, RH) are being measured as well.

Intermittent episodes of “natural” ozone from very large biomass fires in boreal forests (Alaska, Canada, Siberia) can produce short-lived pulses of ozone up to 0.020 ppm that may arrive during the North American ozone season. Present understanding suggests that these are infrequent events at latitudes below about 50N. There are no data documenting such an event in California. Long range transport of anthropogenic ozone may grow as Asian energy consumption increases the continent’s NO_x emissions. Model studies indicate that the Asian ozone increment in North America could double over the next few decades. Assuming the temporal pattern of transport remains unchanged, such an impact could increase mean ozone concentrations by 0.002 to 0.006 ppm. The potential effect on peak transport events is unknown at this time.

2.5.3 Ozone Precursor Emissions

Ozone is an oxidant gas that forms photochemically in the atmosphere when nitrogen oxides (NO_x) and reactive organic gases (ROG) are present under appropriate atmospheric conditions (see Chapter 5). Carbon monoxide (CO) is also an ozone precursor. Both ROG and NO_x are emitted from mobile sources, point sources, and area-wide sources. ROG emissions from anthropogenic sources result primarily from incomplete fuel combustion, and from the evaporation of solvents and fuels, while NO_x and CO emissions result almost entirely from combustion processes.

2.5.4 Monitoring Method

Two measurement methods for ozone are approved for use in the U.S. by the USEPA: one is based on the chemiluminescence that occurs when ozone and ethylene react, and the other on the attenuation of ultraviolet (UV) radiation by ozone. The method based on UV spectrometry is almost universally used in practice. Specifications and criteria for both methods exist in federal regulation. The UV photometry-based method

is approved for use in California for state air quality standards. Both state and federal requirements are applied directly by the ARB and the air districts in the ozone monitoring network in California.

2.5.5 Exposure

During 2001 through 2003, neither the State nor federal 1-hour standard was exceeded in the Lake County Air Basin, North Coast Air Basin, or Northeast Plateau Air Basin. Data for four additional areas, Great Basin Valleys Air Basin, Lake Tahoe Air Basin, North Central Coast Air Basin, and the Upper Sacramento Valley show exceedances of the State standard, but not the federal 1-hour standard (as described earlier, representative data for the Northeast Plateau Air Basin and Great Basin Valleys Air Basin are available for 2002 and 2003 only). Both the State and federal 1-hour standards were exceeded during at least two of the three years in all other areas.

The highest 8-hour average values were found in the South Coast Air Basin and San Joaquin Valley Air Basin. Maximum 8-hour concentrations in the South Coast Air Basin ranged from 0.144 ppm to 0.153 ppm during 2001 through 2003, while maximum 8-hour concentrations in the San Joaquin Valley ranged from 0.120 ppm to 0.132 ppm during the same three-year period. Three other areas, the Mojave Desert Air Basin, the Sacramento Metro Area, and the Salton Sea Air Basin also had a maximum 8-hour concentration above 0.120 ppm during at least one of the three years.

With respect to the federal 8-hour ozone standard, Lake County Air Basin and North Coast Air Basin showed no exceedance days during 2001 through 2003. One area, the Lake Tahoe Air Basin, averaged only one exceedance day for the three-year period, while the North Central Coast Air Basin averaged three 8-hour exceedance days. In contrast, the San Joaquin Valley Air Basin showed the highest average number of exceedance days (123), followed by the South Coast Air Basin (99). The Sacramento Metro Area, Mojave Desert Air Basin, Mountain Counties Air Basin, and Salton Sea Air Basin each averaged between 42 and 68 exceedance days during 2001 through 2003. The remaining four areas averaged between 7 and 25 federal 8-hour exceedance days during the three-year period.

Californians' indoor and personal exposures to ozone are largely determined by the outdoor ozone concentrations in their community. Nonetheless, some Californians experience a substantial exposure to ozone indoors, due to the increasing use of certain types of appliances and equipment that emit ozone. Others, such as many children and those who are employed in outdoor occupations, may experience substantially greater exposures to ozone than the rest of the population, because they spend time outdoors during peak ozone periods.

2.5.6 Welfare Effects

A review of welfare effects, including effects of ozone on forest trees, agricultural crops, and materials is also discussed in this report (Chapter 8). Elevated concentrations of ozone can cause adverse effects on agricultural crops, forest trees and materials at current ambient levels, and the proposed health-based ozone standards should also provide protection to crops, forests and materials. In broad terms, impacts to crops are generally more severe than for forest trees owing to their inherently more vigorous rates

of growth. Discussed in the subsection on crops and the methods used to expose plants to ozone. This is followed by an examination of the physiological basis of ozone damage to plants, with special emphasis on carbon metabolism and the resulting impacts on crop growth and yield. Data collected since the 1950s on mixed conifer forests in the San Bernardino Mountains and the Sierra Nevada indicate that increasing numbers of ponderosa and Jeffrey pines exhibit ozone-specific needle damage due to the pollutant's cumulative effects. Also discussed are the impacts of ozone on materials, including building materials, rubber, paint, and fabrics. Although the proposed ozone standards are based on human health effects, progress toward attaining the proposed standards will provide welfare benefits.

2.5.7 Health Effects

Review of the controlled human exposure, animal toxicology and epidemiologic literature led to the following conclusions as to the health effects of ozone exposure:

1. The lowest ozone concentration at which reduced lung function and increased respiratory and ventilatory symptoms have been observed following 1-hour exposure is 0.12 ppm with moderate to heavy exercise.
2. The lowest ozone concentration at which increased airway hyperreactivity following 2-hour exposure has been reported is 0.18 ppm in exercising subjects.
3. The lowest ozone concentration at which airway inflammation following 2-hour exposure has been reported is 0.20 ppm ozone in exercising subjects
4. Reduced lung function, increased respiratory and ventilatory symptoms, increased airway hyperreactivity, and increased airway inflammation have been reported following 6.6- to 8-hour exposure to 0.08 ppm ozone.
5. Evidence from epidemiological studies of several health endpoints including premature death, hospitalization, respiratory symptoms, and restrictions in activity and lung function.
6. Evidence from epidemiological studies of emergency room visits for asthma suggests a possible threshold concentration between 0.075 and 0.11 ppm from analyses based on a 1-hour averaging time, and a possible threshold concentration between 0.070 and 0.10 ppm from analyses based on an 8-hour averaging time.
7. There is no evidence that children and infants respond to lower ozone concentrations than adults. Their risk is primarily related to their greater ventilation rate and greater exposure duration.
8. The dose-rate of ozone inhalation influences the magnitude of observed effects.

2.6 Summary of Recommendations

Following a detailed review of the scientific literature on the health and welfare effects of ozone, staff is proposing to revise the ambient air quality standard for ozone. The recommended ozone standards are based on scientific information about the health impacts associated with ozone exposure, recognizing the uncertainties in these data. The definition of California ambient air quality standards assumes a threshold below which effects do not occur. However, the extremely wide range of individual

responsiveness to ozone makes identification of a threshold on a population level somewhat problematic. In addition, the Children's Environmental Health Protection Act [Senate Bill 25, Escutia; Stats. 1999, Ch. 731, H&SC section 39606(d)(2)] requires a standard that "adequately protects the health of the public, including infants and children, with an adequate margin of safety." Recognizing the uncertainties in the database, staff makes the following recommendations.

1. Ozone will continue to be the pollutant addressed by the standard.
2. One-hour ambient air quality standard: staff recommends retaining the current 1-hour ozone standard at a concentration of **0.09 ppm**, not to be exceeded, based on several factors. First, at 0.12 ppm, in several studies 10 - 25% of the subjects experienced a decline of 10% or more in FEV1. In one study, these lung function changes were accompanied by increases in cough. At 0.24 ppm, increases were also observed in shortness of breath and pain on deep breath. These lung function and symptom outcomes have been demonstrated and replicated in several carefully controlled human exposure studies. The population at risk for these effects includes children and adults engaged in active outdoor exercise and workers engaged in physical labor outdoors. Thus, a margin of safety is necessary to account for variability in human responses. In addition, the chamber studies, by design, do not include potentially vulnerable populations (e.g., people with moderate to severe asthma, Chronic Obstructive Pulmonary Disease or COPD, and heart disease) who may be incorporated in the epidemiologic studies.

Second, chamber studies indicate that bronchial responsiveness and pulmonary inflammation occur with 1-hour exposure to 0.18 to 0.20 ppm. Bronchial responsiveness can aggravate pre-existing chronic respiratory disease. The ultimate impact of the inflammatory response is unclear but repeated exposures to high ozone levels may result in restructuring of the airways, fibrosis, and possibly permanent respiratory injury. These latter outcomes are supported by animal toxicology studies, which also suggest the possibility of decreases in lung defense mechanisms.

Third, epidemiological studies completed over the last 10 years indicate the potential for severe adverse health outcomes including premature death, hospitalizations, and emergency room visits. These studies include concentrations to which the public is currently being exposed. It is possible that some of these associations are due to relatively short-term exposures, for example less than two hours, since people at risk of experiencing these endpoints are unlikely to be engaged in multi-hour periods of moderate or heavy work or exercise outdoors. However, since there is high temporal correlation between 1-, 8-, and 24-hour average ozone concentrations, the averaging time of concern cannot be discerned from these studies.

Viewing all of the evidence, staff recommends retention of the 1-hour standard of 0.09 ppm, not to be exceeded, as being protective of public health with an adequate margin of safety.

3. Eight-hour ambient air quality standard: We recommend establishing a new 8-hour average standard of **0.070 ppm**, not to be exceeded. Our recommendation for the 8-hour standard is based primarily on the chamber studies that have been conducted

over the last 15 years, supported by the important health outcomes reported in many of the epidemiologic studies. With exposure for 6.6 to 8-hours to an ozone concentration of 0.08 ppm, several studies have reported statistically significant group effects on lung function changes, ventilatory and respiratory symptoms, airway hyperresponsiveness, and airway inflammation in healthy, exercising individuals. A substantial fraction of subjects in these studies exhibited particularly marked responses in lung function and symptoms. Consequently, a concentration of 0.08 ppm ozone for an 8-hour averaging time can not be considered adequately protective of public health, and does not include any margin of safety, based on the definitions put forth in State law. The one published multi-hour study investigating a concentration below 0.08 ppm showed no statistically significant group mean decrement in lung function or symptoms at 0.04 ppm compared to a baseline of clear air. In addition, all individual subjects had changes in FEV1 of less than 10%. One unpublished multi-hour study at 0.06 ppm (Adams 1998) reported no statistically significant group mean changes, relative to clean air, in either lung function or symptoms including pain on deep inhalation and total symptom score. Therefore, staff has recommended an 8-hour concentration of 0.070 ppm. Many of the studies, and issues and concerns associated with the epidemiological studies listed above concerning the 1-hour standard are also relevant to the 8-hour standard. As discussed above, it may be that the health effects, often correlated with 1-hour exposures in the epidemiologic studies, are actually associated with 8-hour (or other) average exposures. Therefore, these epidemiologic findings were factored into the margin of safety for the 8-hour average.

It should be noted that the recommended 8-hour average concentration has three rather than two decimal places. Staff initially considered selection of 0.07 ppm. However, rounding conventions applied to air quality data (see Section 7.1.4) are such that any measured value up to and including 0.074 ppm would round down to 0.07 ppm. The available data suggested that selection of 0.07 ppm would not include an adequate margin of safety, as required by State law. The one available study at 0.06 ppm did not find a group mean effect. Staff is recommending that the 8 hour average standard have three decimal places, 0.070 ppm, to ensure an adequate margin of safety. Section 6.3 discusses issues related to precision and accuracy of the monitored data.

4. Monitoring method for ozone: Staff recommends retention of the current monitoring method for ozone which uses the ultraviolet (UV) absorption method for determining compliance with the state Ambient Air Quality Standard for ozone. Incorporate by reference all federally approved UV methods for ozone as California Approved Samplers for ozone. This will not change current air monitoring practices, but will align state monitoring requirements with federal requirements.

2.6.1 Consideration of Infants and Children

The Children's Environmental Health Protection Act [Health and Safety Code section 39606 (b)] requires that air pollution effects on children and infants be specifically considered in selection of ambient air quality standards. Children have a higher ventilation rate relative to body weight at rest and during activity than adults. Children also tend to spend more time outside and be more active than adults. Consequently,

virtue of their higher ventilation rates and outdoor behavior patterns, they are likely to inhale larger total doses of ozone than the general population. However, the chamber studies of exercising children suggest that they have responses generally similar to adults, pointing to a similar degree of responsiveness. Epidemiologic studies that have examined both children and adults do not show clear evidence for greater sensitivity in children. Studies in animals at high exposure concentrations (0.5 ppm and higher, 8 hrs/day for several consecutive days) indicate that developing lungs of infant animals are adversely affected by ozone. The recommended standards are well below that level of exposure. Two studies have shown evidence of lower lung function in young adults raised in high ozone areas (Kunzli et al. 1997; Galizia and Kinney 1999). The study by Kunzli et al. (1997) suggested that exposure to ozone prior to age 6 was associated with lower attained lung function. Examination of data for the Los Angeles basin from the early 1980s, show summer averages of the 1-hour maximum to be above 0.10 ppm. This is considerably above present levels and above the recommended 1-hour standard. There is also evidence that children who play three or more sports are at higher risk of developing asthma if they also live in high ozone communities in Southern California. This study needs to be repeated before the effect can be attributed to ozone exposure with greater certainty, but the finding is of concern. The warm season daily 8-hour maximum concentrations of ozone measured in these high ozone areas, over the four years of study, was 0.084 ppm. The proposed 8-hour standard of 0.070 ppm, therefore, should protect most children from asthma induction that may be associated with ozone exposure. Collectively, this body of evidence suggests that although children appear to be similarly responsive to a given dose of ozone as adults, they are at greater risk than adults of experiencing adverse responses to ozone by virtue of their higher level of outdoor activity, and consequently greater total exposure.

2.7 Estimated Health Impacts Benefits

Exposure to ozone at current ambient levels has substantial health impacts, including, but not limited to, death, hospitalization, emergency room visits, and symptoms of respiratory irritation. Staff estimates that the annual health impact of exposure to ozone at current levels, compared to attainment of the proposed State 8-hour and 1-hour ozone standards throughout California includes :

~~It is estimated that attainment of the proposed ozone standards throughout California would avoid a significant number of adverse health effects each year, specifically:~~

- 630 (310 – 950, probable range) ~~580 (290 – 870, probable range)~~ premature deaths for all ages.
- 4,200 (2,400 – 5,400, 95% CI) ~~3,800 (2,200 – 5,400, 95% confidence interval (CI))~~ hospitalizations due to respiratory diseases for all ages.
- 660 (400 – 920, 600 (360 – 850, 95% CI) emergency room visits for asthma for children under 18 years of age.
- 4.7 million (1,200,000 – 8,600,000, 3.3 million (430,000 – 6,100,000, 95% CI) school absences for children 5 to 17 years of age.
- 3.1 million (1.3 million – 5.0 million, 2.8 million (1.2 million – 4.6 million, 95% CI) minor restricted activity days for adults above 18 years of age.

These health impact estimates are based on the results of epidemiologic studies on the health effects of ozone exposure and an exposure reduction methodology modified from analyses conducted by the U.S. EPA and other investigators (see Appendix B). The health impacts estimates were made for only a small number of the known health effects of ozone exposure and, consequently, underestimate the total public health impact. The health impacts assessment was not used to select the appropriate levels of the proposed ozone standards.

For comparison purposes, we also estimated the health impacts o current ozone levels compared to attainment of the federal 8-hour ozone standard of 0.08 ppm, and the health impacts of current ozone levels compared to attainment of the State 1-hour ozone standard alone. Specifically, we estimate that 360 (180 – 550, probable range) premature deaths annually are related to current ozone levels, compared to statewide attainment of the federal 8-hour standard, and about 540 (270 – 810, probable range) premature deaths annually are related to current ozone levels, compared to statewide attainment of the State 1-hour standard. Results for other health endpoints are in Appendix B.

The differences between the results are the “incremental” impacts of not attaining the State 1-hour and 8-hour standards, compared to the federal 8-hour ozone standard. However, it is more reasonable to consider attainment of the two State standards together, compared to current ozone levels, since it is unlikely that control strategies will be geared to fist attain one standard and then the other. Nonetheless, the current impact of not attaining the federal 8-hour standard is about 360 premature deaths annually, with an additional 270 deaths associated with not attaining the proposed State 8-hour standard, making the total estimated impact of not attaining both standards 630 deaths. Similarly, the current impact of not attaining the State 1-hour standard is about 540 premature deaths annually, with an additional 90 deaths associated with not attaining the proposed 8-hour standard, making the total estimated impact of not attaining both the State 1-hour and 8-hour ozone standards 630 premature deaths. More detailed discussion of this analysis is available in Appendix B.

As discussed in Appendix B, there are a several important assumptions and uncertainties in this analysis. Some concern the study design, statistical methods, and choice of epidemiological studies used to develop the concentration-response (CR) functions used in the analysis. Few studies have investigated the shape of the CR function, or whether there is a population response threshold for health endpoints other than emergency room visits for asthma. Further uncertainty is added by assumptions in the statewide exposure assessment. It should also be noted that since several health effects related to acute exposure, and effects of chronic ozone exposure, are not included in the estimates noted above, the health benefits associated with lowering ozone exposure are likely underestimated.

2.8 Public Outreach and Review

A draft Staff Report containing staff’s preliminary findings was released to the public on June 21, 2004 titled, “Review of California Ambient Air Quality Standard for Ozone”. Public outreach for the standard review involved dissemination of information through various outlets to include the public in the regulatory process. In an ongoing effort to

include the public in the review of the ozone standard, the ARB and OEHHA integrated outreach into public meetings, workshop presentations, electronic “list serve” notification systems, and various web pages. Notification of release of the Staff Report, the schedule for public meetings and workshops, and invitations to submit comments on the Staff Report were made through the “list serve” notification system. Public workshops on the proposed ozone standard were held on July 14 – 16, 2004 in Sacramento, El Monte, and Fresno. An additional public workshop was held on August 24, 2004 in Sacramento.

Individuals or parties interested in signing up for an electronic e-mail “list serve” notification on the ~~PM~~ozone standards, as well as any air quality-related issue, may self-enroll at the following location: www.arb.ca.gov/listserv/aaqs/aaqs.htm. Additional information on the standards review process is also available at the ozone standards review schedule website at: www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm.

2.9 Air Quality Advisory Committee Review

The Air Quality Advisory Committee, an external scientific peer review committee that was appointed by the President of the University of California, met January 11 and 12, 2005, in Berkeley, California to review the initial Staff Report and public comments, and to ensure that the scientific basis of the recommendations for the ozone standard are based upon sound scientific knowledge, methods, and practices. The AQAC held a public meeting, which provided time for oral public comments, and discussed their review of the draft Staff Report and the draft recommendations, and provided comments for improving the draft Staff Report. Final findings were received on February 24, 2005.

The AQAC determined that the staff recommendations were well founded on the scientific literature, and voted to endorse them. The Committee made suggestions for minor changes to the draft Staff Report to increase clarity, requested more detailed discussion of several topics, and inclusion of several additional scientific papers. The AQAC findings ~~is~~are included in this Initial Statement of Reasons as Appendix C, in Volume IV.

2.10 Environmental and Economic Impacts

The proposed ambient air quality standards are scientific in nature, and will in and of themselves have no environmental or economic impacts. Standards simply define clean air. Once adopted, local air pollution control or air quality management districts are responsible for the adoption of rules and regulations to control emissions from stationary sources to assure their achievement and maintenance. The Board is responsible for adoption of emission standards for mobile sources. A number of different implementation measures are possible, and each could have its own environmental and/or economic impact. These impacts must be evaluated when the control measure is proposed. Any environmental or economic impacts associated with the imposition of future measures will be considered if and when specific measures are proposed.

2.11 Environmental Justice

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies (Senate Bill 115, Solis; Stats 1999, Ch. 690; Government Code §65040.12(c)). The Board established a framework for incorporating environmental justice into the ARB's programs consistent with the directives of State law (ARB, 2001). The policies developed apply to all communities in California, but recognize that environmental justice issues have been raised more in the context of low-income and minority communities, which sometimes experience higher exposures to some pollutants as a result of the cumulative impacts of air pollution from multiple mobile, commercial, industrial, areawide, and other sources.

Because ambient air quality standards simply define clean air, all of California's communities will benefit from the proposed health-based standards, as progress is made to attain the standards. Over the past twenty years, the ARB, local air districts, and federal air pollution control programs have made substantial progress towards improving the air quality in California. However, some communities continue to experience higher exposures than others as a result of the cumulative impacts of air pollution from multiple mobile and stationary sources and thus may suffer a disproportionate level of adverse health effects. Since the same ambient air quality standards apply to all regions of the State, these communities will benefit by a wider margin and receive a greater degree of health improvement from the revised standards than less affected communities, as progress is made to attain the standards. Moreover, just as all communities would benefit from new, stricter standards, alternatives to the proposed recommendations, such as not proposing an eight-hour ozone standard, would adversely affect many communities.

While it is possible that residents in environmental justice communities may be particularly sensitive to ozone, only one study investigated whether socioeconomic status (SES) alters responses to ozone exposure, and those results were difficult to explain. Hence, the study did not allow inferences as to whether socioeconomic status impacts on sensitivity to ozone. Moreover, other controlled studies investigating whether gender, ethnicity or environmental factors contribute to the responses to ozone exposure could not convincingly demonstrate a link with responsiveness. Therefore, the database is insufficient to conclude whether differences in ozone susceptibility exist in environmental justice communities. These studies are discussed in more detail in Section 9.6.8.

Once ambient air quality standards are adopted, the ARB and the local air districts will propose emission standards and other control measures designed to result in a reduction of ambient ozone levels. The environmental justice aspects of each proposed control measure will be evaluated in a public forum at this time.

As additional relevant scientific evidence becomes available, the ozone standards will be reviewed again to make certain that the health of the public is protected with an adequate margin of safety.

2.12 References

Adams WC. 1998. Dose-response effects of varied equivalent minute ventilation rates on pulmonary function responses during exposure to ozone. Final Report to the American Petroleum Institute. Washington D.C.

Air Resources Board. Ambient Air Quality Standard for Ozone: Health and Welfare Effects. Staff Report. September 1987. Sacramento, CA.

Air Resources Board and Office of Environmental Health Hazard Assessment. Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. 2000.

Air Resources Board (2001). Policies and Actions for Environmental Justice, December 13, 2001.

Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ Health Perspect* 107:675-679.

Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. 1997. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen – results of a pilot study. *Environ Res* 72:8-23.

McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386-391.

World Health Organization (2000). Air Quality Guidelines for Europe, Second Edition. (WHO regional publications, European series, No. 91.)

Appendix B

Quantifying the Health ~~Benefits of Reducing~~Impacts of Ozone Exposure

Appendix B

Quantifying the Health Benefits of Reducing Impacts of Ozone Exposure

Quantifying the Health Benefits Impacts of Reducing Ozone Exposure

~~The objectives of this appendix are to quantify the adverse health effects of current ozone levels in California by estimating the health benefits that would accrue from a hypothetical control strategy that achieves the proposed ambient air quality standards for ozone. This health effects assessment is not being used to set the health standards or in any formal cost-benefit analysis. As such, the results from this appendix are provided for public information about the expected benefits of attaining the proposed standards and do not include monetary values.~~

~~There have been several recent published efforts to estimate the health benefits associated with reducing population exposures to ozone (U.S. Environmental Protection Agency 1999; Levy et al. 2000). Numerous epidemiologic studies conducted in the United States and other countries point to the adverse health effects from exposure to ozone. The effects from short-term exposure include, but are not limited to: hospital admissions for respiratory causes, emergency-room visits for asthma, minor restricted activity days, acute respiratory symptoms, exacerbation of asthma, and premature mortality (National Research Council, 2002; U.S. Environmental Protection Agency, 2004). In addition, there is more limited evidence that long-term exposure to ozone may result in new cases of asthma and premature mortality.~~

~~Below we describe the methods, data, results and uncertainties involved with estimating the health benefits of the proposed~~The objective of this appendix is to quantify a subset of adverse health effects attributable to current ozone levels in California to illustrate some of the health impacts of continued ozone exposures. This health impacts assessment was not used to select the appropriate levels of health-based ambient air quality or to perform a cost-benefit analysis. Rather, the results from this assessment provide information to the public on some of the health impacts of current ozone levels. While this section presents the results of our analysis as the impacts of exposure to current levels of ozone, the results can also be viewed as the public health benefits expected to accrue with attainment of the proposed standards.

Specifically, we present results of an analysis of the impacts of the current ozone levels compared to attaining both the proposed State 8-hour standard and the federal 8-hour standard. In addition, we compare the benefits from attaining the proposed State 8-hour standard to the state 1-hour standard.

There have been several recent published efforts to estimate the impacts of ozone on public health, and the health benefits likely to be associated with reducing population exposures to ozone (U.S. Environmental Protection Agency 1999; Levy et al. 2000, Hubbell et al. 2005). This appendix presents a specific analysis of the health impacts of ozone exposure. It describes the methods, data, results and uncertainties involved with estimating the health impacts associated with exposures to current ambient

concentrations of ozone.

The reader should note that health impacts estimates were made for only a small number of known effects of ozone exposure, and consequently this analysis is an underestimate of the total public health impact of current ozone levels.

Health Effects Estimation Approach

Section 812 of the federal Clean Air Act ~~required~~requires the U.S. EPA to periodically conduct an analysis of the health benefits of current federal air pollution regulations, which resulted in a report to the U.S. Congress (U.S. EPA, 1999). These efforts have undergone years of public review and comment as well as full peer review by the U.S. EPA's independent Science Advisory Board and by the National Research Council (2002). We have, therefore, drawn considerably from these prior efforts at the federal level, particularly in the development of concentration-response functions. We have also added new studies ~~published~~ from around the world that have appeared since publication of the most recent U.S. EPA report. ~~The selection~~Selection of the studies and functions ~~to include~~included in our analysis has undergone review by several independent experts on the subject of air pollution and health.

Estimating the health ~~benefits~~impacts associated with ~~reductions in~~current levels of ambient ozone involves four elements:

1. Estimates of the changes in ozone concentrations due to a hypothetical ~~control strategy.~~ ozone reduction control strategy that achieves attainment of a standard.
2. Estimates of the number of people exposed to ozone.
3. Baseline incidence of the adverse health outcomes associated with ozone.
4. Concentration-response (CR) functions that link changes in ozone concentrations with changes in the incidence of adverse health effects. These functions ~~produce~~come from epidemiological studies and are expressed in terms of a beta coefficient, indicating the percent reduction in a given health outcome due to a unit change in ozone.

Health effects results from epidemiological studies are based on various ozone averaging times: 24-hour, 8-hour and 1-hour. As a result, we converted the 8-hour and 24-hour epidemiological results into equivalent values on a 1-hour scale to allow direct comparison of the different studies.

Ultimately, the product of ~~these~~the above four elements generates estimates ~~of the expected number of avoided adverse health outcomes associated with a hypothetical control strategy to reduce current levels of ozone to the proposed standard.~~ that represent the current impact of ozone on public health, compared to attainment of the proposed 8-hour standard, since that is the more stringent of the two standards proposed. Each of these elements is discussed below. Our methods make use of U.S. EPA's ~~development of the Environmental Benefits Mapping and Analysis Program~~

(BenMAP), with modifications where appropriate to reflect the application to California. In addition, we incorporated several recent studies that were not available when the most recent version of BenMAP was California's setting and more recent studies released (Bell et al., 2004; Gryparis et al., 2004). All methods and results presented herein are consistent with those methods used by U.S. EPA in their health benefits assessment (Hubbell et al., 2005). In addition, we have derived substantial material from other previous health impact studies including the U.S. EPA estimates of health benefits of the Clean Air Act (U.S. EPA, 1999), the World Health Organization (WHO) meta-analysis of ozone health effects (Anderson et al. 2004), and the Levy et al. (2001) analysis of the public health benefits of reducing ozone impacts of current ozone concentrations.

Exposure Estimation and Assumptions

The estimation of ozone exposure involves two key elements: assessing changes in ozone concentrations, and estimating the population exposed to these changes in ozone levels.

To assess the changes in the current ozone concentrations necessary to achieve the proposed standards, we first determined the design value, the benchmark used for attainment status. The design value is the Expected Peak Day Concentration, thea value that reflects the highest concentration expected to occur on any given year occur, once per year on average, based on the past three years of air quality monitoring data. The use of three years of data reduces the effect of an anomalous year. Details on how the design values are calculated are presented in Chapter 7. Because the designations of the air quality standards are done mostly generally made at the air basin level, the design value for the basin was used for all counties within the basin.

Monitoring data for 2001 to 2003 were used from all monitors in the State meeting quality assurance criteria for valid data, and were extracted from the ARB ADAM database (ARB, 2004). Chapter 7 provides detailed analyses of exposure to ozone in California.

To calculate changes in exposure to ozone that reflect a hypothetical attainment of the proposed ambient air quality standards, a proportional linear rollback procedure was used. Under real-world conditions, control strategies will likely have some impact on days with low and moderate levels of ozone, as well as on days with high levels. Our rollback procedure reflects this observation. Details on the changes in the distribution of ozone concentrations over time are provided in the Supplement to this appendix.

Design Value Rollback Method

Rollback factors from the 1-hour and 8-hour ozone design values to the applicable standard were calculated for each air basin to assess the daily reductions in current ozone concentrations estimated to result at all monitoring sites when the standards are achieved. ~~achieved, rollback factors from the 1-hour and 8-hour ozone design values to the applicable standard were calculated for each air basin. The ozone design value selected~~ Rollback factors were based on design values for 2003. The design values reflect measured air quality for three years, 2001 through 2003. The design value was determined for each monitoring site according to the relevant regulatory specifications. For example, the Federal 8-hour design value is the three-year average of the annual 4th highest 8-hour ozone measurements. The design values for the state 1-hour and 8-hour standards are Expected Peak Day Concentrations (the statistically derived value expected to be exceeded once per year, on average – details are in Section 7.1.2 of ~~was the highest for the three-year period (2001 to 2003).~~ Chapter 7). Design values for basins are simply the highest design value at any site within the basin. The basin design values typically determine attainment of each ozone standard. An uncontrollable ozone concentration of 0.04 ppm (see Chapter 4) was factored into the calculation of the rollback factor (see below). This represents the average daily one-hour maximum background ozone concentration. The rollback factor was assumed to apply to each site in the air basin for every day in a given year.

~~This~~ Our methodology assumed that under the hypothetical attainment ~~setting, condition~~ all ozone observations within an air basin were subjected to the same percentage rollback factor based on the basin's three-year high value. To investigate the plausibility of this assumption, we examined the trends in the annual distributions of the 1-hour and 8-hour concentrations of ozone in the South Coast Air Basin (SoCAB). Due to its population and current ozone levels, a significant proportion of current statewide health ~~benefits are projected to accrue~~ impacts occur in the SoCAB. For this region, the estimated downward trend was consistent for both 1-hour and 8-hour concentration reductions from the 1980s to the current levels period. The maximum, the 90th, 80th, 70th, 60th, 50th and 40th percentiles from the annual distribution of the basin's daily high concentrations, as well as the individual site's daily highs, show a consistent downward trend ~~from~~ since the 1980s. More importantly, when we examined the rate of change in the concentrations above background ~~from~~ since the 1980s, it was similar among the percentiles. This analysis ~~justifies~~ supports our application of a constant percentage rollback to all sites within ~~a~~ each air basin. Results for several representative sites used in this analysis of ozone trends can be found in the Supplement to this appendix.

Roll-Back Procedure

For each ~~monitoring site~~ air basin in the State, the rollback factor necessary to move ~~from~~ the basin-high design value to the proposed standard was calculated for both the 1- and

8-hour averages. We assumed that only the portion of each ozone value above a background of 40 ppb will decrease as progress toward attainment takes place. For example, in the table of hypothetical values below (Table B-1), suppose the basin-high design value would need to be reduced by 50% for the portion above 40 ppb to achieve the standard. Thus, an ozone measurement of 100 ppb today would face a reduction of $(100 - 40) \times 50\% = 30$ ppb. Hence the projected value at attainment would be $30 \text{ ppb} + 40 \text{ ppb} = 70$ ppb. The effective rollback rate of moving from 100 ppb to 70 ppb is 30%. Similarly, a value close to 40 ppb like 50 ppb would face a reduction of $(50 - 40) \times 50\% = 5$ ppb. With the projected value at attainment of $5 \text{ ppb} + 40 \text{ ppb} = 45$ ppb, the effective rollback rate is 10%, calculated from 50 ppb today. In summary, while both measurements in this hypothetical example (100 ppb and 50 ppb) are subject to the same rollback rate of 50%, the effective rollback rate differs due to the rollback procedure taking into account the background of 40 ppb

Table B-1: Example of Proportional Roll-Back Procedure

<u>Current ozone</u>		<u>Rollback Rate for Portion Above 40 ppb</u>	<u>Future ozone</u>		<u>Effective Rollback Rate</u>
<u>Measured (ppb)</u>	<u>Above 40 ppb</u>		<u>Above 40 ppb</u>	<u>Projected</u>	
<u>100</u>	<u>60</u>	<u>50%</u>	<u>30</u>	<u>70</u>	<u>30%</u>
<u>50</u>	<u>10</u>	<u>50%</u>	<u>5</u>	<u>45</u>	<u>10%</u>

For the 1-hour standard, rollback factors based on 1-hour design values were used to project 1-hour observations assuming a scenario where the 1-hour standard of 0.09 ppm was attained. Specifically, the rollback factor for an air basin was: $(0.094 \text{ ppm} - 0.04 \text{ ppm}) / (1\text{-hour Design Value} - 0.04 \text{ ppm})$. The concentration of 0.094 was used since this is the highest value considered in attainment (after being rounded to 0.09), based on rounding conventions. This procedure produced basin-specific rollback factors, which were applied to daily maximum 1-hour ozone values at all sites in the applicable basin, considering the background of 0.04 ppm. Basin-specific ratios were used for this purpose since the ratios can be significantly different from one basin to another. For example, in the South Coast Air Basin, the 1-hour design value was 0.178 ppm, so the rollback factor was $(0.094 - 0.04) / (0.178 - 0.04) = 39\%$.

For the 8-hour standard, we used basin-specific 1-hour and 8-hour design values to convert the 1-hour data into a form useable for rollback calculations for the 8-hour standard. One-hour ozone concentrations are highly correlated with 8-hour concentrations. Therefore, we calculated the 1-hour value when the 8-hour standard is attained in each air basin and defined this as the “equivalent design value” for the 8-hour standard in that basin. The target for the 8-hour standard (0.070 ppm) was converted to a basin-specific 1-hour equivalent so that the CR functions that were all converted to 1-hour averaging times could be utilized. The conversion was based on the

assumption that the future ratio between 1-hour and 8-hour design values will be similar to its current value. For example, in the South Coast, the 1-hour and 8-hour design values are 0.178 and 0.146 ppm, respectively. To attain the 8-hour standard of 0.070 ppm, the equivalent 1-hour target was projected to be $0.070 \times (0.178/0.146) = 0.086$ ppm. Therefore, 0.086 is the equivalent 1-hour target for attainment of the proposed State 8-hour standard in the South Coast.

Rollback factors for attainment of the 8-hour standard are calculated as: (Equivalent Target – 0.040 ppm) / (1-hour Design Value – 0.04 ppm). So, for the South Coast Air Basin, the rollback factor was $(0.086 - 0.04) / (0.178 - 0.040) = 33\%$.

TheseThe roll-back procedure toward a 0.040 ppm background reflects currently observed rates of change in all parts of the ozone concentration distribution. The rollback factors werethen applied on a site-by-site basis to the ozone readingsfor everymonitoring data for each day. The difference between the observed value and the rolled-back value was calculated for each day of the year in terms of 1-hour maximum ozone (for both standards), thus avoiding the uncertainty associated with converting CR functions into an 8-hour maximum ozone scale.

Similar calculations were made for the federal 8-hour standard. The concentration of 0.084 ppm was used in the rollback since this is the highest value considered in attainment (after being rounded to 0.08), based on rounding conventions.

Health effectsimpacts were then estimated for each day in a given year, summed across sites over the year, and then averaged over the three years of data. We also ensured that no benefitsimpacts would be calculated for any day with an average concentration at or below the assumed background ozone level of 0.04 ppm. For the technical reader, the mathematical formulae for our rollback procedure and evidence forsupporting the rollback assumptions are provided in the Supplement to this appendix.

Estimation of Exposed Population

To estimate the number of people exposed to the ozone changes observed at each monitoring site, the county population was divided by the number of monitoring sites in a given county. This assumes that the population is equally distributed around each monitoring site within a county. We used county population data from the year 2000 census. For further details, see the Supplement to this appendix. We also examined the sensitivity of this assumption by considering two alternative methods for estimating exposure to ozone: census tract interpolation and county averaging of monitored concentrations. Details of these sensitivity analyses are provided below.

Estimates of the Baseline Incidence of Adverse Health Outcomes

The health effect baseline incidences are the number of health events per year per unit population. In this analysis, all baseline incidence rates except those for school absenteeism were taken from U.S. EPA's BenMAP software program.

~~For mortality, the~~Mortality incidence rates were obtained from the U.S. Centers for Disease Control (CDC), as derived from ~~the~~ U.S. death records and the U.S. Census Bureau. Regional hospitalization counts were obtained from the National Center for Health Statistics (NCHS) National Hospital Discharge Survey (NHDS). Per capita hospitalizations were calculated by dividing these counts by the estimated county population estimates derived from the U.S. Census Bureau and the population projections used by NHDS. Hospitalization rates for "all respiratory causes" included ICD-9 codes 460-519. Similarly, regional asthma emergency room visit counts were obtained from the National Ambulatory Medical Care Survey (NHAMCS), combined with population estimates from the 2000 U.S. Census to obtain rates. Illness-related school loss baseline incidence rates were based on Hall et al. (2003). Ostro and Rothschild (1989) provided the estimated rate for minor restricted activity days.

The assumed incidence rates are summarized in Table ~~B-17~~B-19 in the Supplement to this appendix. All counties and sites within each county were assumed to have the same incidence rate for a given population age group.

Concentration-Response Functions

Concentration-response (CR) functions are equations developed from epidemiologic studies that relate the change in the number of adverse health effect incidences in a population to a change in pollutant concentration experienced by that population. As reviewed in Chapter 9 (Controlled Exposure 40, Studies) and Chapter 10 (Epidemiologic Studies), a wide range of adverse health effects has been associated with exposure to current ambient concentrations of ozone. However, we only used CR functions derived

from epidemiologic studies in this analysis. Developing concentration-response functions from this vast and not fully consistent literature is a difficult task and ultimately involves subjective evaluations. In this section, we aim to provide a fair and accurate reflection of the current scientific literature. We also aim to provide enough detail so that others may fully evaluate our assumptions and methodology. Below, we provide CR functions for effects of short-term ozone exposure on premature mortality, hospital admissions for respiratory disease, emergency room visits for asthma, school absenteeism, and minor restrictions in activity. Although epidemiologic studies also ~~other effects have been related to~~ report other adverse effects associated with ozone exposure – such as asthma exacerbations, respiratory symptoms, hospital admissions for cardiovascular disease with short-term ozone exposures, and mortality and asthma onset associated with long-term exposure (i.e., several years) – we determined that the existing evidence was either insufficient or too uncertain to serve as a basis for making quantitative ~~CR function~~ impacts estimates. A good example is asthma exacerbations for which several studies have reported associations with ozone. However, different subgroups of asthmatics and different outcome measures were used, making it difficult to develop consensus estimates.

In this appendix, the primary studies used in the health ~~benefit~~ impacts assessment are ~~generally~~ epidemiological. There are a number of reasons for using epidemiological studies. While human chamber studies have the merit of being able to carefully control for dose and response, they usually involve small sample sizes that ~~do~~ may not include the most sensitive subpopulations, and cannot capture severe outcomes like hospitalization or premature death. Lagged or cumulative effects are similarly omitted, and only a limited range of exposures is examined. In short, human chamber studies are helpful to support causality and to determine effects of short-term exposure on measures like lung function ~~in generally healthy individuals,~~ and airways inflammation, but they do not necessarily ~~provide the information on~~ general population responses to exposure to ozone. For the latter purpose, epidemiological studies which incorporate varying subgroups, exposure scenarios, behaviors, and health outcomes will best serve to ~~determine~~ estimate the overall potential human ~~response to a particular pollutant~~ health impact of air pollutants and be the source of CR functions used for quantitative ~~estimates for~~ health impact assessment.

Besides the primary studies, some CR functions were developed from previous estimates of the health impacts of ozone exposures. Sources for these studies include the U.S. EPA estimates of the health effects associated with the Clean Air Act under Section 812 (U.S. EPA, 1999), the World Health Organization (WHO) meta-analyses on ozone (Anderson et al., 2004), and the Levy et al. (2001) analysis of the public health benefits of reducing ozone.

This section discusses some factors that impact health effect estimates and outlines the epidemiological studies that were used for the basis of the CR functions.

Conversions for Ozone Measurements of Various Averaging Times

Most health studies considered in our analysis were conducted with ozone levels measured as 1-hour maximum or 8-hour maximum. However, there were some studies that measured ozone averaged over other time increments. Since these studies were conducted throughout the United States and other parts of the world, a national average of adjustment factors were used to convert all measurements to 1-hour and 8-hour averages (Schwartz 1997). The 1-hour maximum was assumed to be 2.5 times the 24-hour average, and 1.33 times the 8-hour average concentration. These conversion factors have been used in previous meta-analyses of the ozone epidemiological literature (Levy et al., 2001; Thurston and Ito 2001). Our examination of California monitoring data for 2001-2003 in the San Francisco Bay Area and South Coast indicates that the ratios in California are similar. To reduce the uncertainty associated with converting the results into both the 1-hour and 8-hour time scales, we converted the epidemiological results into a common 1-hour scale only. Because the majority of studies report findings in term of ppb, CR functions were calculated per ppb, and air quality measurements were converted from ppm to ppb accordingly in the calculation of health effects.

Thresholds

Assumptions regarding the appropriateness of applying thresholds, and at what level, can have a major effect on health ~~effects~~impacts estimates. One important issue in estimating ~~ozone health effects~~ozone-related health impacts is whether it is valid to apply the CR functions throughout the range of predicted changes in ambient concentrations, even changes occurring at levels approaching the natural background concentration (without any human activity).

As reviewed in Chapter 10, most of the epidemiologic studies include very low ozone concentrations in their analysis and no clear threshold for effects has been reported, although the issue has not been fully investigated except with reference to ER visits for asthma. These latter studies, reviewed in Section 10.2.3 suggest a population threshold in the range of 0.075 to 0.110 ppm for 1-hour exposures, and 0.056 to 0.084 ppm (using a ratio of 1.33) for 8-hour exposures (see pg. 8-14; figure 8-1). In our approach of applying a constant percent change rollback to all of the basin-wide monitors, many of the reductions in ozone concentrations will occur below the proposed standard. Thus, for some days, our estimate of ~~benefits~~impacts of ozone exposure will be based on ozone concentrations that are within the range of the original epidemiologic studies, but below the proposed standards. In our ~~base case~~base-case model, we assumed that ~~there was no effect threshold was in evidence and~~concentration, and therefore we used the background level of 0.04 ppm as the no effects level. ~~As an alternative for a~~For the sensitivity analysis, we assumed ~~a several~~no effects level at 0.075 ppm levels but adjusted the remaining slope to account for application of a threshold to the concentration-response function. This is described in greater detail below.

Developing the Concentration-Response Function

Most of the epidemiologic studies used in our estimates have used a log-linear model to ~~represent~~describe the relationship between ozone exposure and the health endpoint. In this case, the relationship between ozone levels and the natural logarithm of the health effect is estimated by a linear regression. This regression model generates a beta coefficient that relates the percent change in the health outcome to a unit ~~increase~~change in ozone. Existing studies have reported either a beta coefficient for a unit change in exposure or a relative risk (RR) for a specified change in ozone concentrations, such as 10 ppb ~~4-hour~~1-hour maximum. The RR is defined as the ratio of the health effect predicted from the higher exposure relative to some baseline exposure. Health effect estimates presented in a given study as RR for a specified change in ozone, ΔO_3 , were converted into an estimated beta using the equation:

$$\beta = \ln(RR) / \Delta O_3$$

.The daily change in ozone at each monitoring site i.e., the difference between current ozone and the standard (= ΔO_3) was used to calculate RR:

$$RR = \exp(\beta \Delta O_3)$$

Then, the RR estimates were used to determine the population attributable risk (PAR), which represents the proportion of the health effects in the whole population that may be prevented if the cause (ozone pollution in our case) is reduced by a given amount. Specifically,

$$PAR = (RR - 1) / RR$$

Ultimately, the estimated impact on the health outcome is calculated as follows:

$$\Delta y = PAR \times y_0 \times pop$$

where:

Δy = changes in the incidence of a health endpoint corresponding to a particular change in ozone,

y_0 = baseline incidence rate/person within a defined at-risk subgroup, and

pop = population size of the group exposed.

The parameters in the functions differ depending on the study. For example, some studies considered only members of a particular subgroup of the population, such as individuals 65 and older or children, while other studies considered the entire population in the study location. When using a CR function from an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in ozone in a location, it is important to use the appropriate parameters for the CR function. That is, the ozone averaging time, the subgroup studied, and the health endpoint should be the same as, or as close as possible to, those used in the study that

estimated the CR function.

In some cases, results from several studies of the same health endpoint were combined to estimate the health effect. An inverse-variance weighting scheme was used to pool results from these studies, allowing studies with greater statistical power to receive more weight in the pooled assessment. This approach implicitly assumes that all studies are equally valid and representative of the population in question, and is the standard approach applied in many impact analysis settings.

Mortality from Short-Term Exposure

Chapter 10 concludes that there is sufficient evidence for an effect of daily exposure to ozone (possibly with a lag response of a day or two) on premature mortality. These effects are based on daily time-series studies of counts of daily all-cause mortality within a given city reviewed over several years. The studies control for most other factors that may impact daily mortality such as weather, time trends, seasonality, day of week, and other pollutants. In addition, the studies have been undertaken over a wide range of weather conditions, seasonal patterns, covarying pollutants, baseline population characteristics. Chapter 10 reviews the uncertainties inherent in these studies. The U.S. EPA ~~is currently funding~~ has funded several meta-analyses ~~of the ozone-mortality~~ investigating the association but this information is currently not ~~between ozone and mortality, but the results of these analyses are not yet~~ available. Therefore, below we present the effect estimates from the available literature and develop our rationale for a central estimate and probable bounds that reflect the observed range of effect estimates. Figure 1 summarizes the most relevant meta-analytic studies to date. Additional information about these studies is provided in Chapter 10.

The World Health Organization (~~WHO~~) (Anderson et al., 2004) conducted a meta-analysis of the 15 cities in Europe (Anderson et al. 2004). Their meta-estimates indicate a relative risk of 1.003 (95% CI = 1.001 – 1.004) for a 10 $\mu\text{g}/\text{m}^3$ change in 8-hour ozone. For standard pressure (1 atmosphere) and temperature (25° C), 1 ppb ozone equals 1.96 $\mu\text{g}/\text{m}^3$. We have assumed the ratio between 1-hour and 8-hour ozone of 1.33 and between 1-hour and 24-hour of 2.5 (Schwartz 1997). Making the conversions, the WHO estimate implies a 1.13% change (95% CI = 0.38 - 1.51) in daily mortality per 10 ppb change in ~~24-hour~~ 24-hour average ozone. The WHO also provided an estimate correcting for possible publication bias using a trim and fill technique. Under an assumption that bias was present, the adjusted estimate is 0.75 % (95% CI = 0.19 – 1.32) per 10-ppb change in 24-hour average ozone.

This estimate is very similar to that produced by Levy et al. (2001). In their meta-analysis they began with 50 time-series analyses from 39 published articles. A set of very strict inclusion criteria was applied, which eliminated all but four studies. Reasons for exclusion included: studies outside the US, use of linear temperature terms (versus non-linear and better modeled temperature), lack of quantitative estimates, and failure to include particulate matter (PM) in the regression models. Ultimately, their analysis

generated an estimate of 0.98% (95% CI = 0.59 – 1.38) per 10 ppb change in 24-hour average ozone. If the criteria are loosened to include eleven more studies, the pooled estimate decreases to 0.80 (0.60 – 1.00). Stieb et al. (2002) also reported a similar effect estimate based on 109 previous studies (including those with single- and multi-pollutant models) of 1.12 (0.32 – 1.92). Thurston and Ito (2001) reviewed studies published prior to the year 2000. When the authors focused on seven studies that more carefully specified the effect of a possible confounder, daily temperature, by using non-linear functional forms, the resulting meta-estimate was 1.37% (95% CI = 0.78 – 1.96). Relaxing this constraint to include all 19 available studies, the resulting risk estimate was 0.89% (95% CI = 0.56 – 1.22) per 10-ppb change in 24-hour ozone.

Two more recent meta-analyses have been published that provide lower effect estimates. Gryparis et al. (2004) is an analysis of 23 European cities from the ~~APEHA2~~APEAH2 study. The study controlled for potential confounders by including average daily temperature and humidity, respiratory epidemics, day of week in the regression model. The overall full-year estimate was 0.5% (95% CI = -0.38 – 1.30) per 10-ppb change in 24-hour average ozone. A meta-analysis was also conducted using summer-only data. Presumably this estimate will be less confounded by seasonality and also represent a time when the population would be spending more time outdoors. The summer-only estimate was 1.65% (95% CI = 0.85 – 2.60) per 10-ppb change in 24-hour average ozone. This summer-specific estimate might be particularly relevant for California due to its ~~milder~~ climate. A meta-analysis of the 95 largest U.S. cities from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data base provided estimates using a similar natural spline model for every city (Bell et al., 2004). Ultimately, the model suggested an effect estimate of 0.25% (95% CI = 0.12 – 0.39) per 10-ppb change in 24-hour average ozone. The NMMAPS study may generate an underestimate of the impact of mortality due to the modeling methodology used to control weather factors. Specifically, this effort included four different controls for temperature and dewpoint, where most other times-series analyses used only two or modeled extreme weather events more carefully and used city-specific models to ensure the best fits. In comparing the results for particulate matter (PM) for a given city with studies of individual cities by other researchers, the NMMAPS results are usually lower (Samet al ~~et., et al.~~, 2000). This estimate was based on a lag consisting of today's and yesterday's ozone concentrations. When a longer period 7-day lag was used the estimate increased to 0.52% (95% CI = 0.27 – 0.77) per 10-ppb change in 24-hour ozone.

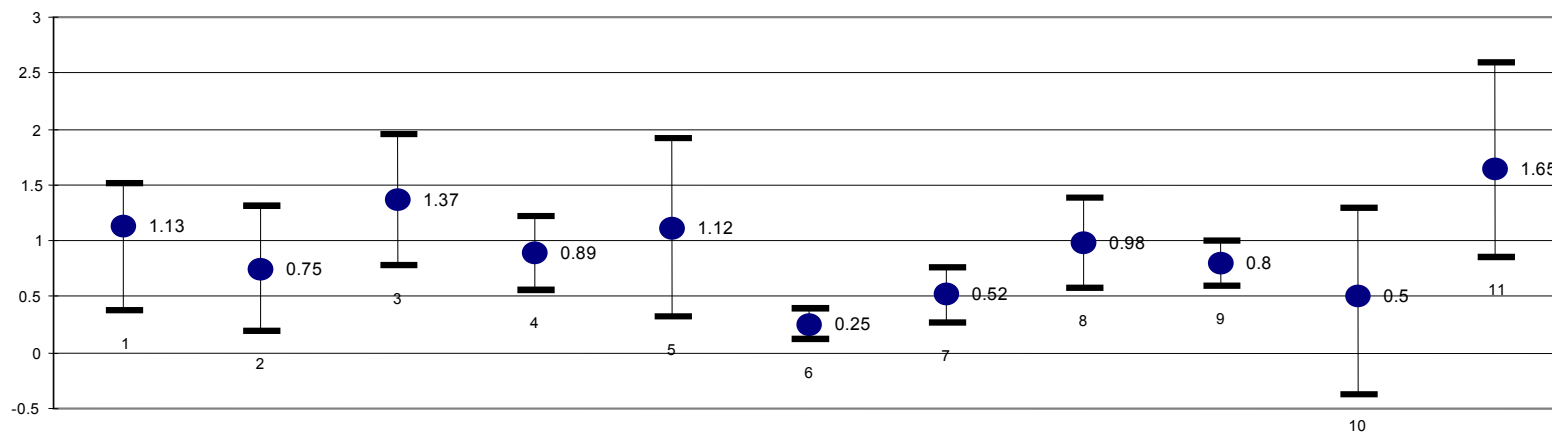
Our estimates for the effects of ozone on mortality ~~attempt to~~ reflect the range provided in the ~~above-cited~~above-cited studies. Table B-2 summarizes the effect estimates reported in these studies. Figure B-1 provides a graphical summary of the range of effect estimates and our suggested central, low and high estimates. A low estimate of 0.5% per 10 ppb, 24-hour average ozone, corresponds to ~~the best~~ estimates from the NMMAPS study (using a one-week cumulative lag) and the ~~APEHA2~~APEAH2 European study, but is below most of the other central estimates. ~~A central estimates~~The central estimate of 1% per 10 ppb is very similar to the central estimate generated by WHO (Anderson et al., 2004), Levy et al. (2001), and Stieb (2003). Finally,

as a high estimate, we use 1.5% per 10 ppb, which reflects the central estimates of Thurston and Ito (using non-linear functions for temperature) and the summer-only estimates of Gryparis et al. (2004). ~~Bates (personal communication, 2005) suggested that these concentration response relationships may be underestimated.~~ Our range of estimates is applied to all age groups.

On the 1-hour scale, a 1% change per 10 ppb of 24-hour ozone is about 0.4% per 10 ppb change in 1-hour daily maximum ozone based on an assumed the ratio between 1-hour and 8-hour ozone of 1.33 and between 1-hour and 24-hour of 2.5 (Schwartz 1997). Specifically,

$$\frac{1\%}{10 \text{ ppb}24\text{hr}} = \frac{1\%}{10 \text{ ppb}24\text{hr}} \times \frac{1 \text{ ppb}24\text{hr}}{2.5 \text{ ppb}1\text{hr}} = \frac{0.40\%}{10 \text{ ppb}1\text{hr}}$$

Figure B-1: Percent Change in Mortality Associated with Ozone (per 10 ppb 24-hour average)



<u>Study #</u>	<u>Author</u>	<u># of studies</u>	<u>Comment</u>
1	Anderson (2004)	15	European
2	Anderson (2004)	20	Euro, corrected for possible publication bias
3	Thurston+Ito (2001)	7	<u>Studies using non-linear temperature</u>
4	Thurston+Ito (2001)	19	All studies
5	Stieb et al. (2003)	109	All studies
6	Bell et. al. (2004)	95	NMMAAPS, lag(01)
7	Bell et. al. (2004)	95	NMMAAPS, lag(06)
8	Levy et al. (2001)	4	Strict criteria
9	Levy et al. (2001)	15	Less strict criteria
10	Gryparis et al. (2004)	23	All year Europe
11	Gryparis et al. (2004)	23	Summer Europe

Hospital Admissions for Respiratory Diseases

Studies of a possible ozone-hospitalization relationship have been conducted for a number of locations in the United States, including California. These studies use a daily time-series design and focus on hospitalizations with a first-listed discharge diagnosis attributed to diseases of the circulatory system (ICD9-CM codes 390-459) or diseases associated with the respiratory system (ICD9-CM codes 460-519). Various age groups are also considered, which vary across studies. For ~~this~~our estimate, we ~~rely~~relied on the meta-analysis by Thurston and Ito (1999). These authors used a random effects model ~~using~~based on three studies from North America. The studies were Burnett et al. (1994), Thurston et al. (1994), and Burnett et al. (1997). The category of all respiratory admissions for all ages yielded an estimate of relative risk of 1.18 (95% CI = 1.10 – 1.26) per 100 ppb change in daily 1-hour maximum ozone. This category includes hospital admissions for asthma and bronchitis, so separate estimates of these outcomes are not necessary. The estimate converts to a 1.65% change in hospital admissions (95% CI = 0.95 – 2.31%) per 10 ppb change in 1-hour daily maximum ozone. This estimate was applied to all age groups. Additional studies of respiratory admissions for specific diseases or subpopulations provide additional support for the above relationship, but are not quantified to avoid double counting. For example, Anderson et al. (1997) reported a relative risk of 1.04 (95% CI = 1.02-1.07) for hospital admissions for COPD for all ages for a ~~50 $\mu\text{g}/\text{m}^3$~~ 50 $\mu\text{g}/\text{m}^3$ change in 24-hour ozone. This converts to ~~2.05%~~0.63% per 10 ppb change in 1-hour maximum ozone. Burnett et al. (2001) investigated respiratory hospitalizations in children under age 2, and reported a ~~relative risk of 1.348 (95% CI = 1.193 – 1.523), which converts to a 6.6%~~7.8% increase in hospital admissions per 10 ppb change in five-day moving average of 1-hour daily maximum ozone- ozone concentrations.

Emergency Room Visits for Asthma

Some studies have examined the relationship between air pollution and emergency room (ER) visits for pediatric asthma. Because most ER visits do not result in an admission to the hospital, we ~~treated~~evaluated hospital admissions and ER visits separately, taking into account ~~of~~ the fraction of ER patients that were admitted to the hospital. Our estimate is based on five studies which provide CR functions across the full range of ozone concentrations: Tolbert et al. (2000), Friedman et al. (2001), Jaffe et al. (2003), Romieu et al. (1995), and Stieb et al. (1996). Tolbert et al. (2000) report an association between pediatric emergency room visits (age < 16) for asthma and ozone in Atlanta during the summers of 1993-1995. The authors report a relative risk of 1.04 (95% CI = 1.008 – 1.074) per 20 ppb change in 8-hour ozone. Friedman et al. (2001) reported an association between daily counts for asthma in two pediatric emergency departments (age 1 to 16) and ozone in Atlanta during the summer of 1996. They report a RR of 1.2 (95% CI = 0.99 – 1.56) per 50 ppb change in 1-hour maximum ozone. This model included PM10 as a co-pollutant. Jaffe et al. (2003) reported an association between ozone and emergency room visits for asthma (ages 5 to 34) among Medicaid recipients in three cities in Ohio for the summer months from 1991- 1996. Estimates for the combined three cities indicate a RR of 1.03 (1.00 – 1.06) for a 10 ppb change in the

8-hour average of ozone. Romieu et al. (1995) reported results for emergency visits for asthma (age < 16) in Mexico City from January to June, 1990. A RR of 1.43 (95% CI= 1.24 – 1.66) was obtained for a 50 ppb change in 1-hour maximum ozone. Finally, Stieb et al. (1996) reported a beta of 0.0035 (95% CI = 0.00 –0.0070) for a 1 ppb change in 1 hour maximum ozone for ER visits for asthma in Saint John, New Brunswick, Canada.

Using an inverse variance weight for these five studies, we obtained a meta-analytic result of 2.4% per 10 ppb in daily 1-hour maximum ozone with a 95% CI = 1.46 to 3.34%. This estimate was applied over the entire range of ozone concentrations to children under 18. Several studies on ER visits for asthma report a non-linear response consistent with an effect threshold (see Section 8.3.3.2 and Figure 8-1, and Section 10.2.5). The threshold level appears to be somewhere between 0.075 and 0.110 ppm for a 1-hour average (or, using a ratio of 1.33, an 8-hour average of 0.056 to 0.084-ppm). This threshold may be due to lower power in detecting effects at low concentrations. In addition, the studies indicate some increased risks observed at below threshold concentrations. Regardless, if a zero slope (implying a threshold) is applied to the lower portion of the data, the concentration-response function for the remaining portion of the data must be larger than the slope for the entire data set. Below we use some of the available information on describe how to adjust the slope in order of the CR function to investigate the implications of imposing a threshold on the CR function.

School Absences

In addition to hospital admissions and ER visits, there is considerable scientific research that has reported significant relationships between elevated ozone levels and other morbidity effects. Controlled human studies have established relationships between ozone and symptoms such as cough, pain on deep inhalation, shortness of breath, and wheeze. In addition, epidemiological research has found relationships between ozone exposure and acute infectious diseases (e.g., bronchitis, and sinusitis) and a variety of “symptom-day” categories. Some “symptom-day” studies examine excess incidences of days with identified symptoms such as wheeze, cough, or other specific upper or lower respiratory symptoms. Other studies estimate relationships with a more general description of days with adverse health impacts, such as “respiratory restricted activity days” or work loss days. We selected a few endpoints that reflect some minor morbidity effects and carefully adjusted estimates to avoid double counting (e.g., adjusted minor restricted activity days by number of asthma-related emergency room visits).

One of these studies demonstrated that absence from school was associated with ozone concentrations in a study of 1,933 fourth grade students from 12 southern California communities participating in the Children’s Health Study (Gilliland et al. 2001). For illness-related absences, verified through telephone contact, further questions assessed whether the illness was respiratory or gastrointestinal, with respiratory including runny nose/sneeze, sore throat, cough, earache, wheezing, or asthma attack. Associations were observed between 8-hour average ozone and school absenteeism due to several different respiratory-related illnesses. Specifically, the authors report a 62.9% (95% CI = 18.4 -124.1%) change in new episodes of absences from all illnesses

associated with a 20 ppb change in 8-hour average ozone. This provides the basis for our quantitative estimate, which was applied to all schoolchildren aged 5-17.

On the 1-hour scale, 62.9% change per 20 ppb change of 8-hour ozone is about 21.2% per 10 ppb change in 1-hour daily maximum ozone using the assumed ratio between 1-hour and 8-hour ozone of 1.33 and between 1-hour and 24-hour of 2.5 (Schwartz 1997). Thus, we estimated:

$$\frac{62.9\%}{20 \text{ ppb}8\text{hr}} \times \frac{1 \text{ ppb}8\text{hr}}{1.33 \text{ ppb}1\text{hr}} = \frac{21.2\%}{10 \text{ ppb}8\text{hr}}$$

In calculating the change ~~in school loss days~~, in episodes of school loss, we assumed children did not attend school during weekends and holidays, that about 20% of students attended year-round schools, and adjusted the attendance rate for each month of the year. The baseline absence rate reported by Hall et al. (2003), based on a telephone survey of school districts, was applied.

To convert episodes of school loss into days, we estimated 1.265 days as the average duration of an illness-related school absence, the result of dividing the average daily school loss rate from BenMAP by the episodic absence rate from Gilliland et al. (2001).

Minor Restricted Activity Days

Ostro and Rothschild (1989) estimated the impact of PM_{2.5} on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for PM_{2.5}, two-week average ozone concentration has a highly variable but statistically significant association with MRADs but not with RRADs. MRADs are days where people reduced their activity, but did not miss work, and can therefore be viewed as relatively minor and transient symptom days.

For our MRAD estimate, we initially reanalyzed on an individual year basis each of the six years of data from Ostro and Rothschild (1989) using their multi-pollutant model that included PM_{2.5}. We then used an inverse variance-weighted meta-analysis to combine the six individual year results. This resulted in an estimate of a 0.112% change (95%CI 0.046 – 0.178%) per $\mu\text{g}/\text{m}^3$ of 1-hour maximum ozone. Conversion to ppb yielded an effect estimate of 2.24% change (95%CI = 0.92 – 3.56%) per 10 ppb change in 1-hour maximum ozone concentration. This estimate was applied to all adults above age 18.

Sensitivity Analysis~~Analyses~~

~~Several~~We also performed several additional analyses ~~were run to indicate~~to evaluate the sensitivity of the results to our assumptions. In our first analysis, we considered two alternative ways to characterize ozone exposure and population. First, we ~~re-estimated the assignment of exposure for each of the county residents~~estimated ozone concentration at the census-tract level. Specifically, we used population data from the

year 2000 census and determined the population centroid for every census tract in the state. The assigned ozone concentration at each centroid was determined using the inverse square distance weighted interpolation of the ozone concentrations observed at the monitors within a 50-kilometer radius of the centroid. This value was then assigned to each resident in the census tract. Second, we averaged the observed concentrations at the monitors within each county and assigned the county average concentration to the entire county population.

As a second sensitivity analysis, we imposed a threshold on all of the CR functions and accompanied this assumption with a re-estimated, higher CR function for the remaining data. Most of existing studies assume a non-threshold model, either linear or logistic, over the entire range of ozone concentrations. If one were to impose a threshold or no-effects level over the lower range of the data, the remaining slope estimate would have to increase to fit the remaining observations. Unfortunately, there is only limited data to suggest the magnitude of the increase in the slope. Specifically, several of the studies of emergency room visits for asthma estimated a slope for both the full range and for an upper portion of the data. Therefore, as a sensitivity analysis, we attempted to draw inference about how the slope would increase, drawing on both the direct and indirect evidence. evidence, described below.

Stieb et al. (1996) examined the effects of ozone on emergency department (ED) visits for asthma in Saint John, Canada. In the basic analysis, they report a beta coefficient for the full population of 0.0035 for a change of 1 ppb in 1-hr maximum average of ozone, using a lag of 0 and 1 day. When a dichotomous model was developed to examine the effect of concentrations above versus below 75 ppb, the beta increased to 0.45. Based on graphical and descriptive data presented in the paper, the mean concentrations above and below 75 ppb were assumed to be 95 and 35 ppb, a difference of 60. This results in a beta of 0.0076 and a ratio of the slope using the highest quartile, where effects are observed, versus the slope for the full range of data of approximately 2.16.

Tolbert et al. (2000) examined the effects of ozone on pediatric ED visits in Atlanta. In the basic analysis, a relative risk (RR) of 1.042 was reported for a 20 ppb change in the 8-hour maximum daily ozone. This relates to a beta of 0.00206 ($= \ln(1.042)/20$) ($\beta = \ln(1.042)/20$) or converting to a 1-hr maximum using a ratio of 1.33, a beta of 0.0015. The authors also report an RR of 1.23 for concentrations above 100 ppb range versus low concentrations (< 50 ppb) of ozone. Assuming the mean for concentrations above 100 ppb was 105 ppb and the mean concentrations for values below 50 ppb was 40 ppb, the resulting beta coefficient is 0.00318 ($= \beta = \ln(1.23)/(105-40)$) for an 8-hour change in ozone or 0.0024 for a 1-hour change which is 1.6 times the slope using all of the ozone data.

Finally, Romieu et al. (1995) studied ozone and pediatric ED visits in Mexico City. The authors report an RR of 1.43 for a 50 ppb change in 1-hour maximum ozone, using a one-day lag. This relates to a beta of 0.00715 for a 1-hour change in ozone. However, when they examined multiple days with high peaks greater than 110 ppb, the RR increased to 1.68 for a cumulative lag of 0 and 1 and to a RR of 2.33 for a cumulative

lag of 1 and 2 days. Based on personal communication with the authors, the mean concentration for days below 110 ppb was 67 ppb versus a mean for days above 110 ppb of 127 ppb. Thus, the resultant betas become 0.0086 ($=\ln(1.68)/60$) and 0.0141 ($=\ln(2.33)/60$), $(\beta = \ln(1.68)/60)$ and 0.0141 ($\beta = \ln(2.33)/60$), respectively. This suggests a ratio of the slope based on data above a threshold relative to the slope for the full data of between 1.21 and 1.97.

Overall, the empirical evidence confirms the logical expectation that the slope for only the upper end of the distribution of concentrations will be much larger than that for the entire distribution. The existing evidence, however, involves different cutpoints for the higher end and different averaging times, which clearly will affect the ultimate slope. However, given these results, it appears that for a sensitivity analysis, an increase of 40% in the slope above a threshold of 60 ppb (8-hour average) is a reasonable approximation. We also examined a presumed threshold of 50 ppb (8-hour average) using a slope increase of 100%. As additional sensitivity analysis, we determined, assuming a 40% increase in the slope in the upper segment of the data, what the threshold concentration would have to be to generate effects similar to those from a non-threshold model. Finally, we determined what the increase in the slope would have to be in the upper segment, given a threshold of 70 ppb 8-hour average, to generate effects similar to a non-threshold model.

Note, however, that these presumed threshold values are well within the range of concentrations observed in most, if not all, of the original epidemiologic studies. In fact, these values are often in the upper end of the range of values, rendering this assumption somewhat unlikely. Nevertheless, it is of interest to examine the effects of such an assumption.

Health Effects Results

Table B-2B-3 presents the estimated statewide annual health benefits from reducing the impacts of current (2001-2003) levels of ozone to achieve the 1-hour standard of 0.09 ozone, compared to attainment of the Federal 8-hour standard of 0.08 ppm. For most of the endpoints, the 95% confidence intervals around each central estimate reflects the uncertainty associated reflect the uncertainty associated with the beta coefficient derived from the epidemiological studies used in the calculation. As discussed above, for mortality, the uncertainty was based on the range of estimates generated from several meta-analyses with the beta coefficient derived from the epidemiological studies used in the calculation. For mortality, the uncertainty was based on the range of estimates generated from several meta-analyses. For example, the results indicate that the impact of not attaining the federal 8-hour standard of 0.08 full attainment of the proposed 1-hour standard would result in 580 fewer ppm statewide is 360 cases of premature mortality (probable range = 290 – 870), 3,800 fewer 180 – 550), 2,400 hospital admissions (95% CI = 2,200 – 5,400) and 3,300,000 fewer days of 1,400 – 3,500), 380 emergency room visits for asthma (95% CI = 230-530), 2.5 million days of illness-related school loss (95% CI = 690,000 – 4,200,000), and 1.8 million (95% CI

430,000—6,100,000)= 730,000 – 2,800,000) minor restricted activity days per year. Since the results for premature mortality due to short-term exposures were derived from examining the evidence from several papers, rather than combining the results into a confidence interval, we use the terminology of a “probable range,” rather than 95% confidence interval.

Table B-4 presents the estimated annual statewide health impacts of current (2001-2003) levels of ozone, compared to attainment of the State 1-hour standard of 0.09 ppm. Again, for most of the endpoints, the 95% confidence intervals around each central estimate reflect the uncertainty associated with the beta coefficient derived from the epidemiological studies used in the calculation. As discussed above, for mortality, the uncertainty was based on the range of estimates generated from several meta-analyses. The results indicate that the current impact of ozone compared to attainment of the proposed 1-hour standard of 0.09 ppm statewide is 540 cases of premature mortality (probable range = 270 – 810), 3,600 hospital admissions (95% CI = 2,000 – 5,000), 560 emergency room visits for asthma (95% CI = 340-790), 3.8 million days of illness-related school loss (95% CI = 1,040,000 – 6,900,000), and 2.6 million (95% CI = 1,100,000 – 4,200,000) minor restricted activity days per year.

Similar to Table B-2, Table B-3 presents statewide results from achieving B-4, Table B-5 presents the impact of current ozone levels compared to attainment of the proposed State 8-hour standard of 0.070 ppm. Generally speaking, the health benefits from impacts of not attaining the 4-hour 8-hour standard are greater than those associated with not attaining the 8-hour standard. Since 1-hour and 8-hour standard. For example, the results indicate that the impact of current ozone levels, compared to full attainment of the proposed 8-hour standard statewide is 630 cases of premature mortality (probable range = 310 – 950), 4,200 hospital admissions (95% CI = 2,400 – 5,800), 660 emergency room visits for asthma (95% CI = 400-920), 4.7 million days of illness-related school loss (95% CI = 1,200,000 – 8,600,000), and 3.1 million (95% CI = 1,300,000 – 5,000,000) minor restricted activity days per year.

concentrations are highly correlated, it is not appropriate to add the estimated benefits from Tables B-2 and B-3 together. Tables B-4 and B-5 Tables B-6, B-7 and B-8 present estimates of the annual health benefits of impacts of not attaining the federal 8-hour, and the proposed State 1-hour and 8-hour standards, respectively, by air basin.

Table B-6 presents estimates of the annual health benefits of attaining either of the standards, whichever provides the greatest amount of control.

Incremental Impacts Discussion

The differences between the results in Tables B-3 (federal 8-hour standard), B-4 (State 1-hour standard) and B-5 (State 8-hour standard) are the “incremental” impacts of not attaining the State 1-hour and 8-hour standards, compared to the federal 8-hour ozone standard, respectively. However, it is more reasonable to consider attainment of the two state standards together, compared to current ozone levels, since it is unlikely that control strategies will be geared to first attain one standard and then the other. Therefore, the impacts are not separable and should not be treated as such. Nonetheless, the following discussion can be helpful in understanding the incremental

impacts.

Comparing Tables B-3 and B-5, the current impact of not attaining the federal 8-hour standard is about 360 premature deaths, with an additional 270 deaths associated with not attaining the proposed State 8-hour standard, making the total estimated impact of not attaining both standards 630 premature deaths. Similarly, statewide about 2,400 hospital admissions annually are associated with nonattainment of the federal 8-hour standard, and an additional 1,800 hospitalizations are due to nonattainment of the proposed State 8-hour standard, making the total estimated impact of not attaining both the federal 8-hour and State 8-hour standards 4,200 hospital admissions. For ER visits, we estimate that 380 cases are associated with nonattainment of the federal 8-hour standard, and an additional 280 cases with nonattainment of the proposed State 8-hour standard, for a total of 660 cases associated annually with nonattainment of the proposed 8-hour standard. For school loss, 2.5 million days are estimated to be associated with the nonattainment of the federal 8-hour standard, and an additional 2.2 million days, a total of 4.7 million days, associated with nonattainment of the proposed State 8-hour standard. Lastly, we estimated that the impact of current ozone levels is about 1.8 million minor restricted activity days due to nonattainment of the federal 8-hour standard, and an additional 1.3 million, for a total of 3.1 million days, associated with nonattainment of the proposed State 8-hour standard.

A similar examination of Tables B-4 and B-5 reveals the incremental impacts of not attaining the proposed State 8-hour standard compared to the State 1-hour standard. The current impact of not attaining the State 1-hour standard is about 540 premature deaths, with an additional 90 deaths associated with not attaining the proposed 8-hour standard, making the total estimated impact of not attaining both the State 1-hour and 8-hour standards 630 premature deaths. Similarly, statewide about 3,600 hospital admissions annually are associated with nonattainment of the State 1-hour standard, and an additional 600 hospitalizations are due to nonattainment of the proposed 8-hour standard, making the total impact of not attaining both the State 1-hour and 8-hour standards 4,200 hospital admissions. For ER visits, we estimate that 560 cases are associated with nonattainment of the 1-hour standard, and an additional 100 cases with nonattainment of the proposed 8-hour standard, for a total of 660 cases associated annually with nonattainment of the proposed 8-hour standard. For school loss, 3.8 million days are estimated to be associated with the nonattainment of the State 1-hour standard, and an additional 900,000 days, a total of 4.7 million days, associated with nonattainment of the proposed 8-hour standard. Lastly, we estimated that the impact of current ozone levels is about 2.6 million minor restricted activity days due to nonattainment of the 1-hour standard, and an additional 500,000, for a total of 3.1 million days, associated with nonattainment of the proposed 8-hour standard.

Figure B-2: Incremental Impacts of Ozone Exposures Compared to Attainment of Ozone Standards for Premature Deaths and School Absences (Annual statewide cases avoided with attainment of ozone standards. Details are given in the text.)

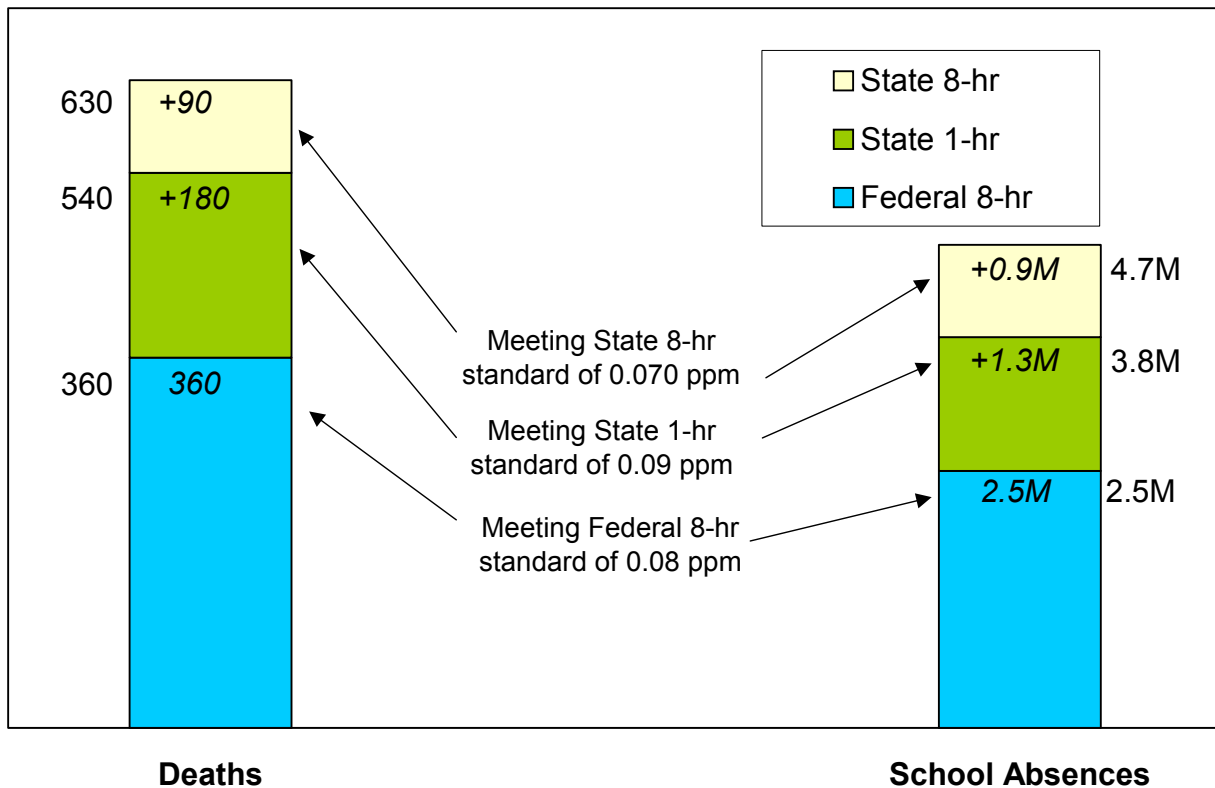


Figure B-2 summarizes the results of the above incremental impacts discussion for two endpoints: premature death and school absences. The numbers within each stacked bar represent the incremental impacts analysis. For premature death, attainment of the federal 8-hour standard would avoid 360 deaths annually in California. An additional 180 deaths, as indicated inside the stacked bar, would be avoided with attainment of the State 1-hour standard, and another 90 deaths would be avoided with attainment of the proposed State 8-hour standard, for a total of 630 deaths avoided annually at full attainment, as indicated on the side next to the bar. For illness-related school absences, the incremental analysis estimates annual avoidance of about 2.5 million school absences with attainment of the federal 8-hour standard, an additional 1.3 million avoided school absences with attainment of the current State 1-hour standard, and another 900,000 (indicated by +0.9M inside the stacked bar) with attainment of the proposed 8-hour standard, for a total of 4.7 million fewer school absences.

Sensitivity Analysis Results

We performed several sensitivity analyses to investigate two key assumptions in our analysis. Here, we discuss results on both the State 8-hour standard and the 1-hour

standard. Our first sensitivity analysis examined the implications of an alternative exposure assessments. Specifically, in the first reassessment, we interpolated concentrations for each census tract using nearby monitoring data. The exposure of the population within each census tract was determined using the inverse square distance weighted interpolation of the ozone concentrations observed at the monitors within a 50-kilometer radius of the centroid. The results for mortality are similar to those obtained using the base-case approach. Attaining the 1-hour ozone standard statewide. Not attaining the proposed California 8-hour ozone standard using census-tract interpolations led to 570 an estimated current impact of 610 deaths compared to 580 deaths avoided 630 deaths using our base-case approach. Not attaining the State 1-hour ozone standard using census-tract interpolations lead to an estimated current impact of 530 deaths compared to 540 deaths using our base-case approach. In the second assessment using reassessment of exposure, we assigned residents to their county-wide average ozone concentrations, similar results, about 630 deaths, are obtained concentrations. Similar impacts of about 680 deaths was obtained for the proposed 8-hour standard and 580 deaths was obtained for the State 1-hour standard.

In our second sensitivity analysis, we examined the implications of assuming alternative threshold models. If we assumed a threshold of 60 ppb and a 40% increase of the slope of the remaining higher concentrations, it resulted in about a 10% to 14% decrease in estimated health outcomes impacts. For example, the estimated mortality impact would decrease from 580 to 520 630 to 540 comparing current ozone levels with attainment of the proposed 8-hour standard, and from 540 to 490 comparing current ozone levels with attainment of the 1-hour standard. The breakeven point associated with a 40% increase in the slope would be about 55 ppb for both the 1-hour and 8-hour standards. In other words, if the slope at the higher end of exposure was 40% greater than the slope for the full range of exposures, we would obtain the same number of cases impact estimate as in the base case, if the higher slope estimate was applied to concentrations greater than 55 ppb. For an assumed threshold of 70 ppb, the slope would have to increase by about 140% to get the same number of cases as in 150% for the 8-hour standard or about 130% for the 1-hour standard to obtain about the same impact as in the base case, non-threshold model. If we assumed a threshold at 50 ppb with a 100% increase in the remaining slope, the estimate number of case estimated impact would increase by about 70% for the 8-hour standard and about 75% for the 1-hour standard.

Uncertainties and Limitations

There are a number of uncertainties involved in quantitatively estimating the impacts on health benefits associated with reductions in outdoor ozone air pollution. Over time, some of these will be reduced as new research is conducted. However, some uncertainty will remain in any estimate. Below, we briefly discuss some of the major uncertainties and limitations of these estimated health benefits impacts. These issues are discussed in more detail in Chapter 10 (also see Levy et al., 2001; Thurston and Ito, 1999).

Developing concentration-response functions

A primary uncertainty is the choice of the specific studies and concentration-response

functions used in ~~this~~the quantification. Several challenges and unresolved issues present themselves with respect to designing and interpreting time-series studies of ~~ozone-related~~ozone-related health effects. The principal challenge facing the analyst in ~~the daily time series context~~ is to remove bias due to confounding by short-term temporal factors operating over time scales from days to seasons. The correlation of ozone with these confounding terms tends to be higher than that for PM or other gaseous pollutants. Thus, model specifications that may be appropriate for PM, the primary focus of much of the available literature, may not necessarily be adequate for ozone. Few studies to date have thoroughly investigated these potential effects with reference to ozone, introducing an element of uncertainty into ~~the health benefits~~ analysis.

Of particular importance is the strong seasonal cycle for ozone, high in summer and low in winter, ~~which is~~ opposite to the usual cycle in daily mortality and morbidity, which is typically high in winter and low in summer. Inadequate control for seasonal patterns in time series analyses leads to biased effect estimates. In the case of ozone, inadequate seasonal pattern control generally yields statistically significant inverse associations between ozone and health outcomes. In contrast, for winter-peaking pollutants such as CO and NO₂, the bias is toward overly positive effect estimates. Also, temporal cycles in daily hospital admissions or emergency room visits are often considerably more episodic and variable than is usually the case for daily mortality. As a result, smoothing functions that have been developed and tuned for analyses of daily mortality data may not work as well at removing cyclic patterns from morbidity analyses.

Potential confounding by daily variations in co-pollutants and weather is another analytical issue to be considered. With respect to co-pollutants, daily variations in ozone tends not to correlate highly with most other criteria pollutants (e.g., CO, NO₂, SO₂, PM₁₀), but may be more correlated with secondary fine particulate matter (e.g., PM_{2.5}) measured during the summer months. Assessing the independent health effects of two pollutants that are somewhat correlated over time is problematic. However, much can be learned from the classic approach of first estimating the effects of each pollutant individually, and then estimating their effects in a two-pollutant model. For this reason, we have emphasized use of studies that have also controlled for PM.

The choice of the studies and concentration-response functions used for health impact assessment can affect the ~~benefits~~impact estimates. Because of differences, likely related to study location, subject population, study size and duration, and analytical methods, effect estimates differ somewhat between studies. We have addressed this issue by emphasizing meta-analyses and multi-city studies, and also by presenting estimates derived from several studies.

To a substantial degree, the growing literature on acute ozone effects is an artifact of interest in studying acute PM effects. For example, of the 84 time-series mortality studies published between 1995 and mid-2004, 35 studies examined PM but not ozone; 47 studies examined both PM and ozone; and only 2 studies examined ozone but not PM. In many of the multi-pollutant studies, ozone is treated primarily as a potential

confounder of the PM effects under study. As a result, many of these studies lack specific hypotheses regarding mortality effects of ozone, and fail to provide the range and depth of analyses, including sensitivity analyses, that would be most useful in judging whether ozone is an independent risk factor for acute mortality. This is in contrast to morbidity studies where hypotheses regarding ozone effects on respiratory symptoms, lung function, hospitalization and ER visits, etc. have been studied with ozone treated as a key pollutant. Fortunately, studies of short-term exposure and mortality have been replicated in many cities throughout the world, under a wide range of exposure conditions, climates and covarying pollutants. As a result, the evidence of an effect of ozone on premature mortality is compelling. Nevertheless, uncertainty remains about the actual magnitude of the effect and the appropriate confidence interval.

Thresholds

A second major uncertainty relates to the general shape of the concentration-response function and the existence of a threshold. This is discussed in detail earlier, with the conclusion that there is little evidence for a threshold. An important consideration in determining if a safe level of ozone can be identified is whether the CR relationship is linear across the full concentration range or instead shows evidence of a threshold. Among the ozone epidemiology literature, only a few studies of hospital admissions and emergency room visits have examined the shape of the CR function. These studies also provide the only epidemiologic investigations into whether or not there is an ozone effect threshold. Since only a few studies have investigated whether there is an effect threshold, and the few studies available do not cover all endpoints, the epidemiologic literature does not provide a basis for concluding whether or not there is a population level effect threshold. However, many of the available studies were conducted at fairly low concentrations of ambient ozone, so we are never extrapolating beyond the range of the studies. Therefore, for this analysis, we have assumed that there is no threshold for ozone effects and we estimated ~~benefits~~impacts down to an assumed background concentration of 0.04 ppm. To the extent that there may not be health effects below the proposed ozone standard, the analysis may overestimate the impacts of ~~reducing~~ambient ozone. However, we also conducted a sensitivity analysis ~~with an assumption of~~assuming several different possible thresholds. In doing so, we also adjusted the slope of the upper segment of the ozone concentrations to conform with the implications of a threshold model. If we had assumed zero ~~benefits~~impacts accrue below the proposed standards and provided no adjustment to the concentration-response functions, our estimates would be reduced by about 80%.

A related issue is that limited data suggest that ozone effects may be seasonal. While analysis of year round data suggests positive associations between a number of endpoints and ozone exposure, some data sets that have been analyzed seasonally report positive RR estimates for summer and negative RR estimates for winter. The cause of this phenomenon has not been adequately investigated, but may be related to thresholds, differences in personal exposure between seasons, or to co-pollutant exposures. In light of this uncertainty, this analysis used year-round effect ~~estimates~~.

~~In~~estimates, ~~addition,~~although the relatively long, warm season in California may make the summer estimates more relevant than those of the winter season.

Assumptions about rollback

A further uncertainty concerns the process used to design and implement strategies for controlling ozone-producing compounds. Such control strategies have been designed with the objective of reducing ozone episodes during worst-case meteorological conditions. In addition, basin-wide strategies have focused on the ozone concentrations at the highest (design) site in each basin. How these strategies would affect other sites during dissimilar episodes cannot be answered with certainty. Site-by-site analyses almost always have found that trends for multiple sites within a basin are very similar to each other. Similarly, monthly trends within a basin have usually proved to be similar, while the prevalence of different episode types may be markedly different for different months during the overall ozone season. (See trend analysis in the Supplement).

Unquantified adverse effects

An additional limitation in this analysis is the inability to quantify all possible ~~health benefits that could be~~impacts that are associated with current ozone concentrations, achieving the proposed ozone standards, since estimates are provided for only a subset of possible adverse outcomes. For example, estimates of the effects of ozone on asthma exacerbation, asthma exacerbation, induction, respiratory symptoms, airway inflammation, and acute and long-term changes in lung function are not presented. Although there is some evidence for such effects, the available data were either too inconsistent or sparse to justify quantification of possible ~~benefits of achieving the proposed ozone standards,~~impacts of not achieving the proposed ozone standards, or the evidence comes from controlled exposure studies that can not be used to make population level effects estimates. To the extent that certain important health outcomes were excluded, we may have underestimated the health ~~benefits~~impacts of the proposed standards.

Baseline rates of mortality and morbidity

There is also uncertainty in the baseline rates for the investigated health outcomes in the studied population. Often, one must assume a baseline incidence level for the city or country of interest. In addition, incidence can change over time as health habits, income and other factors change.

Exposure assessment

There are likely uncertainties in the statewide exposure assessment, and in whether the existing monitoring network provides representative estimates of exposure for the general population. We have attempted to reproduce the same relationship between monitor readings and exposure as in the original epidemiological studies. Most of these studies use population-oriented, background, fixed site monitors, often aggregated to

the county level. The available epidemiological studies have used multiple pollutant averaging times, and we have proposed conversion ratios for 1-hour to 8-hour and 24-hour ozone concentrations based on national estimates. A preliminary examination of the California monitoring data indicates that the ratios are similar to those found in the highly populated areas of the State. However, uncertainty is added to the estimated benefits of attainment of impacts of not attaining the proposed standards to the extent the converted concentration bases differ from monitored concentrations.

Summary

The purpose of this appendix is to provide quantitative estimates of some of the health benefits that may accrue from impacts of current levels of ozone, compared to those that would occur under a hypothetical control strategy that brings the State into attainment with the proposed ozone standards. This assessment should not be regarded as exhaustive, since we have provided estimates only for a selection of the most plausible effects for which there were high quality studies from which to derive CR functions: subset of health effects endpoints. However, the results presented support the conclusion that significant public health benefits would result from statewide impacts are associated with current ozone concentrations in California that would not occur with attainment of the proposed ambient air quality standards for ozone. ozone standards. It is estimated that attainment of the proposed ozone standards throughout California would avoid a significant significantly reduce the number of ozone-related adverse health effects each year. The higher central estimate between the values calculated for 1-hour and 8-hour averaging times is given below.

Specifically, we estimate that the current impact of exposures above the proposed standard is:

- 580 (290 – 870, 630 (310 – 950, probable range) premature deaths for all ages.
- 3,800 (2,200 – 5,400, 4,200 (2,400 – 5,800, 95% confidence interval (CI)) hospitalizations due to respiratory diseases for all ages.
- 600 (360 – 850, 660 (400 – 920, 95% CI) emergency room visits for asthma for children under 18 years of age.
- 3.3 million (430,000 – 6,100,000, 95% CI) 4.7 million (1,200,000 – 8,600,000, 95% CI) illness-related school absences for children 5 to 17 years of age.
- 2.83.1 million (1.2(1.3 million – 4.65.0 million, 95% CI) minor restricted activity days for adults above 18 years of age.

These estimates are based on attainment of the proposed State 8-hour standard, as it is the more stringent of the two (1-hour and 8-hour) proposed standards. The reader is cautioned that since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated benefits from Tables B-2 and B-3 together. impacts from Tables B-3, B-4 and B-5 together. Instead, the estimates above represent the total impacts estimated to accrue from attaining both the proposed 8-hour and 1-hour standards. For a discussion of the incremental impacts of current ozone levels

compared to attainment of the proposed 8-hour standard over the State 1-hour standard and the federal 8-hour standard, see page B-19.

As noted above, there are several important assumptions and uncertainties in this analysis. Some concern the study design, the statistical modeling methodologies used, and the selection of studies from which the CR functions are derived. Few studies have investigated the shape of the CR function, or whether there is a population response threshold for health endpoints other than emergency room visits for asthma. Further, but likely small, uncertainty is added by assumptions in the statewide exposure assessment. Nonetheless, when new evidence on mortality from short-term exposures to ozone is published from the recent meta-analyses sponsored by the US EPA, we will update our estimates and use the census-tract interpolation to characterize ambient ozone exposure. It should also be noted that since several health effects related to acute exposure, and ~~effects of chronic ozone exposure, exposures~~ are not included in the estimates, the health ~~benefits~~impacts associated with lowering ozone exposure are likely underestimated.

Table B-1:B-2: Summary of Meta-Analyses Linking Daily Ozone to Mortality (for 10 ppb change in 24-hour average ozone)

Study Number	Author	# of studies	% Change in Mortality (95% CI)	comment
1	Anderson (2004)	15	1.13 (0.38 - 1.51)	European studies only
2	Anderson (2004)	20	0.75 (0.19 – 1.32)	European studies corrected for possible publication bias
3	Thurston+Ito (2001)	7	1.37 (0.78 – 1.96)	Earlier studies using non-linear specification for temperature
4	Thurston+Ito (2001)	19	0.89 (0.56 – 1.22)	All earlier studies
5	Stieb et al. (2003)	109	1.12 (0.32 – 1.92)	Meta-analysis including single and multi-pollutant models
6	Bell et. al. (2004)	95	0.25 (0.12 – 0.39)	NMMAPS, using lag(01)
7	Bell et. al. (2004)	95	0.52 (0.27 – 0.77)	NMMAPS,lag(06)
8	Levy et al. (2001)	4	0.98 (0.59 – 1.38)	Using relatively stringent inclusion criteria
9	Levy et al. (2001)	15	0.80 (0.60 – 1.00)	Using less stringent inclusion criteria
<u>10</u>	<u>Gryparis et al. (2004)</u>	<u>23</u>	<u>0.5 (-0.38 – 1.30)</u>	<u>APEHA2/APEAH2 studies in Europe, all year</u>
<u>11</u>	<u>Gryparis et al. (2004)</u>	<u>23</u>	<u>1.65 (0.85 – 2.60)</u>	<u>APEHA2/APEAH2 studies in Europe, summer only</u>

Table B-2:B-3: California Annual Health Benefits from Attaining a 1-hour Impacts of Current Ozone Concentrations Compared to the Federal 8-hour Ozone Standard of 0.090.08 ppm*

<u>Health Endpoint</u>	<u>Population</u>	<u>Estimated Beta (% per 10 ppb) (95% Confidence Interval)-Estimated Beta** (% per 10 ppb 1-hour ozone) (95% Confidence Interval)</u>	<u>Avoided Incidence (cases/year) Mean 95% Confidence Interval Incidence (cases/year) (95% Confidence Interval)</u>
<u>Premature Mortality due to Short-term Exposures</u>	<u>All ages</u>	<u>0.0040 (0.0020 - 0.0060) **</u>	<u>580 (290 – 870) ** 360 (180 – 550) ***</u>
<u>Hospital Admissions for Respiratory Diseases</u>	<u>All ages</u>	<u>0.0164 (0.0095 - 0.0228)</u>	<u>3,800 (2,200 – 5,400) 2,400 (1,400 – 3,500)</u>
<u>Emergency Room Visits for Asthma</u>	<u>Age < 18</u>	<u>0.0237 (0.01446 – 0.0329)</u>	<u>600 (360 – 850)-380 (230 – 530)</u>
<u>School Loss Days</u>	<u>Age 5-17</u>	<u>0.2123 (0.0334 – 0.3295) 0.2123 (0.06672 – 0.3295)</u>	<u>3,300,000 (430,000 – 6,100,000) 2,500,000 (690,000 – 4,200,000)</u>
<u>Minor Restricted Activity Days</u>	<u>Age > 18</u>	<u>0.0222 (0.0092 - 0.0350)</u>	<u>2,800,000 (1,200,000 – 4,600,000) 1,800,000 (730,000 – 2,800,000)</u>

*Base period 2001-2003. Since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated impacts from Tables B-3, B-4 and B-5 together. Due to rounding conventions in the process of determining attainment of the federal 8-hour standard, the concentration of 0.084 ppm was used since this is the highest value considered in attainment.

**As discussed in detail in the text, the evaluation of impacts of not attaining the 8-hour standard was based on the equivalent 1-hour concentration. Therefore, the beta coefficients here are in 1-hour scale.

benefits from Tables B-2 and B-3 together. ~~**Results~~***Results for premature mortality represent a probable range of likely values rather than a 95% confidence interval since the coefficients were derived from examining the evidence from several studies separately rather than combining their results in a formal meta-analysis.

Table B-3:B-4: California Annual Health Benefits from Attaining an 8-hour Impacts of Current Ozone Concentrations Compared to the State 1-hour Ozone Standard of 0.0700.09 ppm*

<u>Health Endpoint</u>	<u>Population</u>	<u>Estimated Beta (% per 10 ppb) (95% Confidence Interval)-Estimated Beta (% per 10 ppb 1-hour ozone) (95% Confidence Interval)</u>	<u>Avoided Incidence (cases/year) Mean (95% Confidence Interval)-Incidence (cases/year) (95% Confidence Interval)</u>
Premature Mortality due to Short-term Exposures	All ages	0.0053 (0.0027 – 0.0079)** 0.0040 (0.0020 - 0.0060)	540 (270 – 810) **
Hospital Admissions for Respiratory Diseases	All ages	0.0218 (0.0126 – 0.0302) 0.0164 (0.0095 - 0.0228)	3,600 (2,000 – 5,000)
Emergency Room Visits for Asthma	Age < 18	0.0314 (0.0192 – 0.0434) 0.0237 (0.01446 – 0.0329)	570 (340 – 800) 560 (340 – 790)
School Loss Days	Age 5-17	0.2440 (0.0844 – 0.4034) 0.2123 (0.06672 – 0.3295)	2,600,000 (760,000 – 5,200,000) 3,800,000 (1,040,000 – 6,900,000)
Minor Restricted Activity Days	Age > 18	0.0294 (0.0121 – 0.0462) 0.0222 (0.0092 - 0.0350)	2,600,000 (1,100,000 – 4,200,000)

*Base period 2001-2003. Since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated impacts from Tables B-3, B-4 and B-5 together. Due to rounding conventions in the process of determining attainment of the 1-hour standard, the concentration of 0.094 benefits from Tables B-2 and B-3 together. ppm was used since this is the highest value considered in attainment.

**Results for premature mortality represent a probable range of likely values rather than a 95% confidence interval since the coefficients were derived from examining the evidence from several studies separately rather than combining their results. results in a formal meta-analysis.

Table B-5: California Annual Health Impacts of Current Ozone Concentrations Compared to the State 8-hour Ozone Standard of 0.070 ppm*

<u>Health Endpoint</u>	<u>Population</u>	<u>Estimated Beta** (% per 10 ppb 1-hour ozone) (95% Confidence Interval)</u>	<u>Incidence (cases/year) (95% Confidence Interval)</u>
<u>Premature Mortality due to Short-term Exposures</u>	<u>All ages</u>	<u>0.0040 (0.0020 - 0.0060)</u>	<u>630 (310 – 950) ***</u>
<u>Hospital Admissions for Respiratory Diseases</u>	<u>All ages</u>	<u>0.0164 (0.0095 - 0.0228)</u>	<u>4,200 (2,400 – 5,800)</u>
<u>Emergency Room Visits for Asthma</u>	<u>Age < 18</u>	<u>0.0237 (0.01446 – 0.0329)</u>	<u>660 (400 – 920)</u>
<u>School Loss Days</u>	<u>Age 5-17</u>	<u>0.2123 (0.06672 – 0.3295)</u>	<u>4,700,000 (1,200,000 – 8,600,000)</u>
<u>Minor Restricted Activity Days</u>	<u>Age > 18</u>	<u>0.0222 (0.0092 - 0.0350)</u>	<u>3,100,000 (1,300,000 – 5,000,000)</u>

*Base period 2001-2003. Since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated impacts from Tables B-3, B-4 and B-5 together.

**As discussed in detail in the text, the evaluation of impacts of not attaining the 8-hour standard was based on the equivalent 1-hour concentration. Therefore, the beta coefficients here are in 1-hour scale.

***Results for premature mortality represent a probable range of likely values rather than a 95% confidence interval since the coefficients were derived from examining the evidence from several studies separately rather than combining their results in a formal meta analysis.

Table B-4: Annual Health Benefits from Attaining 1-hour Ozone Standard of Air Basin of 0.09 ppm **Table B-6: Annual Health Impacts of Current Ozone Concentrations Compared to the Federal 8-hour Ozone Standard of 0.08 ppm by Air Basin (cases/year).**

<u>Air Basin</u>	<u>Mortality</u>	<u>Hospital Admissions</u>	<u>Emergency Room Visits</u>	<u>School Absences</u>	<u>Minor Restricted Activity Days</u>
<u>Great Basin Valley</u>	<1	<1	<1	340 <u>280</u>	370 <u>250</u>
<u>Lake County</u>	0	0	0	0	0
<u>Lake Tahoe</u>	<1	3 <1	<1	2,500 <u>450</u>	2,400- <u>360</u>
<u>Mountain Counties</u>	40 <u>7</u>	52 <u>36</u>	7 <u>5</u>	40,000 <u>33,000</u>	41,000 <u>29,000</u>
<u>Mojave Desert</u>	43 <u>26</u>	300 <u>180</u>	50 <u>29</u>	280,000 <u>190,000</u>	220,000 <u>130,000</u>
<u>North Coast</u>	<4 <u>0</u>	<4 <u>0</u>	<4 <u>0</u>	370 <u>0</u>	330 <u>0</u>
<u>North Central Coast</u>	4 <u>0</u>	40 <u>0</u>	2 <u>0</u>	9,000- <u>0</u>	7,700- <u>0</u>
<u>Northeast Plateau</u>	0	0	0	0	0
<u>South Coast</u>	300 <u>220</u>	2,100- <u>1,500</u>	330 <u>230</u>	1,700,000 <u>1,400,000</u>	1,600,000 <u>1,100,000</u>
<u>South Central Coast</u>	46 <u>7</u>	440 <u>48</u>	46 <u>7</u>	97,000 <u>50,000</u>	83,000 <u>36,000</u>
<u>San Diego</u>	24 <u>11</u>	460 <u>71</u>	22 <u>10</u>	120,000 <u>67,000</u>	120,000 <u>55,000</u>
<u>San Francisco Bay</u>	23 <u>1</u>	450 <u>5</u>	21 <u>1</u>	400,000 <u>3,700</u>	420,000 <u>3,900</u>
<u>San Joaquin Valley</u>	95 <u>62</u>	640 <u>400</u>	110 <u>70</u>	650,000 <u>480,000</u>	440,000 <u>280,000</u>
<u>Salton Sea</u>	20 <u>15</u>	420- <u>86</u>	20 <u>14</u>	420,000 <u>105,000</u>	88,000 <u>62,000</u>
<u>Sacramento Valley</u>	39 <u>19</u>	220 <u>103</u>	32 <u>15</u>	470,000 <u>94,000</u>	470,000 <u>78,000</u>
<u>Statewide</u>	580 <u>360</u>	3,800- <u>2,400</u>	600 <u>380</u>	3,300,000 <u>2,500,000</u>	2,800,000 <u>1,800,000</u>

Note: Some columns may not add up to the statewide totals due to rounding. Since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated benefits/impacts from

Tables ~~B-4 and B-5~~B-6, B-7 and B-8 together. Table ~~B-6~~B-8 should be used to estimate the maximum health ~~benefit~~impact per air basin. The uncertainty behind the mortality estimates is on the order of +/- 50% and varies for other endpoints.

Table B-5:B-7: Annual Health Benefits from Attaining 8-hour Impacts of Current Ozone Concentrations Compared to the State 1-hour Ozone Standard of 0.0700.09 ppm by Air Basin (cases/year).

<u>Air Basin</u>	<u>Mortality</u>	<u>Hospital Admissions</u>	<u>Emergency Room Visits</u>	<u>School Absences</u>	<u>Minor Restricted Activity Days</u>
<u>Great Basin Valley</u>	<1	4 <1	<1	650 300	820 260
<u>Lake County</u>	<1 0	<1 0	<1 0	35 0	53 0
<u>Lake Tahoe</u>	4 <1	9 2	4 <1	6,100 1,900	6,600 1,400
<u>Mountain Counties</u>	12 9	62 48	8 6	41,000 45,000	50,000 38,000
<u>Mojave Desert</u>	54 40	350 270	59 45	280,000 310,000	260,000 200,000
<u>North Coast</u>	<1	4 <1	<1	720 0	750 0
<u>North Central Coast</u>	2 1	12 7	2 1	9,100 7,600	8,800 5,300
<u>Northeast Plateau</u>	<1 0	<1 0	<1 0	88 0	140 0
<u>South Coast</u>	260 290	1,700 2,000	270 310	1,100,000 2,000,000	1,300,000 1,500,000
<u>South Central Coast</u>	17 14	120 97	18 14	92,000 106,000	91,000 73,000
<u>San Diego</u>	25 21	170 140	23 19	110,000 130,000	130,000 100,000
<u>San Francisco Bay</u>	14 20	192 40	13 18	51,000 110,000	72,000 110,000
<u>San Joaquin Valley</u>	400 89	670 570	120 100	600,000 740,000	470,000 410,000
<u>Salton Sea</u>	24 19	130 110	24 19	110,000 140,000	91,000 81,000
<u>Sacramento Valley</u>	39	220 200	32 29	140,000 200,000	160,000 150,000
<u>Statewide</u>	540	3,600	570 560	2,600,000 3,800,000	2,600,000

Note: Some columns may not add up to the statewide totals due to rounding. Since 1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated benefits/impacts from

Tables ~~B-4 and B-5~~B-6, B-7 and B-8 together. Table ~~B-6~~B-8 should be used to estimate the maximum health ~~benefit~~impacts per air basin. The uncertainty behind the mortality estimates is on the order of +/- 50% and varies for other endpoints.

Table B-6: Annual Health Benefits from Attaining Both 1-hour and 8-hour Ozone Standards by Air Basin

Table B-8: Annual Health Impacts of Current Ozone Concentrations Compared to the State 8-hour Ozone Standard of 0.070 ppm by Air Basin (cases/year).

<u>Air Basin</u>	<u>Mortality</u>	<u>Hospital Admissions</u>	<u>Emergency Room Visits</u>	<u>School Absences</u>	<u>Minor Restricted Activity Days</u>
<u>Great Basin Valley</u>	<1	4 <1	<1	650 1,100	820 870
<u>Lake County</u>	<1 0	<1 0	<1 0	35 49	53 48
<u>Lake Tahoe</u>	4 <1	9 8	4 <1	6,100 8,600	6,600 6,400
<u>Mountain Counties</u>	42 10	62 61	8	41,000 61,000	50,000 49,000
<u>Mojave Desert</u>	51 50	350 340	59 58	280,000 420,000	260,000 250,000
<u>North Coast</u>	<1	1	<1	720 1,400	750 990
<u>North Central Coast</u>	2	42 17	2 3	9,100 19,000	8,800 12,700
<u>Northeast Plateau</u>	<1 0	<1 0	<1 0	88 270	140 270
<u>South Coast</u>	300 320	2,100 2,200	330 350	4,700,000 2,300,000	1,600,000
<u>South Central Coast</u>	47 19	420 130	48 19	97,000 150,000	91,000 97,000
<u>San Diego</u>	25 33	470 220	23 30	420,000 220,000	430,000 170,000
<u>San Francisco Bay</u>	23 21	450 140	24 19	400,000 120,000	420,000 110,000
<u>San Joaquin Valley</u>	100	670 650	420 110	650,000 880,000	470,000 460,000
<u>Salton Sea</u>	24 24	430 150	24 24	420,000 200,000	91,000 110,000
<u>Sacramento Valley</u>	39 45	220 250	32 37	470,000 250,000	470,000 190,000
<u>Statewide</u>	630	4,200	660	4,700,000	3,100,000

Note: The higher central estimate for the benefit values (either 1-hour or 8-hour averaging times) is given above for each endpoint by Some columns may not add up to the statewide totals due to rounding. Since

1-hour and 8-hour concentrations are highly correlated, it is not appropriate to add the estimated impacts from Tables B-6, B-7 and B-8 together. Table B-8 should be used to estimate the maximum health impacts per air basin. The uncertainty behind the mortality estimates is on the order of +/- 50% and varies for other endpoints.

References

ARB (2004) Aerometric Data Analysis and Management System (ADAM)
<http://www.arb.ca.gov/adam/welcome.html>

Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. 2004. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone. Report of a WHO task group. World Health Organization. (<http://www.euro.who.int/document/e82792.pdf>)

Anderson HR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Ponka A, Bacharova L, Schwartz J, Katsouyanni K. 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *Eur Respir J* 10:1064-71.

~~Bates, DV. 2005. Personal statement made at Air Quality Advisory Committee Meeting, January 12, 2005.~~

Bell M, McDermott A, Zeger S, Samet J, Dominici F. 2004. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *JAMA* 292, 19:2372-2378.

Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environ Res* 72:24-31.

Burnett RT, Dales RE, Raizenne ME, Krewski D, Summers PW, Roberts GR, Raad-Young M, Dann T, Brook J. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. *Environ Res* 65:172-94.

Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, Leech JA, Cakmak S, Krewski D. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 153:444-52.

Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG. 2001. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA* 285:897-905.

Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 12:43-54.

Gryparis A, Forsberg, B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Somoli, E, Medina S, Anderson R, Niciu, E, Wichmann H, Kriz B, Kosnik M, Skordovsky J, Vonk J, Dorbudak Z. 2004. Acute effects of ozone on mortality from the "Air Pollution and Health: A European Approach" project. *Am J Respir Crit Care Med* 170: 1080-1087.

Hall JV, Brajer V, Lurmann FW. 2003. Economic valuation of ozone-related school absences in the south coast air basin of California. *Contemporary Economic Policy* 21:407-417.

Hubbell, BJ, Halberg A, McCubbin, DR, Post, E. 2005. Health-related benefits of attaining the 8-hr ozone standard. *Environ Health Perspect* 113:73-82.

Jaffe DH, Singer ME, Rimm AA. 2003. Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. *Environ Res* 91:21-8.

Levy JI, Carrothers TJ, Tuomisto JT, Hammitt JK, Evans JS. 2001. Assessing the public health benefits of reduced ozone concentrations. *Environ Health Perspect* 109:1215-26.

National Research Council. 2002. Estimating the public health benefits of proposed air pollution regulations. Washington, D.C.: National Academy Press.

Ostro BD, Rothschild S. 1989. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. *Environ Res* 50 :238-47.

Romieu I, Meneses F, Sienra-Monge JJ, Huerta J, Ruiz Velasco S, White MC, Etzel RA, Hernandez-Avila M. 1995. Effects of urban air pollutants on emergency visits for childhood asthma in Mexico City. *Am J Epidemiol* 141:546-53.

Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW *et al.* (2000). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. *Health Effects Institute* (94 Pt 2).

Schwartz J. 1997 Health effects of air pollution from traffic: ozone and particulate matter. *Health at the crossroads: transport policy and urban health*. New York: John Wiley.

Stieb DM, Burnett RT, Beveridge RC, Brook JR. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect* 104:1354-60.

Stieb DM, Judek S, Burnett RT. 2003. Meta-analysis of time-series studies of air pollution and mortality: update in relation to the use of generalized additive models. *J Air Waste Manag Assoc* 53:258-261.

Thurston, G.T. and Ito, K. 1999. Epidemiologic studies of ozone exposure effects. In: *Air Pollution and Health* (Holgate ST, Samet JM, Koren Hs, Maynard RL, eds.) London:Academic Press.

Thurston GD, Ito K. 2001. Epidemiological studies of acute ozone exposures and mortality. *J Expo Anal Environ Epidemiol* 11: 286-94.

Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res* 65: 271-90.

Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, White MC. 2000. Air

quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *Am J Epidemiol* 151:798-810.

U.S. Environmental Protection Agency. 1999. The benefits and costs of the clean air act 1990 to 2010: EPA report to Congress. Washington, D.C.: Office of Air and Radiation and Office of Policy. Report No.: EPA-410-R-99-001, November. (<http://www.epa.gov/air/sect812/copy99.html>).

U.S. Environmental Protection Agency. 2004. Advisory on plans for health effects analysis in the analytical plan for EPA's second prospective analysis benefits and costs of the clean air act, 1990-2020; advisory by the health effects subcommittee of the advisory council on clean air compliance analysis. Washington, D.C. Report No.: EPA-SAB-COUNCIL-ADV-04-002 Environmental. (http://www.epa.gov/sab/pdf/council_adv_04002.pdf)

Supplement to Appendix B

Rollback Formulae

For the technical reader, the mathematical formulae for our rollback procedure follow. Denote:

OzCurrent = current daily ozone observed value,
BasinMax = design value based on three years of measured data,
BG = background ozone of 0.04 ppm,
Std = ~~proposed standard (0.09 ppm for 1-hour and 0.070 ppm for 8-hour),~~ 0.094 ppm for 1-hour, or basin-specific equivalent 1-hour design value for 8-hour standard of 0.070 ppm, or basin-specific equivalent 1-hour design value for federal 8-hour standard of 0.084 ppm, and
OzAttain = rolled-back ozone value in the “attainment” scenario.

First, the reduction percentage (or reduction factor RF) was calculated for each basin as follows:

If BasinMax > Std, then $RF = (BasinMax - Std) / (BasinMax - BG)$.
If BasinMax ≤ Std, then RF = 0.

The rollback factor, 1-RF, is applied as follows. For all sites within the basin, the portion of the site’s current ozone levels above background was adjusted:

If OzCurrent > BG, then $OzAttain = BG + (1 - RF) \times (OzCurrent - BG)$.
If OzCurrent ≤ BG, then OzAttain = OzCurrent.

The change in ozone concentrations is OzCurrent – OzAttain, calculated at the daily level for each site, which is the difference between the observed value and the rolled-back value for each site on each day of the year.

Note that we used the actual levels of the standards, 0.09 and 0.070 ppm, in the rollback rather than the maximal values that round to the standards as is done with air quality modeling. Such modeling usually assumes worst-case meteorology, unlike our methodology of using the three-year high value.

Rollback Method Development

The assumption of a constant rollback factor applied to an entire air basin was justified through an empirical analysis of the trends in the percentiles at South Coast Air Basin monitoring sites. This air basin was selected for the analysis since the air quality trends were clear, there is a range of coastal and inland environments, and a majority of benefits are projected to occur in that air basin. ~~Figures B-2 through B-11 and Tables B-7 through B-16~~ B-3 through B-12 and Tables B-9 through B-18 provide examples of the results from that analysis, and the materials are representative of the results used for development of the rollback factor applied in the benefits analysis. In the graphs, the

dotted line indicates the ozone standard, and the dashed line represents the assumed background level. Due to space limitations, the legend for every percentile line was not provided. However, the reader is advised to examine the solid lines in each graph, from top to bottom, to represent the maximum, 90th percentile, 80th percentile, 70th percentile, 60th percentile, 50th percentile, and 40th percentile of the annual distribution of ozone measurements.

Briefly, the analysis showed that since 1980, the trend in the monitored values associated with the distribution of percentiles was consistently downward, and that the relationships were relatively parallel and linear. Consequently, we assumed a constant rollback factor based on a basin's three-year high value, and applied it to all daily high values at all sites within the basin. In other words, when a control strategy is geared towards reducing the highest ozone levels in an air basin, its impact on days with low and moderate ozone levels is comparable to those days with high ozone levels.

Estimation of Exposed Population

To estimate the number of people exposed to the ozone changes observed at each monitoring site, the county population was divided by the number of monitoring sites in a given county. For example, suppose a county has N monitoring stations and population POP according to year 2000 census. Then we would estimate that (POP/N) persons were exposed to ozone levels at each of the N monitors within this county. The health incidences were then calculated based on the concentration-response functions relating changes in ozone concentrations and exposed population for each day at each monitor. The sensitivity of this methodology is discussed in detail on page ~~B-15~~B-16.

**Trends in Annual Percentiles of the
Daily Max. 1-Hr Ozone in the South Coast Air Basin**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

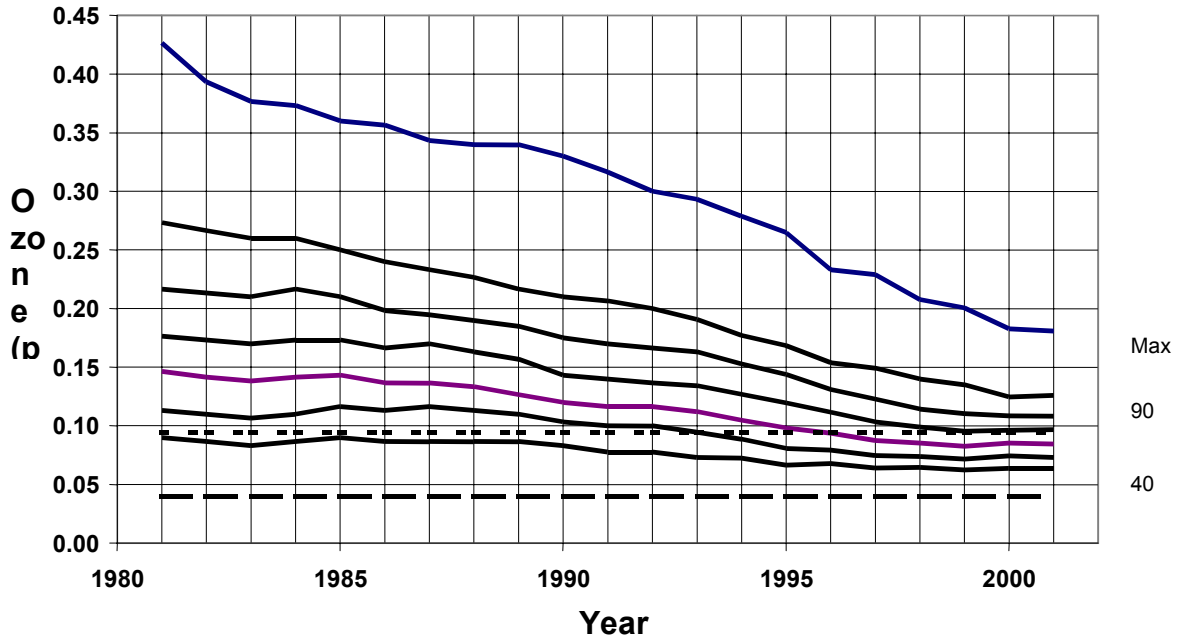


Figure B-2-B-3: Trends in Annual Percentiles of Daily Max 1-hour Ozone in the South Coast Air Basin

Table B-7:B-9: Summary of Trends in Annual Percentiles of the Daily Max. 1-Hr Ozone in the South Coast Air Basin

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.427	0.317	0.183
Δ% above background		28%	63%
90th Percentile	0.273	0.207	0.125
Δ% above background		29%	64%
80th Percentile	0.217	0.170	0.109
Δ% above background		26%	61%
70th Percentile	0.177	0.140	0.096
Δ% above background		27%	59%
60th Percentile	0.147	0.117	0.086
Δ% above background		28%	57%
50th Percentile	0.113	0.100	0.075
Δ% above background		18%	53%
40th Percentile	0.090	0.078	0.064
Δ% above background		24%	52%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

Trends in Annual Percentiles of the Daily Max. 8-Hr Ozone in the South Coast Air Basin

(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

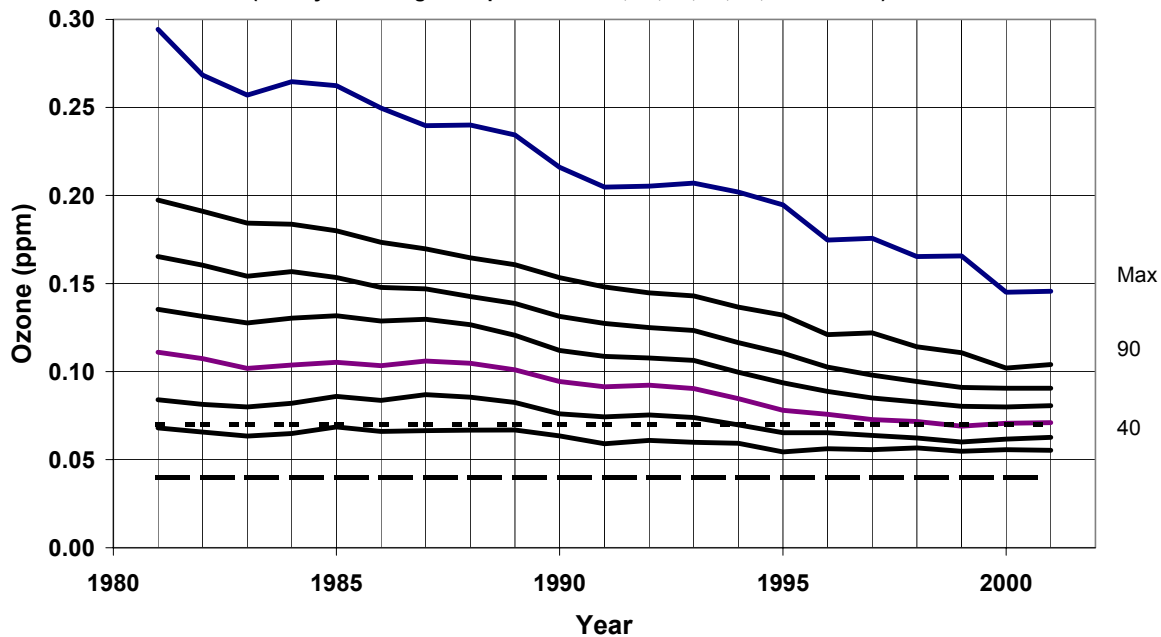


Figure B-3:B-4: Trends in Annual Percentiles of Daily Max 8-hour Ozone in the South Coast Air Basin

Table B-8:B-10: Summary of Trends in Annual Percentiles of the Daily Max. 8-hr Ozone in the South Coast Air Basin

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.294	0.205	0.145
Δ% above background		35%	59%
90th Percentile	0.197	0.148	0.102
Δ% above background		31%	61%
80th Percentile	0.165	0.127	0.091
Δ% above background		30%	60%
70th Percentile	0.135	0.109	0.080
Δ% above background		28%	58%
60th Percentile	0.111	0.091	0.071
Δ% above background		28%	57%
50th Percentile	0.084	0.074	0.062
Δ% above background		22%	51%
40th Percentile	0.068	0.059	0.056
Δ% above background		32%	44%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 1-Hr Ozone at N. Long Beach**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

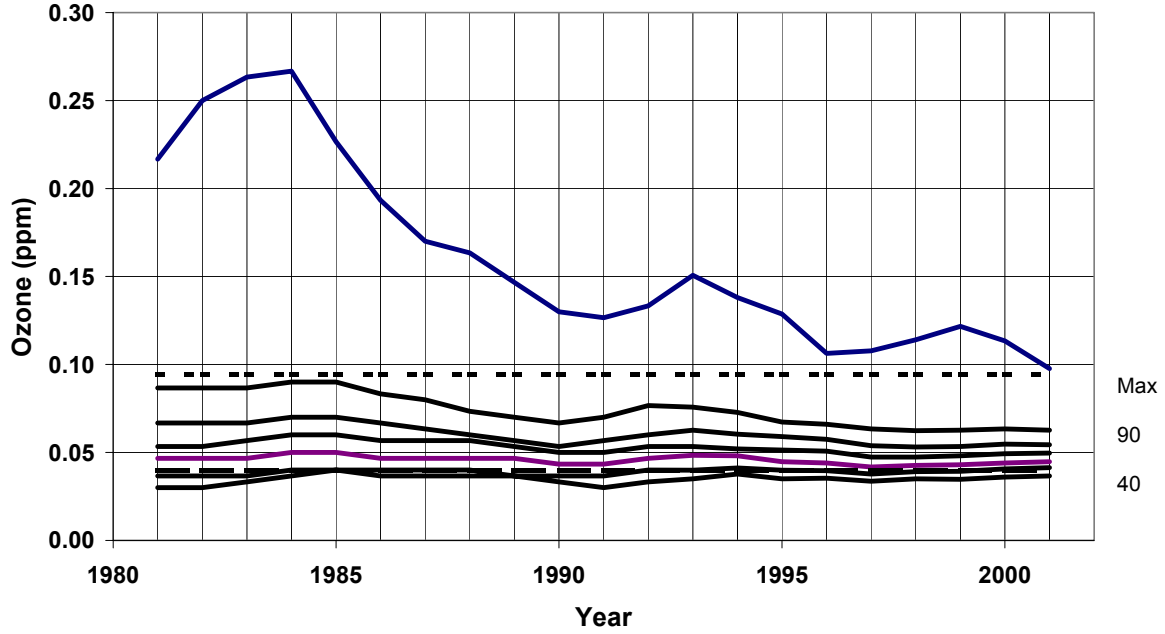


Figure B-4: Trends in Annual Percentiles of Daily Max 1-hour Ozone at N. Long Beach

Table B-9:B-11: Summary of Trends in Annual Percentiles of the Daily Max 1-hour Ozone at N. Long Beach

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.217	0.127	0.113
Δ% above background		51%	58%
90th Percentile	0.087	0.070	0.063
Δ% above background		36%	50%
80th Percentile	0.067	0.057	0.055
Δ% above background		38%	45%
70th Percentile	0.053	0.050	0.049
Δ% above background		25%	30%
60th Percentile	0.047	0.043	0.044
Δ% above background		50%	40%
50th Percentile	0.037	0.037	0.041
Δ% above background		Percentiles are below background.	
40th Percentile	0.030	0.030	0.036
Δ% above background		Percentiles are below background.	
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 8-Hr Ozone at N. Long Beach**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

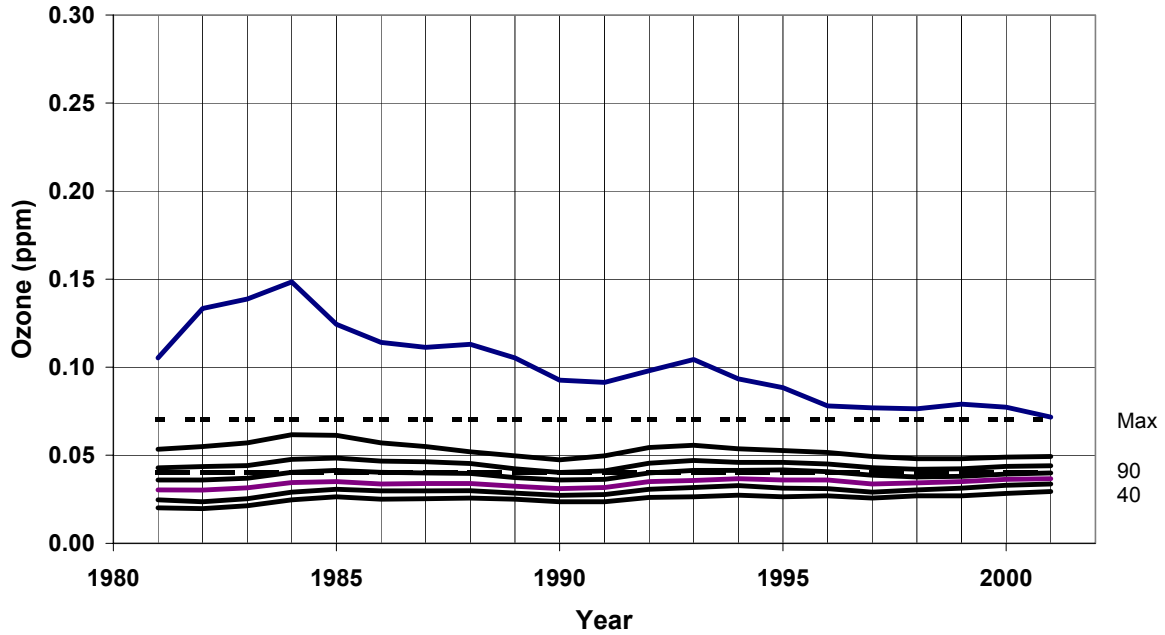
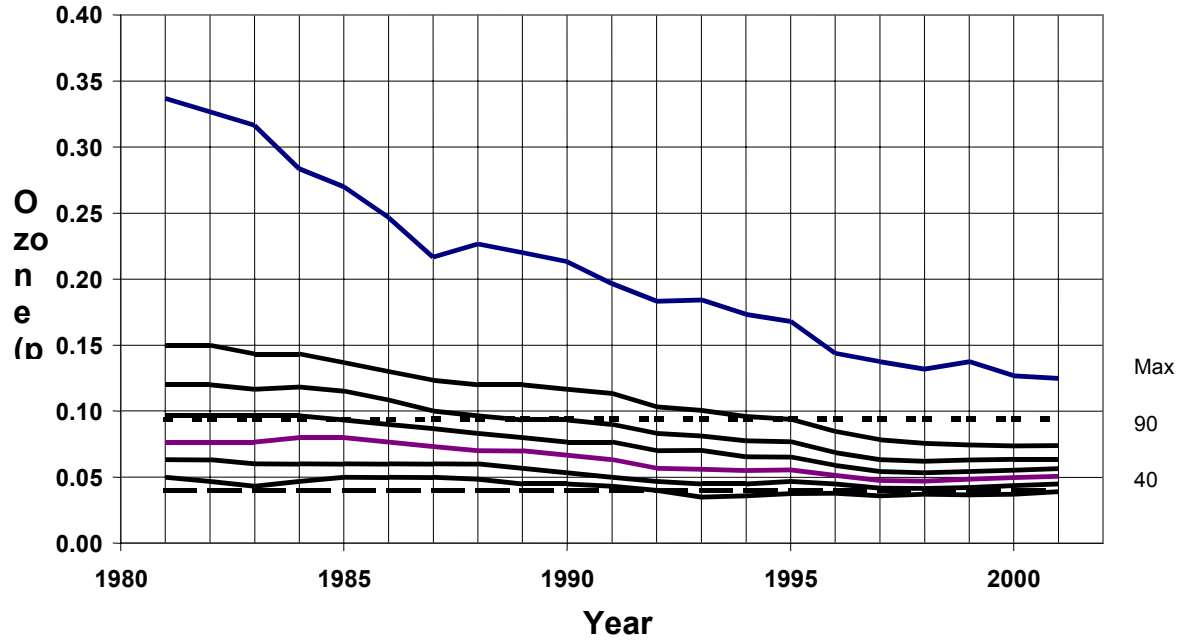


Figure B-5;B-6: Trends in annual percentiles of daily max 8-hour ozone at N. Long Beach

Table B-10:B-12: Summary of Trends in Annual Percentiles of the Daily Max 8-hour Ozone at N. Long Beach

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.105	0.091	0.077
Δ% above background		21%	43%
90th Percentile	0.053	0.050	0.049
Δ% above background		28%	33%
80th Percentile	0.043	0.041	0.044
Δ% above background		59%	-29%
70th Percentile	0.036	0.036	0.039
Δ% above background		Percentiles are below background.	
60th Percentile	0.030	0.032	0.036
Δ% above background		Percentiles are below background.	
50th Percentile	0.025	0.028	0.033
Δ% above background		Percentiles are below background.	
40th Percentile	0.020	0.024	0.028
Δ% above background		Percentiles are below background.	
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 1-Hr Ozone at L.A. - N. Main**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)



**Figure B-6:B-7: Trends in annual percentiles of daily max 1-hour ozone
L.A. – N. Main**

**Table B-11:B-13: Summary of Trends in Annual Percentiles of the Daily
Max 1-hour Ozone at L.A. - N. Main**

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.337	0.197	0.127
Δ% above background		47%	71%
90th Percentile	0.150	0.113	0.074
Δ% above background		33%	69%
80th Percentile	0.120	0.090	0.064
Δ% above background		38%	70%
70th Percentile	0.097	0.077	0.055
Δ% above background		35%	73%
60th Percentile	0.077	0.063	0.050
Δ% above background		36%	74%
50th Percentile	0.063	0.050	0.044
Δ% above background		57%	84%
40th Percentile	0.050	0.043	0.037
Δ% above background		67%	100%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 8-Hr Ozone at L.A. - N. Main**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

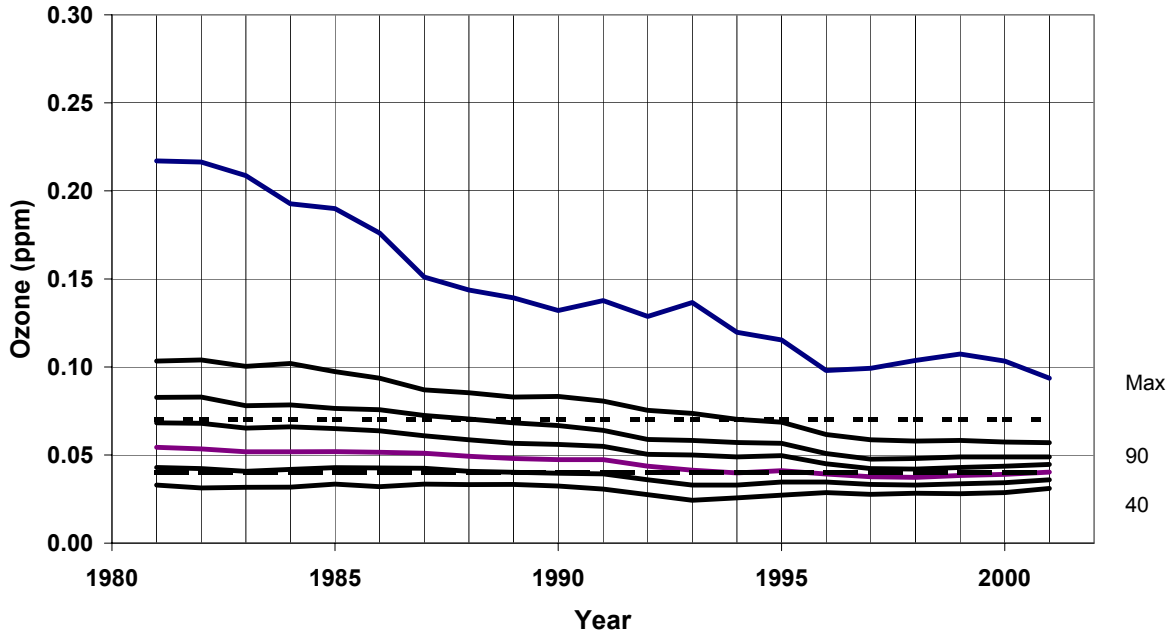
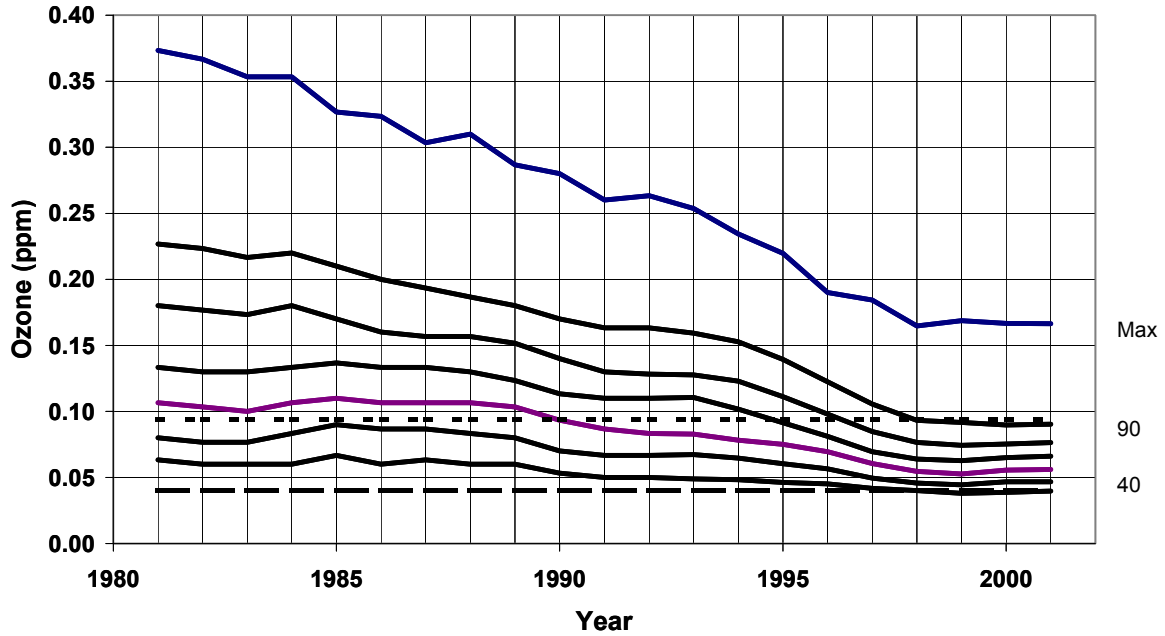


Figure B-7:B-8: Trends in annual percentiles of daily max 8-hour ozone at L.A.-N. Main

Table B-12:B-14: Summary of Trends in Annual Percentiles of the Daily Max 8-hour Ozone at L.A. - N. Main

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.217	0.138	0.103
Δ% above background		45%	64%
90th Percentile	0.103	0.081	0.057
Δ% above background		36%	73%
80th Percentile	0.083	0.064	0.049
Δ% above background		44%	79%
70th Percentile	0.068	0.055	0.044
Δ% above background		47%	87%
60th Percentile	0.054	0.047	0.039
Δ% above background		49%	100%
50th Percentile	0.043	0.039	0.034
Δ% above background		100%	100%
40th Percentile	0.033	0.031	0.029
Δ% above background		Percentiles are below background.	
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 1-Hr Ozone at Azusa**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

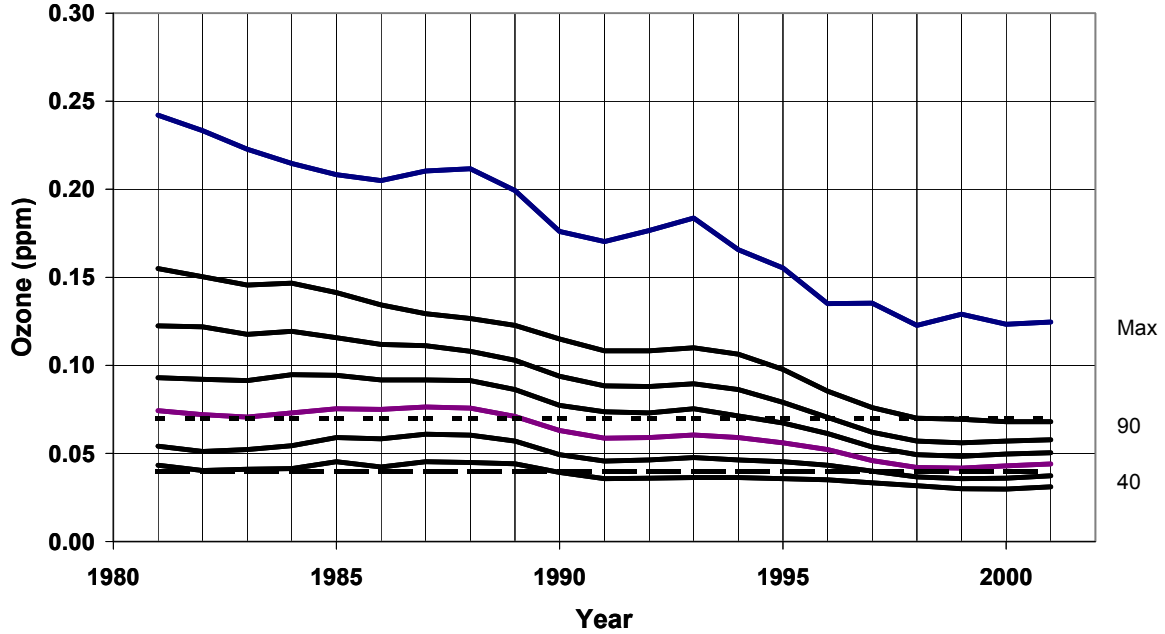


**Figure B-8:B-9: Trends in annual percentiles of daily max 1-hour ozone at
Azusa**

Table B-13:B-15: Summary of Trends in Annual Percentiles of the Daily Max 1-hour Ozone at Azusa

Summary of Trends in Annual Percentiles of the Daily Max. 1-Hr Ozone at Azusa			
Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.373	0.260	0.167
Δ% above background		34%	62%
90th Percentile	0.227	0.163	0.090
Δ% above background		34%	73%
80th Percentile	0.180	0.130	0.075
Δ% above background		36%	75%
70th Percentile	0.133	0.110	0.065
Δ% above background		25%	73%
60th Percentile	0.107	0.087	0.056
Δ% above background		30%	77%
50th Percentile	0.080	0.067	0.047
Δ% above background		33%	83%
40th Percentile	0.063	0.050	0.039
Δ% above background		57%	100%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 8-Hr Ozone at Azusa**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)



**Figure B-9:B-10: Trends in annual percentiles of daily max 8-hour ozone at
Azusa**

Table B-14:B-16: Summary of Trends in Annual Percentiles of the Daily Max 8-hour Ozone at Azusa

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.242	0.170	0.123
Δ% above background		35%	59%
90th Percentile	0.155	0.108	0.068
Δ% above background		41%	76%
80th Percentile	0.123	0.088	0.057
Δ% above background		41%	79%
70th Percentile	0.093	0.074	0.050
Δ% above background		36%	82%
60th Percentile	0.074	0.059	0.043
Δ% above background		46%	100%
50th Percentile	0.054	0.046	0.036
Δ% above background		60%	100%
40th Percentile	0.043	0.036	0.030
Δ% above background		100%	100%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

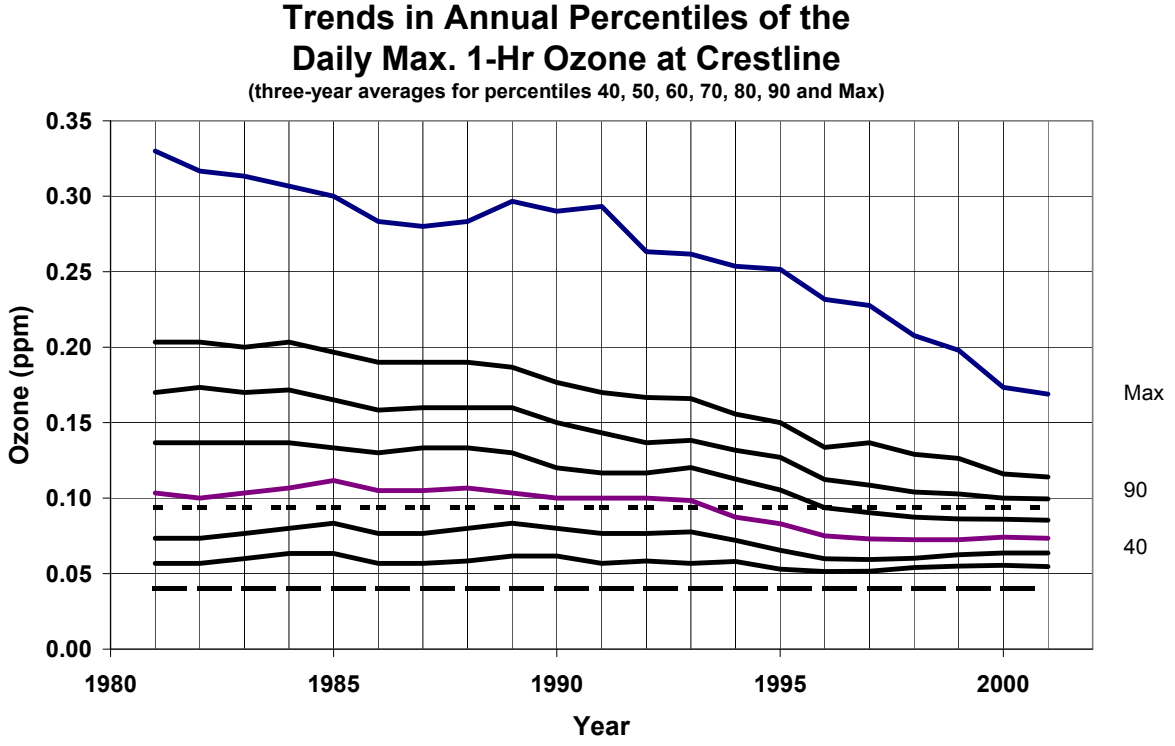


Figure B-10:B-11: Trends in annual percentiles of daily max 1-hour ozone at Crestline

Table B-15:B-17: Summary of Trends in Annual Percentiles of the Daily Max 1-hour Ozone at Crestline

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.330	0.293	0.173
Δ% above background		13%	54%
90th Percentile	0.203	0.170	0.116
Δ% above background		20%	53%
80th Percentile	0.170	0.143	0.100
Δ% above background		21%	54%
70th Percentile	0.137	0.117	0.086
Δ% above background		21%	52%
60th Percentile	0.103	0.100	0.074
Δ% above background		5%	46%
50th Percentile	0.073	0.077	0.064
Δ% above background		-10%	29%
40th Percentile	0.057	0.057	0.056
Δ% above background		0%	7%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

**Trends in Annual Percentiles of the
Daily Max. 8-Hr Ozone at Crestline**
(three-year averages for percentiles 40, 50, 60, 70, 80, 90 and Max)

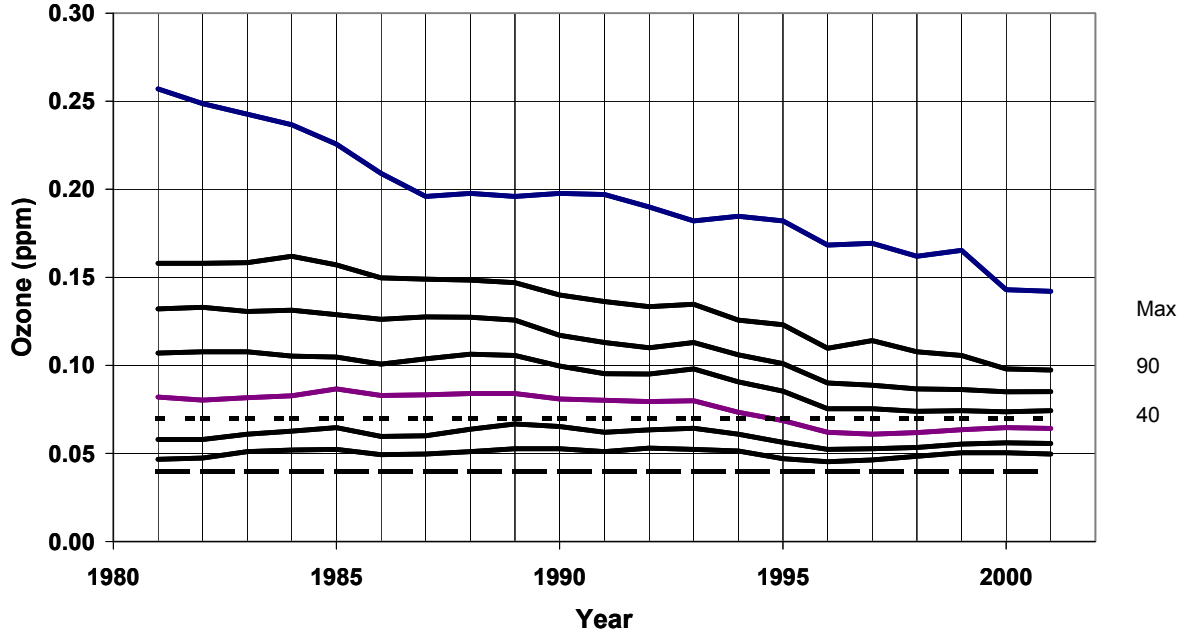


Figure B-11:2: Trends in annual percentiles of daily max 8-hour ozone at Crestline

Table B-16:B-18: Summary of Trends in Annual Percentiles of the Daily Max 8-hour Ozone at Crestline

Indicator	Average Value During Period		
	1980-1982	1990-1992	2000-2002
Maximum	0.257	0.197	0.143
Δ% above background		28%	53%
90th Percentile	0.158	0.136	0.098
Δ% above background		18%	51%
80th Percentile	0.132	0.113	0.085
Δ% above background		21%	51%
70th Percentile	0.107	0.095	0.074
Δ% above background		17%	50%
60th Percentile	0.082	0.080	0.065
Δ% above background		4%	41%
50th Percentile	0.058	0.062	0.056
Δ% above background		-22%	11%
40th Percentile	0.047	0.051	0.050
Δ% above background		-65%	-55%
note: Delta % above background is the change in the portion of measured ozone since 1980-82 above "background", where background is defined as 0.04 ppm.			

Table B-17:B-19: Baseline Incidence Rates (Incidence/1000 Persons/Year)

County Name	Mortality (Short-Term Exposures) Non-Accidental, All Ages	Hospital Admissions, All Respiratory, All Ages	ER Visits for Asthma, Age Under 18	School Loss Days, All Illness, Age 5-17	MRAD Age>18
Alameda County	6.60	10.13	3.81	5990.10	7805.39
Alpine County	7.40	10.13	3.81	5990.10	7805.39
Amador County	9.99	10.13	3.81	5990.10	7805.39
Butte County	10.40	10.13	3.81	5990.10	7805.39
Calaveras County	8.90	10.13	3.81	5990.10	7805.39
Colusa County	7.10	10.13	3.81	5990.10	7805.39
Contra Costa County	6.78	10.13	3.81	5990.10	7805.39
Del Norte County	8.41	10.13	3.81	5990.10	7805.39
El Dorado County	6.29	10.13	3.81	5990.10	7805.39
Fresno County	6.41	10.13	3.81	5990.10	7805.39
Glenn County	7.71	10.13	3.81	5990.10	7805.39
Humboldt County	8.51	10.13	3.81	5990.10	7805.39
Imperial County	5.44	10.13	3.81	5990.10	7805.39
Inyo County	11.81	10.13	3.81	5990.10	7805.39
Kern County	6.60	10.13	3.81	5990.10	7805.39
Kings County	5.66	10.13	3.81	5990.10	7805.39
Lake County	13.13	10.13	3.81	5990.10	7805.39
Lassen County	5.75	10.13	3.81	5990.10	7805.39
Los Angeles County	6.08	10.13	3.81	5990.10	7805.39
Madera County	6.35	10.13	3.81	5990.10	7805.39
Marin County	7.47	10.13	3.81	5990.10	7805.39
Mariposa County	9.48	10.13	3.81	5990.10	7805.39
Mendocino County	8.89	10.13	3.81	5990.10	7805.39
Merced County	6.29	10.13	3.81	5990.10	7805.39
Modoc County	11.62	10.13	3.81	5990.10	7805.39
Mono County	3.87	10.13	3.81	5990.10	7805.39
Monterey County	5.88	10.13	3.81	5990.10	7805.39
Napa County	10.45	10.13	3.81	5990.10	7805.39
Nevada County	8.56	10.13	3.81	5990.10	7805.39
Orange County	5.68	10.13	3.81	5990.10	7805.39

Placer County	7.00	10.13	3.81	5990.10	7805.39
Plumas County	10.08	10.13	3.81	5990.10	7805.39
Riverside County	7.37	10.13	3.81	5990.10	7805.39
Sacramento County	7.14	10.13	3.81	5990.10	7805.39
San Benito County	5.06	10.13	3.81	5990.10	7805.39
San Bernardino County	6.10	10.13	3.81	5990.10	7805.39
San Diego County	6.41	10.13	3.81	5990.10	7805.39
San Francisco County	8.78	10.13	3.81	5990.10	7805.39
San Joaquin County	6.98	10.13	3.81	5990.10	7805.39
San Luis Obispo County	7.87	10.13	3.81	5990.10	7805.39
San Mateo County	6.77	10.13	3.81	5990.10	7805.39
Santa Barbara County	6.80	10.13	3.81	5990.10	7805.39
Santa Clara County	5.19	10.13	3.81	5990.10	7805.39
Santa Cruz County	6.56	10.13	3.81	5990.10	7805.39
Shasta County	9.50	10.13	3.81	5990.10	7805.39
Sierra County	9.26	10.13	3.81	5990.10	7805.39
Siskiyou County	10.42	10.13	3.81	5990.10	7805.39
Solano County	5.90	10.13	3.81	5990.10	7805.39
Sonoma County	8.17	10.13	3.81	5990.10	7805.39
Stanislaus County	7.22	10.13	3.81	5990.10	7805.39
Sutter County	7.43	10.13	3.81	5990.10	7805.39
Tehama County	9.90	10.13	3.81	5990.10	7805.39
Trinity County	10.73	10.13	3.81	5990.10	7805.39
Tulare County	6.71	10.13	3.81	5990.10	7805.39
Tuolumne County	9.50	10.13	3.81	5990.10	7805.39
Ventura County	5.76	10.13	3.81	5990.10	7805.39
Yolo County	6.37	10.13	3.81	5990.10	7805.39
Yuba County	7.26	10.13	3.81	5990.10	7805.39

Reference Lists
Chapters 1-11, Appendix B and Appendix G

Chapter 1

1. Air Resources Board and Office of Environmental Health Hazard Assessment (2000). Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. Sacramento, CA. Available at <http://www.arb.ca.gov/ch/programs/sb25/airstandards.htm>.

Chapter 2

2. Adams WC. 1998. Dose-response effects of varied equivalent minute ventilation rates on pulmonary function responses during exposure to ozone. Final Report to the American Petroleum Institute. Washington D.C.
3. Air Resources Board. Ambient Air Quality Standard for Ozone: Health and Welfare Effects. Staff Report. September 1987. Sacramento, CA.
4. Air Resources Board and Office of Environmental Health Hazard Assessment. Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. 2000.
5. Air Resources Board (2001). Policies and Actions for Environmental Justice, December 13, 2001.
6. Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ Health Perspect* 107:675-679.
7. Kunzli N, Lurmann F, Segal M, Ngo L, Balme J, Tager IB. 1997. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen – results of a pilot study. *Environ Res* 72:8-23.
8. McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386-391.
9. World Health Organization (2000). Air Quality Guidelines for Europe, Second Edition. (WHO regional publications, European series, No. 91.)

Chapter 3

10. Air Resources Board/Planning and Technical Support Division. 2002. California Ambient Air Quality Data – 1980 – 2001. Sacramento, CA.: December. Data CD Number: PTSD-02-017-CD

11. Chameides WL, Fehsenfeld F, Rodgers MO, Cardelino C, Martinez J, Parrish D, Lonneman W, Lawson DR, Rasmussen RA, Zimmerman P, Greenberg J, Middleton P, Wang T. 1992. Ozone Precursor Relationships in the Ambient Atmosphere. *Journal of Geophysical Research* 97:6037-55.
12. Croes BE, Dolislager LJ, Larsen LC, Pitts JN. 2003. The O₃ "Weekend Effect" and NO_x Control Strategies -- Scientific and Public Health Findings and Their Regulatory Implications. *Environ Manager* July:27-35.
13. Finlayson-Pitts BJ, Pitts JN. 2000. *Chemistry of the Upper and Lower Atmosphere - Theory, Experiments, and Applications*. Academic Press, San Diego, CA.
14. National Research Council. 1991. *Rethinking the Ozone Problem in Urban and Regional Air Pollution*, National Academy Press, Washington, DC.
15. Smith TB, Lehrman DE, Knuth WR, Johnson D. 1997. *Monitoring in Ozone Transport Corridors*. Final report prepared for ARB/RD (contract # 94-316).
16. Whitten GZ, 1993. The Chemistry of Smog Formation: A Review of Current Knowledge. *Environment International*. 9:447-463.

Chapter 4

17. Berntsen, T., Karlsdottir, S., Jaffe, D. 1999. Influence of Asian emissions on the composition of air reaching the North Western United States, *Geophys. Res. Lett.* 26(14): 2,171-2,174.
18. Bertschi, I. T., D. A. Jaffe, L. Jaegle, H. U. Price, J. B. Dennison. 2004. PHOBEA/ITCT 2002 airborne observations of transpacific transport of ozone, CO, volatile organic compounds, and aerosols to the northeast Pacific: Impacts of Asian anthropogenic and Siberian boreal fire emissions, *J. Geophys. Res.* 109: D23S12, doi:10.1029/2003JD004328.
19. Bojkov, R. 1986. Surface ozone during the second half of the nineteenth century. *Mon. Weather Rev.* 25: 353-352.
20. Browell, E. et al. 2003. Ozone, aerosol, potential vorticity, and trace gas trends observed at high latitudes over North America from February to May 2000. *J. Geophys. Res.* 108(D4).

21. California Department of Forestry and Fire Protection (CDFFP). 2004a. 2003 Fire Summary
http://WWW.FIRE.CA.GOV/php/2003fireseasonstats_v2.asp.
22. California Department of Forestry and Fire Protection (CDFFP). 2004b. 2003 Large Fires.
http://WWW.FIRE.CA.GOV/php/fire_er_content/downloads/2003LargeFires.pdf
23. Crutzen, P. 1995. My life with O₃, NO_x and other YZO_xs, Nobel Lecture, December 8, 1995.
24. Derognat, C., M. Beekmann, M. Baeumle, D. Martin, H. Schmidt. 2003. Effect of biogenic volatile organic compound emissions on tropospheric chemistry during the Atmospheric Pollution Over the Paris Area (ESQUIF) campaign in the Ile-de-France region. *J. Geophys. Res.* 108(D17).
25. Dreyfus, G. B., Schade, G. W., Goldstein, A.H. 2002. Observational constraints on the contribution of isoprene oxidation to ozone production on the western slope of the Sierra Nevada, CA. *J. Geophys. Res.* 107(D19).
26. Fiore, A. et al. 2002. Background ozone over the United States in summer: Origin, trend, and contribution to pollution episodes. *J. Geophys. Res.* 107(D15).
27. Galani, E., et al. 2003. Observations of stratosphere-to-troposphere transport events over the eastern Mediterranean using a ground-based lidar system. *J. Geophys. Res.* 108(D12).
28. Galanter, M., Levy, H., Carmichael, G. 2000. Impacts of biomass burning on tropospheric CO, NO_x, and O₃. *J. Geophys. Res.* 105(D5): 6,633-6,653.
29. Hauglustaine, D., Brasseur, G. 2001. Evolution of tropospheric ozone under anthropogenic activities and associated radiative forcing of climate. *J. Geophys. Res.* 106(D23).
30. Jaegle, L., Jaffe, D., Price, H., Weiss-Penzias, P., Palmer, P., Evans, M., Jacob, D., Bey, I. 2003. Sources and budgets for CO and O₃ in the northeastern Pacific during the spring of 2001: Results from the PHOBEA-II Experiment. *J. Geophys. Res.* 108(D20).
31. Jacob, D., Logan, J., Murti, P. 1999. Effect of rising Asian emissions on surface ozone in the United States. *Geophys. Res. Lett.* 26(14): 2,175-2,178.
32. Jaffe, D., I. Bertsch, L. Jaegle, P. Novelli, J. S. Reid, H. Tanimoto, R. Vingarzan, D. L. Westphal. 2004. Long-range transport of Siberian biomass burning emissions and impact on surface ozone in western North America. *Geophys. Res. Lett.*, 31, L16106, doi:10.1029/2004GL020093.
33. Jaffe, D., et al. 1999. Transport of Asian Air Pollution to North America, *Geophys Res. Lett.* 26(6): 711-714.

34. Jaffe, D., Weiss-Penzias, P., Dennison, J., Bertschi, I., Westphal, D. 2003a. Siberian Biomass Burning Plumes Across the Pacific: Impact on Surface Air Quality in the Pacific Northwest, *Eos Trans. AGU*, 84(46), Fall Meet. Suppl., Abstract A11I-02.
35. Jaffe, D., Price, H., Parrish, D., Goldstein, A., Harris, J. 2003b. Increasing background ozone during spring on the west coast of North America, *Geophys. Res. Lett.* 30(12).
36. Jaffe, D., McKendry, I., Anderson, T., Price, H. 2003. Six 'new' episodes of trans-Pacific transport of air pollutants. *Atmos. Env.* 37m 391-404.
37. Jenkins, G., Ryu, J-H. 2003. Linking horizontal and vertical transport of biomass fire emissions to the Tropical Atlantic Ozone Paradox during the Northern Hemisphere winter season: climatology. *Atmos. Chem. Phys. Discuss.* 3: 5,061-5,098.
38. Lawrence, M., vonKuhlmann, R., Salzmann, M., Rasch, P. 2003. The balance of effects of deep convective mixing on tropospheric ozone, *Geophys. Res. Lett.* 30(18).
39. Lelieveld, J., Dentener, F. 2000. What controls tropospheric ozone?, *J. Geophys. Res.* 105(D3).
40. Lefohn, A., Oltmans, S., Dann, T., Singh, H 2001. Present-day variability of background ozone in the lower troposphere. *J. Geophys. Res.* 106(D9).
41. Levine, J. 1985. The Structure of the Atmosphere. In: Levine, J., ed. *The Photochemistry of Atmospheres.*, Academic Press, Orlando, FL. pp. 7-9.
42. Lin, C-Y, Jacob, D., Munger, J., Fiore, A. 2000. Increasing background ozone in surface air over the United States. *Geophys. Res. Lett.* 27(21): 3,465-3,468.
43. Lisac, I., Grubilic, V. 1991. An analysis of surface ozone data measured at the end of the 19th century in Zagreb, Yugoslavia. *Atmos. Env.* 25A(2): 481-486.
44. Newchurch, M. J., et al. 2003. Vertical distribution of ozone at four sites in the United States. *J. Geophys. Res.* 108(D1).
45. Pochanart, P., Akimoto, H., Kajii, Y., Potemkin, V., Khodzher, T. 2003. Regional background ozone and carbon monoxide variations in remote Siberia/East Asia. *J. Geophys. Res.* 108(D1).
46. Seo, K.-H., K. P. Bowman. 2002. Lagrangian estimate of global stratosphere-troposphere mass exchange. *J. Geophys. Res.* 107(D21).

47. STACCATO. 2003. Influence of Stratosphere-Troposphere Exchange in a Changing Climate on Atmospheric Transport and Oxidation Capacity (STACCATO). Lehrstuhl für Bioklimatologie und Immissionsforschung, Technische Universität München, Germany, Web Report, <http://www.forst.tu-muenchen.de/EXT/LST/METEO/staccato/>
48. St. John, J., Chameides, W., Saylor, R. 1998. The Role of anthropogenic NO_x and VOC as ozone precursors: A case study from the SOS Nashville/Middle Tennessee Ozone Study. *J. Geophys. Res.* 103(D17): 22,415–22,423.
49. Thouret, V., et al. 2001. Tropospheric ozone layers observed during PEM-Tropics B. *J. Geophys. Res.* 106(D23): 32,527–32,538.
50. Trickl, T., Cooper, O., Eisele, H., James, P., Mucke, R., Stohl, A. 2003. Intercontinental transport and its influence on the ozone concentrations over central Europe: Three case studies. *J. Geophys. Res.* 108(D12).
51. Turco, R. 1985. The Photochemistry of the Stratosphere. In: Levine, J., ed. *The Photochemistry of Atmospheres*. Academic Press, Orlando, FL. pp. 77-128.
52. VanCuren, R., Cahill, T. 2002. Asian aerosols in North America: Frequency and concentration of fine dust. 107(D24).
53. Weiss-Penzias, P., Jaffe, D., McClintick, A., Jaegle, L., Liang, Q. 2003. The influence of long-range transport of pollution on the annual cycles of carbon monoxide and ozone. *Eos Trans. AGU*, 84(46), Fall Meet. Suppl., Abstract A31D-0077.
54. Wernli H., Bourqui, M. A. 2002. Lagrangian “1-year climatology” of (deep) cross tropopause exchange in the extra-tropical Northern Hemisphere. *J. Geo. Res.* 107: 4021.

Chapter 5

55. Planning and Technical Support Division, Air Resources Board. The 2003 California Almanac of Emissions and Air Quality.
56. Emission Inventory Branch, Air Resources Board. May 1996. Emission Inventory Procedure Manual, Volume I, Inventory Development Process.

Chapter 6

57. Air Resources Board. September, 1987. Effects of ozone on health, technical support document, pages 5-13.

58. Code of Federal Regulations. Title 40, Part 50, Appendix D, Measurement principles and calibration procedure for the measurement of ozone in the atmosphere.
59. Code of Federal Regulation. Title 40, Part 53, Revised requirements for designation of reference and equivalent methods for PM_{2.5} and ambient air quality surveillance for particulate matter. Pgs 1-28.
60. McElroy F, Dennis M, Monica N. 1997. Determination of ozone by ultraviolet analysis. A new method for Volume II, Ambient air specific methods, quality assurance handbook for air pollution measurement systems.
61. USEPA. 1996. Air Quality Criteria for ozone and related photochemical oxidants, EPA/600/P-93/004q-cF, pages 3-90 to 3-102. <http://cfpub.epa.gov/ncea/cfm/ozone.cfm>
62. Title 17, Barclay California Code of Regulation, section 70200.
63. USEPA/ORD (2002). List of designated reference and equivalent methods. <http://www.epa.gov/ttn/amtic/criteria.html>
64. Appendix A (1995). Dasibi model 1003 AH ozone analyzer. Volume II: standard operating procedures for air monitoring, air monitoring quality assurance, California Air Resources Board. <http://www.arb.ca.gov/aaqm/qmosqual/qamannual/qamannual.htm>
65. USEPA, 1984. Special Report, Issues Concerning the Use of Precision and Accuracy Data, EPA-450/4-84-006, pages 2 – 23. <http://www.epa.gov/ttn/amtic/cpreldoc.html>
66. USEPA, 1983. Guideline on the Meaning and Use of Precision and Accuracy Data Required By 40 CFR Part 58, Appendices A and B, EPA-600/4-83-023, pages 6-33. <http://www.epa.gov/ttn/amtic/cpreldoc.html>

Chapter 7

67. Air Resources Board. December 2002. Area Designations and Maps 2002.
68. Air Resources Board. December 2002. California Ambient Air Quality Data 1980-2001 Data CD.
69. Air Resources Board. January 20, 2005. ARB warns, danger from popular “air purifying” machines. Available from: <http://www.arb.ca.gov/newsrel/nr012005.htm>
70. Air Resources Board. January 2004. California Ambient Air Quality Data 1980-2002.
71. Air Resources Board. September 1993. Guidance for Using Air Quality-Related Indicators in Reporting Progress in Attaining the State Ambient Air Quality Standards.

72. Air Resources Board. December 2003. Proposed Amendments to the Area Designation Criteria and Area Designations for State Ambient Air Quality Standards and Maps of Area Designations for State and National Ambient Air Quality Standards.
73. Allen RJ, Wadden RA, Ross ED. 1978. Characterization of potential indoor sources of ozone. *Am Ind Hyg Assoc J* 39:466-471.
74. Atkinson R, and Arey J, 2003. Gas-phase tropospheric chemistry of biogenic volatile organic compounds: a review. *Atmospheric Environment* 37 Supp. 2: S197-S1219.
75. Avol EL, Navidi WC, Colome SD. 1998. Modeling ozone levels in and around Southern California homes. *Environ Sci Technol* 32:463-8.
76. Boeniger MF. 1995. Use of ozone generating devices to improve indoor air quality. *Am Ind Hyg Assoc J* 56: 590-8.
77. Brauer M, Brook JR. 1995. Personal and fixed-site ozone measurements with a passive sampler. *J Air Waste Manag Assoc* 45:529-37.
78. Brown SK, 1999. Assessment of Pollutant Emissions from Dry-Process Photocopiers, *Indoor Air, Denmark*, 9:259-267.
79. California Department of Health Services (DHS). 1997. News release (April 1997) available at <http://www.applications.dhs.ca.gov/pressreleases/store/PressReleases/27-97.html>
80. Chen, W and Zhang, JS. 2004. Effectiveness of Portable Room Air Cleaners for Control of Volatile Organic Compounds in Indoor Air, presented at CIB World Building Congress, Toronto.
81. Consumers Union. 1992. Household air cleaners. *Consum Rep Oct.*:657-62.
82. Delfino RJ, Coate BD, Zeiger RS, Seltzer JM, Street DH, Koutrakis P. 1996. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. *Am J Respir Crit Care Med* 154:633-41.
83. Geyh AS, Xue J, Ozkaynak H, Spengler JD. 2000. The Harvard Southern California Chronic Ozone Exposure Study: assessing ozone exposure of grade-school-age children in two southern California communities. *Environ Health Perspect* 108:265-70.
84. Gold DR, Allen G, Damokosh A, Serrano P, Hayes C, Castillejos M. 1996. Comparison of outdoor and classroom ozone exposures for school children in Mexico City. *J Air Waste Manag Assoc* 46:335-42.
85. Hansen RB, Andersen B. 1986. Ozone and other air pollutants from photocopying machines. *Am Ind Hyg Assoc J* 47:659-65.
86. Hayes SR. 1989. Estimating the effect of being indoors on total personal exposure to outdoor air pollution. *JAPCA* 39:1453-61.

87. Hayes SR. 1991. Use of an indoor air quality model (IAQM) to estimate indoor ozone levels. *Journal of Air and Waste Management Association* 41:161-70.
88. Health Canada, 1999. News release (April 13, 1999) available at http://www.hc-sc.gc.ca/english/protection/warnings/1999/99_62e.htm
89. Jakobi G, Fabian P. 1997. Indoor/outdoor concentrations of ozone and peroxyacetyl nitrate (PAN). *Int J Biometerol*. May 40:162-5.
90. Jenkins PL, Phillips TJ, Mulberg EJ, Hui SP. 1992. Activity patterns of Californians: use of and proximity to indoor pollutant sources. *Atmos Environ* 26:2141-2148.
91. Kissel JC. 1993. Potential impact of deliberately introduced ozone on indoor air quality. *J Expo Anal Environ Epidemiol* 3:155-64.
92. Kleno JG, Clausen PA, Weschler CJ, Wolkoff P. 2001. Determination of ozone removal rates by selected building products using the FLEC emission cell. *Environ Sci Technol*; 35:2548-2553.
93. Lee K, Vallarino J, Dumyahn T, Ozkaynak H, Spengler JD. 1999 . Ozone decay rates in residences. *J Air Waste Manag Assoc* 49(12):1238-44.
94. Lee K, Xue J, Geyh AS, Ozkaynak H, Leaderer BP, Weschler CJ, Spengler JD. 2002. Nitrous acid, nitrogen dioxide, and ozone concentrations in residential environments. *Environ Health Perspect* 110:145-50.
95. Leovic KW, Sheldon LS, Whitaker DA, Hates RG, Calcagni JA, Baskir JN. 1996. Measurement of indoor air emissions from dry-process photocopy machines. *J Air Waste Manag Assoc* 46:821-29.
96. Liou PJ. 1989. Exposure assessment of oxidant gases and acidic aerosols. *Annu Rev Public Health* 10:9-84.
97. Liu LJ, Delfino R, Koutrakis P. 1997. Ozone exposure assessment in a southern California community. *Environ Health Perspect* 105:58-65.
98. Liu LJ, Koutrakis P, Leech J, Broder I. 1995 . Assessment of ozone exposures in the greater metropolitan Toronto area. *J Air Waste Manag Assoc* 45:223-34.
99. Liu LJ, Koutrakis P, Suh HH, Mulik JD, Burton RM. 1993. Use of personal measurements for ozone exposure assessment: a pilot study. *Environ Health Perspec* 101:318-24.
100. Mason MA, Sparks LE, Moore SA, Dolgov I, and Perry RB, 2000. Characterization of ozone emissions from air cleaners equipped with ozone generators and sensor and feedback control circuitry. *Proceedings of the Engineering Solutions to Indoor Air Quality Problems Symposium, VIP-98, A&WMA*, pp. 254-269.

101. Moriske HJ, Ebert G, Konieczny L, Menk G, Schondube M. 1998. Concentrations and decay rates of ozone in indoor air in dependence on building and surface materials. *Toxicol Lett* 96-97:319-323.
102. Phillips TJ, Jenkins PL, Mulberg EJ. 1991. Children in California: activity patterns and presence of pollutant sources. Proceedings of the 84th Annual Meeting and Exhibition, Air & Waste Management Association 17: Paper no. 91-172.5, June 1991.
103. Phillips TJ, Bloudoff DP, Jenkins PL, Stroud KR. 1999. Ozone emissions from a personal air purifier. *J Expo Anal Environ Epidemiol* 9:594-601.
104. Reiss R, Ryan PB, Koutrakis P, Tibbetts SJ. 1995a. Ozone reactive chemistry on interior latex paint. *Environ Sci Technol*; 29:1906-12.
105. Reiss R, Ryan PB, Tibbetts SJ, Koutrakis P. 1995b. Measurement of organic acids, aldehydes, and ketones in residential environments and their relation to ozone. *J Air Waste Manag Assoc* 45:811-22.
106. Reiss R, Ryan PB, Koutrakis P. 1994. Modeling ozone deposition onto indoor residential surfaces. *Environ Sci Technol* 28:504-13.
107. Selway MD, Allen RJ, Wadden RA. 1980. Ozone production from photocopying machines. *Am Ind Hyg Assoc J* 41:455-9.
108. Shaughnessy RJ, Mcdaniels TJ, Weschler CJ. 2001. Indoor chemistry: ozone and volatile organic compounds found in tobacco smoke. *Environ Sci Technol*. 35:2758-64.
109. The Freedomia Group, 2004. Consumer Water Purification and Air Cleaning Systems to 2008—Market Size, Market Share, Market Leaders, Demand Forecast and Sales. Cleveland. <http://www.freedomiagroup.com>
110. Tuomi T, Engstrom B, Niemela R, Svinhufvud J, Reijula K. 2000. Emission of ozone and organic volatiles from a selection of laser printers and photocopiers. *Appl Occup Environ Hyg* 15:629-34.
111. U.S. Environmental Protection Agency. 1995. Office equipment: design, indoor air emissions, and pollution prevention opportunities. Washington, DC: Office of Research and Development. Report no. EPA/600/R-95/045.
112. United States Environmental Protection Agency. 1998. Guideline on Data Handling Conventions for the 8-Hour Ozone NAAQS: Appendix I to 40 CFR, Part 50, Interpretation of the 8-Hour Primary and Secondary National Ambient Air Quality Standards for Ozone. December 1998.our PRIMARY AND SECONDARY National Ambient Air Quality Standards for Ozone.
113. United States Environmental Protection Agency. 1986. Guideline on the Identification and Use of Air Quality Data Affected by Exceptional Events. July 1986. Publication No. EPA-450/4-86-007.

114. Wainman T, Zhang J, Weschler CJ, Liroy PJ. 2000. Ozone and limonene in indoor air: a source of submicron particle exposure. *Environ Health Perspect* 108:1139-45.
115. Weschler CJ, Shields HC, Naik DV. 1989. Indoor ozone exposures. *JAPCA* 39: 562-8.
116. Weschler CJ, Shields HC. 1999. Indoor ozone/terpene reactions as a source of indoor particles. *Atmos Environ* 33:2301-2312.
117. Weschler CJ, Shields HC. 2000. The influence of ventilation on reactions among indoor pollutants: modeling and experimental observations. *Indoor Air* 10:92-100.
118. Weschler CJ. 2000. Ozone in indoor environments: concentration and chemistry. *Indoor Air* 10:269-88.
119. Weschler, CJ, Hodgson, AT, Wooley, JD. 1992. Indoor chemistry: ozone, volatile organic compounds and carpets. *Environ Sci Technol* 26:2371-2377.
120. Weschler, CJ, Shields HC, Naik DV. 1994. Indoor chemistry involving O₃, NO, and NO₂ as evidenced by 14 months of measurements at a site in Southern California. *Environ Sci Technol* 28:2120-2132.
121. Wiley JA, Robinson JP, Cheng YT, Piazza T, Stork L, Pladsen K. 1991a. Activity patterns of California residents. Final report, ARB contract A733-149.
122. Wiley JA, Robinson JP, Piazza T, Garrett K, Cirksena K, Martin G. 1991b. Activity patterns of California residents. Final report, ARB contract A6-177-33.
123. Wilkins CK, Clausen PA, Wolkoff P, Larsen ST, Hammer M, Larsen K, Hansen V, and Nielsen GD, 2001. Formation of strong airway irritants in mixtures of isoprene/ozone and isoprene/ozone/nitrogen dioxide. *Environ Health Perspect* 109:937-941.
124. Wolkoff P, Johnsen C, Franck C, Wilhardt P, Albrechtsen O. 1992. A study of human reactions to office machines in a climatic chamber. *J Expo Anal Environ Epidemiol (Suppl.1)*:71-96.
125. Zhang J, Liroy PJ. 1994a. Ozone in residential air: concentrations, I/O ratios, indoor chemistry, and exposures. *Indoor Air* 2:95-105.
126. Zhang J, Wilson WE, Liroy PJ. 1994b. Sources of organic acids in indoor air: a field study. *J Expo Anal Environ Epidemiol* 4:25-47.

Chapter 8

127. Aber JD, Nadelhoffer KJ, Steudler P, Melillo JM. 1989. Nitrogen saturation in northern forest ecosystems. *Bioscience* 39:378-386.
128. Adams MB, Edwards NT, Taylor GE Jr, Skaggs BL. 1990. Whole-plant ¹⁴C-photosynthate allocation in *Pinus taeda*: Seasonal patterns at ambient and elevated ozone levels. *Can J For Res* 152-158.
129. Adams MB, Kelly JM, Edwards NT. 1988. Growth of *Pinus taeda* L. seedlings varies with family and ozone exposure level. *Water Air Soil Pollut* 38:137-150.
130. Adams RM, Glycer JD, Mccarl BA. 1988. The NCLAN economic assessment: approach, findings and implications. In: Heck WW, Taylor OC, Tingey DT, Eds. *Assessment Of Crop Loss From Air Pollutants: Proceedings Of An International Conference; October 1987; Raleigh, NC.* New York, NY: Elsevier Applied Science 473-504.
131. Adams, R.M., Hamilton, S.A., McCarl, B.A. 1986. The benefits of pollution control: the case of ozone and U.S. agriculture. *American Journal of Agricultural Economics* 68:886-893.
132. Air Resources Board, 1987. *Effect of Ozone On Vegetation And Possible Alternative Ambient Air Quality Standards: Technical Support Document* March 1987.
133. Air Resources Board (ARB). 2005. *The California Almanac of Emissions and Air Quality.* Sacramento, CA.
134. Ajax, R.L.; C.J. Conlee; J.B. Upham. 1967. The Effects of Air Pollution on the Fading of Dyed Fabrics, *JAPCA*, 17(4): 220-224.
135. Alscher RG, Amundson RG, Cumming JR, Fellows S, Fincher J, Rubin G, van Leuken P, Weinstein LH. 1989. Seasonal changes in pigments, carbohydrates, and growth of red spruce as affected by ozone. *New Phytol.* 113: 211-223.
136. Alscher, R. G.; Wellburn, A. R., Eds. 1994. *Plant Responses To The Gaseous Environment. Molecular, Metabolic And Physiological Aspects.* London, UK: Chapman and Hall.
137. Alscher, R., Donahue, J.L., Cramer, C.L., 1997. Reactive Oxygen Species And Antioxidants: Relationships In Green Cells. *Physiologia Plantarum* 100, 224-233.
138. Amthor JS, Cumming JR. 1988. Low Levels Of Ozone Increase Bean Leaf Maintenance Respiration. *Can J Bot*; 66: 724-726.
139. Amundson RG, Alscher RG, Fellows S, Rubin G, Fincher J, Van Leuken P, Weinstein LH. 1991. Seasonal Changes In The Pigments, Carbohydrates And Growth Of Red Spruce As Affected By Exposure To Ozone For Two Growing Seasons. *New Phytol* 118: 127-137.

140. Andersen CP. 2003. Source-sink balance and carbon allocation below ground in plants exposed to ozone. *New Phytol.* 157: 213-228.
141. Andersen, CP, Hogsett, WE, Wessling, R, Plocher, M, 1991. Ozone Decreases Spring Root-Growth And Root Carbohydrate Content In Ponderosa Pine The Year Following Exposure. *Canadian Journal of Forest Research* 21: 1288-1291.
142. Arbaugh MJ, Miller PR, Carroll JJ, Takemoto B, Procter T. 1998. Relationships of ozone exposure to pine injury in the Sierra Nevada and San Bernardino Mountains of California, USA. *Environ Pollut.* 101: 291-301.
143. Arbaugh MJ, Peterson DL, Miller PR. 1999. Air pollution effects on growth on ponderosa pine, Jeffrey pine, and Bigcone Douglas-fir. In: Miller PR, McBride JR, editors. *Oxidant air pollution impacts in the montane forests of southern California: a case study of the San Bernardino mountains.* New York: Springer. *Ecological Studies*, Vol 134, p. 179-207.
144. Arndt, U. 1995. Air Pollutants and Pheromones - A Problem? *Chemosphere* 30: 1023-1031.
145. Ashmore, M. R.; Bell, J. N. B.; Mimmack, A. 1988. Crop Growth Along A Gradient Of Ambient Air Pollution. *Environ. Pollut.* 53: 99-121.
146. Badiani, M.; Fuhrer, J.; Paolacci, A. R.; Sermanni, G. G.; Sermanni 1996. Deriving Critical Levels for Ozone Effects On Peach Trees (*Prunus Persica* (L.) Batsch) Grown In Open-Top Chambers In Central Italy. *Fresenius Envir. Bull* 5: 592-597.
147. Bahl, A.; Loitsch, S.M.; Kahl, G. 1995. Transcriptional Activation Of Plant Defence Genes By Short Term Air Pollutant Stress. *Environ. Pollut.* 89: 221-227
148. Balaguer, L., Barnes, J.D., Panicucci, A., Borland, A.M., 1995. Production And Utilization Of Assimilates In Wheat (*Triticum Aestivum* L.) Leaves Exposed To Elevated CO₂ and/or O₃. *New Phytol.* 129, 557-568.
149. Baldocchi, D.D., Hicks, B.B., Camara, P. 1987. A Canopy Stomatal Resistance Model for Gaseous Deposition To Vegetated Surfaces. *Atmos Environ.* 21, 91-101.
150. Barnard JE, Lucier AA, Brooks RT, Johnson AH, Dunn PH, and Karnosky DF. 1991. Changes in forest health and productivity in the United States and Canada. NAPAP Report 16. IN: Irving PM , ed. *Acidic Deposition: State of Science and Technology.* Vol. III. Terrestrial, Materials, Health and Visibility Effects. Washington, DC.: NAPAP.
151. Barnes JD, Davison AW. 1988. The influence of ozone on the winter hardiness of Norway spruce [*Picea abies* (L.) Karst.] *New Phytol.* 108: 159-166.

152. Barnes JD, Eamus D, Brown KA. 1990. The influence of ozone, acid mist, and soil nutrient status on Norway spruce [*Picea abies* (L.) Karst.] II. Photosynthesis, dark respiration, and soluble carbohydrates of trees during late autumn. *New Phytol.* 115: 149-156.
153. Barnes RL. 1972. Effects of chronic exposure to ozone on photosynthesis and respiration of pines. *Environ Pollut.* 3: 133-138.
154. Barnes, J.D., Davison, A.W., Booth, T.A., 1998. Ozone Accelerates Structural Degradation Of Epicuticular Wax On Norway Spruce Needles. *New Phytologist*, 110, 309-318.
155. Barnes, J.D., Ollerenshaw, J.H., Whitfield. C.P., 1995. Effects Of Elevated CO₂ and/or O₃ On Growth, Development And Physiology Of Wheat (*Triticum Aestivum* L.). *Global Change Biology* 1, 129-142.
156. Bastrup-Birk A, Brandt J, Zlatev Z. 1997. Studying cumulative ozone exposures in Europe during a 7-year period. *J Geophys Res.* 102(D20): 23,917-23,935.
157. Bauer MR, Hultman NE, Panek JA, Goldstein AH. 2000. Ozone deposition to a ponderosa pine plantation in the Sierra Nevada Mountains (CA): a comparison of two different climatic years. *J Geophys Res.* 105(D17): 22,123-22,136.
158. Beare, J. A.; Archer, S. A.; Bell, J. N. B. 1999. Marssonina Leafspot Disease of Poplar Under Elevated Ozone: Pre-Fumigated Host And In Vitro Studies. *Environ. Pollut.* 105: 409-417.
159. Bell, J. N. B., Marshall, F.M. 2000. Field Studies on Impacts of Air Pollution on Agricultural Crops. In: Agrawal, S.B. and Agrawal, M., eds. *Environmental Pollution and Plant Responses*. Lewis Publishers, London. Pp. 99-110.
160. Bell, J. N. B.; Treshow, M., Eds. 2002. *Air Pollution and Plant Life*. 2nd Ed. Chichester, UK: John Wiley & Sons.
161. Beloin, N.J. 1972. A Field Study: Fading of Dyed Fabrics by Air Pollution, *Textile Chem. Colorist*, 4:77-78.
162. Beloin, N.J. 1973. Fading of Dyed Fabrics Exposed to Air Pollutants, *Textile Chem. Colorist*, 5:128-133.
163. Bender, J.; Herstein, U.; Black, C. R. 1999. Growth and Yield Responses Of Spring Wheat To Increasing Carbon Dioxide, Ozone And Physiological Stresses: A Statistical Analysis Of 'SPACE-Wheat' Results. *Eur. J. Agron.* 10: 185-195.
164. Bender, J.; Weigel, H.-J.; Jäger, H.J. 1990. Regression Analysis to Describe Yield and Metabolic Responses of Beans (*Phaseolus Vulgaris*) to Chronic Ozone Stress. *Angew Bot* 64: 329-343.
165. Benner P, Wild A. 1987. Measurement of photosynthesis in spruce trees with various degrees of damage. *J Plant Physiol.* 129: 59-72.

166. Bennett, J.P., Oshima, R.J., Lippert, L.F. 1979. Effects of Ozone on Injury and Dry Matter Partitioning in Pepper Plants. *Environmental and Experimental Botany* 19: 33-39.
167. Benton, J.; Fuhrer, J.; Gimeno, B. S.; Skarby, L.; Palmer-Brown, D.; Balls, G.; Roadknight, C.; Mills, G. 2000. An International Cooperative Programme Indicates the Widespread Occurrence of Ozone Injury on Crops. *Agric. Ecosys. Environ.* 78: 19-30.
168. Bergweiler, C.J.; Manning, W.J. 1999. Inhibition Of Flowering and Reproductive Success in Spreading Dogbane (*Apocynum Androsaemifolium*) by Exposure to Ambient Ozone. *Environ. Pollut.* 105:333-339.
169. Beyers JL, Riechers GH, Temple PJ. 1992. Effects of Long-Term Ozone Exposure and Drought on the Photosynthetic Capacity of Ponderosa Pine (*Pinus Ponderosa* Laws.). *New Phytol.* 122: 81-90.
170. Bielenberg, D. G.; Lynch, J. P.; Pell, E. J. 2001. A Decline in Nitrogen Availability Affects Plant Responses to Ozone. *New Phytol.* 151: 413-425.
171. Black, V. J.; Black, C. R.; Roberts, J. A.; Stewart, C. A. 2000. Impact of Ozone on the Reproductive Development of Plants. *New Phytol.* 147: 421-447.
172. Blum, U., Mrozek Jr., E., Johnson. E., 1983. Investigation Of Ozone (O₃) Effects on ¹⁴C Distribution in Ladino Clover. *Environmental and Experimental Botany.* 23: 369-378.
173. Böhm M, McCune B, Vandetta T. 1991. Diurnal curves of tropospheric ozone in the western United States. *Atmos Environ.* 25A(8): 1,577-1,590.
174. Booker, F.L., 2000. Influence of Carbon Dioxide Enrichment, Ozone and Nitrogen Fertilization on Cotton (*Gossypium Hirsutum* L.) Leaf and Root Composition. *Plant Cell and Environment.* 23: 573-583.
175. Broadmeadow, M.S.J., Jackson, S.B. 2000. Growth Responses of *Quercus Petraea*, *Fraxinus Excelsior* and *Pinus Sylvestris* to Elevated Carbon Dioxide, Ozone and Water Supply. *New Phytol.* 146: 437-451.
176. Browning, V.D., Taylor, H.M., Huck, M.G., Klepper, B. 1975. Water Relations of Cotton: A Rhizotron Study. Auburn University, Agricultural Experiment Station Bulletin No. 467.
177. Butler, L.K., Tibbits, T.W. 1979. Stomatal Mechanisms Determining Genetic Resistance to Ozone in *Phaseolus Vulgaris* L. *Journal of The American Society Of Horticultural Science.* 104: 213-216.
178. Bytnerowicz A, Miller PR, Olszyk DM, Dawson PJ, Fox CA. 1987. Gaseous and particulate air pollution in the San Gabriel mountains of southern California. *Atmos Environ.* 21(8): 1,805-1,814.

179. Bytnerowicz A, Olszyk DM, Dawson PJ, Morrison L. 1989a. Effects of air filtration on concentration and deposition of gaseous and particulate air pollutants in open-top field chambers. *J Environ Qual.* 18(3); 268-273.
180. Bytnerowicz A, Olszyk DM, Huttunen S, Takemoto B. 1989b. Effects of photochemical smog on growth, injury, and gas exchange of pine seedlings. *Can J Bot.* 67: 2,175-2,181.
181. Bytnerowicz A, Takemoto BK. 1989. Effects of photochemical smog on the growth and physiology of ponderosa pine seedlings grown under nitrogen and magnesium deficiencies. In: Olson RK, Lefohn AS, editors. *Effects of air pollution on western forests.* APCA Trans Ser. 16: 455-467.
182. Cahill TA, Annegarn H, Ewell D, Pedersen B. 1989. *Monitoring of Atmospheric Particles and Ozone in Sequoia National Park: 1985-1987.* Sacramento, CA. ARB Final Report No. A5-180-32. NTIS No. PB90157512.
183. California Department of Forestry and Fire Protection (CDF). 1988. *California's Forests and Rangelands: Growing Conflict over Changing Uses.* Sacramento, CA. Forest and Rangeland Resources Assessment Program.
184. Cardoso-Vilhena, J.; Barnes, J. 2001. Does Nitrogen Supply Affect the Response Of Wheat (*Triticum Aestivum* Cv. Hanno) To The Combination Of Elevated CO₂ and O₃? *J. Exp. Bot.* 52: 1901-1911.
185. Carroll JJ, Dixon AJ. 1993. *Sierra Cooperative Ozone Impact Assessment Study: Year 3.* Sacramento, CA. ARB Final Report No. A132-188. NTIS No. PB94208865.
186. Carroll JJ, Dixon AJ. 1995. *Sierra Cooperative Ozone Impact Assessment Study: Year 4. Volume 1.* Sacramento, CA. ARB Final Report No. 92-346. NTIS No. PB96195912.
187. Carroll JJ. 1992. *Sierra Cooperative Ozone Impact Assessment Study: Year 2.* Sacramento, CA. ARB Final Report, No. A032-129. NTIS No. PB93210292.
188. Carroll JJ. 1991. *Sierra Ozone Impact Assessment Study.* Sacramento, CA. ARB Final Report, No. A933-097. NTIS No. PB92104660.
189. Castillo F J ; Heath R L. 1990a. Active and Passive Calcium Transport Are Affected by Ozone Exposure in Leaf Plant Membranes. *Physiologia Plantarum.* 79: A124.
190. Castillo F J; Heath R L. 1990b. Calcium Ion Transport in Membrane Vesicles from Pinto Bean Leaves and Its Alteration after Ozone Exposure. *Plant Physiology (Rockville).* 94(2). 788-795.
191. Chapin FS III, Bloom AJ, Field CB, Waring RH. 1987. Plant responses to multiple environmental factors. *Bioscience.* 37: 49-57.

192. Chappelka, A. H.; Freer-Smith, P. H. 1995. Predisposition of Trees by Air Pollutants to Low Temperatures and Moisture Stress. *Environ. Pollut.* 87: 105-117.
193. Chappelka, A. H.; Samuelson, L. J. 1998. Ambient Ozone Effects on Forest Trees of The Eastern United States: A Review. *New Phytol.* 139: 91-108.
194. Chappelka, A.H., Chevone, B.I. 1988. Growth and Physiological Responses of Yellow-Poplar Seedlings Exposed to Ozone and Simulated Acidic Rain. *Environmental Pollution.* 49: 1-18.
195. Chappelka, A.H., Chevone, B.I., Burk, T.E. 1988. Growth Response of Green and White Ash Seedlings to Ozone, Sulphur Dioxide and Simulated Acid Rain. *Forest Science.* 34: 1016-1029.
196. Colbeck I, Harrison RM. 1985. Dry deposition of ozone: Some measurements of deposition velocity and of vertical profiles to 100 metres. *Atmos Environ.* 19: 1,807-1,818.
197. Coleman, M.D., Dickson, R.E., Isebrands, J.G., Karnosky, D.F. 1995. Carbon Allocation and Partitioning in Aspen Clones Varying in Sensitivity to Tropospheric Ozone. *Tree Physiology.* 15: 593-604.
198. Colls, J. J.; Baker, C. K. 1988. The methodology of open-field fumigation. In: Mathy, P., ed. *Air Pollution and Ecosystems, Proceedings of an International Symposium, Grenoble, France, 18-22 May, 1987.* Reidel Publishing, Dordrecht. pp. 361-371.
199. Constantinidou HA, Kozlowski TT. 1979. Effects of Sulfur Dioxide and Ozone on *Ulmus Americana* Seedlings: II. Carbohydrates, Proteins and Lipids. *Can J Bot.* 57(2): 176-184.
200. Cooley DR, Manning WJ. 1987. The Impact of Ozone on Assimilate Partitioning in Plants: A Review. *Environmental Pollution.* 47(2). 95-113.
201. Cooley, DR. Manning, W. J. 1988. Ozone Effects on Growth and Assimilate Partitioning in Alfalfa, *Medicago Sativa* L. *Environ. Pollut.* 49: 19-36.
202. Coyne PI, Bingham GE. 1981. Comparative ozone dose response of gas exchange in a ponderosa pine stand exposed to long-term fumigations. *J Air Pollut Contr Assoc.* 31(1): 38-41.
203. Coyne PI, Bingham GE. 1982. Variation in photosynthesis and stomatal conductance in an ozone-stressed ponderosa pine stand: Light response. *For Sci.* 28(2): 257-273.
204. Craigon, J., Fangmeier, A., Jones, M, Donnelly, A., Bindi, M., De Temmerman, L., Persson, K., Ojanpera, K. 2002. Growth and marketable-yield responses of potato to increased CO₂ and O₃. *European Journal of Agronomy.* 17: 273-289.
205. Craker LE, Starbuck JS. 1972. Metabolic Changes Associated With Ozone Injury Of Bean Leaves. *Can J Plant Sci* 52: 589-597.

206. Crisosto, C. H.; Retzlaff, W. A.; Williams, L. E.; Dejong, T. M.; Zoffoli, J. P. 1993. Postharvest Performance Evaluation of Plum (*Prunus Salicina* Lindel., 'Casselman') Fruit Grown Under Three Ozone Concentrations. *J. Amer. Soc. Hort. Sci.* 118 (4): 497-502.
207. Dale JW, technical coordinator. 1996. California Forest Health in 1994 and 1995. San Francisco, CA: USDA, Forest Service, Pacific Southwest Region. Report R-5-FPM-PR-002.
208. Danielsson, H.; Karlsson, G. P.; Karisson, P. E.; Pleijel, J. 2003. Ozone Uptake Modelling and Flux-Response Relationships--An Assessment of Ozone-Induced Yield Loss in Spring Wheat. *Atmos. Environ.* 37: 475-485.
209. Dann, M.S., Pell, E.J. 1989. Decline of Activity and Quantity of Ribulose Bisphosphate Carboxylase Oxygenase and Net Photosynthesis in Ozone-Treated Potato Foliage. *Plant Physiology.* 91: 427-432.
210. Darrall, N.M., 1989. The Effect of Air Pollutants on Physiological Processes in Plants. *Plant, Cell And Environment.* 12: 1-30.
211. Davison, A.W., Barnes, J.D., 1998. Effects of Ozone on Wild Plants. *New Phytol.* 139:135-151.
212. De Santis, F. 1999. New Directions: Will A New European Vegetation Ozone Standard Be Fair To All European Countries? *Atmos. Environ.* 33: 3873-3874.
213. De Santis, F. 2000. New Directions: Use and Abuse of the AOT40 Concept. *Atmos Environ.* 34:1158-1159.
214. De Temmerman, L.; Vandermeiren, K.; Guns, M. 1992. Effects of Air Filtration Onspring Wheat Grown in Open-Top Field Chambers at a Rural Site. I. Effect on Growth, Yield and Dry Matter Partitioning. *Environ. Pollut.* 77: 1-5
215. Dickson RE, Isebrands JG. 1993. Carbon Allocation Terminology: Should It Be More Rational? *Bull Ecol Soc Am.* 74:175-177.
216. Dickson, R.E., Coleman, M.D., Riemenschneider, D.E., Isebrands, J.G., Hogan, G.D., Einig, W., Lauxmann, U., Hauch, B., Hampp, R., Landolt, W., Maurer, S., Matyssek, R., 1997. Ozone-Induced Accumulation of Carbohydrates Changes Enzyme Activities of Carbohydrate Metabolism in Birch Leaves. *New Phytol.* 137:673-680.
217. Docherty, M.; Salt, D. T.; Holopainen, J. K. 1997. The Impacts of Climate Change and Pollution on Forest Pests. In: Watt, A. D.; Stork, N. E.; Hunter, M. D., Eds. *Forests and Insects.* New York, NY: Chapman and Hall; Pp. 229-247.
218. Dodd, I.C., Stikic, R., Davies, W.J., 1996. Chemical Regulation of Gas Exchange and Growth of Plants in Drying Soil in the Field. *Journal of Experimental Botany.* 47:1475-1490.

219. Dominy, P. J.; Heath, R. L. 1985. Inhibition of the K⁺-Stimulated Atpase of the Plasmalemma of Pinto Bean (*Phaseolus vulgaris*) Leaves by Ozone. *Plant Physiol.* 77: 43-45.
220. Drisko, K.; G.R. Cass, J.R. Druzik. 1984. Fading of Artists' Pigments Due to Atmospheric Ozone. Presented at 77th Annual meeting of the APCA, San Francisco, CA, June 24-29, 1984
221. Drogoudi, P. D; Ashmore, M. R. 2000. Does Elevated Ozone Have Differing Effects in Flowering and Deblossomed Strawberry? *New Phytol.* 147:561-569.
222. Eason, G., Reinert, R. A. 1991. Responses Of Closely Related Bush Blue Lake Snap Bean Cultivars to Increasing Concentrations of Ozone. *J. Am. Soc. Hortic. Sci.* 116: 520-524.
223. Einig, W., Lauxmann, U., Hauch, B., Hampp, R., Landolt, W., Maurer, S., Matyssek, R., 1997. Ozone-Induced Accumulation of Carbohydrates Changes Enzyme Activities of Carbohydrate Metabolism in Birch Leaves. *New Phytol.* 137: 673-680.
224. Eissenstat, D. M.; Syvertsen, J. P.; Dean, T. J.; Yelenosky, G.; Johnson, J. D. 1991a. Sensitivity of frost resistance and growth in citrus and avocado to chronic ozone exposure. *New Phytol.* 118:139-146.
225. Elagoz, V., Manning, W.J. 2002. Ozone and bean plants: morphology matters. *Environmental Pollution* 120: 521-524.
226. Elkiey, T., Ormrod, D.P. 1979. Ozone and/or Sulfur Dioxide Effects On Tissue Permeability Of Petunia Leaves. *Atmos Environ.* 13(8). 1165-1168.
227. Emberson, L.; Ashmore, M. R.; Cambridge, H. M.; Simpson, D Tuovinen, J.-P. 2000. Modelling Stomatal Ozone Flux Across Europe. *Environ. Pollut.* 109: 403-413.
228. Evans LS, Ting IP. 1973. Ozone-Induced Membrane Permeability Changes. *Amer J Bot.* 60(2): 155-162.
229. Evans, P. A.; Ashmore, M. R. 1992. The Effects of Ambient Air on a Semi-Natural Grassland Community. *Agric. Ecosyst. Environ.* 38: 91-97.
230. Fangmeier, A., Brockerhoff, U., Gruters, U., Jager, H.J., 1994b. Growth And Yield Responses Of Spring Wheat (*Triticum Aestivum* L. Cv. Turbo) Grown In Open-Top Chambers to Ozone and Water-Stress. *Environmental Pollution.* 83: 317-325.
231. Fangmeier, A.; Brunschon, S.; Jager, H.-J. 1994a. Time Course of Oxidant Stress Biomarkers in Flag Leaves of Wheat Exposed to Ozone and Drought Stress. *New Phytol.* 126: 63-69.
232. Farage, P.K., Long, S.P. 1995. An In Vivo Analysis of Photosynthesis during Short-Term O₃ Exposure in Three Contrasting Species. *Photosynthesis Research.* 43: 11-18.

233. Farage, P.K., Long, S.P., Lechner, E.G., Baker, N.R., 1991. The Sequence of Change within the Photosynthetic Apparatus of Wheat Following Short-Term Exposure to Ozone. *Plant Physiology*, 95: 529-535.
234. Fenn ME, Poth MA, Johnson DW. 1996. Evidence for Nitrogen Saturation in the San Bernardino Mountains in Southern California. *For Ecol Manage*; 82: 211-230.
235. Fernandez-Bayon, J.M., Barnes, J.D., Ollerenshaw, J.H., Davison, A.W., 1993. Physiological Effects of Ozone on Cultivars of Watermelon (*Citrullus Lanatus*) And Muskmelon (*Cucumis Melo*) Widely Grown in Spain. *Environmental Pollution*, 81: 199-206.
236. Fialho, R.C., Bucker, J. 1996. Changes in Levels of Foliar Carbohydrates and Myoinositol before Premature Leaf Senescence of *Populus Nigra* Induced by a Mixture of O₃ and SO₂. *Canadian Journal Botany* 74: 965-970.
237. Finnan, J. M.; Jones, M. B.; Burke, J. I. 1996a. A Time-Concentration Study on the Effects of Ozone on Spring Wheat (*Triticum Aestivum* L.) .1. Effects on Yield. *Agriculture Ecosystems & Environment* 57: 159-167.
238. Flagler, R.B., 1998. Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas. 2nd Edition. Air and Waste Management Assoc., Pittsburgh.
239. Fluckiger, W.; Braun, S.; Hiltbrunner, E. 2002. Effects of Air Pollutants on Biotic Stress. In: Bell, J. N. B.; Treshow, M., Eds. *Air Pollution and Plant Life*. 2nd Ed. Chichester, UK: John Wiley and Sons. Pp. 379-406.
240. Foot, J. P.; Caporn, S. J. M.; Lee, J. A.; Ashenden, T. W. 1997. Evidence that Ozone Exposure Increases the Susceptibility of Plants to Natural Frosting Episodes. *New Phytol.* 135: 369-374.
241. Foot, J.P., Caporn, S.J.M., Lee, J.A., Ashenden, T.W., 1996. The Effect of Long-Term Ozone Fumigation on the Growth, Physiology and Frost Sensitivity of *Calluna Vulgaris*. *New Phytologist*. 133: 503-511.
242. Forberg, E., Aarnes, H., Nilsen, S., 1987. Effects of Ozone on Net Photosynthesis in Oat (*Avena Sativa*) and Duckweed (*Lemna Gibba*). *Environmental Pollution*. 47: 285-291.
243. Fredericksen, T. S.; Kolb, T. E.; Skelly, J. M.; Steiner, K. C.; Joyce, B. J.; Savage, J. E. 1996a. Light Environment Alters Ozone Uptake per Net Photosynthetic Rate in Black Cherry Trees. *Tree Physiol.* 16: 485-490.
244. Fredericksen, T. S.; Skelly, J. M.; Snyder, K. R.; Steiner, K. C. 1996b. Predicting Ozone Uptake from Meteorological and Environmental Variables. *J. Air Waste Manage. Assoc.* 46: 464-469.
245. Friend, A.L., Tomlinson, P.T., 1992. Mild Ozone Exposure Alters ¹⁴C Dynamics in Foliage of *Pinus Taeda* L. *Tree Physiology.* 11: 215-227.
246. Fuhrer J., Skärby L., Ashmore M.R. 1997. Critical Levels for Ozone Effects on Vegetation in Europe. *Environ Pollut.* 97: 91-106.

247. Fuhrer, J. 1994. Effects of ozone on managed pasture: I. Effects of open-top chambers on microclimate, ozone flux, and plant growth. *Environmental Pollution*. 86: 297-305.
248. Fuhrer, J.; Egger, A.; Lehnherr, B.; Grandjean, A.; Tschannen, W. 1989. Effects of Ozone on The Yield of Spring Wheat (*Triticum Aestivum* L., Cv. Albis) Grown in Open-Top Field Chambers. *Environ. Pollut.* 60: 273-289.
249. Fuhrer, J.; Grimm, A. G.; Tschannen, W.; Shariat-Madari, H. 1992. The Response of Spring Wheat (*Triticum Aestivum* L.) to Ozone at Higher Elevations. *New Phytol.* 121: 211-219.
250. Fumagalli, I.; Ambrogi, R.; Mignanego, L. 2001a. Yield Responses of Plants Exposed to Ambient Ozone in the River Po Valley (Italy). *Agronomie*. 21: 227-233.
251. Fumagalli, I.; Gimeno, B. S.; Velissariou, D.; De Temmerman, L.; Mills, G. 2001b. Evidence of Ozone-Induced Adverse Effects on Crops in the Mediterranean Region. *Atmospheric Environment*. 35: 2583-2587.
252. Gate, I. M.; McNeill, S.; Ashmore, M. R. 1995. Effects of Air Pollution on the Searching Behaviour of an Insect Parasitoid. *Water Air Soil Pollut.* 85: 1425-1430.
253. Gelang, J.; Pleijel, H.; Sild, E.; Danielsson, H.; Younis, S.; Sellden, G. 2000. Rate and Duration of Grain Filling in Relation to Flag Leaf Senescence and Grain Yield in Spring Wheat (*Triticum Aestivum*) Exposed to Different Concentrations of Ozone. *Physiologia Plantarum*. 110: 366-375.
254. Gimeno, B. S.; Bermejo, V.; Reinert, R. A.; Zheng, Y. B.; Barnes, J. D. 1999. Adverse Effects of Ambient Ozone on Watermelon Yield and Physiology at a Rural Site in Eastern Spain. *New Phytologist*. 144: 245-260.
255. Goldschmidt, E.E., Huber, S.C., 1992. Regulation of Photosynthesis by End-Product Accumulation in Leaves of Plants Storing Starch, Sucrose, and Hexose Sugars. *Plant Physiology*. 99: 1443-1448.
256. Goldstein AH, Panek J. ~~YEAR~~ 2002. Modeling ozone flux to forests across an ozone concentration gradient in the Sierra Nevada Mountains, California. Final Report Summary for EPA Agreement No. R826601.
257. Gorissen, A., Van Veen, J.A. 1988. Temporary Disturbances of Translocation of Assimilates in Douglas Firs Caused by Low Levels of Ozone and Sulphur Dioxide. *Plant Physiology*. 88: 559-563.
258. Grandjean, A.; Fuhrer, J. 1989. Growth and Leaf Senescence in Spring Wheat (*Triticum Aestivum*) Grown at Different Ozone Concentrations in Open-Top Field Chambers. *Physiol. Plant.* 77: 389-394.
259. Grantz D.A., Farrar J.F. 2000. Ozone Inhibits Phloem Loading from a Transport Pool: Compartmental Efflux Analysis in Pima Cotton. *Australian Journal of Plant Physiology*. 27(8-9): 859-868.

260. Grantz, D. A., J. H. B. Garner, and D. W. Johnson. 2002. Ecological effects of particulate matter. *Environment International*. 29: 213-239.
261. Grantz, D.A. 1990. Response of Plants to Humidity. *Plant Cell and Environment*. 13:667-679.
262. Grantz, D.A., Farrar, H., 1999. Acute Exposure to Ozone Inhibits Rapid Carbon Translocation from Source Leaves of Pima Cotton. *Journal of Experimental Botany*. 50: 1253-1262.
263. Grantz, D.A., Macpherson, J.I., Massman, W.J. Pederson, J., 1994. Aircraft Study Demonstrates Measurements Demonstrate Ozone Uptake by San Joaquin Valley Crops. *California Agriculture*. 48:9-12.
264. Grantz, D.A., Mccool, P.H., 1992. Effect of Ozone on Pima and Acala Cottons in the San Joaquin Valley. In: Herber, D.J., Richter, D.A., (Eds.), *Proceedings 1992 Beltwide Cotton Conferences, Vol 3*. National Cotton Council of America, Memphis, TN. Pp. 1082-1084.
265. Grantz, D.A., Yang, S., 2000. Ozone Impacts on Allometry and Root Hydraulic Conductance Are Not Mediated by Source Limitation Nor Developmental Age. *Journal of Experimental Botany*. 51: 919-927.
266. Grantz, D.A., Yang, S., 1996. Effects of O₃ on Hydraulic Architecture in Pima Cotton. Biomass Allocation and Water Transport Capacity of Roots and Shoots. *Plant Physiology*. 112: 1649-1657.
267. Grantz, D.A., Zhang, X., Carlson, T.N., 1999. Observations and Model Simulations Link Stomatal Inhibition to Impaired Hydraulic Conductance Following Ozone Exposure in Cotton. *Plant Cell and Environment*. 22: 1201-1210.
268. Grantz, D.A., Zhang, X.J., Massman, W.J., Delany, A., Pederson, J.R., 1997. Ozone Deposition to a Cotton (*Gossypium Hirsutum* L.) Field: Stomatal and Surface Wetness Effects during the California Ozone Deposition Experiment. *Agricultural and Forest Meteorology*. 85: 19-31.
269. Grantz, D.A., Zhang, X.J., Massman, W.J., Den Hartog, G., Neumann, H.H., Pederson, J.R., 1995. Effects of Stomatal Conductance and Surface Wetness on Ozone Deposition in Field-Grown Grape. *Atmospheric Environment*. 29: 3189-3198.
270. Greitner, C.S., Pell, E.J., Winner, W.E. 1994. Analysis of aspen foliage exposed to multiple stresses: ozone, nitrogen deficiency and drought. *New Phytologist*. 127:579-589.
271. Grulke N.E., Andersen C.P., Hogsett W.E. 2001. Seasonal Changes in Above- and Below-ground Carbohydrate Concentrations of Ponderosa Pine Along a Pollution Gradient. *Tree Physiology*. 21(2-3):173-181.
272. Grulke NE, Andersen CP, Fenn ME, Miller PR. 1998. Ozone and nitrogen deposition reduces root biomass of ponderosa pine in the San Bernardino Mountains, California. *Environ Pollut*. 103: 63-73.

273. Grulke NE. 1999. Physiological Responses of Ponderosa Pine to Gradients of Environmental Stressors. In: Miller PR, McBride JR, Editors. Oxidant Air Pollution Impacts in the Montane Forests of Southern California: A Case Study of the San Bernardino Mountains. New York: Springer; Ecological Studies, Vol 134, Pgs. 126-163.
274. Grulke, N.E., Balduman, L., 1999. Deciduous Conifers: High N Deposition and O₃ Exposure Effects on Growth and Biomass Allocation in Ponderosa Pine. *Water, Air and Soil Pollution*. 116: 235-248.
275. Grunhage, L., Jager, H.-J., Haenel, H.-D., Hanewald, K., Krupa, S.V., 1997. PLATIN (Plant-Atmosphere Interaction): II. Co-occurrence of High Ambient O₃ Concentrations and Factors Limiting Plant Absorbed Dose. *Environmental Pollution*. 98: 51-60.
276. Grunhage, L.; Jager, H.-J. 1994. Influence of the Atmospheric Conductivity on the Ozone Exposure of Plants under Ambient Conditions: Considerations for establishing ozone standards to protect vegetation. *Environmental Pollution*. 85: 125-129.
277. Grunhage, L.; Jager, H.-J.; Haenel, H.-D.; Lopmeier, F.-J.; Hanewald, K. 1999. The European Critical Levels for Ozone: Improving Their Usage. *Environ. Pollut.* 105: 163-173.
278. Grunhage, L.; Krause, G. H. M.; Kollner, B.; Bender, J.; Weigel, H. J.; Jager, H. J.; Guderian, R. 2001. A New Flux-Orientated Concept to Derive Critical Levels for Ozone to Protect Vegetation. *Environmental Pollution*. 111: 355-362.
279. Guidi, L., Nali, C., Ciompi, S., Lorenzini, G., Soldatini, G.F. 1997. The Use Of Chlorophyll Fluorescence And Leaf Gas Exchange As Methods For Studying The Different Responses To Ozone Of Two Bean Cultivars. *Journal Of Experimental Botany*, 48, 173-179.
280. Guidi, L.; Tonini, M.; Soldatini, G. F. 2000. Effects of High Light and Ozone Fumigation on Photosynthesis in *Phaseolus Vulgaris*. *Plant Physiol. Biochem.* 38: 717-725.
281. Gunn, S., Bailey, S.J., Farrar, J.F. 1999. Partitioning of Dry Weight and Leaf Area Within Plants of Three Native Species Grown at Elevated CO₂. *Functional Ecology*. 13: 3-11.
282. Hanson, G.P., Stewart, W.S. 1970. Photochemical Oxidants: Effect on Starch Hydrolysis in Leaves. *Science*. 168: 1223-1224.
283. Harris, M.J., Heath, R.L. 1981. Ozone Sensitivity in Sweet Corn (*Zea Mays* L.) Plants: A Possible Relationship to Water Balance. *Plant Physiology*. 68: 885-890.
284. Hausladen A, Madamanchi NR, Alscher RG, Amundson RG, Fellows S. 1990. Seasonal changes in antioxidants in red spruce as affected by ozone. *New Phytol.* 115: 447-458.

285. Haynie, F.H.; J.W. Spence; J.B. Upham. 1976. Effects of Gaseous Pollutants on Materials – A Chamber Study. EPA-600/3-76-015, U.S. EPA, RTP, NC.
286. Haynie, F.H.; Spence, J.W. 1984. Air Pollution to Exterior Household Paints. JAPCA 34: 941-44.
287. Heagle, A. S. 1989. Ozone and Crop Yield. Annu. Rev. Phytopathol. 27: 397-423.
288. Heagle, A. S.; Brandenburg, R. L.; Burns, J. C.; Miller, J. E. 1994. Ozone and Carbon Dioxide Effects on Spider Mites in White Clover and Peanut. J. Environ. Qual. 23: 1168-1176.
289. Heagle, A. S.; Miller, J. E.; Booker, F. L.; Pursley, W. A. 1999. Ozone Stress, Carbon Dioxide Enrichment, And Nitrogen Fertility Interactions In Cotton. Crop Sci. 39: 731-741.
290. Heagle, A. S.; Miller, J. E.; Chevone, B. I.; Dreschel, T. W.; Manning, W. J.; Mccool, P. M.; Morrison, C. L.; Neely, G. E.; Rebbeck, J. 1995. Response of a White Clover Indicator System to Tropospheric Ozone at Eight Locations in the United States. Water Air And Soil Pollution 85: 1373-1378.
291. Heagle, A. S.; Miller, J. E.; Pursley, W. A. 2000. Growth And Yield Responses Of Winter Wheat To Mixtures Of Ozone And Carbon Dioxide. Crop Sci.40: 1656-1664.
292. Heagle, A. S.; Philbeck, R. B.; Ferrell, R. E.; Heck, W. W. 1989a. Design And Performance Of A Large, Field Exposure Chamber To Measure Effects Of Air Quality On Plants. J. Environ. Qual. 18: 361-368.
293. Heagle, A. S.; Heck, W. W.; Lesser, V. M.; Rawlings, J. O.; Mowry, F.L. 1986. Injury and yield response of cotton to chronic doses of ozone and sulfur-dioxide. Journal of Environ. Qual. 15: 375-379.
294. Heagle, A. S.; Philbeck, R. B.; Heck-, W. W. 1973. An Open-Top Chamber to Assess the Impact of Air Pollution on Plants. Journal of Environmental Quality 2: 365-368.
295. Heagle, A. S.; Philbeck, R. B.; Rogers, H. H.; Letchworth, M. B. 1979b. Dispensing and Monitoring Ozone in Open-Top Field Chambers for Plant-Effects Studies. Phytopathology. 69: 15-20.
296. Heagle, A. S.; Rebbeck, J.; Shafer, S. R.; Blum, U.; Heck, W. W. 1989b. Effects of long-term ozone exposure and soil moisture deficit on growth of a ladino clover-tall fescue pasture. Phytopathology. 79: 128-136.

297. Heagle, A. S.; Kress, L. W.; Temple, P. J.; Kohut, R. J.; Miller, J. E.; Heggestad, H. E. 1988a. Factors Influencing Ozone Dose-Yield Response Relationships in Open-Top Field Chamber Studies. In: Heck, W. W.; Taylor, O. C.; Tingey, D. T., Eds. Assessment of Crop Loss from Air Pollutants: Proceedings of an International Conference; October, 1987; Raleigh, NC. New York, NY: Elsevier Applied Science. London: Elsevier Press. Pp. 141-179.
298. Heagle, A.S., Cure, W.W., Rawlings, J.O. 1985. Response of turnips to chronic doses of ozone in open-top chambers. *Environmental Pollution*. 38:305-319.
299. Heagle, A.S., Miller, J.E., Heck, W.W., Patterson, R.P. 1988b. Injury and yield response of cotton to chronic doses of ozone and soil moisture deficit. *Journal of Environmental Quality*. 17: 627-635.
300. Heagle, A.S., Reinert, R.A., Miller, J.E. 1996. Response of white clover to ozone in different environments. *Journal of Environmental Quality*. 25:273-278.
301. Heath, R.L., 1980. Initial Events in Injury to Plants by Air Pollutants. *Annual Review of Plant Physiology*. 31: 395-431.
302. Heath, R.L., 1988. Biomechanical Mechanisms of Pollutant Stress. In: Heck, W.W., Tingey, D.T., Taylor, O.C., Eds. Assessment of Crop Loss from Air Pollutants. Elsevier, London. Pp. 259-286.
303. Heck W.W.; Taylor O C; Adams R; Bingham G; Miller J; Preston E; Weinstein L. 1982. Assessment of Crop Loss from Ozone. *Journal of the Air Pollution Control Association*. 32(4): 353-361.
304. Heck WW, Furiness C.S. 2001. The Effects of Ozone on Ecological Systems: Time for a Full Assessment. *Env. Mgr.* October: 15-24.
305. Heck, W. W.; Dunning, J. A.; Reinert, R. A.; Prior, S. A.; Rangappa, M.; Benepal, P. S. 1988. Differential Responses of Four Bean Cultivars to Chronic Doses of Ozone. *J. Am. Soc. Hortic. Sci.* 113: 46-51.
306. Heck, W. W.; Furiness, C. S.; Cowling, E. B.; Sims, C. K. 1998. Effects of Ozone on Crop, Forest, and Natural Ecosystems: Assessment Of Research Needs. *Env. Mgr.* October: 11-22.
307. Heck, W.W., 1989. Assessment of Crop Loss from Air Pollutants in the United States. In: Mackenzie, J.J. And El-Ashry, M.T. Air Pollution's Toll On Forest And Crops. Yale University Press, New Haven, Chapter 6, Pgs. 235-315.
308. Heck, W.W., Cure, W., Rawlings, J., Zaragoza, L., Heagle, A. et al., 1984. Assessing impacts of ozone on agricultural crops. II. Crop yield functions and alternative exposure statistics. *J. Air Pollut. Control Assoc.* 34:810-817.

309. Heck, W.W., Taylor, O.C., Tingey, D.T. 1988a. Assessment of Crop Loss from Air Pollutants: Proceedings of an International Conference, Raleigh, North Carolina, USA, October 25-29, 1987. Elsevier Applied Science, New York.
310. Heggstad, H.E., Anderson, E.L., Gish, T.J., Lee, E.H. 1988. Effects of Ozone and Soil Water Deficit on Roots and Shoots of Field-Grown Soybeans. *Environmental Pollution*. 50: 259-278.
311. Heggstad, H.E., Gish, T.J., Lee, E.H., Bennett, J.H., Douglass, L.W., 1985. Interaction of Soil Moisture Stress and Ambient Ozone on Growth and Yield of Soybeans. *Phytopathology*. 75: 472-477.
312. Heggstad, H.E., Lee, E.H., 1990. Soybean Root Distribution, Top Growth and Yield Response to Ambient Ozone and Soil Moisture Stress When Grown in Soil Columns in Greenhouses. *Environmental Pollution*. 65: 195-207.
313. Hendrix, D.L., Grange, R.I., 1991. Carbon Partitioning and Export from Mature Cotton Leaves. *Plant Physiology*. 95: 228-233.
314. Hendrix, D.L., Peelen, K.K. 1987. Artifacts in the Analysis of Plant-Tissues for Soluble Carbohydrates. *Crop Science*. 27: 710-715.
315. Hicks, B.B., Baldocchi, D.D., Meyers, T.P., Hosker, R.P., Jr., Man, D.R., 1987. A Preliminary Multiple Resistance Routine for Deriving Dry Deposition Velocities from Measured Quantities. *Water Air and Soil Pollution*. 36: 311-330.
316. Hill, A.C., Littlefield, N., 1969. Ozone: Effect on Apparent Photosynthesis, Rate of Transpiration and Stomatal Closure in Plants. *Environmental Science and Technology*. 3:52-56.
317. Ho, L.C., 1976. The Relationship between the Rates of Carbon Transport and of Photosynthesis in Tomato Leaves. *Journal of Experimental Botany*. 27: 87-97.
318. Hogsett, W.; Tingey, D. T.; Lee, E. H. 1988. Ozone Exposure Indices: Concepts for Development and Evaluation of Their Use. In: Heck, W. W.; Taylor, O. C.; Tingey, D. T., Eds. Assessment of Crop Loss from Air Pollutants: Proceedings of an International Conference; October 1987; Raleigh, NC. New York, NY: Elsevier Applied Science. Pp. 107-138.
319. Holopainen, J.K. 2002. Aphid response to elevated ozone and CO₂. *Entomologia Experimentalis et Applicata*. 104:137-142.
320. Holt, D.A. 1988. Crop Assessment: International Needs and Opportunities, In: Heck, W. W.; Taylor, O. C.; Tingey, D. T., Eds. Assessment of Crop Loss from Air Pollutants: Proceedings of an International Conference; October 1987; Raleigh, NC. New York, NY: Elsevier Applied Science. Pp. 107-138.

321. Hormaza, J.I., Pinney, K., Polito, V.S. 1996. Correlation in the tolerance to ozone between sporophytes and male gametophytes of several fruit and tree nut species (Rosaceae). *Sexual Plant Reproduction*. 9:44-48.
322. Hummel, R.L., Brandenburg, R.L., Heagle, A.S., Arellano, C. 1998. Effect of ozone on reproduction of two spotted spider mite (Acari:Tetranychidae) on white clover. *Environmental Entomology*. 27:388-394.
323. Hunt, R. 1990. *Basic Growth Analysis*. Unwin Hyman, London.
324. Isebrands, J. G.; McDonald, E. P.; Kruger, E.; Hendrey, G.; Percy, K.; Pregitzer, K.; Sober, J.; Karnosky, D. F. 2001. Growth Responses of *Populus Tremuloides* Clones to Interacting Carbon Dioxide and Tropospheric Ozone. *Environ. Pollut.* 115: 359-371.
325. Jarvis, P.G., McNaughton, K.G. 1986. Stomatal Control of Transpiration-Scaling Up from Leaf to Region. *Advances in Ecological Research*. 15: 1-49.
326. Johnson, B. G.; Hale, B. A.; Ormrod, D. P. 1996a. Carbon Dioxide and Ozone Effects on Growth of a Legume-Grass Mixture. *J. Environ. Qual.* 25: 908-916.
327. Jones, H.C., Lacasse, N.L., Liggett, W.S., Weatherford, F. 1977. Experimental air exclusion system for field studies of SO₂ effects on crop productivity. Muscle Shoals, AL, Tennessee Valley Authority, No. E-EP-77-5.
328. Kangasjarvi, J., Talvinen, J., Utriainen, M., Kargalainen, R., 1994. Plant Defense Systems Induced by ozone. *Plant Cell and Environment*. 17: 783-794.
329. Karenlampi, L.; Skarby, L. 1996. Critical Levels for Ozone in Europe: Testing and Finalizing the Concepts. UN-ECE Workshop Report. Kupio, Finland: University Of Kuopio, Department of Ecology and Environmental Science.
330. Karlsson, P.E., Medin, E.L., Wallin, G., Selldén, G., Skärby, L. 1997. Effects of Ozone and Drought Stress on the Growth of Two Clones of Norway Spruce (*Picea Abies*). *New Phytologist*. 136: 265-275.
331. Karnosky, D. F.; Mankovska. B.; Percy, K.; Dickson, R. E.; Podila, G. K.; Sober, J.; Noormets, A.; Hendrey, G.; Coleman, M. D.; Kubiske, M.; Pregitzer K. S.; Isebrands, J. G. 1999. Effects of Tropospheric O₃ on Trembling Aspen and Interaction with CO₂: Results from an O₃ Gradient and a FACE Experiment. *Water Air Soil Pollut.* 116: 311-322.
332. Karnosky, D.F. et al. 1996. Changes in Growth, Leaf Abscission, and Biomass Associated with Seasonal Tropospheric Ozone Exposures of *Populus tremuloides* Clones and Seedlings. *Canadian Journal of Forest Research* 16:23-27

333. Kasana, M.S., Mansfield, T.A., 1986. Effects of Air Pollutants on the Growth and Functioning of Roots. *Proceedings of Indian Academy of Sciences (Plant Sciences)*. 96: 429-441.
334. Kats, G.; Dawson, P. J.; Bytnerowicz, A.; Wolf, J. W.; Thompson, C. R.; Olszyk, D. M. 1985. Effects of Ozone or Sulfur Dioxide on Growth and Yield of Rice. *Agric. Ecosyst. Environ.* 14: 103-117.
335. Kelly JM, Taylor GE Jr, Edwards NT, Adams MB, Edwards GS, Friend AL. 1993. Growth, Physiology, and Nutrition of Loblolly Pine Seedlings Stressed by Ozone and Acidic Precipitation: A Summary of the ROPIS-South Project. *Water Air Soil Pollut.* 69: 363-391.
336. Kerstiens G ; Lenzian K J. 1989a. Interactions between Ozone and Plant Cuticles I. Ozone Deposition and Permeability. *New Phytologist*. 112:13-20.
337. Kerstiens G ; Lenzian K J. 1989b. Interactions between Ozone and Plant Cuticles II. Water Permeability. *New Phytologist*. 112: 21-28.
338. Khan, M. R.; Khan, M. W. 1998. Interactive Effects of Ozone and Root-Knot Nematode on Tomato. *Agric. Ecosyst. Environ.* 70: 97-107.
339. Khan, M. R.; Khan, M. W. 1999. Effects of Intermittent Ozone Exposures on Powdery Mildew of Cucumber. *Environ. Exp. Bot.* 42:163-171.
340. Kickert, R. N.; Krupa, S. V. 1991. Modeling Plant Response to Tropospheric Ozone: A Critical Review. *Environ. Pollut.* 70: 271-383.
341. Kickert, R. N.; Tonella, G.; Simonov, A.; Krupa, S. V. 1999. Predictive Modeling of Effects under Global Change. *Environ. Pollut.* 100: 87-132.
342. Kim, H. J.; Helfand, G. E.; Howitt, R. E. 1998. An Economic Analysis of Ozone Control in California's San Joaquin Valley. *J. Agric. Resource Econ.* 23: 55-70.
343. Kleier, C.; Farnsworth, B.; Winner, W. 2001. Photosynthesis and Biomass Allocation of Radish Cv. "Cherry Belle" in Response to Root Temperature and Ozone. *Environ. Pollut.* 111:127-133.
344. Kobayashi, K.; Okada, M.; Nouchi, I. 1994. A Chamber System for Exposing Rice (*Oryza Sativa* L.) to Ozone in a Paddy Field. *New Phytologist*. 126: 317-325.
345. Kobayashi, K.; Okada, M.; Nouchi, I. 1995a. Effects of Ozone on Dry-Matter Partitioning and Yield of Japanese Cultivars of Rice (*Oryza-Sativa* L.). *Agriculture Ecosystems & Environment*. 53:109-122.
346. Kohut, R. J.; Amundson, R. G.; Laurence, J. A.; Colavito, L.; Van Leuken, P.; King, P. 1987. Effects of Ozone and Sulfur Dioxide on Yield of Winter Wheat. *Phytopathology*. 77: 71-74.
347. Kopp, R.J., Vaughn, W.J., Hazilla, M., Carson, R. 1985. Implications of environmental policy for U.S. agriculture: the case of ambient ozone standards. *Journal of Environmental Management*. 20:321-331.

348. Kostka-Rick, R., Manning, W.J., 1992. Partitioning of Biomass and Carbohydrates in Field-Grown Radish under Ambient Concentrations of Ozone, and Treated with the Anti-Ozonant Ethylene-Diurea (EDU). *New Phytologist*. 121: 187-200.
349. Kostka-Rick, R., Manning, W.J., Buonaccorsi, J.P. 1993. Dynamics of Biomass Partitioning in Field-Grown Radish Varieties Treated with Ethylenediurea. *Environmental Pollution*. 80:133-145.
350. Koziol, M.J., Whatley, F., 1984. *Gaseous Air Pollutants and Plant Metabolism*. Butterworths, London.
351. Kozlowski TT, Kramer PJ, Pallardy SG. *The physiological ecology of woody plants*. San Diego: Academic Press, Inc.; 1981. 657 pp.
352. Krapp, A., Hoffmann, B., Schafer, C., Stitt, M., 1993. Regulation of the Expression of Rbcs and Other Photosynthetic Genes by Carbohydrates – A Mechanism for the Sink Regulation Of Photosynthesis. *Plant Journal*. 3: 817-828.
353. Krapp, A., Quick, W.P., Stitt, M., 1991. Ribulose-1,5-Bisphosphate Carboxylase-Oxygenase, Other Calvin-Cycle Enzymes, and Chlorophyll Decrease When Glucose Is Supplied to Mature Spinach Leaves Via the Transpiration Stream. *Planta*. 186: 58-69.
354. Kress LW, Allen HL, Mudano JE, Stow TK. Impact of ozone on loblolly pine seedling foliage production and retention. *Environ Toxicol Chem*. 1992; 11: 1,115-1,128.
355. Krupa, S.; Kickert, R. 1997. Considerations for Establishing Relationships between Ambient Ozone (O₃) and Adverse Crop Response. *Environmental Reviews*. 5: 55-77.
356. Krupa, S.V., Nosal, M., Legge, A.H. 1994. Ambient ozone and crop loss: Establishing a cause-effect relationship. *Environmental Pollution*. 83:269-276.
357. Kurpius MR, McKay M, Goldstein AH. 2002. Annual ozone deposition to a Sierra Nevada ponderosa pine plantation. *Atmos Environ*. 36: 4,503-4,515.
358. Laisk A; Kull O; Moldau H. 1989. Ozone Concentration In Leaf Intercellular Air Spaces Is Close to Zero. *Plant Physiology (Rockville)*. 90:1163-1167.
359. Landolt, W, Gunthardt-Goerg, M, Pfenninger, I., Scheidegger, C. 1994. Ozone Induced Microscopic Changes and Quantitative Carbohydrate Contents of Hybrid Poplar (*Populus X Euramericana*). *Trees-Structure and Function*. 8:183-190.
360. Landolt, W., Bühlmann, U., Bleuler, P., Bucher, J.B. 2000. Ozone Exposure-Response Relationships for Biomass and Root/Shoot Ratio of Beech (*Fagus Sylvatica*), Ash (*Fraxinus Excelsior*), Norway Spruce (*Picea Abies*) and Scots Pine (*Pinus Sylvestris*). *Environmental Pollution*. 109: 473-478.

361. Larcher W. 1975. *Physiological Plant Ecology*. Berlin: Springer-Verlag.
362. Laurence JA, Kohut RJ, Amundson RG. 1989. Response of red spruce seedlings exposed to ozone and simulated acidic precipitation in the field. *Arch Environ Contam Toxicol*. 18: 285-290.
- ~~363.~~ Laurence, J.A., Amundson, R.G., Friend, A.L., Pell, E.J., Temple, P.J., 1994. Allocation of Carbon in Plants under Stress--An Analysis of the ROPIS Experiments. *Journal of Environmental Quality*. 23:412-417.
- 363.
364. Lawson, T.; Craigon, J.; Black, C. R.; Colls, J. J.; Tulloch, A.-M.; Landon, G. 2001a. Effects of Elevated Carbon Dioxide and Ozone on the Growth and Yield of Potatoes (*Solanum Tuberosum*) Grown in Open-Top Chambers. *Environ. Pollut*. 111:479-491.
365. Lechowicz MJ. 1987. Resource Allocation by Plants under Air Pollution Stress: Implications for Plant-Pest-Pathogen Interactions. *Bot Rev*. 53: 281-300.
366. Lee EH, Bennett JH. 1982. Superoxide Dismutase: A Possible Protective Enzyme against Ozone Injury in Snap Beans (*Phaseolus Vulgaris* L.). *Plant Physiol*. 69: 1,444-1,449.
367. Lee T.T. 1965. Sugar Content and Stomatal Width as Related to Ozone Injury in Tobacco Leaves. *Can J Bot*. 43: 677-685.
368. Lee, E. H.; Hogsett, W. E. 1999. Role of Concentrations and Time of Day In Developing Ozone Exposure Indices for a Secondary Standard. *J. Air Waste Manage. Assoc*. 49: 669-681.
369. Lee, E. H.; Tingey, D. T.; Hogsett, W. E. 1988. Evaluation of Ozone Exposure Indices in Exposure-Response Modeling. *Environ. Pollut*. 53:43-62.
370. Lee, E.H., Hogsett, W.E., Tingey, D.T. 1994. Attainment and effects issues regarding alternative secondary ozone air-quality standards. *Jour. Environ. Qual*. 23:1129-1140.
371. Lee, W.S., Chevone, B.I., Seiler, J.R., 1990. Growth Response and Drought Susceptibility of Red Spruce Seedlings Exposed to Simulated Acidic Rain and Ozone. *Forest Science*. 36: 265-275.
372. Lefohn AS, Krupa SV, Winstanley D. 1990. Surface Ozone Exposures Measured at Clean Locations Around the World. *Environ Pollut*. 63:189-224.
373. Lefohn, A. S.; Laurence, J.A.; Kohut R.J. 1988. A comparison of indices that describe the relationship between exposure to ozone and reduction in the yield of agricultural crops. *Atmos. Environ*. 49: 669-681.
374. Legge, A. H.; Grunhage, L.; Nosal, M.; Jager, H.-J.; Krupa, S. V. 1995. Ambient Ozone and Adverse Crop Response: An Evaluation of North

- American and European Data As They Relate to Exposure Indices and Critical Levels. *Angew. Bot.* 69: 192-205.
375. Lesser, V. M.; Rawlings, J. O.; Spruill, S. E.; Somerville, M. C. 1990. Ozone Effects on Agricultural Crops: Statistical Methodologies and Estimated Dose-Response Relationships. *Crop Sci.* 30: 148-155.
 376. Leuning, R., Neumann, H.H., Thurtell, G.W., 1979a. Ozone Uptake by Corn (*Zea Mays* L.): A General Approach. *Agricultural Meteorology* 20, 115-135.
 377. Leuning, R., Unsworth, M.H., Neumann, H.H., King, K.M., 1979b. Ozone Fluxes to Tobacco and Soil under Field Conditions. *Atmospheric Environment.* 13:1155-1163.
 378. Lorenzini, G.; Medeghini Bonatti, P.; Nali, C.; Baroni Fornasiero, R. 1994. The Protective Effect of Rust Infection against Ozone, Sulphur Dioxide and Paraquat Toxicity Symptoms in Broad Bean. *Physiol. Mol. Plant Pathol.* 45: 263-279.
 379. Lyons, T.M., Barnes, J.D., Davison, A.W., 1997. Relationships between Ozone Resistance and Climate in European Populations of *Plantago Major*. *New Phytologist.* 136: 503-510.
 380. Lyons, T.M.; Barnes, J.D. 1998. Influence of Plant Age on Ozone Resistance in *Plantago Major*. *New Phytologist.* 138:83-89.
 381. Mandl, R. H.; Weinstein, L. H.; Mccune, D. C.; Keveny, M. 1973. A Cylindrical, Open-Top Chamber for the Exposure of Plants to Air Pollutants in the Field. *J. Environ. Qual.* 2: 371-376.
 382. Manning, W. J.; Krupa, S. V. 1992. Experimental Methodology for Studying the Effects of Ozone on Crops and Trees. In: Lefohn, A. S., Ed. *Surface Level Ozone Exposures and Their Effects on Vegetation.* Chelsea, MI: Lewis Publishers, Inc.; Pp. 93-156.
 383. Martin, M.J., Frage, P.K., Humphrie, S.W., Long, S.P., 2000. Can Stomatal Changes Caused by Acute Ozone Exposure be Predicted by Changes Occurring in the Mesophyll? A Simplification for Models of Vegetation Response to the Global Increase in Tropospheric Elevated Ozone Episodes. *Australian Journal of Plant Physiology* 27: 211-219.
 384. Massman WJ, Musselman RC, Lefohn AS. 2000. A Conceptual Ozone Dose-Response Model to Develop a Standard to Protect Vegetation. *Atmos Environ.* 34(5): 745-759.
 385. Massman, W.J., Grantz, D.A., 1995. Estimating Canopy Conductance to Ozone Uptake from Observations of Evapotranspiration at the Canopy Scale and at the Leaf Scale. *Global Change Biology.* 1: 183-198.
 386. Massman, W.J., Pederson, J., Delany, A., Grantz, D., Den Hartog, G., Neumann, H.H., Oncley, S.P, Pearson, R., Jr., Shaw, R.H., 1994. An

- Evaluation Of The Regional Acid Deposition Model Surface Module For Ozone Uptake At Three Sites In The San Joaquin Valley Of California. *J. Geophysical Research*, 99(D4):8281-8294).
387. Materna, J., 1984. Impact Of Atmospheric Pollution On Natural Ecosystems. In: Treshow, M. (Ed.), *Air Pollution And Plant Life*. John Wiley & Sons, Chichester, UK, Pp 397-416.
388. Mauzerall, D.L., Wang, X. 2001. Protecting agricultural crops from the effects of tropospheric ozone exposure: Reconciling science and standard setting in the United States, Europe, and Asia. *Annu. Rev. Energy Environ.* 26: 237-68.
389. McCool, P. M.; Musselman, R. C. 1990. Impact Of Ozone On Growth Of Peach, Apricot And Almond. *Hortscience*, 25(11):1384-1385.
390. McCool, P. M.; Musselman, R. C.; Teso, R. R. 1987. Air Pollutant Yield-Loss Assessment For Four Vegetable Crops. *Agric. Ecosys. And Environ.*, 20: 11-21.
391. McCool, P.M., Menge, J.A. 1983. Influence Of Ozone On Carbon Partitioning In Tomato: Potential Role Of Carbon Flow In Regulation Of The Mycorrhizal Symbiosis Under Under Conditions Of Stress. *New Phytologist*, 94: 241-247.
392. McCrady, J.K., Anderson, C.P., 2000. The Effect Of Ozone On Below-Ground Carbon Allocation In Wheat. *Environmental Pollution*, 107: 465-472.
393. McLaughlin SB, Layton PA, Adams MB, Edwards NT, Hanson PJ, O'Neill EG, Roy WK. 1994. Growth responses of 53 open-pollinated loblolly pine families to ozone and acid rain. *J Environ Qual*, 23: 247-257.
394. McLaughlin SB, McConathy RK. 1983. Effects Of SO₂ And ozone-O₃ On Allocation Of ¹⁴C-Labeled Photosynthate In Phaseolus Vulgaris. *Plant Physiol*, 73: 630-635.
395. McLaughlin SB, Shriner DS. 1980. Allocation Of Resources To Defense And Repair. In: Horsfall JB, Cowling EB, Editors. *Plant Disease*, Vol. 5. New York: Academic Press; P. 407-431.
- ~~396. McLaughlin SB, Shriner DS. Allocation of resources to defense and repair. In: Horsfall JB, Cowling EB, editors. Plant Disease, Vol. 5. New York: Academic Press; 1980, p. 407-431.~~
- ~~397-396.~~ McLaughlin, S.B., McConathy, R.K., Durick, D., Mann, L.K. 1982. Effects Of Chronic Air Pollution Stress On Photosynthesis, Carbon Allocation And Growth Of White Pine Trees. *Forestry Science*, 28: 60-70.
- ~~398-397.~~ McLeod, A. R.; Baker, C. K. 1988. The Use Of Open Field Systems To Assess Yield Response To Gaseous Pollutants. In: Heck, W. W.; Taylor, O. C.; Tingey, D. T., Eds. *Assessment Of Crop Loss*

- ~~From~~ Air Pollutants. London, United Kingdom: Elsevier Applied Science; Pp. 181-210.
- ~~399-398.~~ McLeod, A. R.; Shaw, P. J. A.; Holland, M. R. 1992. The Liphook Forest Fumigation Project: Studies ~~Of~~ Sulphur Dioxide ~~And~~ Ozone Effects ~~On~~ Coniferous Trees. For. Ecol. Manage. 51: 121-127.
- ~~400-399.~~ Mehlhorn H, Seufert G, Schmit A, Kunert KJ. 1986. Effect ~~Of~~ SO_2 ~~And~~ ozone- O_3 ~~On~~ Production ~~Of~~ Antioxidants ~~In~~ Conifers. Plant Physiol. 82: 336-338.
- ~~401-400.~~ Meier S, Grand LF, Schoeneberger MM, Reinert RA, Bruck RI. 1990. Growth, Ectomycorrhizae ~~And~~ Nonstructural Carbohydrates ~~Of~~ Loblolly Pine Seedlings Exposed ~~To~~ Ozone ~~And~~ Soil Water Deficit. Environ Pollut. 64: 11-27.
- ~~402-401.~~ Meinzer, F.C., Grantz, D.A.; 1989. Stomatal Control ~~Of~~ Transpiration ~~From~~ ~~A~~ Developing Sugarcane Canopy. Plant Cell ~~And~~ Environment. 12; 635-642.
- ~~403-402.~~ Meinzer, F.C., Grantz, D.A.; 1990. Stomatal ~~And~~ Hydraulic Conductance ~~In~~ Growing Sugarcane: Stomatal Adjustment ~~To~~ Water Transport Capacity. Plant Cell ~~And~~ Environment. 13; 383-388.
- ~~404-403.~~ Meyer, U., Kollner, B., Willenbrink, J., Krause, G.H.M.; 1997. Physiological Changes ~~On~~ Agricultural Crops Induced ~~By~~ Different Ambient Ozone Exposure Regimes. 1. Effects ~~On~~ Photosynthesis ~~And~~ Assimilate Allocation ~~In~~ Spring Wheat. New Phytologist. 136; 645-652.
- ~~405-404.~~ Miller JE, Vozzo SF, Patterson RP, Pursley WA, Heagle AS. 1995. Effects ~~Of~~ Ozone ~~And~~ Water Deficit ~~On~~ Field-Grown Soybean: II. Leaflet Nonstructural Carbohydrates. J Environ Qual. 24: 670-677.
- ~~406-405.~~ Miller PR, Chow J, Watson JG, editors. 1996a. Assessment of Acidic Deposition and Ozone Effects on Conifer Forests in the San Bernardino Mountains. Sacramento, CA.; ARB; ~~1996a.~~ Final Report, No. A032-180. NTIS No. PB97115612.
- ~~407-406.~~ Miller PR, Chow JC, Watson JG, editors. 1996b. Ecosystem Level Alterations in Soil Nutrient Cycling: An Integrated Measure of Cumulative Effects of Acidic Deposition on a Mixed Conifer Forest in Southern California. Sacramento, CA.; ARB; ~~1996b.~~ Final Report, No. 92-335. NTIS No. PB97106223.
- ~~408-407.~~ Miller PR, Evans LS. Histopathology of oxidant injury and winter fleck injury on needles of western pines. Phytopath 1974; 64: 801-806.
- ~~409.~~ Miller PR, McBride JR, editors. ~~Oxidant air pollution impacts in the montane forests of southern California: a case study of the San Bernardino mountains. New York: Springer; 1999, Ecological Studies, Vol 134.~~

- 410.408. Miller PR, Millecan AA. 1971. Extent of oxidant air pollution damage to some pines and other conifers in California. *Plant Disease R.* 1971; 55(6): 555-559.
- 411.409. Miller PR, Parmeter JR Jr, Flick BH, Martinez CW. 1969. Ozone dosage response of ponderosa pine seedlings. *J Air Pollut Contr Assoc* 1969; 19(6):435-438.
- 412.410. Miller PR, Taylor OC, Poe MP. 1986. Spatial variation of summer ozone concentrations in the San Bernardino mountains. *Mountains*. Minneapolis, MN.: APCA 79th Ann Mtg.; 1986, Paper No. 86-39.2.
- 413.411. Miller PR. 1969. Air pollution and the forests of California. *Calif Air Environ.* 1969; 1: 1-2.
- 414.412. Miller, J.E., 1988. Effects On Photosynthesis, Carbon Allocation, And Plant Growth Associated With Air Pollutant Stress. In: Heck, W.W., Tingey, D.T., Taylor, C.C. (Eds.), *Assessment Of Crop Loss From Air Pollutants*. Elsevier, London; Pp. 287-314.
- 415.413. Miller, J.E., Patterson, R.P., Heagle, A.S., Pursley, W.A., Heck, W.W. 1988. Growth of cotton under chronic ozone stress at two levels of soil moisture. *Journal of Environmental Quality.* 17: 635-643.
- 416.414. Miller, J.E., Patterson, R.P., Pursley, W.A., Heagle, A.S., Heck, W.A., 1989. Response Of Soluble Sugars And Starch In Field-Grown Cotton To Ozone, Water Stress, And Their Combination. *Environmental And Experimental Botany.* 29; 477-486.
- 417.415. Miller, P., McBride J.R., 1999. Oxidant Air Pollution Impacts In The Montane Forests Of Southern California: A Case Study Of The San Bernardino Mountains. *Ecological Studies vol. 134*; Springer; 424 Pages; New York.
- 418.416. Mills, G.; Ball, G.; Hayes, F.; Fuhrer, J.; Skarby, L.; Gimeno, B.S.; de Temmerman, L.; Heagle, A. 2000. Development Of A Multi-Factor Model For Predicting The Critical Level Of Ozone for White Clover. *Environ. Pollut.* 109: 533-542.
- 419.417. Minnocci, A, Panicucci, A., Sebastiana, L., Lorenzini, G., Vitagliano, C., 1999. Physiological And Morphological Responses Of Olive Plants To Ozone Exposure During A Growing Season. *Tree Physiology.* 19; 391-397.
- 420.418. Moldau, H.; Bichele, I. 2002. Plasmalemma Protection By The Apoplast As Assessed From Above-Zero Ozone Concentrations In Leaf Intercellular Air Spaces. *Planta.* 214: 484-487.
- 421.419. Mortensen, L. M. 1990. Effects Of Ozone On Growth Of Triticum Aestivum L. At Different Light, Air Humidity, And CO₂ Levels. *Norw. J. Agric. Sci.* 4: 343-348.

- 422.420. Mortensen, L., Engvild, K.C., 1995. Effects of Ozone on ¹⁴C Translocation Velocity and Growth of Spring Wheat (*Triticum Aestivum* L.) Exposed in Open-Top Chambers. *Environmental Pollution*, 87: 135-140.
- 423.421. Mudd J B. 1996. Biochemical Basis for the Toxicity of Ozone. In: Yunus, M.; Iqbal, M.: Eds. *Plant Response to Air Pollution*. - 267-283.
- 424.422. Mulchi, C.; Rudorff, B.; Lee, E.; Rowland, R.; Pausch, R. 1995. Morphological Responses Among Crop Species to 53 Full-Season Exposures to Enhanced Concentrations of Atmospheric CO₂ and ozone-O₃. *Water Air Soil Pollut.* 85: 1379-1386.
- 425.423. Murphy, J.J.; Deluki, M.A.; Mccubbin D.R.; Kim H.J. 1999. The Cost of Crop Damage Caused By Ozone Air Pollution from Motor Vehicles. *Journal of Environmental Management* 55: 273-289.
- 426.424. Musselman RC, Minnick TJ. 2000. Nocturnal stomatal conductance and ambient air quality standards for ozone. *Atmos Environ.* 2000; 34(5): 719-733.
- 427.425. Musselman, R.C.; Massman, W. J. 1999. Ozone Flux to Vegetation and Its Relationship to Plant Response and Ambient Air Quality Standards. *Atmos Environ.* 33: 65-73.
- 428.426. Mutters, R., Soret, S. 1998. Statewide Potential Crop Losses from Ozone Exposure. Final Report to California Air Resources Board on Contract 94-345.
- 429.427. Mutters, R., Guzy M. Thompson, C.R. 1993. Crop Losses from Air Pollutants—A Computer and Field-Based Assessment Program and Crop and Forest Losses from Air Pollutants—An Assessment Program. Final Report to California Air Resources Board on Contracts A033-174 and A933-190.
- 430.428. Mutters, R., Soret, S. 1995. Crop Losses from Air Pollutants-- A GIS Regional Analysis and Statewide Crop Losses From Air Pollutants. Final Report to California Air Resources Board on Contracts A133-185 and A92-350.
- 431.429. NARSTO (North American Research Strategy for Tropospheric ozone). 2000. The NARSTO ozone-O₃ Assessment—Critical Reviews. *Atmospheric Environment*, 34: 1853-2332.
- 432.430. Nouchi, I., Ito, O., Harazono Y., Kobayashi, K. 1991. Effects of chronic ozone exposure on growth, root respiration and nutrient uptake of rice plants. *Environmental Pollution*, 74: 149-164.
- 433.431. Noormets, A.; Söber, A.; Pell, E. J.; Dickson, R. E.; Podila, G. K.; Söber, J. Isebrands, J. G.; Karnosky, D. F.; 2001a. Stomatal and non-stomatal limitation to photosynthesis in two trembling aspen (*Populus*

tremuloides Michx.) clones exposed to elevated CO₂ and/or O₃. Plant, Cell & Environment, Volume 24: 327-336.

- 434.432. Nussbaum, S., Geissmann, M., Fuhrer, J. 1995. Ozone exposure-response relationships for mixtures of perennial ryegrass and white clover depend on ozone exposure patterns. Atmospheric Environment 29: 989-995.
- 435.433. Ojanpera K.; Patsikka, E.; Ylaranta, T. 1998. Effects of Low Ozone Exposure of Spring Wheat on Net CO₂ Uptake, Rubisco, Leaf Senescence and Grain Filling. New Phytol. 138: 451-460.
- 436.434. Okano, K., Ito, O., Takeba, G., Shimizu, A., Totsuka, T., 1984. Alteration of ¹³C-Assimilate Partitioning in Plants of Phaseolus Vulgaris Exposed to Ozone. New Phytologist, 97: 155-163.
- 437.435. Ollerenshaw, J. H.; Lyons, T. 1999. Impacts of Ozone on the Growth and Yield of Field-Grown Winter Wheat. Environ. Poll. 106:67-72.
- 438.436. Ollerenshaw, J.H., Lyons, T., Barnes, J.D., 1999. Impacts of Ozone on the Growth and Yield of Field-Grown Winter Oilseed Rape. Environmental Pollution, 104: 53-59.
- 439.437. Olson RK, Binkley D, Böhm M, editors. The Response of Western Forests to Air Pollution. New York: Springer-Verlag, New York; 1992.
- 440.438. Olszyk, D. M.; Bytnerowicz, A.; Kats, G.; Dawson, P. J.; Wolf, J.; Thompson, C. R. 1986a. Effects of Sulfur Dioxide and Ambient Ozone on Winter Wheat and Lettuce. J. Environ. Qual. 15: 363-369.
- 441.439. Olszyk, D. M.; Bytnerowicz, A.; Kats, G.; Dawson, P. J.; Wolf, J.; Thompson, C. R. 1986b. Crop Effects from air pollutants in air exclusion systems vs. field chambers. J. Environ. Qual. 15: 417-422.
- 442.440. Olszyk, D. M.; Kats, G. L.; Morrison, C. L.; Dawson, P. J.; Gocka, I.; Wolf, J.; Thompson, C.R. 1990b. 'Valencia' Orange Fruit Yield With Ambient Oxidant or Sulfur Dioxide Exposures. J. Amer. Soc. Hort. Sci. 115(6): 878-883.
- 443.441. Olszyk, D. M.; Tibbitts, T. W.; Hertzberg, W. M. 1980. Environment In Open-Top Field Chambers Utilized for Air Pollution Studies. J. Environ. Qual. 9: 610-615.
- 444.442. Olszyk, D., Bytnerowicz, A., Kats, G., Reagan, C., Hake, S., Kerby, T., Millhouse, D., Roberts, B., Anderson, C., Lee, H., 1993. Cotton Yield Losses and Ambient Ozone Concentrations in California's San Joaquin Valley. Journal of Environmental Quality, 22: 602-611.
- 445.443. Olszyk, D.M., Takemoto, B.K., Kats, G., Dawson, P.J., Morrison, C.L., Preston, J.W., Thompson, C.R. 1992. Effects of open top chambers on "Valencia" orange trees. Journal of Environmental Quality, 21:128-134.

- 446.444. Olszyk, D.M., Takemoto, B.K., Poe, M. 1991. Leaf Photosynthetic And Water Relations Responses For "Valencia" Orange Trees Exposed To Oxidant Air Pollution. Environmental And Experimental Botany. 31: 427-436.
- 447.445. Olszyk, D.M., Wise, C., 1997. Interactive Effects Of Elevated CO₂ And ozone-O₃ On Rice And Flacca Tomato. Agriculture, Ecosystems And Environment. 66: 1-10.
448. Ommen, O. E.; Donnelly, A.; Vanhoutvin, S.; Van Oijen, M.; Manderscheid, R. 1999. Chlorophyll Content Of Spring Wheat Flag Leaves Grown Under Elevated CO₂ Concentrations And Other Environmental Stresses Within The 'ESPACE-Wheat' Project. Eur. J. Agron. 10: 197-203.
446. Oren, R., Werk, K.S., Meyer, J., Schulze, E.-D. 1989. Potentials And Limitations Of Field Studies On Forest Decline Associated With Anthropogenic Pollution. Chapt. 3 in Schulze, E.D., Lange, O.L., Oren, R. (eds.) Ecological Studies Vol. 77 "Forest Decline And Air Pollution: A Study Of Spruce (*Picea Abies*) On Acid Soils" Springer Verlag, Berlin.
447. Oren, R., Werk, K.S., Meyer, J., Schulze, E.-D. 1989. Potentials and Limitations of Field Studies on Forest Decline Associated with Anthropogenic Pollution. In: Schulze, E.-D., Lange, O.L., Oren, R., eds. Ecological Studies Vol. 77. Chapt. 3 "Forest Decline and Air Pollution: A Study Of Spruce (*Picea Abies*) on Acid Soils." Springer Verlag, Berlin.
- 450.448. Ormrod, D.P., Marie, B.A. And Allen, O.B. 1988. Research Approaches To Pollutant Crop Loss Functions, In: Heck, W. W.; Taylor, O. C.; Tingey, D. T., Eds. Assessment Of Crop Loss From Air Pollutants: Proceedings Of An International Conference; October, (1987); Raleigh, NC. New York, NY: Elsevier Applied Science. Pp. 27-44. London: Elsevier Press. Pgs. 27-44.
- 451.449. Oshima, R. J.; Braegelmann, P. K.; Baldwin, D. W.; Van Way, V.; Taylor, O. C. 1977a. Responses Of Five Cultivars Of Fresh Market Tomato To Ozone: A Contrast Of Cultivar Screening With Foliar Injury And Yield. J. Am. Soc. Hortic. Sci. 102: 286-289.
- 452.450. Oshima, R. J.; Poe, M. P.; Braegelmann, P. K.; Baldwin, D. W.; Van Way, V. 1976. Ozone Dosage-Crop Loss Function For Alfalfa: A Standardized Method For Assessing Crop Losses From Air Pollutants. J. Air Pollut. Control Assoc. 326: 861-865.
- 453.451. Oshima, R.J., Bennett, J.P., Braegelmann, P.K., 1978. Effect Of Ozone On Growth And Assimilate Partitioning In Parsley. Journal Of The American Society Of Horticultural Science. 103: 348-350.
- 454.452. Oshima, R.J., Braegelmann, P.K., Baldwin, D.W., Van Way, V., Taylor, O.C., 1977b. Reduction In Tomato Fruit Size And Yield By

- Ozone. Journal Of The American Society Of Horticultural Science, 102: 289-293.
- 455.453. Oshima, R.J., Braegelmann, P.K., Flagler, R.B., Teso, R.R., 1979. The Effects Of Ozone On The Growth, Yield And Partitioning Of Dry Matter In Cotton. Journal Of Environmental Quality, 8: 474-479.
- 456.454. Paakkonen, E.; Holopainen, T.; 1995. Influence of nitrogen supply on the response of clones of birch (*Betula pendula* Roth) to ozone. New Phytologist 129: 595-603.
- 457.455. Palou, L.; Crisosto, C.H.; Smilanick, J.L.; Adaskaveg, J.E.; Zoffoli, J.P. 2002. Effects of continuous 0.3 ppm ozone exposure on decay development and physiological responses of peaches and table grapes in cold storage. Postharvest Biology and Technology, 24: 39-48.
- 458.456. Parker GG. 1990. Evaluation of dry deposition, pollutant damage, and forest health with throughfall studies. In: Lucier AA, Haines GG, editors. Mechanisms of forest response to acidic deposition. New York: Springer-Verlag; 1990. Pgs. 10-61.
- 459.457. Patterson MT, Rundel PW. 1989. Seasonal physiological responses of ozone stressed Jeffrey pine in Sequoia National Park, California. In: Olson RK, Lefohn AS, editors. Effects of air pollution on western forests. APCA Trans Ser. 16. Pgs. 419-427.
- 460.458. Peace, A., Lea, P.J., Darrall, N.M., 1995 The Effect Of Open Fumigation With SO₂ And Ozone-O₃ On Carbohydrate Metabolism In Scots Pine (*Pinus Sylvestris*) And Norway Spruce (*Picea Abies*). Plant Cell And Environment, 18: 277-283.
- 461.459. Pearson, M. 1995. Effects of Ozone on Growth and Gas-Exchange of Eucalyptus-Globulus Seedlings. Tree Physiol. 15: 207-210.
- 462.460. Pell, E. J.; Sinn, J. P.; Eckardt, N.; Johansen, C. V.; Winner, W. E.; Mooney, H.A. 1993. Response Of Radish To Multiple Stresses II. Influence Of Season And Genotype On Plant Response To Ozone And Soil Moisture Deficit. New Phytol. 123: 153-163.
- 463.461. Pell, E. J.; Sinn, J. P.; Johansen C. V. 1995. Nitrogen Supply As A Limiting Factor Determining The Sensitivity Of *Populus Tremuloides* Michx. To Ozone Stress. New Phytol. 130: 437-446.
- 464.462. Pell, E.J., Eckardt, N., Enyedi, A.J., 1992. Timing Of Ozone Stress And Resulting Status Of Ribulose Bisphosphate Carboxylase/Oxygenase And Associated Net Photosynthesis. New Phytologist, 120: 397-405.
- 465.463. Pell, E.J., Pearson, N.S., 1983. Ozone Induced Reduction In Quantity Of Ribulose-1,5-Bisphosphate Carboxylase In Alfalfa Foliage. Plant Physiology, 73: 185-187.

- ~~466~~.464. Pell, E.J., Schlaghaufer, C.D., Arteca, R.N.; 1997. ~~ozone~~O₃-Induced Oxidative Stress: Mechanisms ~~Of~~ Action ~~And~~ Reaction. *Physiologia Plantarum*, 100; 264-273.
- ~~467~~.465. Pell, E.J., Temple, P.J., Friend, A.L., Mooney, H.A., Winner, W.E.; 1994. Compensation ~~Asas~~ As Plant Response ~~To~~ Ozone ~~And~~ Associated Stresses: An Analysis ~~Of~~ ROPIS Experiments. *Journal Of Environmental Quality*, 23; 429-436.
- ~~468~~.466. Peterson DL, Arbaugh MJ, Robinson LJ. 1991. Regional growth changes in ozone-stressed ponderosa pine (*Pinus ponderosa*) in the Sierra Nevada, California, USA. *Holocene*, 1994; 1: 50-61.
- ~~469~~.467. Peterson DL, Arbaugh MJ, Wakefield VA, Miller PR. 1987. Evidence of growth reduction in ozone-injured Jeffrey pine (*Pinus jeffreyi* Grev. and Balf.) in Sequoia and Kings Canyon National Parks. *J Air Pollut Contr Assoc*, 1987; 37: 906-912.
- ~~470~~.468. Peterson DL, Arbaugh MJ. 1988. An evaluation of the effects of ozone injury on radial growth of ponderosa pine (*Pinus ponderosa*) in the southern Sierra Nevada. *J Air Pollut Contr Assoc*, 1988; 38: 921-927.
- ~~471~~.469. Peterson DL, Silsbee DG, Poth M, Arbaugh MJ, Biles FE. 1995. Growth response of big-cone Douglas fir (*Pseudotsuga macrocarpa*) to long-term ozone exposure in southern California. *J Air Waste Manage* 1995; 45: 36-45.
- ~~472~~.470. Pleiejl, H.; Karlsson, G.P.; Danielsson, H.; Sellden, G. 1995. Surface Wetness Enhances Ozone Deposition to a Pasture Canopy. *Atmospheric Environ.* 29: 3391-3393.
- ~~473~~.471. Pleijel, H.; Skarby, L. 1991. Yield And Grain Quality Of Spring Wheat (*Triticum Aestivum* L., Cv. Drabant) Exposed ~~To~~ Different Concentrations ~~Of~~ Ozone ~~in~~ Open-Top Chambers. *Environmental Pollution*, 69: 151-168.
- ~~474~~.472. Pleijel, H.; Skärby, L.; Ojanperä, K.; Selldén, G. 1992. Yield ~~And~~ Quality ~~Of~~ Spring Barley, *Hordeum Vulgare* L., Exposed ~~To~~ Different Concentrations ~~Of~~ Ozone ~~in~~ Open-Top Chambers. *Agriculture, Ecosystems ~~And~~ Environment*, 38: 21-29.
- ~~475~~.473. Pleijel, H.; Wallin, G.; Karlsson, P. E.; Skarby, L.; Sellden, G. 1994b. Ozone Deposition ~~To~~ ~~An~~ Oat Crop (*Avena Sativa* L.) Grown ~~in~~ Open-Top Chambers ~~And~~ ~~in~~ ~~The~~ Ambient Air; *Atmos. Environ.* 28: 1971-1979.
- ~~476~~.474. Poorter, H., Perez-Soba, M. 2001. The growth response of plants to elevated CO₂ under non-optimal environmental conditions. *Oecologia*, 129: 1-20.
- ~~477~~.475. Pye JM. 1988. Impact of ozone on the growth and yield of trees: A review. *J Environ Qual*, 1988; 17(3): 347-360.

- 478.476. ____Rebbeck J, Jensen KF, Greenwood MS. 1993. Ozone effects on grafted mature and juvenile red spruce: photosynthesis, stomatal conductance, and chlorophyll concentration. *Can J For Res.* 23: 450-456.
- 479.477. ____Reich PB, Amundson RG. 1985. Ambient levels of ozone reduce net photosynthesis in tree and crop species. *Science.* 230: 566-570.
- 480.478. ____Reich PB, Schoettle AW, Stroo HF, Troiano J, Amundson RG. 1987. Effects of ozone and acid rain on white pine (*Pinus strobus*) seedlings grown in five soils. I. Net photosynthesis and growth. *Can J Bot* 1987; 65: 977-987.
- 481.479. ____Reich PB. 1987. Quantifying Plant Response To Ozone: A Unifying Theory. *Tree Physiol.* 3: 63-91.
- 482.480. ____Reich, P.H., 1983. Effects Of Low Concentrations Of O₃ ozone On Net Photosynthesis, Dark Respiration, And Chlorophyll Contents In Aging Hybrid Poplar Leaves. *Plant Physiology.* 73; 291-296.
- 483.481. ____Reich, P.H., Amundson, R.G., 1984. Low Level ozone—O₃ And/Or and/or SO₂ Exposure Causes A Linear Decline In Soybean Yield. *Environmental Pollution.* 34; 345-355.
- 484.482. ____Reiling, K., Davison, A.W.; 1992a. The Response Of Native, Herbaceous Species To Ozone: Growth And Fluorescence Screening. *New Phytologist.* 120; 29-37.
- 485.483. ____Reiling, K., Davison, A.W.; 1992b. Spatial Variation In Ozone Resistance Of British Populations Of *Plantago Major* L. *New Phytologist.* 122; 699-708.
- 486.484. ____Reiling, K., Davison, A.W.; 1994. Effects Of Exposure To Ozone To Different Stages In The Development Of *Plantago Major* L. On Chlorophyll Fluorescence And Gas Exchange. *New Phytologist.* 128; 509-514.
- 487.485. ____Reinert, R.A., Ho, M.C.; 1995. Vegetative Growth Of Soybean As Affected By Elevated Carbon Dioxide And Ozone. *Environmental Pollution.* 89; 89-96.
- 488.486. ____Reinert, R.A., Schafer, S.R., Eason, G., Schoeneberger, M.M., Horton, S.J., 1996. Response Of Loblolly Pine To Ozone And Simulated Acidic Rain. *Canadian Journal Of Forest Research.* 26; 1715-1723.
- 489.487. ____Renaud J. P.; Allard, G.; Mauffette, Y. 1997. Effects Of Ozone On Yield, Growth, And Root Starch Concentrations Of Two Alfalfa (*Medicago Sativa* L.) Cultivars. *Environmental Pollution.* 95: 273-281.
- 490.488. ____Retzlaff, W. A.; Arthur, M. A.; Grulke, N. E.; Weinstein, D. A.; Gollands, B. 2000. Use Of A Single-Tree Simulation Model To Predict

Effects Of Ozone And Drought On Growth Of A White Fir Tree. *Tree Physiol.* 20: 195-202.

- ~~491.489.~~ Retzlaff, W. A.; Williams, L. E.; Dejong, T. M. 1997. Growth And Yield Response Of Commercial Bearing-Age 'Casselman' Plum Trees To Various Ozone Partial Pressures. *Journal Of Environmental Quality*, 26(3): 858-865.
- ~~492.490.~~ Retzlaff, W. A.; Williams, L. E.; Dejong, T. M. 1992. Photosynthesis, Growth, And Yield Response Of 'Casselman' Plum To Various Ozone Partial Pressures During Orchard Establishment. *J. Am. Soc. Hortic. Sci.* 117: 703-710.
- ~~493.491.~~ Retzlaff, W.A., Williams, L.E., DeJong, T.M. 1991. The effect of different atmospheric ozone partial pressures on photosynthesis and growth of nine fruit and nut tree species. *Tree Physiology*, 8:93-105.
- ~~494.492.~~ Richards BL Sr, Taylor OC, Edmunds GF Jr. 1968. Ozone needle mottle of pine in southern California. *J Air Pollut Contr Assoc.* 18(2): 73-77.
- ~~495.493.~~ Richardson CJ, Sasek TW, Fendick EA, Kress LW. 1992. Ozone exposure-response relationships for photosynthesis in genetic strains of loblolly pine seedlings. *For Ecol Manage.* 51: 163-178.
- ~~496.494.~~ Roberts, B.R., Cannon, W.N., Jr., 1992. Growth And Water Relationships Of Red Spruce Seedlings Exposed To Atmospheric Deposition And Drought. *Canadian Journal Of Forest Research.* 22: 193-197.
- ~~497.495.~~ Rocchio JE, Ewell DM, Procter CT, Takemoto BK. 1993. Project FOREST: The Forest Ozone Response Study. In: Brown WE, Veirs SD Jr, editors. *Partners in stewardship*. Hancock, MI: Geo Wright Soc; 1993. Pgs p. 112-119.
- ~~498.496.~~ Roper, T.R., Williams, L.E., 1989. Effect Of Ambient And Acute Partial Pressures Of Ozone On Leaf Net CO₂ Assimilation Of Field-Grown *Vitis Vinifera* L. *Plant Physiology*, 91: 1501-1506.
- ~~499.497.~~ Rosen, P.M., Runeckles, V.C. 1976. Interaction of ozone and greenhouse whitefly in plant injury. *Environmental Conservation*, 3:70-71.
- ~~500.498.~~ Rosenbaum BJ, Strickland TC, McDowell MK. 1994. Mapping critical levels of ozone, sulfur dioxide and nitrogen dioxide for crops, forests and natural vegetation in the United States. *Water Air Soil Pollut.* 74: 307-319.
- ~~501.499.~~ Rowe, R.D.; C.G.L. Lauraine Chestnut; D.C. Peterson; C. Miller; R.M. Adamas; W.R. Oliver; and H. Hogo. The Benefits of Air Pollution Control in California, Report for the ARB, 1986

- 502-500. Rudorff B. F. T.; Mulchi, C. L.; Lee, E. H.; Rowland, R.; Pausch, R. 1996a. Effects of Enhanced ozone-O₃ and CO₂ Enrichment on Plant Characteristics in Wheat and Corn. *Environ. Pollut.* 94: 53-60.
- 503-501. Rudorff, B. F. T.; Mulchi, C. L.; Fenny, P.; Lee, E. H.; Rowland, R. 1996c. Wheat Grain Quality Under Enhanced Tropospheric CO₂ and ozone O₃ Concentrations. *Journal of Environmental Quality*, 25: 1384-1388.
- 504-502. Runeckles, V. C.; Wright, E. F.; White, D. 1990. A Chamberless Field Exposure System for Determining the Effects of Gaseous Air Pollutants on Crop Growth and Yield. *Environ. Pollut.* 63: 61-77.
- 505-503. Rusch, H.; Laurence, J. A. 1993. Interactive Effects of Ozone and Powdery Mildew on Pea Seedlings. *Phytopathology*, 83: 1258-1263.
- 506-504. Sah, D. N.; Von Tiedemann, A.; Fehrmann, H. 1993. Effects of Ozone Exposure on Resistance of Wheat Genotypes to *Phenophora Tritici-Repentis*. *J. Phytopathol.* 138: 249-256.
- 507-505. Sakaki, T., Saito, K., Kawaguchi, A., Kondo, N., Yamada, M. 1990. Conversion of Monogalactosyldiacylglycerols to Triacylglycerols in Ozone-Fumigated Spinach Leaves. *Plant Physiology (Rockville)*, 94(2): 766-772.
- 508-506. Sandermann, H., Jr. 1996. Ozone and Plant Health. *Annu. Rev. Phytopathol.* 34: 347-366.
- 509-507. Sanders, G. E.; Clark, A. G.; Colls, J. J. 1991. The influence of open-top chambers on the growth and development of field bean. *New Phytologist*, 117: 439-447.
- 510-508. Sanders, G. E.; Colls, J. J.; Clark, A. G.; Galaup, S.; Bonte, J.; Cantuel, J. 1992b. *Phaseolus vulgaris* and ozone: results from open-top chamber experiments in France and England. *Agriculture, Ecosystems and Environment*, 38: 31-40.
- 511-509. Sanders, G. E.; Robinson, A. D.; Geissler, P. A.; Colls, J. J. 1992a. Yield stimulation of a commonly grown cultivar of *Phaseolus vulgaris* L. at near-ambient ozone concentrations. *New Phytologist*, 122: 63-70.
- 512-510. Schenone, G.; Botteschi, G.; Fumagalli, I.; Montinaro, F. 1992. Effects of Ambient Air Pollution in Open-Top Chambers on Bean (*Phaseolus Vulgaris* L.) I. Effects on Growth and Yield. *New Phytol.* 122: 689-697.
- 513-511. Schenone, G.; Fumagalli, I.; Mignanego, F.; Montinaro, F.; Soldatini, G.F. 1994. Effects of Ambient Air Pollution in Open-Top Chambers on Bean (*Phaseolus vulgaris* L.). II. Effects on Photosynthesis and Stomatal Conductance. *New Phytol.* 126: 309-315.

- ~~514-512.~~ Schulze E-D. 1989. Air pollution and forest decline in a spruce (*Picea abies*) forest. *Science*, 1989; 244: 776-783.
- ~~515-513.~~ Schulze, E-D., Lange, O.L., Oren, R. (Eds.); 1989. *Forest Decline And Air Pollution: A Study Of Spruce (Picea Abies) On Acid Soils*. Springer-Verlag. New York.
- ~~516-514.~~ Shafer SR, Heagle AS, Camberato DM. 1987. Effects of chronic doses of ozone on field-grown loblolly pine: seedling responses in the first year. *J Air Pollut Contr Assoc*; 1987; 37: 1,179-1,184.
- ~~517-515.~~ Shafer SR, Heagle AS. 1989. Growth responses of field-grown loblolly pine to chronic doses of ozone during multiple growing seasons. *Can J For Res*; 1989; 19: 821-831.
- ~~518-516.~~ Shaver, C.L., Igal L.L.; T.H. Nash III, 1983. Lichen Communities on Conifers in Southern California Mountains: An Ecological Survey Relative to Oxidant Air Pollution; *Ecology*, 64(6): 1342-1354, 1983.
- ~~519-517.~~ Shaver, C.L.; Cass, G.R.; Druzik, J.R.; 1983. Ozone and the Deterioration of Works of Art. *Environ. Sci. Technol.*; 17: 749-752; (1983)
- ~~520-518.~~ Shaw, P.J. 1986. The Liphook forest fumigation experiment: description and project plan. TPRD/L/2985/R86: 1-15. Central Electricity Generating Board, technology planning and research division. Kelvin Ave. Leatherhead, Surrey KT22 7SE.
- ~~521-519.~~ Showman, R.E. 1991. A comparison of ozone injury to vegetation during moist and drought years. *Journal of the Air and Waste Management Association*, 41: 63-64.
- ~~522-520.~~ Shriner DS, Heck WW, McLaughlin SB, Johnson DW, Irving PM, Joslin JD, Peterson CE. 1991. Response of vegetation to atmospheric deposition and air pollution. *NAPAP Report 18*. In: Irving PM, editor. *Acidic Deposition: State of Science and Technology. NAPAP Report 18*. Vol. III. Terrestrial, Materials, Health and Visibility Effects. Washington, DC.: NAPAP; 1991.
- ~~523-521.~~ Skärby L, Troeng E, Boström C-Å. 1987. Ozone Uptake And Effects On Transpiration, Net Photosynthesis, And Dark Respiration In Scots Pine. *For Sci*, 33(3): 801-808.
- ~~524-522.~~ Skelly, J.M., Yang, Y-S., Chevonne, B.I., Long, S.J., Nellessen, J.E., Winner, W.E.; 1983. Ozone Concentrations And Their Influence On Forest Species In The Blue Ridge Mountains Of Virginia. In: David, D.D., Miller, A.A., Dochinger, L., (Eds.); *Air Pollution And The Productivity Of The Forest*. Isaac- Walton League Of America; Arlington, VA; Pp. 143-160.
- ~~525-523.~~ Smeulders, S.M., Gorissen, A., Joosten, N.N., Vanveen, J.A.; 1995. Effects Of Short-Term Ozone Exposure On The Carbon Economy Of Mature And Juvenile Douglas Firs [*Pseudotsuga Menziesii* (Mirb) Franco]. *New Phytologist*, 129; 45-53.

- 526-524. Snyder, R. G.; Simon, J. E.; Reinert, R. A.; Simini, M.; Heck, W. W. 1988. Effects Of Air Quality On Foliar Injury, Growth, Yield, And Quality Of Muskmelon. *Environ. Pollut.* 53: 187-196.
- 527-525. Snyder, R. G.; Simon, J. E.; Reinert, R. A.; Simini, M.; Wilcox, G. E. 1991. Effects Of Air Quality On Growth, Yield, And Quality Of Watermelon. *Hortscience-Hortscienc.* 26: 1045-1047.
- 528-526. Soja, G.; Barnes, J. D.; Posch, M.; Vandemieiren, K.; Pleijel, H.; Mills, G. 2000. Phenological Weighting Of Ozone Exposures In The Calculation Of Critical Levels For Wheat, Bean And Plantain. *Environmental Pollution*, 109: 517-524.
- 529-527. Spash, C. L. 1997. Assessing The Economic Benefits To Agriculture From Air Pollution Control. *J. Econ. Surv.* 11: 47-70.
- 530-528. Spence, R.D., Rykiel, E.J., Sharpe, P.J.H., 1990. Ozone Alters Carbon Allocation In Loblolly Pine: Assessment With Carbon-11 Labeling. *Environmental Pollution*, 64, 93-106.
- 531-529. Stewart, C. A.; Black, V. J.; Black, C. R.; Roberts, J. A. 1996. Direct Effects Of Ozone On The Reproductive Development Of Brassica Species. *J. Plant Physiol.* 148: 172-178.
- 532-530. Summers, C. G.; Retzlaff, W. A., Stephenson, S. 1994. The effect of ozone on the mean relative growth rate of *Diuraphis Noxia* (Mordvilko) (Homoptera:Aphididae). *Journal of Agricultural Entomology*, 11: 181-187.
- 533-531. Takemoto BK, Bytnerowicz A, Dawson PJ, Morrison CL, and Temple PJ. 1997. Effects of ozone on *Pinus ponderosa* seedlings: comparison of responses in the first and second growing seasons of exposure. *Can J For Res*, 27: 23-30.
- 534-532. Takemoto BK, Bytnerowicz A, Fenn ME. 2001. Current And Future Effects Of Ozone And Atmospheric Nitrogen Deposition On And Future Effects of Ozone and Atmospheric Nitrogen Deposition on California's Mixed Conifer Forests. *For Ecol Manage*, 144: 159-173.
- 535-533. Takemoto, B. K.; Olszyk, D. M.; Johnson, A. G.; Parada, C. R. 1988b. Yield Responses Of Field-Grown Crops To Acidic Fog And Ambient Ozone. *J. Environ. Qual.* 17:192-197.
- 536-534. Taylor GE Jr, Hanson PJ. 1992. Forest trees and tropospheric ozone: role of canopy deposition and leaf uptake in developing exposure-response relationships. *Agric Ecosys Environ*, 42: 255-273.
- 537-535. Taylor GE Jr, Norby RJ, McLaughlin SB, Johnson AH, Turner RS. 1986. Carbon dioxide assimilation and growth of red spruce (*Picea rubens* Sarg.) seedlings in response to ozone, precipitation chemistry, and soil type. *Oecologia*, 70: 163-171.

- ~~538-536.~~ Taylor GE Jr. 1994. Role of genotype in the response of loblolly pine to tropospheric ozone: effects at the whole-tree, stand, and regional level. *J Environ Qual.* 23: 63-82.
- ~~539-537.~~ Taylor, G. E. Jr.; Tingey, D. T.; Ratsch, H. C. 1982a. Ozone Flux in Glycine Max (L.) Merr.: Sites of Regulation and Relationship to Leaf Injury. *Oecologia.* 53: 179-186.
- ~~540-538.~~ Taylor, G., Davies, W.J., 1990. Root Growth of *Fagus Sylvatica*: Impact of Air Quality and Drought at a Site in Southern Britain. *New Phytologist.* 116: 457-464.
- ~~541-539.~~ Taylor, G., Ferris, R., 1996. Influence of Air Pollution on Root Physiology and Growth. In: Yunus, M., Iqbal, M., Eds. *Plant Response to Air Pollution*. London, J.Wiley and Sons, Pp. 375-394.
- ~~542-540.~~ Taylor, G.E., Johnson, D.J., Andersen, C.P. 1994. Air Pollution and Forest Ecosystems: A Regional to Global Perspective. *Ecological Applications.* 4: 662-689.
- ~~543-541.~~ Temple PJ, Bytnerowicz A. 1993. Growth, Physiological, and Biochemical Responses of Ponderosa Pine (*Pinus ponderosa*) to Ozone. Sacramento, CA.: ARB, 1993. Final Report, on contracts Nos. A733-137, A833-083, and A033-056. NTIS No. PB94131372.
- ~~544-542.~~ Temple PJ, Miller PR. 1994. Foliar ozone injury and radial growth of ponderosa pine. *Can J For Res.* 24: 1,877-1,882.
- ~~545-543.~~ Temple PJ, Riechers GH, Miller PR, Lennox RW. Growth responses of ponderosa pine to long-term exposure to ozone, wet and dry acidic deposition, and drought. *Can J For Res* 1993; 23: 59-66.
- ~~546-544.~~ Temple PJ, Riechers GH, Miller PR. 1992. Foliar injury responses of ponderosa pine seedlings to ozone, wet and dry acidic deposition, and drought. *Environ Exp Bot.* 32(2): 101-113.
- ~~547-545.~~ Temple, P. J. 1991. Variations in Responses of Dry Bean (*Phaseolus Vulgaris*) Cultivars to Ozone. *Agric. Ecosyst. Environ.* 36: 1-11.
- ~~548-546.~~ Temple, P. J., Lennox, R. W.; Bytnerowicz, A.; Taylor, O. C. 1987. Interactive Effects of Simulated Acidic Fog and Ozone on Field-Grown Alfalfa. *Environ. And Exper. Bot.* 27(4): 409-417.
- ~~549-547.~~ Temple, P. J.; Jones, T. E., Lennox, R. W. 1990. Yield Loss Assessments for Cultivars of Broccoli, Lettuce, and Onion Exposed to Ozone. *Environmental Pollution.* 66: 289-299.
- ~~550-548.~~ Temple, P.J. 1990b. Growth and Yield Responses of Processing Tomato (*Lycopersicon Esculentum* Mill.) Cultivars to Ozone. *Environ. And Exper. Bot.* 30(3): 283-291.

- ~~551-549.~~ Temple, P.J., 1986. Stomatal Conductance ~~And~~ Transpirational Responses ~~Of~~ Field-Grown Cotton ~~To~~ Ozone. *Plant Cell ~~And~~ Environment*, 9, 315-321.
- ~~552-550.~~ Temple, P.J., 1990a. Water Relations ~~Of~~ Differentially Irrigated Cotton Exposed ~~To~~ Ozone. *Agronomy Journal*, 82, 800-805.
- ~~553-551.~~ Temple, P.J., 1990c. Growth Form ~~And~~ Yield Responses ~~Of~~ Four Cotton Cultivars ~~To~~ Ozone. *Agronomy Journal*, 82, 1045-1050.
- ~~554-552.~~ Temple, P.J., Benoit, L.F., Lennox, R.W., Reagan, C.A., Taylor, O.C. 1988b. Combined Effects ~~Of~~ Ozone ~~And~~ Water Stress ~~On~~ Alfalfa Growth ~~And~~ Yield. *Journal ~~Of~~ Environmental Quality* 17, 108-113.
- ~~555-553.~~ Temple, P.J., Kupper, R.S., Lennox, R.W., Rohr, K., 1988a. Injury ~~And~~ Yield Responses ~~Of~~ Differentially Irrigated Cotton ~~To~~ Ozone. *Agronomy Journal*, 80, 751-755.
- ~~556.~~ Temple, P.J., Riechers, G.H., Miller, P.R., Lennox, R.W. 1993. Growth Responses ~~Of~~ Ponderosa Pine ~~To~~ Long Term Exposure ~~To~~ Ozone, Wet ~~And~~ Dry Acidic Deposition, ~~And~~ Drought. *Canadian Journal ~~Of~~ Forestry Research* 23, 559-566.
- ~~557-554.~~ Tetlow, I.J., Farrar, J.F. 1993. Apoplastic Sugar Concentration ~~And~~ ~~pH~~ ~~in~~ Barley Leaves Infected ~~With~~ Brown Rust. *Journal ~~Of~~ Experimental Botany*, 44, 929-936.
- ~~558-555.~~ Thompson, C. R. 1985. Effects ~~Of~~ Sulfur Dioxide ~~And~~ Ambient Ozone ~~On~~ Winter Grown Crop ~~in~~ California. California Air Resources Board, Final Report Agreement No. Contract A3-057-33, Pp. 51 ~~And~~ Appendix.
- ~~559-556.~~ Thompson, C.R. Olszyk, D.M. 1986. Crop Loss ~~From~~ Air Pollutants Assessment Program. Interim Report ~~To~~ California Air Resources Board ~~On~~ Contracts A5-031-33 ~~And~~ A4-088-33.
- ~~560-557.~~ Thomson, W.W., Dugger, W.M., Palmer, R.L., 1966. Effect ~~Of~~ Ozone ~~On~~ ~~the~~ Fine Structure ~~Of~~ ~~the~~ Palisade Parenchyma Cells ~~Of~~ Bean Leaves. *Canadian Journal ~~Of~~ Botany*, 44, 1677-1682.
- ~~561-558.~~ Ting IP, Mukerji SK. 1971. Leaf Ontogeny ~~As~~ ~~a~~ Factor ~~in~~ Susceptibility ~~To~~ Ozone: Amino Acid ~~And~~ Carbohydrate Changes ~~During~~ Expansion. *Amer J Bot*, 58(6): 497-504.
- ~~562-559.~~ Tingey, D. T.; Rodecap, K. D.; Lee, E. H.; Hogsett, W. E.; Gregg, J. W. 2002. Pod Development Increases ~~The~~ Ozone Sensitivity ~~Of~~ Phaseolus Vulgaris. *Water Air Soil Pollut.* 139: 325-341.
- ~~563-560.~~ Tingey, D.T., Fites, R.C., Wickliff, C., 1973. Foliar Sensitivity ~~Of~~ Soybeans ~~To~~ Ozone ~~As~~ Related ~~To~~ Several Leaf Parameters. *Environmental Pollution*, 4, 183-192.
- ~~564-561.~~ Tingey, D.T., Heck, W.W., Reinert, R.A., 1971. Effect ~~Of~~ Low Concentrations ~~Of~~ Ozone ~~And~~ Sulfur Dioxide ~~On~~ Foliage Growth

- And and Yield Of Radish. Journal Of The American Society Of Horticultural Science. 96: 369-371.
- 565-562. Tjoelker M.G.; Volin J.C.; Oleksyn J; Reich P.B. 1995b. Interaction Of Ozone Pollution And Light Effects On Photosynthesis In A Forest Canopy Experiment. Plant, Cell & Environment. 18(8): 895-905.
- 566-563. Tjoelker M.G., Volin, J.C., Oleksyn, J., Reich, P.B. 1995a. Light environment alters response to ozone stress in seedlings of *Acer saccharum* Marsh and hybrid *Populus L.* In situ net photosynthesis, dark respiration and growth. New Phytologist. 124: 627-636.
- 567-564. Tonneijck, A. E. G. 1994. Effects Of Various Ozone Exposures On The Susceptibility Of Bean Leaves (Phaseolus Vulgaris L.) To Botrytis Cinerea. Environ. Pollut. 85: 59-65.
- 568-565. Tonneijck, A. E. G.; Leone, G. 1993. Changes In Susceptibility Of Bean Leaves (Phaseolus Vulgaris) To Sclerotinia Sclerotiorum And Botrytis Cinerea By Pre-Inoculative Ozone Exposures. Neth. J. Plant Pathol. 99: 313-322.
- 569-566. Tonneijck, A. E. G.; Van Dijk, C. J. 1998. Responses Of Bean (Phaseolus Vulgaris L. Cv. Pros) To Chronic Ozone Exposure At Two Levels Of Atmospheric Ammonia. Environ. Pollut. 99: 45-51.
- 570-567. Topa, M.A., Vanderklein, D.W., Corbin, A. 2001. Effects of elevated ozone and low light on diurnal and seasonal carbon gain in sugar maple. Plant Cell and Environment. 24:663-677.
- 571-568. Torsethaugen G., Pell, E.J., Assmann, S.M. 1999. Ozone Inhibits Guard Cell K⁺ Channels Implicated In Stomatal Opening. Proceedings National Academy Of Sciences. 96: 13577-13582.
- 572-569. Troughton, A., 1955. The Application Of The Allometric Formula To The Study Of The Relationship Between Roots And Shoots. Agricultural Progress. 30: 59-65.
- 573-570. Turner, N.C., Rich, S., Waggoner, P.E. 1973. Removal Of Ozone By Soil. Journal Of Environmental Quality. 2: 259-264.
- 574-571. Tyree, M.T., Sperry, J.S. 1988. Do Woody Plants Operate Near The Point Of Catastrophic Xylem Dysfunction Caused By Dynamic Water Stress? Plant Physiology. 88: 574-580.
- 575-572. United States Environmental Protection Agency. 1978. Air Quality Criteria For Ozone And Other Photochemical Oxidants. Research Triangle Park, NC: Office Of Health And Environmental Assessment, Environmental Criteria And Assessment Office; Report No. EPA-600/8-78-004. Available From NTIS, Springfield, VA; PB80-124753.
- 576-573. United States Environmental Protection Agency. 1986. Air Quality Criteria For Ozone And Other Photochemical Oxidants. Research Triangle Park, NC: Office Of Health And Environmental Assessment,

Environmental Criteria ~~And~~ Assessment Office; Report No. EPA-600/8-84-020af-Ef. 5 Volumes. Available From NTIS, Springfield, VA; PB87-142949.

~~577-574.~~ United States Environmental Protection Agency. 1992. Summary Of Selected New Information ~~On~~ Effects ~~Of~~ Ozone ~~On~~ Health ~~And~~ Vegetation: Supplement ~~To~~ 1986 Air Quality Criteria ~~For~~ Ozone ~~And~~ Other Photochemical Oxidants. Research Triangle Park, NC: Office ~~Of~~ Health ~~And~~ Environmental Assessment, Environmental Criteria ~~And~~ Assessment Office; Report No. EPA-600/8-88/105F. Available From NTIS, Springfield, VA; PB92-235670.

~~578-575.~~ United States Environmental Protection Agency. 1996a. Air Quality Criteria ~~For~~ Ozone ~~And~~ Other Photochemical Oxidants. Research Triangle Park, NC: Office ~~Of~~ Research ~~And~~ Development; Reports No. EPA-600/AP-93/004af-Cf. 3 Volumes. Available From NTIS, Springfield, VA; PB96-185582, PB96-185590, PB96-185608, And Online At: [Www.Epa.Gov/Ncea/Ozone.Htm](http://www.epa.gov/ncea/ozone.htm).

~~579-576.~~ United States Environmental Protection Agency. 1996b. A Review ~~Of~~ National Ambient Air Quality Standards ~~For~~ Ozone: Assessment ~~Of~~ Scientific ~~And~~ Technical Information. OAQPS ~~s~~Staff Paper. Research Triangle Park, NC: Office ~~Of~~ Air Quality Planning ~~And~~ Standards; Reports No. EPA-452/R-96/007. Available From NTIS, Springfield, VA; PB96-203435/XAB, And Online At [Http://Epa.Gov/Ttn/Oarpg/T1sp.Html](http://epa.gov/ttn/oarpg/T1sp.html).

~~580-577.~~ Unsworth, M.H., Heagle, A.S., Heck, W.W. 1984a. Gas exchange in open-top field chambers. I. Measurement and analysis of atmospheric resistances to gas exchange. *Atmospheric Environment*, 18:373-380.

~~581-578.~~ Unsworth, M.H., Heagle, A.S., Heck, W.W. 1984b. Gas exchange in open-top field chambers. II. Resistances to ozone uptake by soybeans. *Atmospheric Environment*, 18:381-385.

~~582-579.~~ Van Oijen, M., Ewert, F. 1999. The effects of climatic variation in Europe on the yield response of spring wheat cv. Minaret to elevated CO₂ and ~~ozone~~ O₃: an analysis of open-top chamber experiments by means of two crop growth simulation models. *European Journal of Agronomy*, 10:249-264.

~~583-580.~~ Van Ooy DJ, Carroll JJ. 1995. The spatial variation of ozone climatology on the western slope of the Sierra Nevada. *Atmos Environ*, 1995; 29(11): 1,319-1,330.

~~584-581.~~ Volin, J. C.; Reich, P. B.; Givnish, T. J. 1998. Elevated Carbon Dioxide Ameliorates ~~The~~ Effects ~~Of~~ Ozone ~~On~~ Photosynthesis ~~And~~ Growth: Species Respond Similarly Regardless ~~Of~~ Photosynthetic Pathway or Plant Functional Group. *New Phytol*. 138: 315-325.

~~585-582.~~ Vorne, V.; Ojanpera, K.; De Temmerman, L.; Bindi, M.; Hogy, P.; Jones, M. B.; Lawson, T.; Persson, K. 2002. Effects ~~Of~~ Elevated Carbon Dioxide ~~And~~ Ozone ~~On~~ Potato Tuber Quality ~~In~~ ~~The~~ European

- Multiple-Site Experiment 'CHIP-Project'. *European Journal of Agronomy*. 17: 369-381.
- ~~586-583.~~ 586-583. Vozzo, S. F.; Miller, J. E.; Pursley, W. A.; Heagle, A. S. 1995. Effects of Ozone and Water Deficit on Field-Grown Soybean: I. Leaf Gas Exchange. *J. Environ. Qual.* 24: 663-670.
- ~~587-584.~~ 587-584. Wallace RA, King JL, Sanders GP. 1981. Biology: The science of life. Santa Monica: Goodyear Publishing Co., Inc. Santa Monica, CA; 1981. 1,140 pp.
- ~~588-585.~~ 588-585. Warwick, K.R., Taylor, G., 1995. Contrasting Effects of Tropospheric Ozone on Five Native Herbs Which Coexist in Calcareous Grassland. *Global Change Biology*; 1; 143-151.
- ~~589-586.~~ 589-586. Weber JA, Clark CS, Hogsett WE. 1993. Analysis of the relationships among ozone uptake, conductance, and photosynthesis in needles of *Pinus ponderosa*. *Tree Physiol*; 13: 157-172.
- ~~590-587.~~ 590-587. Weinstock, L.; Kender, W. J.; Musselman, R. C. 1982. Microclimate Within Open-Top Air Pollution Chambers and Its Relation to Grapevine Physiology. *J. Am. Soc. Hortic. Sci.* 107: 923-929.
- ~~591-588.~~ 591-588. Welfare, K.; Flowers, T. J.; Taylor, G.; Yeo, A. R. 1996. Additive and antagonistic effects of ozone and salinity on the growth, ion contents, and gas exchange of five varieties of rice (*Oryza sativa* L.). *Environmental Pollution*. 92: 257-266.
- ~~592-589.~~ 592-589. Wesely, M.L., Eastman, J.A., Cook, D.R., Hicks, B.B., 1978. Daytime Variations of Ozone Eddy Fluxes to Maize. *Boundary-Layer Meteorology*. 15; 361-373.
- ~~593-590.~~ 593-590. Whitfield, C.P., Davison, A.W., Ashenden, T.W. 1998. The effects of nutrient limitation on the response of *Plantago major* to ozone. *New Phytologist*. 140: 219-230.
- ~~594-591.~~ 594-591. Whittaker, J. B. (1994). Interactions Between Insects and Air Pollutants. In: Alscher, R. G.; Wellburn, A. R., Eds. *Plant Responses to the Gaseous Environment. Molecular, Metabolic and Physiological Aspects.* London, United Kingdom: Chapman and Hall; London, United Kingdom. Pp. 365-384.
- ~~595-592.~~ 595-592. Wiese, C.B., Pell, E.J., 1997. Influence of Ozone on Transgenic Tobacco Plants Expressing Reduced Quantities of Rubisco. *Plant, Cell and Environment*; 20; 1283-1291.
- ~~596-593.~~ 596-593. Wilbourn, S., Davison, A.W., Ollerenshaw, J.H. 1995. The Use of an Unenclosed Field Fumigation System to Determine the Effects of ozone on a Grass-Clover Mixture. *New Phytologist*. 129, 23-32.
- ~~597-594.~~ 597-594. Wilkinson TG, Barnes RL. 1973. Effects of ozone on ¹⁴CO₂ fixation patterns in pine. *Can J Bot*. 51(9): 1,573-1,578.

- ~~598-595.~~ Williams WT, Williams JA. 1986. Effects of oxidant air pollution on needle health and annual-ring width in a ponderosa pine forest. *Environ Conserv*, 13(3): 229-234.
- ~~599-596.~~ Wiltshire, J. J. J.; Wright, C. J.; Unsworth, M. H. 1992. A New Method For Exposing Mature Trees To Ozone Episodes. *For. Ecol. Manage.* 51: 115-120.
- ~~600-597.~~ Wolfenden J, Wellburn AR. 1991. Effects of summer ozone on membrane lipid composition during subsequent frost hardening in Norway spruce [*Picea abies* (L.) Karst]. *New Phytol*, 118: 323-329.
- ~~601-598.~~ Younglove, T.; Mccool, P. M.; Musselman, R. C.; Kahl, M. E. 1994. Growth-Stage Dependent Crop Yield Response To Ozone Exposure. *Environmental Pollution*, 86: 287-295.
- ~~602-599.~~ Zelitch I. 1982. The close relationship between net photosynthesis and crop yield. *Bioscience*, 32: 796-802.
- ~~603-600.~~ Zhang, J., Davies, WJ. Changes in the concentration of ABA in xylem sap as a function of changing soil water status can account for change in Leaf Conductance And Growth. *Plant Cell And Environment*, 13: 277-285.
- ~~604-601.~~ Zheng, Y., Shimizu, H., Barnes, J.D., 2002. Limitations To CO₂ Assimilation In Ozone-Exposed Leaves Of *Plantago Major*. *New Phytologist*, 155: 67-78.
- ~~605-602.~~ Zheng, Y.; Lyons, T.; Barnes, J. 2000. Effects Of Ozone On The Production And Utilization Of Assimilates In *Plantago Major*. *Environmental And Experimental Botany*, 43: 171-180.
- ~~606-603.~~ Zimmerman R, Oren R, Schulze E-D, Werk KS. 1988. Performance of two *Picea abies* (L.) Karst. stands at different stages of decline. II. Photosynthesis and leaf conductance. *Oecologia*, 76: 513-518.

Chapter 9

- ~~607-604.~~ Adams WC, Brookes KA, Schelegle ES. 1987. Effects of NO₂ alone and in combination with ozone-O₃ on young men and women. *J Appl Physiol* 62:1698-704.
- ~~608-605.~~ Adams WC, Savin WM, Christo AE. 1981. Detection of ozone toxicity during continuous exercise via the effective dose concept. *J Appl Physiol* 51:415-22.
- ~~609-606.~~ Adams WC, Schelegle ES, Shaffrath JD. 1989. Oral and oronasal breathing during continuous exercise produce similar responses to ozone inhalation. *Arch Environ Health* 44:311-6.
- ~~610-607.~~ Adams WC, Schelegle ES. 1983. Ozone and high ventilation

effects on pulmonary function and endurance performance. *J Appl Physiol* 55:805-12.

611-608. Adams WC. 1986. Physiological responses of healthy human subjects consequent to inhalation of NO₂, ~~ozone~~-O₃ and NO₂ plus ozone during heavy, sustained exercise. Sacramento, CA: California Air Resources Board. Sacramento, CA: California Air Resources Board.

612-609. Adams WC. 2000a. Feasibility study of prolonged ozone inhalation exposure via face mask. *Inhal Toxicol* 12:299-313

613-610. Adams WC. 2000b. Ozone dose-response effects of varied equivalent minute ventilation rates. *J Expo Anal Environ Epidemiol* 10: 217-26.

614-611. Adams WC. 2002. Comparison of chamber and face-mask 6.6-hour exposures to ozone on pulmonary function and symptoms responses. *Inhal Toxicol* 14:745-64.

615-612. Adams WC. 2003a. Comparison of chamber and face mask 6.6-hour exposure to 0.08 ppm ozone via square-wave and triangular profiles on pulmonary responses. *Inhal Toxicol* 15:265-81.

616-613. Adams WC. 2003b. Relation of pulmonary responses induced by 6.6-hour exposures to 0.08 ppm ozone and 2-hour exposures to 0.30 ppm ozone via chamber and face-mask inhalation. *Inhal Toxicol* 15:745-59.

617-614. Adamson IY, Vincent R, Bjarnason SG. 1999. Cell injury and interstitial inflammation in rat lung after inhalation of ozone and urban particulates. *Am J Respir Cell Mol Biol* 20:1067-72.

618-615. American Thoracic Society. 1995. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease: definitions, epidemiology, pathophysiology, diagnosis, and staging. 152 :S78-S83.

619-616. Aris R, Christian D, Sheppard D, Balmes JR. 1991. The effects of sequential exposure to acidic fog and ozone on pulmonary function in exercising subjects. *Am Rev Respir Dis* 143:85-91.

620-617. Aris R, Christian D, Tager I, Ngo L, Finkbeiner WE, Balmes JR. 1993a. Effects of nitric acid gas alone or in combination with ozone on healthy volunteers. *Am Rev Respir Dis* 148:965-73.

621-618. Aris RM, Christian D, Hearne PQ, Kerr K, Finkbeiner WE, Balmes JR. 1993b. Ozone-induced airway inflammation in human subjects as determined by airway lavage and biopsy. *Am Rev Respir Dis* 148:1363-72.

622-619. Aris RM, Tager I, Christian D, Kelly T, Balmes JR. 1995.

Methacholine responsiveness is not associated with ozone O_3 -induced decreases in FEV1. Chest 107:621-8.

~~623-620.~~ Arito H, Uchiyama I, Arakawa H, Yokoyama E. 1990. Ozone-induced bradycardia and arrhythmia and their relation to sleep-wakefulness in rats. Toxicol Lett 52:169-78.

~~624-621.~~ Arsalane K, Broeckaert F, Knoop B, Clippe A, Buchet JP, Bernard A. 1999. Increased serum and urinary concentrations of lung Clara cell protein in rats acutely exposed to ozone. Toxicol Appl Pharmacol 159:169-74.

~~625-622.~~ Asplund PT, Ben-Jebria A, Rigas ML, Ultman JS. 1996. Longitudinal distribution of ozone absorption in the lung: effect of continuous inhalation exposure. Arch Environ Health 51:431-8.

~~626-623.~~ Avissar NE, Reed CK, Cox C, Frampton MW, Finkelstein JN. 2000. Ozone, but not nitrogen dioxide, exposure decreases glutathione peroxidases in epithelial lining fluid of human lung. Am J Respir Crit Care Med 162:1342-7.

~~627-624.~~ Avol EL, Linn WS, Shamoo DA, Spier CE, Valencia LM, Venet TG, Trim SC, Hackney JD. 1987. Short-term respiratory effects of photochemical oxidant exposure in exercising children. JAPCA 37:158-62.

~~628-625.~~ Avol EL, Linn WS, Shamoo DA, Valencia LM, Anzar UT, Venet TG, Hackney JD. 1985. Respiratory effects of photochemical oxidant air pollution in exercising adolescents. Am Rev Respir Dis 132:619-22.

~~629-626.~~ Avol EL, Linn WS, Venet TG, Shamoo DA, Hackney JD. 1984. Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. J Air Pollut Control Assoc 34:804-9.

~~630-627.~~ Avol EL, Navidi WC, Rappaport EB, Peters JM. 1998. Acute effects of ambient ozone on asthmatic, wheezy, and healthy children. Res Rep Health Eff Inst ~~iii~~; Report #82. 1-18; discussion 19-30.

~~631-628.~~ Ball BA, Folinsbee LJ, Peden DB, Kehrl H. 1996. Allergen bronchoprovocation of patients with mild allergic asthma after ozone exposure. J Allergy Clin Immunol 98:563-72.

~~632-629.~~ Balmes JR, Aris RM, Chen LL, Scannell C, Tager IB, Finkbeiner W, Christian D, Kelly T, Hearne PQ, Ferrando R, Welch B. 1997. Effects of ozone on normal and potentially sensitive human subjects. Part I: Airway inflammation and responsiveness to ozone in normal and asthmatic subjects. Res Rep Health Eff Inst ~~iv~~; 1-37; discussion 81-99.

~~633-630.~~ Balmes JR, Chen LL, Scannell C, Tager I, Christian D, Hearne PQ,

Kelly T, Aris RM. 1996. Ozone-induced decrements in FEV1 and FVC do not correlate with measures of inflammation. *Am J Respir Crit Care Med* 153:904-9.

~~634-631.~~ Barr BC, Hyde DM, Plopper CG, Dungworth DL. 1988. Distal airway remodeling in rats chronically exposed to ozone. *Am Rev Respir Dis* 137:924-38.

~~635-632.~~ Barr BC, Hyde DM, Plopper CG, Dungworth DL. 1990. A comparison of terminal airway remodeling in chronic daily versus episodic ozone exposure. *Toxicol Appl Pharmacol* 106:384-407.

~~636-633.~~ Barry BE, Crapo JD. 1985. Application of morphometric methods to study diffuse and focal injury in the lung caused by toxic agents. *Crit Rev Toxicol* 14:1-32.

~~637-634.~~ Barry BE, Mercer RR, Miller FJ, Crapo JD. 1988. Effects of inhalation of 0.25 ppm ozone on the terminal bronchioles of juvenile and adult rats. *Exp Lung Res* 14:225-45.

~~638-635.~~ Barry BE, Miller FJ, Crapo JD. 1985. Effects of inhalation of 0.12 and 0.25 parts per million ozone on the proximal alveolar region of juvenile and adult rats. *Lab Invest* 53:692-704.

~~639-636.~~ Bartlett D Jr, Faulkner CS 2nd, Cook K. 1974. Effect of chronic ozone exposure on lung elasticity in young rats. *J Appl Physiol* 37:92-6.

~~640-637.~~ Bascom R, Naclerio RM, Fitzgerald TK, Kagey-Sobotka A, Proud D. 1990. Effect of ozone inhalation on the response to nasal challenge with antigen of allergic subjects. *Am Rev Respir Dis* 142:594-601.

~~641-638.~~ Basha MA, Gross KB, Gwizdala CJ, Haidar AH, Popovich J Jr. 1994. Bronchoalveolar lavage neutrophilia in asthmatic and healthy volunteers after controlled exposure to ozone and filtered purified air. *Chest* 106:1757-65.

~~642-639.~~ Bassett D, Elbon-Copp C, Otterbein S, Barraclough-Mitchell H, Delorme M, Yang H. 2001. Inflammatory cell availability affects ozone-induced lung damage. *J Toxicol Environ Health A* 64:547-65.

~~643-640.~~ Bassett DJ, Elbon-Copp C, Ishii Y, Barraclough-Mitchell H, Yang H. 2000. Lung tissue neutrophil content as a determinant of ozone-induced injury. *J Toxicol Environ Health A* 60:513-30.

~~644-641.~~ Bates DV, Bell GM, Burnham CD, Hazucha M, Mantha J, Pengelly LD, Silverman F. 1972. Short-term effects of ozone on the lung. *J Appl Physiol* 32:176-81.

- 645-642. Bayram H, Rusznak C, Khair OA, Sapsford RJ, Abdelaziz MM. 2002. Effect of ozone and nitrogen dioxide on the permeability of bronchial epithelial cell cultures of non-asthmatic and asthmatic subjects. *Clin Exp Allergy* 32:1285-92.
- 646-643. Beckett WS, McDonnell WF, Horstman DH, House DE. 1985. Role of the parasympathetic nervous system in acute lung response to ozone. *J Appl Physiol* 59:1879-85.
- 647-644. Bedi JF, Drechsler-Parks DM, Horvath SM. 1985. Duration of increased pulmonary function sensitivity to an initial ozone exposure. *Am Ind Hyg Assoc J* 46:731-4.
- 648-645. Bedi JF, Folinsbee LJ, Horvath SM, Ebenstein RS. 1979. Human exposure to sulfur dioxide and ozone: absence of a synergistic effect. *Arch Environ Health* 34:233-9.
- 649-646. Bedi JF, Horvath SM, Drechsler-Parks DM. 1988. Reproducibility of the pulmonary function response of older men and women to a 2-hour ozone exposure. *JAPCA* 38:1016-9.
- 650-647. Bedi JF, Horvath SM, Drechsler-Parks DM. 1989. Adaptation by older individuals repeatedly exposed to 0.45 parts per million ozone for two hours. *JAPCA* 39:194-9.
- 651-648. Bedi JF, Horvath SM, Folinsbee LJ. 1982. Human exposure to sulfur dioxide and ozone in a high temperature-humidity environment. *Am Ind Hyg Assoc J* 43:26-30.
- 652-649. Bell KA, Linn WS, Hazucha M, Hackney JD, Bates DV. 1977. Respiratory effects of exposure to ozone plus sulfur dioxide in Southern Californians and Eastern Canadians. *Am Ind Hyg Assoc J* 38:696-706.
- 653-650. Bergamaschi E, De Palma G, Mozzoni P, Vanni S, Vettori MV, Broeckaert F, et al. 2001. Polymorphism of quinone-metabolizing enzymes and susceptibility to ozone-induced acute effects. *Am J Respir Crit Care Med* 163(6):1426-31.
- 654-651. Berger WE. 2003. Overview of allergic rhinitis. *Ann Allergy Asthma Immunol* 90:7-12.
- 655-652. Bhalla DK, Gupta SK. 2000. Lung injury, inflammation, and inflammatory stimuli in rats exposed to ozone. *J Toxicol Environ Health A* 59:211-28.
- 656-653. Bhalla DK. 1999. Ozone-induced lung inflammation and mucosal barrier disruption: toxicology, mechanisms, and implications. *J Toxicol Environ Health B Crit Rev* 2:31-86.

- 657-654. Biagini RE, Moorman WJ, Lewis TR, Bernstein IL. 1986. Ozone enhancement of platinum asthma in a primate model. *Am Rev Respir Dis* 134:719-25.
- 658-655. Bignami G. 1996. Economical test methods for developmental neurobehavioral toxicity. *Environ Health Perspect* 104 Suppl 2:285-98.
- 659-656. Bignami G, Musi B, Dell'Omo G, Laviola G, Alleva E. 1994. Limited effects of ozone exposure during pregnancy on physical and neurobehavioral development of CD-1 mice. *Toxicol Appl Pharmacol* 129:264-71.
- 660-657. Blomberg A, Mudway IS, Nordenhall C, Hedenstrom H, Kelly FJ, Frew AJ, Holgate ST, Sandstrom T. 1999. Ozone-induced lung function decrements do not correlate with early airway inflammatory or antioxidant responses. *Eur Respir J* 13:1418-28.
- 661-658. Boatman ES, Frank R. 1974. Morphologic and ultrastructural changes in the lungs of animals during acute exposure to ozone. *Chest* 65:Suppl:9S-11S.
- 662-659. Boorman GA, Hailey R, Grumbein S, Chou BJ, Herbert RA, Goehl T, Mellick PW, Roycroft JH, Haseman JK, Sills R. 1994. Toxicology and carcinogenesis studies of ozone and ozone 4-(N-nitrosomethylamino)-1-(3-pyridyl)-1-butanone in Fischer-344/N rats. *Toxicol Pathol* 22:545-54.
- 663-660. Boorman GA, Schwartz LW, Dungworth DL. 1980. Pulmonary effects of prolonged ozone insult in rats. Morphometric evaluation of the central acinus. *Lab Invest* 43:108-15.
- 664-661. Bosson J, Stenfors N, Bucht A, Helleday R, Pourazar J, Holgate ST, Kelly FJ, Sandstrom T, Wilson S, Frew AJ, Blomberg A. 2003. Ozone-induced bronchial epithelial cytokine expression differs between healthy and asthmatic subjects. *Clin Exp Allergy* 33:777-82.
- 665-662. Bouthillier L, Vincent R, Goegan P, Adamson IY, Bjarnason S, Stewart M, Guenette J, Potvin M, Kumarathasan P. 1998. Acute effects of inhaled urban particles and ozone: lung morphology, macrophage activity, and plasma endothelin-1. *Am J Pathol* 153:1873-84.
- 666-663. Brook RD, Brook JR, Urch B, Vincent R, Rajagopalan S, Silverman F. 2002. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation* 105:1534-6.
- 667-664. Brookes KA, Adams WC, Schelegle ES. 1989. 0.35 ppm O_3 ozone exposure induces hyperresponsiveness on 24-h reexposure to 0.20 ppm ozone O_3 . *J Appl Physiol* 66:2756-62.

- 668-665. Brummer MEG, Schwartz LW, McQuillen NK. 1977. A quantitative study of lung damage by scanning electron microscopy. Inflammatory cell response to high-ambient levels of ozone. Chicago, IL: IIT Research Institute. Chicago, IL: IIT Research Institute:513-8.
- 669-666. Bush ML, Asplund PT, Miles KA, Ben-Jebria A, Ultman JS. 1996. Longitudinal distribution of O₃ absorption in the lung: gender differences and intersubject variability. *J Appl Physiol* 81:1651-1657.
- 670-667. Calderon-Garciduenas L, Devlin RB, Miller FJ. 2000a. Respiratory tract pathology and cytokine imbalance in clinically healthy children chronically and sequentially exposed to air pollutants. *Med Hypotheses* 55:373-8.
- 671-668. Calderon-Garciduenas L, Mora-Tiscareno A, Chung CJ, Valencia G, Fordham LA, Garcia R, Osnaya N, Romero L, Acuna H, Villarreal-Calderon A, Devlin RB, Koren HS. 2000b. Exposure to air pollution is associated with lung hyperinflation in healthy children and adolescents in Southwest Mexico City: a pilot study. *Inhal Toxicol* 12:537-61.
- 672-669. California Air Resources Board. 1994. Review of the one-hour ambient air quality standard for sulfur dioxide. Staff Report. August.
- 673-670. California Department of Health Services. 1987. Recommendation for an Ambient Air Quality Standard for Ozone.
- 674-671. Castleman WL, Dungworth DL, Schwartz LW, Tyler WS. 1980. Acute respiratory bronchiolitis: an ultrastructural and autoradiographic study of epithelial cell injury and renewal in rhesus monkeys exposed to ozone. *Am J Pathol* 98:811-40.
- 675-672. Castleman WL, Tyler WS, Dungworth DL. 1977. Lesions in respiratory bronchioles and conducting airways of monkeys exposed to ambient levels of ozone. *Exp Mol Pathol* 26:384-400.
- 676-673. Catalano PJ, Chang LY, Harkema JR, Kaden DA, Last JA, Mellick PW, Parks WC, Pinkerton KE, Radhakrishnamurthy B, Ryan LM, et al. 1995a. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part XI: Integrative Summary. *Res Rep Health Eff Inst*: 1-54; discussion 55-85.
- 677-674. Catalano PJ, Rogus J, Ryan LM. 1995b. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part X: Robust composite scores based on median polish analysis. *Res Rep Health Eff Inst*: 1-57; discussion 59-64.
- 678-675. Centanni S, Di Marco F, Castagna F, Santus P, Guarnieri R, Allegra L. 2001. Atopy prevalence and spirometric performance in

asymptomatic schoolchildren exposed to air pollution. *Monaldi Arch Chest Dis* 56:304-8.

679-676. Centers for Disease Control and Prevention National Center for Environmental Health. 2004. *Asthma: Asthma's Impact on Children and Adolescents*.

680-677. Chang LY, Huang Y, Stockstill BL, Graham JA, Grose EC, Menache MG, Miller FJ, Costa DL, Crapo JD. 1992. Epithelial injury and interstitial fibrosis in the proximal alveolar regions of rats chronically exposed to a simulated pattern of urban ambient ozone. *Toxicol Appl Pharmacol* 115:241-52.

681-678. Chang LY, Stockstill BL, Menache MG, Mercer RR, Crapo JD. 1995. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part VIII: Morphometric analysis of structural alterations in alveolar regions. *Res Rep Health Eff Inst*: 3-39; discussion 99-110.

682-679. Charpin D, Pascal L, Birnbaum J, Armengaud A, Sambuc R, Lanteaume A, Vervloet D. 1999. Gaseous air pollution and atopy. *Clin Exp Allergy* 29:1474-80.

683-680. Cheek JM, Buckpitt AR, Li C, Tarkington BK, Plopper CG. 1994. Ozone injury to alveolar epithelium in vitro does not reflect loss of antioxidant defenses. *Toxicol Appl Pharmacol* 125:59-69.

684-681. Cheek JM, McDonald RJ, Rapalyea L, Tarkington BK, Hyde DM. 1995. Neutrophils enhance removal of ozone-injured alveolar epithelial cells in vitro. *Am J Physiol* 269:L527-35.

685-682. Chen LC, Miller PD, Lam HF, Guty J, Amdur MO. 1991. Sulfuric acid-layered ultrafine particles potentiate ozone-induced airway injury. *J Toxicol Environ Health* 34:337-52.

686-683. Chen LL, Tager IB, Peden DB, Christian DL, Ferrando RE, Welch BS, et al. 2004. Effect of ozone exposure on airway responses to inhaled allergen in asthmatic subjects. *Chest* 125:2328-35.

687-684. Cheng PW, Boat TF, Shaikh S, Wang OL, Hu PC, Costa DL. 1995. Differential effects of ozone on lung epithelial lining fluid volume and protein content. *Exp Lung Res* 21:351-65.

688-685. Chow CK, Plopper CG, Chiu M, Dungworth DL. 1981. Dietary vitamin E and pulmonary biochemical and morphological alterations of rats exposed to 0.1 ppm ozone. *24. 24:315-24*.

689-686. Christian DL, Chen LL, Scannell CH, Ferrando RE, Welch BS,

Balmes JR. 1998. Ozone-induced inflammation is attenuated with multiday exposure. *Am J Respir Crit Care Med* 158:532-7.

~~690-687.~~ Coffey MJ, Wheeler CS, Gross KB, Eschenbacher WL, Sporn PH, Peters-Golden M. 1996. Increased 5-lipoxygenase metabolism in the lungs of human subjects exposed to ozone. *Toxicology* 114:187-97.

~~691-688.~~ Cohen MD, Sisco M, Baker K, Li Y, Lawrence D, van Loveren H, Zelikoff JT, Schlesinger RB. 2002. Effects of inhaled ozone on pulmonary immune cells critical to antibacterial responses in situ. *Inhal Toxicol* 14:599-619.

~~692-689.~~ Cohen MD, Sisco M, Li Y, Zelikoff JT, Schlesinger RB. 2001. Ozone-induced modulation of cell-mediated immune responses in the lungs. *Toxicol Appl Pharmacol* 171:71-84.

~~693-690.~~ Cohen MD, Zelikoff JT, Chen L-C, Schlesinger RB. 1997. Pulmonary retention and distribution of inhaled chromium: effects of particle solubility and coexposure to ozone. *Toxicology* 9:843-65.

~~694-691.~~ Coleridge HM, Coleridge JC, Baker DG, Ginzel KH, Morrison MA. 1978. Comparison of the effects of histamine and prostaglandin on afferent C-fiber endings and irritant receptors in the intrapulmonary airways. *Adv Exp Med Biol* 99:291-305.

~~695-692.~~ Coleridge HM, Coleridge JC, Ginzel KH, Baker DG, Banzett RB, Morrison MA. 1976. Stimulation of 'irritant' receptors and afferent C-fibers in the lungs by prostaglandins. *Nature* 264:451-3.

~~696-693.~~ Coleridge JC, Coleridge HM, Schelegle ES, Green JF. 1993. Acute inhalation of ozone stimulates bronchial C-fibers and rapidly adapting receptors in dogs. *J Appl Physiol* 74:2345-52.

~~697-694.~~ Corradi M, Alinovi R, Goldoni M, Vettori M, Folesani G, Mozzoni P, et al. (2002 Aug). Biomarkers of oxidative stress after controlled human exposure to ozone. *Toxicol Lett* 134(1-3):219-25.

~~698-695.~~ Costa DL, Hatch GE, Highfill J, Stevens MA, Tepper JS. 1988. Pulmonary function studies in the rat addressing concentration versus time relationships of ozone (~~ozone~~ O_3). US Environmental Protection Agency, Health Effects Research Laboratory. US Environmental Protection Agency, Health Effects Research Laboratory (Research Triangle Park, NC).

~~699-696.~~ Costa DL, Hatch GE, Highfill J, Stevens MA, Tepper JS. 1989. Pulmonary function studies in the rat addressing concentration versus time relationships of ozone. Pittsburgh, PA: Air & Waste Management Association. Pittsburgh, PA: Air & Waste Management Association:120-6. In: Schneider, T, Lee SD, Wolters GJR, Grant LD, eds. Atmospheric

ozone research and its policy implications. Proceedings of the Third US – Dutch International Symposium. Nijmegen, The Netherlands, May 9-13, 1988. Elsevier. Amsterdam.

700-697. Costa DL, Tepper JS. 1988. Repeated exposure to ozone (ozone O_3) and chronic lung disease: recent animal data. Presented at the 81st Annual Meeting of the Air Pollution Control Association, June 1988. Dallas, Texas. Paper no. 88-122.3.

701-698. Costello RW, Jacoby DB, Gleich GJ, Fryer AD. 2000. Eosinophils and airway nerves in asthma. *Histol Histopathol* 15:861-8.

702-699. Criqui GI, Solomon C, Welch BS, Ferrando RE, Boushey HA, Balmes JR. 2000. Effects of azithromycin on ozone-induced airway neutrophilia and cytokine release. *Eur Respir J* 15:856-62.

703-700. Currie WD, van Schaik SM, Vargas I, Enhorning G. 1998. Ozone affects breathing and pulmonary surfactant function in mice. *Toxicology* 125 :21-30.

704-701. D'Amato G, Liccardi G, D'Amato M, Cazzola M. 2002. Respiratory allergic diseases induced by outdoor air pollution in urban areas. *Monaldi Arch Chest Dis* 57:161-3.

705-702. D'Amato G, Liccardi G. 1998. Outdoor environmental injury of the airways and development of allergic respiratory diseases. *Pulm Pharmacol Ther* 11:369-74.

706-703. Damji KS, Sherwin RP. 1989. The effect of ozone and simulated high altitude on murine lung elastin: quantitation by image analysis. *Toxicol Ind Health* 5:995-1003.

707-704. David GL, Romieu I, Sienra-Monge JJ, Collins WJ, Ramirez-Aguilar M, del Rio-Navarro BE, et al. (2003 Nov). Nicotinamide adenine dinucleotide (phosphate) reduced:quinone oxidoreductase and glutathione S-transferase M1 polymorphisms and childhood asthma. *Am J Respir Crit Care Med* 168(10):1199-204.

708-705. Dell'Omo G, Fiore M, Petruzzi S, Alleva E, Bignami G. 1995a. Neurobehavioral development of CD-1 mice after combined gestational and postnatal exposure to ozone. *Arch Toxicol* 69:608-16.

709-706. Dell'Omo G, Wolfer D, Alleva E, Lipp HP. 1995b. Developmental exposure to ozone induces subtle changes in swimming navigation of adult mice. *Toxicol Lett* 81:91-9.

710-707. DeMore WB, Romanovsky JC, Feldstein M, Hamming WJ, Mueller PK. 1976 Interagency comparison of iodometric methods for ozone

determination. Calibration in Air Monitoring. Vol. ASTM STP 598. American Society for Testing and Materials, p. 131-143.

- ~~711-708.~~ Depuydt PO, Lambrecht BN, Joos GF, Pauwels RA. 2002. Effect of ozone exposure on allergic sensitization and airway inflammation induced by dendritic cells. *Clin Exp Allergy* 32:391-6.
- ~~712-709.~~ Devlin RB, Folinsbee LJ, Biscardi F, Hatch G, Becker S, Madden MC, Robbins M, Koren HS. 1997. Inflammation and cell damage induced by repeated exposure of humans to ozone. *Inhal Toxicol* 9:211-35.
- ~~713-710.~~ Devlin RB, Koren HS. 1990. The use of quantitative two-dimensional gel electrophoresis to analyze changes in alveolar macrophage proteins in humans exposed to ozone. *Am J Respir Cell Mol Biol* 2:281-8.
- ~~714-711.~~ Devlin RB, McDonnell WF, Becker S, Madden MC, McGee MP, Perez R, Hatch G, House DE, Koren HS. 1996. Time-dependent changes of inflammatory mediators in the lungs of humans exposed to 0.4 ppm ozone for 2 hour: a comparison of mediators found in bronchoalveolar lavage fluid 1 and 18-hour after exposure. *Toxicol Appl Pharmacol* 138:176-85.
- ~~715-712.~~ Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D, Koren HS. 1991. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *Am J Respir Cell Mol Biol* 4:72-81.
- ~~716-713.~~ Devlin RB, McKinnon KP, Noah T, Becker S, Koren HS. 1994. Ozone-induced release of cytokines and fibronectin by alveolar macrophages and airway epithelial cells. *Am J Physiol* 266:L612-9.
- ~~717-714.~~ Dimeo MJ, Glenn MG, Holtzman MJ, Sheller JR, Nadel JA, Boushey HA. 1981. Threshold concentration of ozone causing an increase in bronchial reactivity in humans and adaptation with repeated exposures. *Am Rev Respir Dis* 124:245-8.
- ~~718-715.~~ Dormans JA, Rombout PJ, van Loveren H. 1990. Surface morphology and morphometry of rat alveolar macrophages after ozone exposure. *J Toxicol Environ Health* 31:53-70.
- ~~719-716.~~ Dormans JA, van Bree L, Boere AJ, Marra M, Rombout PJ. 1989. Study of the effects of ozone in emphysematous rats. *J Toxicol Environ Health* 26:1-18.
- ~~720-717.~~ Dormans JA, van Bree L, Boere AJ, Marra M, Rombout PJ. 1999. Interspecies differences in time course of pulmonary toxicity following repeated exposure to ozone. *Inhal Toxicol* 11:309-29.

- 721-718. Dormans JAMA, Boere AJF, van Loveren H, Rombout PJA, Marra M, van Bree L 1996. Age-related toxicity in rat lungs following acute and repeated ozone exposure. *Inhal Toxicol* 8:903-25.
- 722-719. Drechsler-Parks DM, Bedi JF, Horvath SM. 1984. Interaction of peroxyacetyl nitrate and ozone on pulmonary functions. *Am Rev Respir Dis* 130:1033-7.
- 723-720. Drechsler-Parks DM, Bedi JF, Horvath SM. 1987a. Pulmonary function responses of older men and women to ozone exposure. *Exp Gerontol* 22:91-101.
- 724-721. Drechsler-Parks DM, Bedi JF, Horvath SM. 1987b. Pulmonary function desensitization on repeated exposures to the combination of peroxyacetyl nitrate and ozone. *JAPCA* 37:1199-201.
- 725-722. Drechsler-Parks DM, Bedi JF, Horvath SM. 1989. Pulmonary function responses of young and older adults to mixtures of ozone O_3 , NO_2 and PAN-. *Toxicol Ind Health* 5:505-17.
- 726-723. Drechsler-Parks DM, Horvath SM, Bedi JF. 1990. The "effective dose" concept in older adults exposed to ozone. *Exp Gerontol* 25:107-15.
- 727-724. Drechsler-Parks DM. 1987. Effect of nitrogen dioxide, ozone, and peroxyacetyl nitrate on metabolic and pulmonary function. *Res Rep Health Eff Inst* :1-37.
- 728-725. Driscoll KE, Vollmuth TA, Schlesinger RB. 1987. Acute and subchronic ozone inhalation in the rabbit: response of alveolar macrophages-. *J Toxicol Environ Health* 21:27-43.
- 729-726. Dungworth DL, Castleman WL, Chow CK, Mellick PW, Mustafa MG, Tarkington B, Tyler WS. 1975. Effect of ambient levels of ozone on monkeys. *Fed Proc* 34:1670-4.
- 730-727. Dye JA, Madden MC, Richards JH, Lehmann JR, Devlin RB, Costa DL. 1999. Ozone effects on airway responsiveness, lung injury, and inflammation. Comparative rat strain and in vivo/in vitro investigations. *Inhal Toxicol* 11:1015-40.
- 731-728. Dziedzic D, White HJ. 1986. Thymus and pulmonary lymph node response to acute and subchronic ozone inhalation in the mouse. *Environ Res* 41:598-609.
- 732-729. Elsayed NM, Mustafa MG, Postlethwait EM. 1982. Age-dependent pulmonary response of rats to ozone exposure. *J Tox Environ Health* 9:835-848.

- 733-730. Emmons K, Foster WM. 1991. Smoking cessation and acute airway response to ozone. *Arch Environ Health* 46:288-95.
- 734-731. Eschenbacher WL, Ying RL, Kreit JW, Gross KB. 1989. Ozone-induced lung function changes in normal and asthmatic subjects and the effect of indomethacin. In: Schneider T, Lee SD, Wolters GJR, Grant, LD, eds. *Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium, May 1988, Nijmegen, The Netherlands*. Amsterdam, The Netherlands: Elsevier Science Publishers: 493-9.
- 735-732. Eustis SL, Schwartz LW, Kosch PC, Dungworth DL . 1981. Chronic bronchiolitis in nonhuman primates after prolonged ozone exposure. *Am J Pathol* 105:121-37.
- 736-733. Evans MJ, Fanucchi MV, Baker GL, Van Winkle LS, Pantle LM, Nishio SJ, Schelegle ES, Gershwin LJ, Miller LA, Hyde DM, Sannes PL, Plopper CG. 2003. Atypical development of the tracheal basement membrane zone of infant rhesus monkeys exposed to ozone and allergen. *Am J Physiol Lung Cell Mol Physiol* 285:L931-9.
- 737-734. Evans MJ, Johnson LV, Stephens RJ, Freeman G. 1976. Renewal of the terminal bronchiolar epithelium in the rat following exposure to NO₂ or ozone O₃. *Lab Invest* 35:246-57.
- 738-735. Fahy JV, Wong HH, Liu JT, Boushey HA. 1995. Analysis of induced sputum after air and ozone exposures in healthy subjects. *Environ Res* 70 :77-83.
- 739-736. Fakhrzadeh L, Laskin JD, Laskin DL. 2002. Deficiency in inducible nitric oxide synthase protects mice from ozone-induced lung inflammation and tissue injury. *Am J Respir Cell Mol Biol* 26:413-9.
- 740-737. Farrell BP, Kerr HD, Kulle TJ, Sauder LR, Young JL. 1979. Adaptation in human subjects to the effects of inhaled ozone after repeated exposure. *Am Rev Respir Dis* 119:725-30.
- 741-738. Fernandes AL, Molfino NA, McClean PA, Silverman F, Tarlo S, Raizenne M, Slutsky AS, Zamel N. 1994. The effect of pre-exposure to 0.12 ppm of ozone on exercise-induced asthma. *Chest* 106:1077-82.
- 742-739. Folinsbee LJ, Bedi JF, Horvath SM. 1980. Respiratory responses in humans repeatedly exposed to low concentrations of ozone. *Am Rev Respir Dis* 121:431-9.
- 743-740. Folinsbee LJ, Bedi JF, Horvath SM. 1981. Combined effects of ozone and nitrogen dioxide on respiratory function in man. *Am Ind Hyg Assoc J* 42:534-41.

- 744-741. Folinsbee LJ, Bedi JF, Horvath SM. 1984. Pulmonary function changes after 1 h continuous heavy exercise in 0.21 ppm ozone. *J Appl Physiol* 57:984-8.
- 745-742. Folinsbee LJ, Bedi JF, Horvath SM. 1985. Pulmonary response to threshold levels of sulfur dioxide (1.0 ppm) and ozone (0.3 ppm). *J Appl Physiol* 58:1783-7.
- 746-743. Folinsbee LJ, Drinkwater BL, Bedi JF, Horvath SM. 1978a. The influence of exercise on the pulmonary function changes due to exposure to low concentrations of ozone. New York, NY: Academic Press. New York, NY: Academic Press: 125-45.
- 747-744. Folinsbee LJ, Hazucha MJ. 2000. Time course of response to ozone exposure in healthy adult females. *Inhal Toxicol* 12:151-67.
- 748-745. Folinsbee LJ, Horstman DH, Kehrl HOUR, Harder S, Abdul-Salaam S, Ives PJ. 1994. Respiratory responses to repeated prolonged exposure to 0.12 ppm ozone. *Am J Respir Crit Care Med* 149:98-105.
- 749-746. Folinsbee LJ, Horstman DH, Kehrl HOUR, McDonnell WF, Gerrity TR, Seal. E. , Larson R, Hazucha MJ, Abdul-Salaam S, Faucette B, Ives PJ. 1991. Effects of single and repeated prolonged low-level ozone exposure in man. Presented at the annual meeting of the Society for Occupational and Environmental Health, March, Washington, DC.
- 750-747. Folinsbee LJ, Horstman DH, Vorona RD, Prince JM, Berry M. 1986. Determinants of endurance performance during ozone inhalation. 30th International Union of Physiological Sciences Congress, Vancouver, BC, Canada. 16. 16:176.
- 751-748. Folinsbee LJ, Horvath SM, Bedi JF, Delehunt JC . 1978b. Effect of 0.62 ppm NO₂ on cardiopulmonary function in young male nonsmokers. *Environ Res* 15:199-205.
- 752-749. Folinsbee LJ, Horvath SM, Raven PB, Bedi JF, Morton AR, Drinkwater BL, Bolduan NW, Gliner JA. 1977. Influence of exercise and heat stress on pulmonary function during ozone exposure. *J Appl Physiol* 43:409-13.
- 753-750. Folinsbee LJ, Horvath SM. 1986. Persistence of the acute effects of ozone exposure. *Aviat Space Environ Med* 57:1136-43.
- 754-751. Folinsbee LJ, McDonnell WF, Horstman DH. 1988. Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm ozone with moderate exercise. *JAPCA* 38:28-35.
- 755-752. Folinsbee LJ, Silverman F, Shephard RJ. 1975. Exercise

responses following ozone exposure. *J Appl Physiol* 38:996-1001.

~~756-753.~~ Folinsbee LJ. 1989. Human health effects of exposure to airborne acid. *Environ Health Perspect* 79:195-9.

~~757-754.~~ Folinsbee LJ. 1993. Human health effects of air pollution. *Environ Health Perspect* 100:45-56.

~~758-755.~~ Forster J, Kuehr J. 2000. The role of ozone. *Pediatr Allergy Immunol* 11 Suppl 13:23-5.

~~759-756.~~ Foster WM, Brown RH, Macri K, Mitchell CS. 2000. Bronchial reactivity of healthy subjects: 18-20 h postexposure to ozone. *J Appl Physiol* 89:1804-10.

~~760-757.~~ Foster WM, Stetkiewicz PT. 1996. Regional clearance of solute from the respiratory epithelia: 18-20 h postexposure to ozone. *J Appl Physiol* 81:1143-9.

~~761-758.~~ Fouke JM, DeLemos RA, Dunn MJ, McFadden ER Jr. 1990. Effects of ozone on cyclooxygenase metabolites in the baboon tracheobronchial tree. *J Appl Physiol* 69:245-50.

~~762-759.~~ Fouke JM, DeLemos RA, McFadden ER Jr. 1988. Airway response to ultra short-term exposure to ozone. *Am Rev Respir Dis* 137:326-30.

~~763-760.~~ Fouke JM, Wolin AD, McFadden ER Jr. 1991. Effects of ozone on lung mechanics and cyclooxygenase metabolites in dogs. *Prostaglandins* 42:343-53.

~~764-761.~~ Fox SD, Adams WC, Brookes KA, Lasley BL. 1993. Enhanced response to ozone exposure during the follicular phase of the menstrual cycle. *Environ Health Perspect* 101:242-4.

~~765-762.~~ Foxcroft WJ, Adams WC. 1986. Effects of ozone exposure on four consecutive days on work performance and $V_{O_{2max}}$. *J Appl Physiol* 61:960-6.

~~766-763.~~ Frampton MW, Balmes JR, Cox C, Krein PM, Speers DM, Tsai Y, Utell MJ. 1997a. Effects of ozone on normal and potentially sensitive human subjects. Part III: Mediators of inflammation in bronchoalveolar lavage fluid from nonsmokers, smokers, and asthmatic subjects exposed to ozone: a collaborative study. *Res Rep Health Eff Inst* :73-9; discussion 81-99.

~~767-764.~~ Frampton MW, Morrow PE, Cox C, Levy PC, Condemi JJ, Speers D, Gibb FR, Utell MJ. 1995. Sulfuric acid aerosol followed by ozone exposure in healthy and asthmatic subjects. *Environ Res* 69:1-14.

- 768-765. Frampton MW, Morrow PE, Torres A, Voter KZ, Whitin JC, Cox C, Speers DM, Tsai Y, Utell MJ. 1997b. Effects of ozone on normal and potentially sensitive human subjects. Part II: Airway inflammation and responsiveness to ozone in nonsmokers and smokers. *Res Rep Health Eff Inst* :39-72; discussion 81-99.
- 769-766. Frampton MW, Pryor WA, Cueto R, Cox C, Morrow PE, Utell MJ. 1999. Ozone exposure increases aldehydes in epithelial lining fluid in human lung. *Am J Respir Crit Care Med* 159:1134-7.
- 770-767. Frank R, Liu MC, Spannhake EW, Mlynarek S, Macri K, Weinmann GG. 2001. Repetitive ozone exposure of young adults: evidence of persistent small airway dysfunction. *Am J Respir Crit Care Med* 164:1253-60.
- 771-768. Freed AN, Cueto R, Pryor WA. 1999. Antioxidant transport modulates peripheral airway reactivity and inflammation during ozone exposure. *J Appl Physiol* 87:1595-603.
- 772-769. Freeman G, Juhos LT, Furiosi NJ, Mussenden R, Stephens RJ, Evans MJ. 1974. Pathology of pulmonary disease from exposure to interdependent ambient gases (nitrogen dioxide and ozone). *Arch Environ Health* 29:203-10.
- 773-770. Freeman G, Stephens RJ, Coffin DL, Stara JF. 1973. Changes in dogs' lung after long-term exposure to ozone: light and electron microscopy. *Arch Environ Health* 26:209-16.
- 774-771. Frischer T, Studnicka M, Gartner C, Tauber E, Horak F, Veiter A, Spengler J, Kuhr J, Urbanek R. 1999. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med* 160:390-6.
- 775-772. Fu J, Chen P, Xiang X. 2002. [Eotaxin in induced sputum in patients with asthma and chronic bronchitis: relationship with airway inflammation]. *Zhonghua Jie He He Hu Xi Za Zhi* 25:29-32.
- 776-773. Fujinaka LE, Hyde DM, Plopper CG, Tyler WS, Dungworth DL, Lollini LO. 1985. Respiratory bronchiolitis following long-term ozone exposure in bonnet monkeys: a morphometric study. *Exp Lung Res* 8:167-90.
- 777-774. Galbo H. 1983. Hormonal and metabolic adaptation to exercise. New York, NY: Thieme and Straton.
- 778-775. Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ Health Perspect* 107:675-9.

- ~~779-776.~~ Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. 2002. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med* 166:76-84.
- ~~780-777.~~ Gauvreau GM, Watson RM, O'Byrne PM. 1999. Kinetics of allergen-induced airway eosinophilic cytokine production and airway inflammation. *Am J Respir Crit Care Med* 160:640-7.
- ~~781-778.~~ Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, Leaderer BP. 2003. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* 290:1859-67.
- ~~782-779.~~ Gerrity TR, McDonnell WF, House DE. 1994. The relationship between delivered ozone dose and functional responses in humans. *Toxicol Appl Pharmacol* 124:275-83.
- ~~783-780.~~ Gerrity TR, Weaver RA, Berntsen J, House DE, O'Neil JJ. 1988. Extrathoracic and intrathoracic removal of ozone— O_3 in tidal-breathing humans. *J Appl Physiol* 65:393-400.
- ~~784-781.~~ Gerrity TR. 1995. Regional deposition of gases and particles in the lung: implications for mixtures. *Toxicology* 105:327-34.
- ~~785-782.~~ Gershwin LJ, Osebold JW, Zee YC. 1981. Immunoglobulin E-containing cells in mouse lung following allergen inhalation and ozone exposure. *Int Arch Allergy Appl Immunol* 65:266-77.
- ~~786-~~Gertner A, Bromberger-Barnea B, Traystman R, Berzon D, Menkes H. 1983a. Responses of the lung periphery to ozone and histamine. *J Appl Physiol* 54:640-6.
- 783.
- ~~784.~~ Gertner A, Bromberger-Barnea B, Traystman R, Menkes H. 1983b. Effects of ozone on peripheral lung reactivity. *J Appl Physiol* 55:777-84.
- ~~788-785.~~ Gibbons SI, Adams WC. 1984. Combined effects of ozone exposure and ambient heat on exercising females. *J Appl Physiol* 57:450-6.
- ~~789-786.~~ Gilmour MI, Hmieleski RR, Stafford EA, Jakab GJ. 1991. Suppression and recovery of the alveolar macrophage phagocytic system during continuous exposure to 0.5 ppm ozone. *Exp Lung Res* 17:547-58.
- ~~790-787.~~ Gilmour MI, Park P, Doerfler D, Selgrade MK. 1993. Factors that influence the suppression of pulmonary antibacterial defenses in mice exposed to ozone. *Exp Lung Res* 19:299-314.

- ~~791-788.~~ Gilmour MI, Selgrade MK. 1993. A comparison of the pulmonary defenses against streptococcal infection in rats and mice following ozone O_3 exposure: differences in disease susceptibility and neutrophil recruitment. *Toxicol Appl Pharmacol* 123:211-8.
- ~~792-789.~~ Gilmour MI. 1995. Interaction of air pollutants and pulmonary allergic responses in experimental animals. *Toxicology* 105:335-42.
- ~~793-790.~~ Gliner JA, Horvath SM, Folinsbee LJ. 1983. Preexposure to low ozone concentrations does not diminish the pulmonary function response on exposure to higher ozone concentrations. *Am Rev Respir Dis* 127:51-5.
- ~~794-791.~~ Golden JA, Nadel JA, Boushey HA. 1978. Bronchial hyperirritability in healthy subjects after exposure to ozone. *Am Rev Respir Dis* 118:287-94.
- ~~795-792.~~ Gong H Jr, Bedi JF, Horvath SM. 1988. Inhaled albuterol does not protect against ozone toxicity in nonasthmatic athletes. *Arch Environ Health* 43:46-53.
- ~~796-793.~~ Gong H Jr, Bradley PW, Simmons MS, Tashkin DP. 1986. Impaired exercise performance and pulmonary function in elite cyclists during low-level ozone exposure in a hot environment. *Am Rev Respir Dis* 134:726-33.
- ~~797-794.~~ Gong H Jr, McManus MS, Linn WS. 1997a. Attenuated response to repeated daily ozone exposures in asthmatic subjects. *Arch Environ Health* 52:34-41.
- ~~798-795.~~ Gong H Jr, Shamoo DA, Anderson KR, Linn WS. 1997b. Responses of older men with and without chronic obstructive pulmonary disease to prolonged ozone exposure. *Arch Environ Health* 52:18-25.
- ~~799-796.~~ Gong H Jr, Wong R, Sarma RJ, Linn WS, Sullivan ED, Shamoo DA, Anderson KR, Prasad SB. 1998. Cardiovascular effects of ozone exposure in human volunteers. *Am J Respir Crit Care Med* 158:538-46.
- ~~800-797.~~ Goodman JW, Peter-Fizaine FE, Shipcock SG, Hall EA, Fahmie DJ. 1989. Immunologic and hematologic consequences in mice of exposure to ozone. *J Environ Pathol Toxicol Oncol* 9:243-52.
- ~~801-798.~~ Gordon RE, Park E, Laskin D, Schuller-Levis GB-. 1998. Taurine protects rat bronchioles from acute ozone exposure: a freeze fracture and electron microscopic study. *Exp Lung Res* 24:659-74.
- ~~802-799.~~ Gould HJ, Sutton BJ, Beavil AJ, Beavil RL, McCloskey N, Coker HA, Fear D, Smurthwaite L. 2003. The biology of IGE and the basis of allergic disease. *Annu Rev Immunol* 21:579-628.

- 803-800. Graham D, Henderson F, House D. 1988. Neutrophil influx measured in nasal lavages of humans exposed to ozone. Arch Environ Health 43:228-33.
- 804-801. Graham DE, Koren HS. 1990. Biomarkers of inflammation in ozone-exposed humans. Comparison of the nasal and bronchoalveolar lavage. Am Rev Respir Dis 142:152-6.
- 805-802. Graham JA, Gardner DE, Blommer EJ, House DE, Menache MG, Miller FJ. 1987. Influence of exposure patterns of nitrogen dioxide and modifications by ozone on susceptibility to bacterial infectious disease in mice. J Toxicol Environ Health 21:113-25.
- 806-803. Green RH, Brightling CE, McKenna S, Hargadon B, Parker D, Bradding P, Wardlaw AJ, Pavord ID. 2002. Asthma exacerbations and sputum eosinophil counts: a randomised controlled trial. Lancet 360:1715-21.
- 807-804. Grievink L, van't Veer JSM, Brunekreef B. 1998. Acute effects of ozone on pulmonary function of cyclists receiving antioxidant supplements. Occup Environ Med 55:13-17.
- 808-805. Grose EC, Stevens MA, Hatch GE, Jaskot RH, Selgrade MJK, Stead AG, et al. 1989 The impact of a 12-month exposure to a diurnal pattern of ozone on pulmonary function, antioxidant biochemistry and immunology. In: Schneider T, Lee SD, Wolters GJR, Grant, LD, eds. Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium May 1988, Nijmegen. Amsterdam, The Netherlands: Elsevier Science Publishers, p. 535-543.
- 809-806. Gross KB, Sargent NE. 1992. Increases in bronchial hyperresponsiveness induced by ozone and the effects of selected non-steroidal anti-inflammatory agents. 145. 145:A97.
- 810-807. Gross KB, White HJ. 1986. Pulmonary functional and morphological changes induced by a 4-week exposure to 0.7 ppm ozone followed by a 9-week recovery period. J Toxicol Environ Health 17:143-57.
- 811-808. Gross KB, White HJ. 1987. Functional and pathologic consequences of a 52-week exposure to 0.5 PPM ozone followed by a clean air recovery period. Lung 165:283-95.
- 812-809. Gunnison AF, Weideman PA, Sobo M, Koenig KL, Chen LC (1992 Apr). Age-dependence of responses to acute ozone exposure in rats. Fundam Appl Toxicol 18(3):360-9.
- 813-810. Guyton AC, Hall JE 1996. Textbook of Medical Physiology. Philadelphia, PA : WB Saunders Company.

- 814-811. Hacker AD, Mustafa MG, Ospital JJ, Elsayed NM, Lee SD. 1986. Relationship of age to rat lung collagen synthesis in response to ozone exposure. *Age* 9:1-5.
- 815-812. Hackney JD, Linn WS, Avol EL. 1989. Acid fog: effects on respiratory function and symptoms in healthy and asthmatic volunteers. *Environ Health Perspect* 79:159-62.
- 816-813. Hackney JD, Linn WS, Buckley RD, Hislop HJ. 1976. Studies in adaptation to ambient oxidant air pollution: effects of ozone exposure in Los Angeles residents vs. new arrivals. *Environ Health Perspect* 18:141-6.
- 817-814. Hackney JD, Linn WS, Law DC, Karuza SK, Greenberg H, Buckley RD, Pedersen EE. 1975a. Experimental studies on human health effects of air pollutants. III. Two-hour exposure to ozone alone and in combination with other pollutant gases. *Arch Environ Health* 30:385-90.
- 818-815. Hackney JD, Linn WS, Mohler JG, Collier CR. 1977. Adaptation to short-term respiratory effects of ozone in men exposed repeatedly. *J Appl Physiol* 43:82-5.
- 819-816. Hackney JD, Linn WS, Mohler JG, Pedersen EE, Breisacher P, Russo A. 1975b. Experimental studies on human health effects of air pollutants. II. Four-hour exposure to ozone alone and in combination with other pollutant gases. *Arch Environ Health* 30:379-84.
- 820-817. Hanania NA, Tarlo SM, Silverman F, Urch B, Senathirajah N, Zamel N, Corey P. 1998. Effect of exposure to low levels of ozone on the response to inhaled allergen in allergic asthmatic patients. *Chest* 114:752-6.
- 821-818. Harkema JR, Mauderly JL. 1994. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part V: Effects on pulmonary function. *Res Rep Health Eff Inst* :3-17; discussion 19-26.
- 822-819. Harkema JR, Plopper CG, Hyde DM, St George JA, Dungworth DL. 1987a. Effects of an ambient level of ozone on primate nasal epithelial mucosubstances. Quantitative histochemistry. *Am J Pathol* 127:90-6.
- 823-820. Harkema JR, Plopper CG, Hyde DM, St George JA, Wilson DW, Dungworth DL. 1987b. Response of the macaque nasal epithelium to ambient levels of ozone. A morphologic and morphometric study of the transitional and respiratory epithelium. *Am J Pathol* 128:29-44.
- 824-821. Harkema JR, Plopper CG, Hyde DM, St George JA, Wilson DW, Dungworth DL. 1993. Response of macaque bronchiolar epithelium to ambient concentrations of ozone. *Am J Pathol* 143:857-66.

- ~~825-822.~~ Haro R, Paz C. 1993. Effects of ozone exposure during pregnancy on ontogeny of sleep in rats. *Neurosci Lett* 164:67-70.
- ~~826-823.~~ Hassett C, Mustafa MG, Coulson WF, Elashoff RM . 1985. Murine lung carcinogenesis following exposure to ambient ozone concentrations. *J Natl Cancer Inst* 75:771-7.
- ~~827-824.~~ Hatch GE, Slade R, Harris LP, McDonnell WF, Devlin RB, Koren HS, Costa DL, McKee J. 1994. Ozone dose and effect in humans and rats. A comparison using oxygen-18 labeling and bronchoalveolar lavage. *Am J Respir Crit Care Med* 150:676-83.
- ~~828-825.~~ Hazbun ME, Hamilton R, Holian A, Eschenbacher WL. 1993. Ozone-induced increases in substance P and 8-epi-prostaglandin F2 alpha in the airways of human subjects. *Am J Respir Cell Mol Biol* 9:568-72.
- ~~829-826.~~ Hazucha M, Bates DV. 1975. Combined effect of ozone and sulphur dioxide on human pulmonary function. *Nature* 257:50-1.
- ~~830-827.~~ Hazucha M, Silverman F, Parent C, Field S, Bates DV. 1973. Pulmonary function in man after short-term exposure to ozone. *Arch Environ Health* 27:183-8.
- ~~831-828.~~ Hazucha MJ, Bates DV, Bromberg PA. 1989. Mechanism of action of ozone on the human lung. *J Appl Physiol* 67:1535-41.
- ~~832-829.~~ Hazucha MJ, Folinsbee LJ, Seal E Jr. 1992. Effects of steady-state and variable ozone concentration profiles on pulmonary function. *Am Rev Respir Dis* 146:1487-93.
- ~~833-830.~~ Hazucha MJ, Folinsbee LJ, Seal E, Bromberg PA. 1994. Lung function response of healthy women after sequential exposures to NO₂ and ozone O₃. *Am J Respir Crit Care Med* 150:642-7.
- ~~834-831.~~ Hazucha MJ, Madden M, Pape G, Becker S, Devlin R, Koren HS, Kehrl H, Bromberg PA. 1996. Effects of cyclo-oxygenase inhibition on ozone-induced respiratory inflammation and lung function changes. *Eur J Appl Physiol Occup Physiol* 73:17-27.
- ~~835-832.~~ Hazucha MJ. 1987. Relationship between ozone exposure and pulmonary function changes. *J Appl Physiol* 62:1671-80.
- ~~836-833.~~ Henderson FW, Dubovi EJ, Harder S, Seal E Jr, Graham D. 1988. Experimental rhinovirus infection in human volunteers exposed to ozone. *Am Rev Respir Dis* 137:1124-8.
- ~~837-834.~~ Herbert RA, Hailey JR, Grumbein S, Chou BJ, Sills RC, Haseman JK, Goehl T, Miller RA, Roycroft JH, Boorman GA. 1996. Two-year and

lifetime toxicity and carcinogenicity studies of ozone in B6C3F1 mice. *Toxicol Pathol* 24:539-48.

~~838-835.~~ Hiltermann JT, Lapperre TS, van Bree L, Steerenberg PA, Brahim JJ, Sont JK, Sterk PJ, Hiemstra PS, Stolk J. 1999. Ozone-induced inflammation assessed in sputum and bronchial lavage fluid from asthmatics: a new noninvasive tool in epidemiologic studies on air pollution and asthma. *Free Radic Biol Med* 27:1448-54.

~~839-836.~~ Hiltermann TJ, Peters EA, Alberts B, Kwikkers K, Borggreven PA, Hiemstra PS, Dijkman JH, van Bree LA, Stolk J. 1998. Ozone-induced airway hyperresponsiveness in patients with asthma: role of neutrophil-derived serine proteinases. *Free Radic Biol Med* 24:952-8.

~~840-837.~~ Hiltermann TJ, Stolk J, Hiemstra PS, Fokkens PH, Rombout PJ, Sont JK, Sterk PJ, Dijkman JH. 1995. Effect of ozone exposure on maximal airway narrowing in non-asthmatic and asthmatic subjects. *Clin Sci (Lond)* 89:619-24.

~~841-838.~~ Holtzman MJ, Cunningham JH, Sheller JR, Irsigler GB, Nadel JA, Boushey HA. 1979. Effect of ozone on bronchial reactivity in atopic and nonatopic subjects. *Am Rev Respir Dis* 120:1059-67.

~~842-839.~~ Holz O, Jorres RA, Timm P, Mucke M, Richter K, Koschyk S, Magnussen H. 1999. Ozone-induced airway inflammatory changes differ between individuals and are reproducible. *Am J Respir Crit Care Med* 159:776-84.

~~843-840.~~ Holz O, Mucke M, Paasch K, Bohme S, Timm P, Richter K, Magnussen H, Jorres RA. 2002. Repeated ozone exposures enhance bronchial allergen responses in subjects with rhinitis or asthma. *Clin Exp Allergy* 32:681-9.

~~844-841.~~ Hornof WJ, Schelegle E, Kammerman M, Gunther RA, Fisher PE, Cross CE (1989 Sep). Ozone-induced accelerated lung clearance of 99mTc-DTPA aerosol in conscious sheep. *Respir Physiol* 77(3):277-90.

~~845-842.~~ Horstman DH, Ball BA, Brown J, Gerrity T, Folinsbee LJ. 1995. Comparison of pulmonary responses of asthmatic and nonasthmatic subjects performing light exercise while exposed to a low level of ozone. *Toxicol Ind Health* 11:369-85.

~~846-843.~~ Horstman DH, Folinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF. 1990. Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. *Am Rev Respir Dis* 142:1158-63.

~~847-844.~~ Horvath SM, Bedi JF, Drechsler-Parks DM. 1986. Effects of

peroxyacetyl nitrate alone and in combination with ozone in healthy young women. *J Air Pollut Control Assoc* 36:265-70.

848-845. Horvath SM, Bedi JL, Drechsler-Parks DM, Williams RE. 1991. Alterations in pulmonary function parameters during exposure to 80 ppb ozone for 6.6 hours in healthy middle aged individuals. Pittsburgh, PA: Air and Waste Management Association. Pittsburgh, PA: Air and Waste Management Association:59-70.

849-846. Horvath SM, Folinsbee LJ, Bedi JF. 1987. Combined effect of ozone and sulfuric acid on pulmonary function in man. *Am Ind Hyg Assoc J* 48:94-8.

850-847. Horvath SM, Gliner JA, Folinsbee LJ. 1981. Adaptation to ozone: duration of effect. *Am Rev Respir Dis* 123:496-9.

851-848. Horvath SM, Gliner JA, Matsen-Twisdale JA. 1979. Pulmonary function and maximum exercise responses following acute ozone exposure. *Aviat Space Environ Med* 50:901-5.

852-849. Hotchkiss JA, Harkema JR, Henderson RF. 1991. Effect of cumulative ozone exposure on ozone-induced nasal epithelial hyperplasia and secretory metaplasia in rats. *Exp Lung Res* 17:589-600.

853-850. Hotchkiss JA, Harkema JR, Kirkpatrick DT, Henderson RF. 1989a. Response of rat alveolar macrophages to ozone: quantitative assessment of population size, morphology, and proliferation following acute exposure. *Exp Lung Res* 15:1-16.

854-851. Hotchkiss JA, Harkema JR, Sun JD, Henderson RF . 1989b. Comparison of acute ozone-induced nasal and pulmonary inflammatory responses in rats. *Toxicol Appl Pharmacol* 98:289-302.

855-852. Housley DG, Eccles R, Richards RJ. 1996. Gender difference in the concentration of the antioxidant uric acid in human nasal lavage. *Acta Otolaryngol* 11:751-754.

856-853. Hu SC, Ben-Jebria A, Ultman JS. 1992a. Simulation of ozone uptake distribution in the human airways by orthogonal collocation on finite elements. *Comput Biomed Res* 25:264-78.

857-854. Hu SC, Ben-Jebria A, Ultman JS. 1992b. Longitudinal distribution of ozone absorption in the lung: quiet respiration in healthy subjects. *J Appl Physiol* 73:1655-61.

858-855. Huffman LJ, Judy DJ, Brumbaugh K, Frazer DG, Reynolds JS, McKinney WG, Goldsmith WT. 2001. Hyperthyroidism increases the risk of ozone-induced lung toxicity in rats. *Toxicol Appl Pharmacol* 173:18-26.

- ~~859-856.~~ Hyde DM, Miller LA, McDonald RJ, Stovall MY, Wong V, Pinkerton KE, Wegner CD, Rothlein R, Plopper CG. 1999. Neutrophils enhance clearance of necrotic epithelial cells in ozone-induced lung injury in rhesus monkeys. *Am J Physiol* 277:L1190-8.
- ~~860-857.~~ Hynes B, Silverman F, Cole P, Corey P. 1988. Effects of ozone exposure: a comparison between oral and nasal breathing. *Arch Environ Health* 43:357-60.
- ~~861-858.~~ Ichinose T, Sagai M. 1992. Combined exposure to NO₂, ~~ozone-O₃~~ and H₂SO₄-aerosol and lung tumor formation in rats. *Toxicology* 74:173-84.
- ~~862-859.~~ Iijima MK, Kobayashi T, Kamada H, Shimojo N. 2001. Exposure to ozone aggravates nasal allergy-like symptoms in guinea pigs. *Toxicol Lett* 123:77-85.
- ~~863-860.~~ Islam MS, ~~Grosskurth D,~~ Ulmer WT. 1979. Long-term exposure against a gas mixture of SO₂, NO₂ and O₃ in three-times MIC on lung function and reactivity of the bronchial system on healthy persons. Effect of ozone (0.1-0.15 ppm) on ventilatory function in human volunteers. *2-Wissenschaft und Umwelt.* 4:186-190. ~~2:67-73.~~
- ~~864-861.~~ Iwasaki T, Takahashi M, Saito H, Arito H. 1998. Adaptation of extrapulmonary responses to ozone exposure in conscious rats. *Ind Health* 36:57-60.
- ~~865-862.~~ Jabbour AJ, Altman LC, Wight TN, Luchtel DL. 1998. Ozone alters the distribution of beta1 integrins in cultured primate bronchial epithelial cells. *Am J Respir Cell Mol Biol* 19:357-65.
- ~~866-863.~~ Jakab GJ, Bassett DJ. 1990. Influenza virus infection, ozone exposure, and fibrogenesis. *Am Rev Respir Dis* 141:1307-15.
- ~~867-864.~~ Janssen LJ, O'Byrne PM, Daniel EE. 1991. Mechanism underlying ozone-induced in vitro hyperresponsiveness in canine bronchi. *Am J Physiol* 261:L55-62.
- ~~868-865.~~ Jenkins HS, Devalia JL, Mister RL, Bevan AM, Rusznak C, Davies RJ. 1999. The effect of exposure to ozone and nitrogen dioxide on the airway response of atopic asthmatics to inhaled allergen: dose- and time-dependent effects. *Am J Respir Crit Care Med* 160:33-9.
- ~~869-866.~~ Joad JP, Bric JM, Weir AJ, Putney L, Hyde DM, Postlethwait EM, Plopper CG. 2000. Effect of respiratory pattern on ozone injury to the airways of isolated rat lungs. *Toxicol Appl Pharmacol* 169:26-32.
- ~~870-867.~~ Johansen H, Dutta M, Mao Y, Chagani K, Sladeczek I. 1992. An investigation of the increase in preschool-age asthma in Manitoba, Canada.

Health Rep 4:379-402.

871-868. Johnson HG, Stout BK, Ruppel PL. 1988. Inhibition of the 5-lipoxygenase pathway with piroprost (U-60,257) protects normal primates from ozone-induced methacholine hyperresponsive small airways. *Prostaglandins* 35:459-66.

872-869. Johnston CJ, Oberdorster G, Gelein R, Finkelstein JN. 2000. Newborn mice differ from adult mice in chemokine and cytokine expression to ozone, but not to endotoxin. *Inhal Tox* 12:205-224.

873-870. Johnson NF, Hotchkiss JA, Harkema JR, Henderson RF. 1990. Proliferative responses of rat nasal epithelia to ozone. *Toxicol Appl Pharmacol* 103 :143-55.

874-871. Jonsson LM, Edlund T, Marklund SL, Sandstrom T . 2002. Increased ozone-induced airway neutrophilic inflammation in extracellular-superoxide dismutase null mice. *Respir Med* 96:209-14.

875-872. Jorres R, Nowak D, Magnussen H. 1996. The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis. *Am J Respir Crit Care Med* 153:56-64.

876-873. Jorres RA, Holz O, Zachgo W, Timm P, Koschyk S, Muller B, Grimminger F, Seeger W, Kelly FJ, Dunster C, Frischer T, Lubec G, Waschewski M, Niendorf A, Magnussen H. 2000. The effect of repeated ozone exposures on inflammatory markers in bronchoalveolar lavage fluid and mucosal biopsies. *Am J Respir Crit Care Med* 161:1855-61.

877-874. Kabel JR, Ben-Jebria A, Ultman JS. 1994. Longitudinal distribution of ozone absorption in the lung: comparison of nasal and oral quiet breathing. *J Appl Physiol* 77:2584-92.

878-875. Kagawa J, Tsuru K. 1979. [Effects of ozone and smoking alone and in combination on bronchial reactivity to inhaled acetylcholine aerosol in normal subjects (author's transl)]. *Nihon Kyobu Shikkan Gakkai Zasshi* 17:703-9.

879-876. Kagawa J. 1983. Respiratory effects of two-hour exposure with intermittent exercise to ozone, sulfur dioxide and nitrogen dioxide alone and in combination in normal subjects. *Am Ind Hyg Assoc J* 44:14-20.

880-877. Kagawa J. 1986. Experimental studies on human health effects of aerosol and gaseous pollutants. Chelsea, MI: Lewis Publishers, Inc. Chelsea, MI: Lewis Publishers, Inc.: 683-97.

881-878. Kavlock R, Daston G, Grabowski CT. 1979. Studies on the developmental toxicity of ozone. I. Prenatal effects. *Toxicol Appl Pharmacol*

48:19-28.

~~882-879.~~ Kavlock RJ, Meyer E, Grabowski CT. 1980. Studies on the developmental toxicity of ozone: postnatal effects. *Toxicol Lett* 5:3-9.

~~883-880.~~ Keefe MJ, Bennett WD, DeWitt P, Seal E, Strong AA, Gerrity TR. 1991. The effect of ozone exposure on the dispersion of inhaled aerosol boluses in healthy human subjects. *Am Rev Respir Dis* 144:23-30.

~~884-881.~~ Kehrl HOUR, Hazucha MJ, Solic JJ, Bromberg PA. 1985. Responses of subjects with chronic obstructive pulmonary disease after exposures to 0.3 ppm ozone. *Am Rev Respir Dis* 131:719-24.

~~885-882.~~ Kehrl HOUR, Peden DB, Ball B, Folinsbee LJ, Horstman D. 1999. Increased specific airway reactivity of persons with mild allergic asthma after 7.6 hours of exposure to 0.16 ppm ozone. *J Allergy Clin Immunol* 104:1198-204.

~~886-883.~~ Kerr HD, Kulle TJ, McIlhany ML, Swidersky P. 1975. Effects of ozone on pulmonary function in normal subjects. An environmental-chamber study. *Am Rev Respir Dis* 111:763-73.

~~887-884.~~ Kimmel TA, Chen LC, Bosland MC, Nadziejko C. 1997. Influence of acid aerosol droplet size on structural changes in the rat lung caused by acute exposure to sulfuric acid and ozone. *Toxicol Appl Pharmacol* 144:348-55.

~~888-885.~~ Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, El-Fawal H, Devlin RB, Rom WN. 1996. Biomarkers of lung inflammation in recreational joggers exposed to ozone. *Am J Respir Crit Care Med* 154:1430-5.

~~889-886.~~ Kirichenko A, Li L, Morandi MT, Holian A. 1996. 4-hydroxy-2-nonenal-protein adducts and apoptosis in murine lung cells after acute ozone exposure. *Toxicol Appl Pharmacol* 141:416-24.

~~890-887.~~ Kleeberger SR, Ohtsuka Y, Zhang LY, Longphre M. 2001. Airway responses to chronic ozone exposure are partially mediated through mast cells. *J Appl Physiol* 90:713-23.

~~891-888.~~ Kleinman MT. 1991. Effects of ozone on pulmonary function: the relationship of response to dose. *J Expos Anal Environ Epidemiol* 1:309-325.

~~892-889.~~ Kleinman MT, Bailey RM, Chang YT, Clark KW, Jones MP, Linn WS, Hackney JD. 1981. Exposures of human volunteers to a controlled atmospheric mixture of ozone, sulfur dioxide and sulfuric acid. *Am Ind Hyg Assoc J* 42:61-9.

- ~~893-890.~~ ___Kodavanti UP, Costa DL, Richards J, Crissman KM, Slade R, Hatch GE. 1996. Antioxidants in bronchoalveolar lavage fluid cells isolated from ozone--exposed normal and ascorbate-deficient guinea pigs. *Exp Lung Res* 22:435-48.
- ~~894-891.~~ ___Kodavanti UP, Hatch GE, Starcher B, Giri SN, Winsett D, Costa DL. 1995. Ozone-induced pulmonary functional, pathological, and biochemical changes in normal and vitamin C-deficient guinea pigs. *Fundam Appl Toxicol* 24:154-64.
- ~~895-892.~~ ___Koenig JQ, Covert DS, Hanley QS, van Belle G, Pierson WE. 1990. Prior exposure to ozone potentiates subsequent response to sulfur dioxide in adolescent asthmatic subjects. *Am Rev Respir Dis* 141:377-80.
- ~~896-893.~~ ___Koenig JQ, Covert DS, Marshall SG, Van Belle G, Pierson WE. 1987. The effects of ozone and nitrogen dioxide on pulmonary function in healthy and in asthmatic adolescents. *Am Rev Respir Dis* 136:1152-7.
- ~~897-894.~~ ___Koenig JQ, Covert DS, Morgan MS, Horike M, Horike N, Marshall SG, Pierson WE. 1985. Acute effects of 0.12 ppm ozone or 0.12 ppm nitrogen dioxide on pulmonary function in healthy and asthmatic adolescents. *Am Rev Respir Dis* 132:648-51.
- ~~898-895.~~ ___Koenig JQ, Covert DS, Pierson WE, Hanley QS, Rebolledo V, Dumler K, McKinney SE. 1994. Oxidant and acid aerosol exposure in healthy subjects and subjects with asthma. Part I: Effects of oxidants, combined with sulfuric or nitric acid, on the pulmonary function of adolescents with asthma. *Res Rep Health Eff Inst* :1-36.
- ~~899-896.~~ ___Koenig JQ, Covert DS, Smith MS, van Belle G, Pierson WE. 1988a. The pulmonary effects of ozone and nitrogen dioxide alone and combined in healthy and asthmatic adolescent subjects. *Toxicol Ind Health* 4:521-32.
- ~~900-897.~~ ___Koenig JQ, Pierson WE, Covert DS, Marshall SG, Morgan MS, van Belle G. 1988b. The effects of ozone and nitrogen dioxide on lung function in healthy and asthmatic adolescents. *Res Rep Health Eff Inst* :5-24.
- ~~901-898.~~ ___Koren HS, Devlin RB, Becker S, Perez R, McDonnell WF. 1991. Time-dependent changes of markers associated with inflammation in the lungs of humans exposed to ambient levels of ozone. *Toxicol Pathol* 19:406-11.
- ~~902-899.~~ ___Koren HS, Devlin RB, Graham DE, Mann R, McDonnell WF. 1989a. The inflammatory response in human lung exposed to ambient levels of ozone. Amsterdam, The Netherlands-: Elsevier Science Publishers B.V., p. 745-753.
- ~~903-900.~~ ___Koren HS, Devlin RB, Graham DE, Mann R, McGee MP, Horstman

DH, Kozumbo WJ, Becker S, House DE, McDonnell WF, et al. 1989b. Ozone-induced inflammation in the lower airways of human subjects. *Am Rev Respir Dis* 139:407-15.

904-901. Koren HS, Hatch GE, Graham DE. 1990. Nasal lavage as a tool in assessing acute inflammation in response to inhaled pollutants. *Toxicology* 60:15-25.

905-902. Koren HS. 1995. Associations between criteria air pollutants and asthma. *Environ Health Perspect* 103 Suppl 6:235-42.

906-903. Kotlikoff MI, Jackson AC, Watson JW. 1984. Oscillatory mechanics of the respiratory system in ozone-exposed rats. *J Appl Physiol* 56:182-6.

907-904. Kreit JW, Gross KB, Moore TB, Lorenzen TJ, D'Arcy J, Eschenbacher WL. 1989. Ozone-induced changes in pulmonary function and bronchial responsiveness in asthmatics. *J Appl Physiol* 66:217-22.

908-905. Krishna MT, Springall D, Meng QH, Withers N, Macleod D, Biscione G, Frew A, Polak J, Holgate S. 1997. Effects of ozone on epithelium and sensory nerves in the bronchial mucosa of healthy humans. *Am J Respir Crit Care Med* 156:943-50.

909-906. Kulle TJ, Kerr HD, Farrell BP, Sauder LR, Bermel MS. 1982. Pulmonary function and bronchial reactivity in human subjects with exposure to ozone and respirable sulfuric acid aerosol. *Am Rev Respir Dis* 126:996-1000.

910-907. Kulle TJ, Milman JH, Sauder LR, Kerr HD, Farrell BP, Miller WR. 1984. Pulmonary function adaptation to ozone in subjects with chronic bronchitis. *Environ Res* 34:55-63.

911-908. Kulle TJ, Sauder LR, Hebel JR, Chatham MD. 1985. Ozone response relationships in healthy nonsmokers. *Am Rev Respir Dis* 132:36-41.

912-909. Kulle TJ, Sauder LR, Kerr HD, Farrell BP, Bermel MS, Smith DM. 1982. Duration of pulmonary function adaptation to ozone in humans. *Am Ind Hyg Assoc J* 43:832-7.

913-910. Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. 1997. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen--results of a pilot study. *Environ Res* 72:8-23.

914-911. Larsen RI, McDonnell WF, Horstman DH, Folinsbee LJ. 1991. An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 11. A lognormal model relating human lung function decrease to ozone exposure. *J Air Waste Manage Assoc* 41:455-9.

- 915-912. Larson SD, Schelegle ES, Walby WF, Gershwin LJ, Fanucchi MV, Evans MJ, Joad JP, Tarkington BK, Hyde DM, Plopper CG. 2004. Postnatal remodeling of the neural components of the epithelial-mesenchymal trophic unit in the proximal airways of infant rhesus monkeys exposed to ozone and allergen. *Toxicol Appl Pharmacol* 194:211-20.
- 916-913. Laskin DL, Heck DE, Laskin JD. 1998. Role of inflammatory cytokines and nitric oxide in hepatic and pulmonary toxicity. *Toxicol Lett* 102-103:289-93.
- 917-914. Laskin DL, Pendino KJ, Punjabi CJ, Rodriguez del Valle M, Laskin JD. 1994. Pulmonary and hepatic effects of inhaled ozone in rats. *Environ Health Perspect* 102 Suppl 10:61-4.
- 918-915. Last JA, Gelzleichter T, Harkema J, Parks WC, Mellick P. 1993. Effects of 20 months of ozone exposure on lung collagen in Fischer 344 rats. *Toxicology* 84:83-102.
- 919-916. Last JA, Greenberg DB, Castleman WL. 1979. Ozone-induced alterations in collagen metabolism of rat lungs. *Toxicol Appl Pharmacol* 51:247-58.
- 920-917. Last JA, Pinkerton KE. 1997. Chronic exposure of rats to ozone and sulfuric acid aerosol: biochemical and structural responses. *Toxicology* 116 :133-46.
- 921-918. Last JA, Reiser KM, Tyler WS, Rucker RB. 1984. Long-term consequences of exposure to ozone. I. Lung collagen content. *Toxicol Appl Pharmacol* 72:111-8.
- 922-919. Last JA, Reiser KM. 1984. Collagen biosynthesis. *Environ Health Perspect* 55:169-77.
- 923-920. Last JA, Warren DL, Pecquet-Goad E, Witschi H. 1987. Modification by ozone of lung tumor development in mice. *J Natl Cancer Inst* 78:149-54.
- 924-921. Lategola MT, Melton CE, Higgins EA. 1980. Pulmonary and symptom threshold effects of ozone in airline passenger and cockpit crew surrogates. *Aviat Space Environ Med* 51:878-84.
- 925-922. Lauritzen SK, Adams WC. 1985. Ozone inhalation effects consequent to continuous exercise in females: comparison to males. *J Appl Physiol* 59:1601-6.
- 926-923. Lee LY, Dumont C, Djokic TD, Menzel TE, Nadel JA. 1979. Mechanism of rapid, shallow breathing after ozone exposure in conscious dogs. *J Appl Physiol* 46:1108-14.

- 927-924. Leikauf GD, Zhao Q, Zhou S, Santrock J. 1993. Ozonolysis products of membrane fatty acids activate eicosanoid metabolism in human airway epithelial cells. *Am J Respir Cell Mol Biol* 9:594-602.
- 928-925. Leikauf GD, Zhao Q, Zhou S, Santrock J. 1995. Activation of eicosanoid metabolism in human airway epithelial cells by ozonolysis products of membrane fatty acids. *Res Rep Health Eff Inst* :1-15; discussion 19-26.
- 929-926. Li AF, Richters A. 1991. Ambient level ozone effects on subpopulations of thymocytes and spleen T lymphocytes. *Arch Environ Health* 46:57-63.
- 930-927. Li L, Hamilton RF Jr, Holian A. 2000. Protection against ozone-induced pulmonary inflammation and cell death by endotoxin pretreatment in mice: role of HO-1. *Inhal Toxicol* 12:1225-38.
- 931-928. Li Z, Daniel EE, Lane CG, Arnaout MA, O'Byrne PM. 1992. Effect of an anti-Mo1 MAb on ozone-induced airway inflammation and airway hyperresponsiveness in dogs. *Am J Physiol* 263:L723-6.
- 932-929. Linder J, Herren D, Monn C, Wanner HU. 1988. [Effect of ozone on physical performance]. *Schweiz Z Sportmed* 36:5-10.
- 933-930. Linn WS, Anderson KR, Shamoo DA, Edwards SA, Webb TL, Hackney JD, Gong H Jr. 1995. Controlled exposures of young asthmatics to mixed oxidant gases and acid aerosol. *Am J Respir Crit Care Med* 152:885-91.
- 934-931. Linn WS, Avol EL, Shamoo DA, Peng RC, Valencia LM, Little DE, Hackney JD. 1988. Repeated laboratory ozone exposures of volunteer Los Angeles residents: an apparent seasonal variation in response. *Toxicol Ind Health* 4 :505-20.
- 935-932. Linn WS, Avol EL, Shamoo DA, Spier CE, Valencia LM, Venet TG, Fischer DA, Hackney JD. 1986 . A dose-response study of healthy, heavily exercising men exposed to ozone at concentrations near the ambient air quality standard. *Toxicol Ind Health* 2:99-112.
- 936-933. Linn WS, Buckley RD, Spier CE, Blessey RL, Jones MP, Fischer DA, Hackney JD. 1978. Health effects of ozone exposure in asthmatics. *Am Rev Respir Dis* 117:835-43.
- 937-934. Linn WS, Fischer DA, Medway DA, Anzar UT, Spier CE, Valencia LM, Venet TG, Hackney JD. 1982. Short-term respiratory effects of 0.12 ppm ozone exposure in volunteers with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 125:658-63.

- 938-935. Linn WS, Gong H Jr, Shamoo DA, Anderson KR, Avol EL. 1997. Chamber exposures of children to mixed ozone, sulfur dioxide, and sulfuric acid. *Arch Environ Health* 52:179-87.
- 939-936. Linn WS, Jones MP, Bachmayer EA, Clark KW, Karuza SK, Hackney JD. 1979. Effect of low-level exposure to ozone on arterial oxygenation in humans. *Am Rev Respir Dis* 119:731-40.
- 940-937. Linn WS, Jones MP, Bachmayer EA, Spier CE, Mazur SF, Avol EL, Hackney JD. 1980. Short-term respiratory effects of polluted ambient air: a laboratory study of volunteers in a high-oxidant community. *Am Rev Respir Dis* 121:243-52.
- 941-938. Linn WS, Medway DA, Anzar UT, Valencia LM, Spier CE, Tsao FS, Fischer DA, Hackney JD. 1982-. Persistence of adaptation to ozone in volunteers exposed repeatedly for six weeks. *Am Rev Respir Dis* 125:491-5.
- 942-939. Linn WS, Shamoo DA, Anderson KR, Peng RC, Avol EL, Hackney JD. 1994. Effects of prolonged, repeated exposure to ozone, sulfuric acid, and their combination in healthy and asthmatic volunteers. *Am J Respir Crit Care Med* 150:431-40.
- 943-940. Linn WS, Shamoo DA, Venet TG, Spier CE, Valencia LM, Anzar UT, Hackney JD. 1983. Response to ozone in volunteers with chronic obstructive pulmonary disease. *Arch Environ Health* 38:278-83.
- 944-941. Long NC, Suh J, Morrow JD, Schiestl RH, Murthy GG, Brain JD, Frei B. 2001. Ozone causes lipid peroxidation but little antioxidant depletion in exercising and nonexercising hamsters. *J Appl Physiol* 91:1694-700.
- 945-942. Longphre M, Zhang L, Harkema JR, Kleeberger SR-. 1999. Ozone-induced pulmonary inflammation and epithelial proliferation are partially mediated by PAF. *J Appl Physiol* 86:341-9.
- 946-943. Mannino DM, Homa DM, Akinbami LJ, Moorman JE, Gwynn C, Redd SC. 2002. Surveillance for asthma--United States, 1980-1999. *MMWR Surveill Summ* 51:1-13.
- 947-944. Mariassy AT, Abraham WM, Phipps RJ, Sielczak MW, Wanner A. 1990. Effect of ozone on the postnatal development of lamb mucociliary apparatus. *J Appl Physiol* 68:2504-10.
- 948-945. Mariassy AT, Sielczak MW, McCray MN, Abraham WM, Wanner A. 1989. Effects of ozone on lamb tracheal mucosa. Quantitative glycoconjugate histochemistry. *Am J Pathol* 135:871-9.
- 949-946. Martin CJ, Boatman ES, Ward G. 1983. Mechanical properties of

alveolar wall after pneumonectomy and ozone exposure. *J Appl Physiol* 54:785-8.

950-947. Matricardi PM. 2001. Prevalence of atopy and asthma in eastern versus Western Europe: why the difference? *Ann Allergy Asthma Immunol* 87(6 Suppl 3):24-27.

951-948. Matsumura Y. 1970a. The effects of ozone, nitrogen dioxide, and sulfur dioxide on the experimentally induced allergic respiratory disorder in guinea pigs. I. The effect on sensitization with albumin through the airway. *Am Rev Respir Dis* 102:430-7.

952-949. Matsumura Y. 1970b. The effects of ozone, nitrogen dioxide, and sulfur dioxide on the experimentally induced allergic respiratory disorder in guinea pigs. II. The effects of ozone on the absorption and the retention of antigen in the lung. *Am Rev Respir Dis* 102:438-43.

953-950. Mautz WJ, Bufalino C. 1989. Breathing pattern and metabolic rate responses of rats exposed to ozone. *Respir Physiol* 76:69-77.

954-951. Mautz WJ, Kleinman MT, Phalen RF, Crocker TT. 1988. Effects of exercise exposure on toxic interactions between inhaled oxidant and aldehyde air pollutants. *J Toxicol Environ Health* 25:165-77.

955-952. Mautz WJ, McClure TR, Reischl P, Phalen RF, Crocker TT. 1985. Enhancement of ozone-induced lung injury by exercise. *J Toxicol Environ Health* 16:841-54.

956-953. Mautz WJ. 2003. Exercising animal models in inhalation toxicology: interactions with ozone and formaldehyde. *Environ Res* 92: 14-26.

957-954. McBride DE, Koenig JQ, Luchtel DL, Williams PV, Henderson WR Jr. 1994. Inflammatory effects of ozone in the upper airways of subjects with asthma. *Am J Respir Crit Care Med* 149:1192-7.

958-955. McDonnell WF 3rd, Chapman RS, Leigh MW, Strobe GL, Collier AM. 1985a. Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am Rev Respir Dis* 132:875-9.

959-956. McDonnell WF 3rd, Horstman DH, Abdul-Salaam S, House DE. 1985b. Reproducibility of individual responses to ozone exposure. *Am Rev Respir Dis* 131:36-40.

960-957. McDonnell WF, Horstman DH, Abdul-Salaam S, Raggio LJ, Green JA. 1987. The respiratory responses of subjects with allergic rhinitis to ozone exposure and their relationship to nonspecific airway reactivity. *Toxicol Ind Health* 3:507-17.

- 961-958.____McDonnell WF, Horstman DH, Hazucha MJ, Seal E Jr, Haak ED, Salaam SA, House DE. 1983. Pulmonary effects of ozone exposure during exercise: dose-response characteristics. *J Appl Physiol* 54:1345-52.
- 962-959.____McDonnell WF, Kehrl HOUR, Abdul-Salaam S, Ives PJ, Folinsbee LJ, Devlin RB, O'Neil JJ, Horstman DH. 1991. Respiratory response of humans exposed to low levels of ozone for 6.6 hours. *Arch Environ Health* 46: 145-50.
- 963-960.____McDonnell WF, Muller KE, Bromberg PA, Shy CM. 1993. Predictors of individual differences in acute response to ozone exposure. *Am Rev Respir Dis* 147:818-25.
- 964-961.____McDonnell WF, Smith MV. 1994. Description of acute ozone response as a function of exposure rate and total inhaled dose. *J Appl Physiol* 76:2776-84.
- 965-962.____McDonnell WF, Stewart PW, Andreoni S, Seal E Jr, Kehrl HOUR, Horstman DH, Folinsbee LJ, Smith MV. 1997. Prediction of ozone-induced FEV1 changes. Effects of concentration, duration, and ventilation. *Am J Respir Crit Care Med* 156:715-22.
- 966.McDonnell WF, Stewart PW, Andreoni S, Smith MV-. 1995. Proportion of moderately exercising individuals responding to low-level, multi-hour ozone exposure. *Am J Respir Crit Care Med* 152:589-96.
- 963.
- 964.McDonnell WF, Stewart PW, Smith MV, Pan WK, Pan J. 1999. Ozone-induced respiratory symptoms: exposure-response models and association with lung function. *Eur Respir J* 14:845-53.
- 968-965.____McGee MP, Devlin R, Saluta G, Koren H. 1990. Tissue factor and factor VII messenger RNAs in human alveolar macrophages: effects of breathing ozone. *Blood* 75:122-7.
- 969-966.____McKittrick T, Adams WC. 1995. Pulmonary function response to equivalent doses of ozone consequent to intermittent and continuous exercise. *Arch Environ Health* _ 50:153-8.
- 970-967.____Mellick PW, Dungworth DL, Schwartz LW, Tyler WS. 1977. Short term morphologic effects of high ambient levels of ozone on lungs of rhesus monkeys. *Lab Invest* 36:82-90.
- 971-968.____Messineo TD, Adams WC. 1990. Ozone inhalation effects in females varying widely in lung size: comparison with males. *J Appl Physiol* 69:96-103.

- 972-969. Miller FJ, Overton JH Jr, Jaskot RH, Menzel DB-. 1985. A model of the regional uptake of gaseous pollutants in the lung. I. The sensitivity of the uptake of ozone in the human lung to lower respiratory tract secretions and exercise. *Toxicol Appl Pharmacol* 79:11-27.
- 973-970. Miller LA, Hyde DM, Gershwin LJ, Schelegle ES, Fanucchi MV, Evans MJ, Gerriets JE, Putney LF, Stovall MY, Tyler NK, Usachenko JL, Plopper CG. 2003. The effect of house dust mite aeroallergen and air pollutant exposures during infancy. *Chest* 123:434S.
- 974-971. Miller PD, Ainsworth D, Lam HF, Amdur MO. 1987. Effect of ozone exposure on lung functions and plasma prostaglandin and thromboxane concentrations in guinea pigs. *Toxicol Appl Pharmacol* 88:132-40.
- 975-972. Miller PD, Ainsworth D, Lam HF, Amdur MO. 1988. Indomethacin and cromolyn sodium alter ozone-induced changes in lung function and plasma eicosanoid concentrations in guinea pigs. *Toxicol Appl Pharmacol* 93:175-86.
- 976-973. Mills PC, Roberts CA, Smith NC. 1996. Effects of ozone and airway inflammation on glutathione status and iron homeostasis in the lungs of horses. *Am J Vet Res* 57:1359-63.
- 977-974. Moffatt RK, Hyde DM, Plopper CG, Tyler WS, Putney LF. 1987. Ozone-induced adaptive and reactive cellular changes in respiratory bronchioles of bonnet monkeys. *Exp Lung Res* 12:57-74.
- 978-975. Molfino NA, Wright SC, Katz I, Tarlo S, Silverman F, McClean PA, Szalai JP, Raizenne M, Slutsky AS, Zamel N. 1991. Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet* 338:199-203.
- 979-976. Moore PF, Schwartz LW. 1981. Morphological effects of prolonged exposure to ozone and sulfuric acid aerosol on the rat lung. *Exp Mol Pathol* 35:108-23.
- 980-977. Morgan MS, Meyer P, Holub R, Frank R. 1986. Overall and regional lung function in dogs exposed acutely to ozone. *Environ Res* 41:546-57.
- 981-978. Murlas CG, Lang Z, Williams GJ, Chodimella V. 1992. Aerosolized neutral endopeptidase reverses ozone-induced airway hyperreactivity to substance P. *J Appl Physiol* 72:1133-41.
- 982-979. Murlas CG, Murphy TP, Chodimella V. 1990. ~~ozone~~ O₃-induced mucosa-linked airway muscle hyperresponsiveness in the guinea pig. *J Appl Physiol* 69:7-13.

- 983-980. Musi B, Dell'Omo G, Ricceri L, Santucci D, Laviola G, Bignami G, Alleva E. 1994. Effects of acute and continuous ozone (ozoneO₃) exposure on activity/exploration and social behavior of CD-1 mice. *Neurotoxicology* 15:827-35.
- 984-981. Nayak AS. 2003. The asthma and allergic rhinitis link. *Allergy Asthma Proc* 24:395-402.
- 985-982. Neuhaus-Steinmetz U, Uffhausen F, Herz U, Renz H. 2000. Priming of allergic immune responses by repeated ozone exposure in mice. *Am J Respir Cell Mol Biol* 23:228-33.
- 986-983. Nicolai T. 2002. Pollution, environmental factors and childhood respiratory allergic disease. *Toxicology* 181-182:317-21.
- 987-984. Nightingale JA, Rogers DF, Barnes PJ. 1999. Effect of inhaled ozone on exhaled nitric oxide, pulmonary function, and induced sputum in normal and asthmatic subjects. *Thorax* 54:1061-9.
- 988-985. Nightingale JA, Rogers DF, Fan Chung K, Barnes PJ. 2000. No effect of inhaled budesonide on the response to inhaled ozone in normal subjects. *Am J Respir Crit Care Med* 161:479-86.
- 989-986. Nikula KJ, Wilson DW, Giri SN, Plopper CG, Dungworth DL. 1988. The response of the rat tracheal epithelium to ozone exposure. Injury, adaptation, and repair. *Am J Pathol* 131:373-84.
- 990-987. Nishikawa M, Suzuki S, Ikeda H, Fukuda T, Suzuki J, Okubo T. 1990. Dose-response relationship of ozone-induced airway hyperresponsiveness in unanesthetized guinea pigs. *J Toxicol Environ Health* 30:123-34.
- 991-988. O'Byrne P. 1998. Asthma pathogenesis and allergen-induced late responses. *J Allergy Clin Immunol* 102:S85-9-.
- 992-989. Oosting RS, van Golde LM, Verhoef J, Van Bree L. 1991. Species differences in impairment and recovery of alveolar macrophage functions following single and repeated ozone exposures. *Toxicol Appl Pharmacol* 110:170-8.
- 993-990. Osebold JW, Gershwin LJ, Zee YC. 1980. Studies on the enhancement of allergic lung sensitization by inhalation of ozone and sulfuric acid aerosol. *J Environ Pathol Toxicol* 3:221-34.
- 994-991. Osebold JW, Zee YC, Gershwin LJ. 1988. Enhancement of allergic lung sensitization in mice by ozone inhalation. *Proc Soc Exp Biol Med* 188:259-64.

- ~~995-992.~~ Oshima Y, Ishizaki T, Miyamoto T, Kabe J, Makino S. 1964. A study of Tokyo-Yokohama asthma among Japanese. *Am Rev Respir Dis* 90:632-4.
- ~~996-993.~~ Ostro BD, Lipsett MJ, Jewell NP. 1989. Predicting respiratory disease from pulmonary function tests: a reanalysis of ozone chamber studies. *JAPCA* 39:1313-8.
- ~~997-994.~~ Otto-Knapp R, Jurgovsky K, Schierhorn K, Kunkel G (2003 Feb). Antioxidative enzymes in human nasal mucosa after exposure to ozone. Possible role of GSTM1 deficiency. *Inflamm Res* 52(2):51-5.
- ~~998-995.~~ Overton JH, Graham RC, Miller FJ. 1987. A model of the regional uptake of gaseous pollutants in the lung. II. The sensitivity of ozone uptake in laboratory animal lungs to anatomical and ventilatory parameters. *Toxicol Appl Pharmacol* 88:418-32.
- ~~999-996.~~ Ozawa M, Fujimaki H, Imai T, Honda Y, Watanabe N. 1985. Suppression of IgE antibody production after exposure to ozone in mice. *Int Arch Allergy Appl Immunol* 76:16-9.
- ~~1000-997.~~ Paige RC, Plopper CG. 1999. Acute and chronic effects of ozone in animal models. 531-57.
- ~~1001-998.~~ Paige RC, Royce FH, Plopper CG, Buckpitt AR. 2000. Long-term exposure to ozone increases acute pulmonary centriacinar injury by 1-nitronaphthalene: I. Region-specific enzyme activity. *J Pharmacol Exp Ther* 295:934-41.
- ~~1002-999.~~ P'an AY, Jeiger Z. 1972. Trypsin protein esterase in relation to ozone-induced vascular damage. *Arch Environ Health* 24:233-6.
- ~~1003-1000.~~ Paquette NC, Zhang LY, Ellis WA, Scott AL, Kleeberger SR. 1996. Vitamin A deficiency enhances ozone-induced lung injury. *Am J Physiol* 270:L475-82.
- ~~1004-1001.~~ Passannante AN, Hazucha MJ, Bromberg PA, Seal E, Folinsbee L, Koch G. 1998. Nociceptive mechanisms modulate ozone-induced human lung function decrements. *J Appl Physiol* 85:1863-70.
- ~~1005-1002.~~ Paz C. 1997. Some consequences of ozone exposure on health. *Arch Med Res* 28:163-70.
- ~~1006-1003.~~ Peden DB, Boehlecke B, Horstman D, Devlin R. 1997. Prolonged acute exposure to 0.16 ppm ozone induces eosinophilic airway inflammation in asthmatic subjects with allergies. *J Allergy Clin Immunol* 100:802-8.

- ~~1007~~.1004. Peden DB, Setzer RW Jr, Devlin RB. 1995. Ozone exposure has both a priming effect on allergen-induced responses and an intrinsic inflammatory action in the nasal airways of perennially allergic asthmatics. *Am J Respir Crit Care Med* 151:1336-45.
- ~~1008~~.1005. Peters EA, Hiltermann JT, Stolk J. 2001. Effect of apocynin on ozone-induced airway hyperresponsiveness to methacholine in asthmatics. *Free Radic Biol Med* 31:1442-7.
- ~~1009~~.1006. Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, Thomas DC. 1999. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am J Respir Crit Care Med* 159:768-75.
- ~~1010~~.1007. Petruzzi S, De Acetis L, Chiarotti F, Sorace A, Alleva E. 1999. Limited changes in handedness and morphine reactivity in CD-1 mice after pre- and postnatal ozone exposure. *Acta Neurobiol Exp (Wars)* 59:115-22.
- ~~1011~~.1008. Petruzzi S, Fiore M, Dell'Omo G, Bignami G, Alleva E. 1995. Medium and long-term behavioral effects in mice of extended gestational exposure to ozone. *Neurotoxicol Teratol* 17:463-70.
- ~~1012~~.1009. Phalen RF, Crocker TT, McClure TR, Tyler NK (1986). Effect of ozone on mean linear intercept in the lung of young beagles. *J Toxicol Environ Health* 17(2-3):285-96.
- ~~1013~~.1010. Pickrell JA, Hahn FF, Rebar AH, Horoda RA, Henderson RF. 1987. Changes in collagen metabolism and proteinolysis after repeated inhalation exposure to ozone. *Exp Mol Pathol* 46:159-67.
- ~~1014~~.1011. Pinkerton KE, Brody AR, Miller FJ, Crapo JD. 1989. Exposure to low levels of ozone results in enhanced pulmonary retention of inhaled asbestos fibers. *Am Rev Respir Dis* 140:1075-81.
- ~~1015~~.1012. Pinkerton KE, Joad JP. 2000. The mammalian respiratory system and critical windows of exposure for children's health. *Environ Health Perspect* 108 Suppl 3:457-62.
- ~~1016~~.1013. Pinkerton KE, Menache MG, Plopper CG. 1995. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part IX: Changes in the tracheobronchial epithelium, pulmonary acinus, and lung antioxidant enzyme activity. *Res Rep Health Eff Inst* 41-98; discussion 99-110.
- ~~1017~~.1014. Pinkerton KE, Mercer RR, Plopper CG, Crapo JD. 1992. Distribution of injury and microdosimetry of ozone in the ventilatory unit of the rat. *J Appl Physiol* 73:817-24.

- ~~1018~~.1015. Pinkerton KE, Weller BL, Menache MG, Plopper CG. 1998. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part XIII. A comparison of changes in the tracheobronchial epithelium and pulmonary acinus in male rats at 3 and 20 months. *Res Rep Health Eff Inst.* 1-32; discussion 33-7.
- ~~1019~~.1016. Pino MV, McDonald RJ, Berry JD, Joad JP, Tarkington BK, Hyde DM. 1992. Functional and morphologic changes caused by acute ozone exposure in the isolated and perfused rat lung. *Am Rev Respir Dis* 145:882-9.
- ~~1020~~.1017. Plopper CG, Chang AM, Pang A, Buckpitt AR. 1991. Use of microdissected airways to define metabolism and cytotoxicity in murine bronchiolar epithelium. *Exp Lung Res* 17:197-212.
- ~~1021~~.1018. Plopper CG, Chow CK, Dungworth DL, Brummer M, Nemeth TJ. 1978. Effect of low level of ozone on rat lungs. II. Morphological responses during recovery and re-exposure. *Exp Mol Pathol* 29:400-11.
- ~~1022~~.1019. Plopper CG, Chow CK, Dungworth DL, Tyler WS. 1979. Pulmonary alterations in rats exposed to 0.2 and 0.1 ppm ozone: a correlated morphological and biochemical study. *Arch Environ Health* 34:390-5.
- ~~1023~~.1020. Plopper CG, Chu FP, Haselton CJ, Peake J, Wu J, Pinkerton KE. 1994a. Dose-dependent tolerance to ozone. I. Tracheobronchial epithelial reorganization in rats after 20 months' exposure. *Am J Pathol* 144:404-20.
- ~~1024~~.1021. Plopper CG, Duan X, Buckpitt AR, Pinkerton KE. 1994b. Dose-dependent tolerance to ozone. IV. Site-specific elevation in antioxidant enzymes in the lungs of rats exposed for 90 days or 20 months. *Toxicol Appl Pharmacol* 127:124-31.
- ~~1025~~.1022. Plopper CG, Hatch GE, Wong V, Duan X, Weir AJ, Tarkington BK, Devlin RB, Becker S, Buckpitt AR. 1998. Relationship of inhaled ozone concentration to acute tracheobronchial epithelial injury, site-specific ozone dose, and glutathione depletion in rhesus monkeys. *Am J Respir Cell Mol Biol* 19:387-99.
- ~~1026~~.1023. Postlethwait EM, Joad JP, Hyde DM, Schelegle ES, Bric JM, Weir AJ, Putney LF, Wong VJ, Velsor LW, Plopper CG. 2000. Three-dimensional mapping of ozone-induced acute cytotoxicity in tracheobronchial airways of isolated perfused rat lung. *Am J Respir Cell Mol Biol* 22:191-9.
- ~~1027~~.1024. Pryor WA, Squadrito GL, Friedman M. 1995. The cascade mechanism to explain ozone toxicity: the role of lipid ozonation products. *Free Radic Biol Med* 19:935-41.
- ~~1028~~.1025. Pryor WA. 1992. How far does ozone penetrate into the pulmonary

air/tissue boundary before it reacts? Free Radic Biol Med 12:83-8.

~~1029-1026.~~ Rahman I, Massaro GD, Massaro D. 1992. Exposure of rats to ozone: evidence of damage to heart and brain. Free Radic Biol Med 12:323-6.

~~1030-1027.~~ Raub JA, Miller FJ, Graham JA (1983). Effects of low-level ozone exposure on pulmonary function in adult and neonatal rats. Advances in Modern Environmental Toxicology 5:363-7.

~~1031-1028.~~ Redd SC, Mokdad AH. 2002. Invited commentary: obesity and asthma--new perspectives, research needs, and implications for control programs. Am J Epidemiol 155:198-202.

~~1032-1029.~~ Reinhart PG, Gupta SK, Bhalla DK. 1999. Attenuation of ozone-induced lung injury by interleukin-10. Toxicol Lett 110:35-42.

~~1033-1030.~~ Reisenauer CS, Koenig JQ, McManus MS, Smith MS, Kusic G, Pierson WE. 1988. Pulmonary response to ozone exposures in healthy individuals aged 55 years or greater. JAPCA 38:51-5.

~~1034-1031.~~ Reiser KM, Tyler WS, Hennessy SM, Dominguez JJ, Last JA. 1987. Long-term consequences of exposure to ozone. II. Structural alterations in lung collagen of monkeys. Toxicol Appl Pharmacol 89:314-22.

~~1035-1032.~~ Riedel F, Kramer M, Scheibenbogen C, Rieger CH-. 1988. Effects of SO₂ exposure on allergic sensitization in the guinea pig. J Allergy Clin Immunol 82:527-34.

~~1036-1033.~~ Rigas ML, Ben-Jebria A, Ultman JS. 1997. Longitudinal distribution of ozone absorption in the lung: effects of nitrogen dioxide, sulfur dioxide, and ozone exposures. Arch Environ Health 52:173-8.

~~1037-1034.~~ Rivas-Arancibia S, Vazquez-Sandoval R, Gonzalez-Kladiano D, Schneider-Rivas S, Lechuga-Guerrero A. 1998. Effects of ozone exposure in rats on memory and levels of brain and pulmonary superoxide dismutase. Environ Res 76 :33-9.

~~1038-1035.~~ Rivas-Manzano P, Paz C. 1999. Cerebellar morphological alterations in rats induced by prenatal ozone exposure. Neurosci Lett 276:37-40.

~~1039-1036.~~ Rombout PJA, Lioy PJ, Goldstein BD. 1986. Rationale for and eight-hour ozone standard. 36. 36(8):913-7.

~~1040-1037.~~ Rombout PJA, van Bree L, Heisterkamp SH, Marra M. 1989. The need for an eight hour ozone standard. Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international

symposium May 1988; Nijmegen, The Netherlands. Amsterdam, The Netherlands: Elsevier Science Publishers B. V., p. 701-710.

~~1041-1038.~~ Romero-Velazquez RM, Alfaro-Rodriguez A, Gonzalez-Pina R, Gonzalez-Maciel A (2002). Effect of ozone prenatal exposure on postnatal development of cerebellum. *Proc West Pharmacol Soc* 45:65-7.

~~1042-1039.~~ Romieu I, Meneses F, Ramirez M, Ruiz S, Perez Padilla R, Sienna JJ, Gerber M, Grievink L, Dekker R, Walda I, Brunekreef B. 1998. Antioxidant supplementation and respiratory functions among workers exposed to high levels of ozone. *Am J Respir Crit Care Med* 158:226-232.

~~1043-1040.~~ Romieu I, Sienna-Monge JJ, Ramirez-Aguilar M, Moreno-Macias H, Reyes-Ruiz NI, Estela del Rio-Navarro B, et al. 2004. Genetic polymorphism of GSTM1 and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City. *Thorax* 59:8-10.

~~1044-1041.~~ Romieu I, Sienna-Monge JJ, Ramirez-Aguilar M, Tellez-Rojo MM, Moreno-Macias H, Reyes-Ruiz NI, del Rio-Navarro BE, Ruiz-Navarro MX, Hatch G, Slade R, Hernandez-Avila M. 2002. Antioxidant supplementation and lung functions among children with asthma exposed to high levels of air pollutants. *Am J Respir Crit Care Med* 166:703-709.

~~1045-1042.~~ Rossiter CE, Weill H. 1974. Ethnic differences in lung function: evidence for proportional differences. *Int J Epidemiol* 3:55-61.

~~1046-1043.~~ Roux E, Hyvelin JM, Savineau JP, Marthan R. 1999. Human isolated airway contraction: interaction between air pollutants and passive sensitization. *Am J Respir Crit Care Med* 160:439-45.

~~1047-1044.~~ Salvi S. 2001. Pollution and allergic airways disease. *Curr Opin Allergy Clin Immunol* 1:35-41.

~~1048-1045.~~ Samet JM, Hatch GE, Horstman D, Steck-Scott S, Arab L, Bromberg PA, Levine M, McDonnell WF, Devlin RB. 2001. Effect of antioxidant supplementation on ozone-induced lung injury in human subjects. *Am J Respir Crit Care Med* 164:819-25.

~~1049-1046.~~ Sarangapani R, Gentry PR, Covington TR, Teeguarden JG, Clewell HJ 3rd. 2003. Evaluation of the potential impact of age- and gender-specific lung morphology and ventilation rate on the dosimetry of vapors. *Inhal Toxicol* 15:987-1016.

~~1050-1047.~~ Sasaki K, Nadel JA, Hahn HL. 1987. Effect of ozone on breathing in dogs: vagal and nonvagal mechanisms. *J Appl Physiol* 62:15-26.

~~1051-1048.~~ Savin WM, Adams WC. 1979. Effects of ozone inhalation on work

performance and VO_{2max} . J Appl Physiol 46:309-14.

~~1052-1049.~~ Scannell C, Chen L, Aris RM, Tager I, Christian D, Ferrando R, Welch B, Kelly T, Balmes JR. 1996. Greater ozone-induced inflammatory responses in subjects with asthma. Am J Respir Crit Care Med 154: 24-9.

~~1053-1050.~~ Schelegle ES, Adams WC, Giri SN, Siefkin AD. 1989. Acute ozone exposure increases plasma prostaglandin F2 alpha in ozone-sensitive human subjects. Am Rev Respir Dis 140:211-6.

~~1054-1051.~~ Schelegle ES, Adams WC, Siefkin AD. 1987. Indomethacin pretreatment reduces ozone-induced pulmonary function decrements in human subjects. Am Rev Respir Dis 136:1350-4.

~~1055-1052.~~ Schelegle ES, Adams WC. 1986. Reduced exercise time in competitive simulations consequent to low level ozone exposure. Med Sci Sports Exerc 18:408-14.

~~1056-1053.~~ Schelegle ES, Carl ML, Coleridge HM, Coleridge JC, Green JF. 1993. Contribution of vagal afferents to respiratory reflexes evoked by acute inhalation of ozone in dogs. J Appl Physiol 74:2338-44.

~~1057-1054.~~ Schelegle ES, Eldridge MW, Cross CE, Walby WF, Adams WC. 2001. Differential effects of airway anesthesia on ozone-induced pulmonary responses in human subjects. Am J Respir Crit Care Med 163:1121-7.

~~1058-1055.~~ Schelegle ES, Miller LA, Gershwin LJ, Fanucchi MV, Van Winkle LS, Gerriets JE, Walby WF, Mitchell V, Tarkington BK, Wong VJ, Baker GL, Pantle LM, Joad JP, Pinkerton KE, Wu R, Evans MJ, Hyde DM, Plopper CG. 2003a. Repeated episodes of ozone inhalation amplifies the effects of allergen sensitization and inhalation on airway immune and structural development in Rhesus monkeys . Toxicol Appl Pharmacol 191:74-85.

~~1059-1056.~~ Schelegle ES, Siefkin AD, McDonald RJ. 1991. Time course of ozone-induced neutrophilia in normal humans. Am Rev Respir Dis 143:1353-8.

~~1060-1057.~~ Schelegle ES, Walby WF, Alfaro MF, Wong VJ, Putney L, Stovall MY, Sterner-Kock A, Hyde DM, Plopper CG. 2003b. Repeated episodes of ozone inhalation attenuates airway injury/repair and release of substance P, but not adaptation. Toxicol Appl Pharmacol 186:127-42.

~~1061-1058.~~ Schierhorn K, Hanf G, Fischer A, Umland B, Olze H, Kunkel G. 2002. Ozone-induced release of neuropeptides from human nasal mucosa cells. Int Arch Allergy Immunol 129:145-51.

~~1062-1059.~~ Schlesinger RB, Cohen M, Gordon T, Nadziejko C, Zelikoff JT, Sisco M, Regal JF, Menache MG. 2002. Ozone-induced modulation of

airway hyperresponsiveness in guinea pigs. Res Rep Health Eff Inst :1-40; discussion 41-51.

~~1063-1060.~~ Schlessinger RB, Gorczynski JE, Dennison J, Richards L, Kinney PL, Bosland MC. 1992a. Long-term intermittent exposure to sulfuric acid aerosol, ozone, and their combination: alterations in tracheobronchial mucociliary clearance and epithelial secretory cells. Exp Lung Res 18:505-34.

~~1064-1061.~~ Schlessinger RB, Zelikoff JT, Chen LC, Kinney PL. 1992b. Assessment of toxicologic interactions resulting from acute inhalation exposure to sulfuric acid and ozone mixtures. Toxicol Appl Pharmacol 115:183-90.

~~1065-1062.~~ Schonfeld BR, Adams WC, Schelegle ES. 1989. Duration of enhanced responsiveness upon re-exposure to ozone. Arch Environ Health 44:229-36.

~~1066-1063.~~ Schuller-Levis GB, Gordon RE, Park E, Pendino KJ, Laskin DL. 1995. Taurine protects rat bronchioles from acute ozone-induced lung inflammation and hyperplasia. Exp Lung Res 21:877-88.

~~1067-1064.~~ Schwartz LW, Dungworth DL, Mustafa MG, Tarkington BK, Tyler WS. 1976. Pulmonary responses of rats to ambient levels of ozone: effects of 7-day intermittent or continuous exposure. Lab Invest 34:565-78.

~~1068-1065.~~ Seal E Jr, McDonnell WF, House DE, Salaam SA, Dewitt PJ, Butler SO, Green J, Raggio L. 1993. The pulmonary response of white and black adults to six concentrations of ozone. Am Rev Respir Dis 147:804-10.

~~1069-1066.~~ Seal E Jr, McDonnell WF, House DE. 1996. Effects of age, socioeconomic status, and menstrual cycle on pulmonary response to ozone. Arch Environ Health 51:132-7.

~~1070-1067.~~ Selgrade MK, Cooper KD, Devlin RB, van Loveren H, Biagini RE, Luster MI. 1995. Immunotoxicity--bridging the gap between animal research and human health effects. Fundam Appl Toxicol 24:13-21.

~~1071-1068.~~ Selgrade MK, Hatch GE, Grose EC, Stead AG, Miller FJ, Graham JA, Stevens MA, Hardisty JF. 1990. Pulmonary effects due to subchronic exposure to oil fog. Toxicol Ind Health 6:123-43.

~~1072-1069.~~ Selgrade MK, Illing JW, Starnes DM, Stead AG, Menache MG, Stevens MA. 1988. Evaluation of effects of ozone exposure on influenza infection in mice using several indicators of susceptibility. Fundam Appl Toxicol 11:169-80.

~~1073-1070.~~ Seltzer J, Bigby BG, Stulbarg M, Holtzman MJ, Nadel JA, Ueki IF,

- Leikauf GD, Goetzi EJ, Boushey HA. 1986. ~~ozone~~ O₃-induced change in bronchial reactivity to methacholine and airway inflammation in humans. *J Appl Physiol* 60:1321-6.
- ~~1074-1071.~~ Sherwin RP, Richters V. 1985. Effect of 0.3 ppm ozone exposure on type II cells and alveolar walls of newborn mice: an image-analysis quantitation. *J Toxicol Environ Health* 16:535-46.
- ~~1075-1072.~~ Shore SA, Abraham JH, Schwartzman IN, Murthy GG, Laporte JD. 2000. Ventilatory responses to ozone are reduced in immature rats. *J Appl Physiol* 88:2023-30.
- ~~1076-1073.~~ Shore SA, Johnston RA, Schwartzman IN, Chism D, Krishna Murthy GG. 2002. Ozone-induced airway hyperresponsiveness is reduced in immature mice. *J Appl Physiol* 92:1019-28.
- ~~1077-1074.~~ Sienra-Monge JJ, Ramirez-Aguilar M, Moreno-Macias H, Reyes-Ruiz NI, del Rio-Navarro BE, Ruiz-Navarro MX, Hatch G, Crissman K, Slade R, Devlin RB, Romieu I. 2004. Antioxidant supplementation and nasal inflammatory responses among young asthmatics exposed to high levels of ozone. *Clin Exp Immunol* 138:317-322.
- ~~1078-1075.~~ Silbaugh SA, Mauderly JL. 1986. Effects of ozone and sulfuric acid aerosol on gas trapping in the guinea pig lung. *J Toxicol Environ Health* 18:133-41.
- ~~1079-1076.~~ Sills RC, Hong HL, Greenwell A, Herbert RA, Boorman GA, Devereux TR. 1995. Increased frequency of K-ras mutations in lung neoplasms from female B6C3F1 mice exposed to ozone for 24 or 30 months. *Carcinogenesis* 16:1623-8.
- ~~1080-1077.~~ Silverman F, Folinsbee LJ, Barnard J, Shephard RJ. 1976. Pulmonary function changes in ozone-interaction of concentration and ventilation. *J Appl Physiol* 41:859-64.
- ~~1081-1078.~~ Silverman F. 1979. Asthma and respiratory irritants (ozone). *Environ Health Perspect* 29:131-6.
- ~~1082-1079.~~ Smiler KL, Anver MR, Brady AN. 1988. Histopathological effects of chronic exposure of Fischer-344 rats to low-levels of ozone. ~~Research publication no. GMR-6355. research publication no. GMR-6355.~~
- ~~1083-1080.~~ Smith BW, Kolb EJ, Phelps HW, Weiss HA, Hollinden AB. 1964. Tokyo-Yokohama asthma, an area specific air pollution disease. *Arch Environ Health* 81:805-17.
- ~~1084-1081.~~ Solic JJ, Hazucha MJ, Bromberg PA. 1982. The acute effects of 0.2 ppm ozone in patients with chronic obstructive pulmonary disease. *Am Rev*

Respir Dis 125:664-9.

~~1085~~.1082. Sorace A, de Acetis L, Alleva E, Santucci D. 2001. Prolonged exposure to low doses of ozone: short- and long-term changes in behavioral performance in mice. *Environ Res* 85:122-34.

~~1086~~.1083. Spannhake EW. 1996. Down-regulation of canine airway mast cell function following exposure to ozone in vivo. *Exp Lung Res* 22:163-78.

~~1087~~.1084. Stacy RW, Seal E Jr, House DE, Green J, Roger LJ, Raggio L. 1983. A survey of effects of gaseous and aerosol pollutants on pulmonary function of normal males. *Arch Environ Health* 38:104-15.

~~1088~~.1085. Stenfors N, Pourazar J, Blomberg A, Krishna MT, Mudway I, Helleday R, Kelly FJ, Frew AJ, Sandstrom T. 2002. Effect of ozone on bronchial mucosal inflammation in asthmatic and healthy subjects. *Respir Med* 96:352-8.

~~1089~~.1086. Stephens RJ, Buntman DJ, Negi DS, Parkhurst RM, Thomas DW. 1983. Tissue levels of vitamin E in the lung and the cellular response to injury resulting from oxidant gas exposure. ~~83 (suppl.)~~. *Chest* 83 (suppl.):37S-9S.

~~1090~~.1087. Stephens RJ, Evans MJ, Sloan MF, Freeman G. 1974. A comprehensive ultrastructural study of pulmonary injury and repair in the rat resulting from exposures to less than one PPM ozone. *Chest* 65:Suppl:11S-13S.

~~1091~~.1088. Stephens RJ, Freeman G, Stara JF, Coffin DL. 1973. Cytologic changes in dogs lungs induced by chronic exposure to ozone. *Am J Pathol* 73:711-26.

~~1092~~.1089. Stephens RJ, Sloan MF, Evans MJ, Freeman G. 1974a. Early response of lung to low levels of ozone. *Am J Pathol* 74:31-58.

~~1093~~.1090. Stephens RJ, Sloan MF, Evans MJ, Freeman G. 1974b. Alveolar type 1 cell response to exposure to 0.5 PPM ~~ozone~~ O₃ for short periods. *Exp Mol Pathol* 20:11-23.

~~1094~~.1091. Stephens RJ, Sloan MF, Groth DG, Negi DS, Lunan KD. 1978. Cytologic responses of postnatal rat lungs to ~~ozone~~ O₃ or NO₂ exposure. *Am J Pathol* 93:183-200.

~~1095~~.1092. Stephens RJ, Sloan MF, Groth DG. 1976. Effects of long-term, low-level exposure of NO₂ or ~~ozone~~ O₃ on rat lungs. ~~46~~. *Environ Health Perspect* 16:178-9.

~~1096~~.1093. Sterling TD, Phair JJ, Pollack SV, Schumsky DA, DeGroot I. 1966.

Urban ~~disease morbidity~~ and air pollution. A first report. Arch Environ Health 13:158-70 contd.

~~1097-1094.~~ Sterner-Kock A, Kock M, Braun R, Hyde DM. 2000. Ozone-induced epithelial injury in the ferret is similar to nonhuman primates. Am J Respir Crit Care Med 162:1152-6.

~~1098-1095.~~ Sterner-Kock A, Vesely KR, Stovall MY, Schelegle ES, Green JF, Hyde DM. 1996. Neonatal capsaicin treatment increases the severity of ozone-induced lung injury. Am J Respir Crit Care Med 153:436-43.

~~1099-1096.~~ Stiles J, Tyler WS. 1988. Age-related morphometric differences in responses of rat lungs to ozone. Toxicol Appl Pharmacol 92:274-85.

~~1100-1097.~~ Sulakvelidze I, Inman MD, Rerecich T, O'Byrne PM. 1998. Increases in airway eosinophils and interleukin-5 with minimal bronchoconstriction during repeated low-dose allergen challenge in atopic asthmatics. Eur Respir J 11:821-7.

~~1101-1098.~~ Sumitomo M, Nishikawa M, Fukuda T, Kaneko T, Ikeda H, Suzuki S, Okubo T. 1990. Effects of ozone exposure on experimental asthma in guinea pigs sensitized with ovalbumin through the airway. Int Arch Allergy Appl Immunol 93:139-47.

~~1102-1099.~~ Suzuki E, Takahashi Y, Aida S, Kimura Y, Ito Y, Miura T. 1992. Alteration in surface structure of Clara cells and pulmonary cytochrome P-450b level in rats exposed to ozone. Toxicology 71:223-32.

~~1103-1100.~~ Takebayashi T, Abraham J, Murthy GG, Lilly C, Rodger I, Shore SA. 1998. Role of tachykinins in airway responses to ozone in rats. J Appl Physiol 85:442-50.

~~1104-1101.~~ Tepper JS, Costa DL, Lehmann JR, Weber MF, Hatch GE. 1989. Unattenuated structural and biochemical alterations in the rat lung during functional adaptation to ozone. Am Rev Respir Dis 140:493-501.

~~1105-1102.~~ Tepper JS, Wiester MJ, Weber MF, Fitzgerald S, Costa DL. 1991. Chronic exposure to a simulated urban profile of ozone alters ventilatory responses to carbon dioxide challenge in rats. Fundam Appl Toxicol 17:52-60.

~~1106-1103.~~ Tepper JS, Wiester MJ, Weber MF, Menache MG. 1990. Measurements of cardiopulmonary response in awake rats during acute exposure to near-ambient concentrations of ozone. J Appl Toxicol 10:7-15.

~~1107-1104.~~ Torres A, Utell MJ, Morow PE, Voter KZ, Whitin JC, Cox C, Looney RJ, Speers DM, Tsai Y, Frampton MW. 1997. Airway inflammation in smokers and nonsmokers with varying responsiveness to ozone. Am J

Respir Crit Care Med 156:728-36.

~~1108-1105.~~ Trenga CA, Koenig JQ, Williams PV. 2001. Dietary antioxidants and ozone-induced bronchial hyperresponsiveness in adults with asthma. Arch Environ Health 56:242-9.

~~1109-1106.~~ Tyler WS, Tyler NK, Last JA, Barstow TJ, Magliano DJ, Hinds DM. 1987. Effects of ozone on lung and somatic growth. Pair fed rats after ozone exposure and recovery periods. Toxicology 46:1-20.

~~1110-1107.~~ Tyler WS, Tyler NK, Last JA, Gillespie MJ, Barstow TJ. 1988. Comparison of daily and seasonal exposures of young monkeys to ozone. Toxicology 50:131-44.

~~1111-1108.~~ Tyler WS, Tyler NK, Magliano DJ, Hinds DM, Tarkington B, Julian MD, et al. 1991 Effects of ozone inhalation on lungs of juvenile monkeys. Morphometry after a 12-month exposure and following and following a 6 month post-exposure period. In: Berglund RL, Lawson DR, McKee DJ, eds. Tropospheric ozone and the environment: Papers from an international conference, March 1990, Los Angeles, CA. Pittsburgh, PA: Air and Waste Management Assoc., p. 151-160.

~~1112-1109.~~ Tyson CA, Lunan KD, Stephens RJ. 1982. Age-related differences in GSH-shuttle enzymes in NO₂- or ozone O₃-exposed rat lungs. Arch Environ Health 37:167-76.

~~1113-1110.~~ Uchida DA, Ballowe CA, Irvin CG, Larsen GL. 1992. Assessment of airway responsiveness to inhaled methacholine and the effects of short-term ozone exposure in aged Fischer-344 rats. 1. 1:20-3.

~~1114-1111.~~ Umezu T, Shimojo N, Tsubone H, Suzuki AK, Kubota K, Shimizu A. 1987. Effect of ozone toxicity in the drinking behavior of rats. Arch Environ Health 42:58-62.

~~1115-1112.~~ US EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Vol. 3.

~~1116-1113.~~ Utell MJ, Frampton MW, Morrow PE, Cox C, Levy PC, Speers DM, Gibb FR. 1994. Oxidant and acid aerosol exposure in healthy subjects and subjects with asthma. Part II: Effects of sequential sulfuric acid and ozone exposures on the pulmonary function of healthy subjects and subjects with asthma. Res Rep Health Eff Inst. 37-93, discussion 95-112.

~~1117-1114.~~ Vagaggini B, Taccola M, Cianchetti S, Carnevali S, Bartoli ML, Bacci E, Dente FL, Di Franco A, Giannini D, Paggiaro PL. 2002. Ozone exposure increases eosinophilic airway response induced by previous allergen challenge. Am J Respir Crit Care Med 166:1073-7.

- 1118-1115. Vagaggini B, Taccola M, Conti I, Carnevali S, Cianchetti S, Bartoli ML, Bacci E, Dente FL, Di Franco A, Giannini D, Paggiaro PL. 2001. Budesonide reduces neutrophilic but not functional airway response to ozone in mild asthmatics. *Am J Respir Crit Care Med* 164:2172-6.
- 1119-1116. Van Bree L, Dormans JA, Boere AJ, Rombout PJ. 2001. Time study on development and repair of lung injury following ozone exposure in rats. *Inhal Toxicol* 13:703-18.
- 1120-1117. Van Bree L, Marra M, van Scheindelen HJ, Fischer PH, de Loos S, Buringh E, Rombout PJ. 1995. Dose-effect models for ozone exposure: tool for quantitative risk estimation. *Toxicol Lett* 82-83:317-21.
- 1121-1118. Van Bree L, Rombout PJA, Rietjens IMCM, Dormans JAMA, Marra M. 1989. Pathochemical effects in rat lung related to episodic ozone exposure. Nijmegen, The Netherlands. Nijmegen, The Netherlands (Elsevier Science Publishers, Amsterdam, The Netherlands):723-32.
- 1122-1119. Van Loveren H, Krajnc EI, Rombout PJ, Blommaert FA, Vos JG. 1990. Effects of ozone, hexachlorobenzene, and bis(tri-n-butyltin)oxide on natural killer activity in the rat lung. *Toxicol Appl Pharmacol* 102:21-33.
- 1123-1120. Van Loveren H, Rombout PJ, Wagenaar SS, Walvoort HC, Vos JG. 1988. Effects of ozone on the defense to a respiratory *Listeria monocytogenes* infection in the rat. Suppression of macrophage function and cellular immunity and aggravation of histopathology in lung and liver during infection. *Toxicol Appl Pharmacol* 94:374-93.
- 1124-1121. Vargas MH, Romero L, Sommer B, Zamudio P, Gustin P, Montano LM. 1998. Chronic exposure to ozone causes tolerance to airway hyperresponsiveness in guinea pigs: lack of SOD role. *J Appl Physiol* 84:1749-55.
- 1125-1122. Vargas MH, Segura P, Campos MG, Hong E, Montano LM. 1994. Effect of ozone exposure on antigen-induced airway hyperresponsiveness in guinea pigs. *J Toxicol Environ Health* 42:435-42.
- 1126-1123. Vesely KR, Hyde DM, Stovall MY, Harkema JR, Green JF, Schelegle ES. 1999a. Capsaicin-sensitive C-fiber-mediated protective responses in ozone inhalation in rats. *J Appl Physiol* 86:951-62.
- 1127-1124. Vesely KR, Schelegle ES, Stovall MY, Harkema JR, Green JF, Hyde DM. 1999b. Breathing pattern response and epithelial labeling in ozone-induced airway injury in neutrophil-depleted rats. *Am J Respir Cell Mol Biol* 20:699-709.
- 1128-1125. Victorin K. 1996. Genotoxicity and carcinogenicity of ozone. *Scand J Work Environ Health* 22 Suppl 3:42-51.

- ~~1129-1126.~~ Vincent R, Adamson IY. 1995. Cellular kinetics in the lungs of aging Fischer 344 rats after acute exposure to ozone. *Am J Pathol* 146:1008-16.
- ~~1130-1127.~~ Vincent R, Bjarnason SG, Adamson IY, Hedgecock C, Kumarathasan P, Guenette J, Potvin M, Goegan P, Bouthillier L. 1997. Acute pulmonary toxicity of urban particulate matter and ozone. *Am J Pathol* 151:1563-70.
- ~~1131-1128.~~ Wagner JG, Hotchkiss JA, Harkema JR. 2002. Enhancement of nasal inflammatory and epithelial responses after ozone and allergen coexposure in Brown Norway rats. *Toxicol Sci* 67:284-94.
- ~~1132-1129.~~ Warren JB, Dalton N. 1983. A comparison of the bronchodilator and vasopressor effects of exercise levels of adrenaline in man. *Clin Sci (Lond)* 64:475-9.
- ~~1133-1130.~~ Watkinson WP, Campen MJ, Nolan JP, Costa DL. 2001. Cardiovascular and systemic responses to inhaled pollutants in rodents: effects of ozone and particulate matter. *Environ Health Perspect* 109 Suppl 4:539-46.
- ~~1134-1131.~~ Watkinson WP, Gordon CJ. 1993. Caveats regarding the use of the laboratory rat as a model for acute toxicological studies: modulation of the toxic response via physiological and behavioral mechanisms. *Toxicology* 81:15-31.
- ~~1135-1132.~~ Watt KC, Plopper CG, Weir AJ, Tarkington B, Buckpitt AR. 1998. Cytochrome P450 2E1 in rat tracheobronchial airways: response to ozone exposure. *Toxicol Appl Pharmacol* 149:195-202.
- ~~1136-1133.~~ Wayne WS, Wehrle PF, Carroll RE. 1967. Oxidant air pollution and athletic performance. *JAMA* 199:901-4.
- ~~1137-1134.~~ Weinman GG, Liu MC, Proud D, Weidenbach-Gerbase M, Hubbard W, Frank R. 1995a. Ozone exposure in humans: inflammatory, small and peripheral airway responses. *Am J Respir Crit Care Med* 152: 1175-1182.
- ~~1138-1135.~~ Weinmann GG, Weidenbach-Gerbase M, Foster WM, Zacur H, Frank R. 1995b. Evidence for ozone-induced small-airway dysfunction: lack of menstrual-cycle and gender effects. *Am J Respir Crit Care Med* 152:988-96.
- ~~1139-1136.~~ Weymer AR, Gong H Jr, Lyness A, Linn WS. 1994. Pre-exposure to ozone does not enhance or produce exercise-induced asthma. *Am J Respir Crit Care Med* 149:1413-9.
- ~~1140-1137.~~ Whittemore AS, Korn EL. 1980. Asthma and air pollution in the Los Angeles Area. *Am J Public Health* 70:687-96.

- ~~1141-1138.~~ Wiester MJ, Tepper JS, Doerfler DL, Costa DL. 1995. Ozone adaptation in rats after chronic exposure to a simulated urban profile of ozone. *Fundam Appl Toxicol* 24:42-51.
- ~~1142-1139.~~ Wiester MJ, Tepper JS, Winsett DW, Crissman KM, Richards JH, Costa DL. 1996a. Adaptation to ozone in rats and its association with ascorbic acid in the lung. *Fundam Appl Toxicol* 31:56-64 .
- ~~1143-1140.~~ Wiester MJ, Watkinson WP, Costa DL, Crissman KM, Richards JH, Winsett DW, Highfill JW. 1996b. Ozone toxicity in the rat. III. Effect of changes in ambient temperature on pulmonary parameters. *J Appl Physiol* 81:1691-700.
- ~~1144-1141.~~ Wiester MJ, Winsett DW, Richards JH, Jackson MC, Crissman KM, Costa DL. 2000. Ozone adaptation in mice and its association with ascorbic acid in the lung. *Inhal Toxicol* 12:577-90.
- ~~1145-1142.~~ Wilson DW, Plopper CG, Dungworth DL. 1984. The response of the macaque tracheobronchial epithelium to acute ozone injury. A quantitative ultrastructural and autoradiographic study. *Am J Pathol* 116:193-206.
- ~~1146-1143.~~ Witschi H, Breider MA, Schuller HM. 1993a. Failure of ozone and nitrogen dioxide to enhance lung tumor development in hamsters. *Res Rep Health Eff Inst.* 1-25; discussion 27-38.
- ~~1147-1144.~~ Witschi H, Espiritu I, Pinkerton KE, Murphy K, Maronpot RR. 1999. Ozone carcinogenesis revisited. *Toxicol Sci* 52:162-7.
- ~~1148-1145.~~ Witschi H, Wilson DW, Plopper CG. 1993b. Modulation of N-nitrosodiethylamine-induced hamster lung tumors by ozone. *Toxicology* 77:193-202.
- ~~1149-1146.~~ Wright E, Gross K, Smiler K. 1989. Continuous chronic exposure to ozone: biochemical, functional and histopathologic effects in rat lung. Presented at: Proceedings of the 82nd annual meeting of the Air and Waste Management Association, June 25-30, 1989, Anaheim, CA Paper no. 89-89:12.2.
- ~~1150-1147.~~ Wright ES, White DM, Smiler KL. 1990. Effects of chronic exposure to ozone on pulmonary lipids in rats. *Toxicology* 64:313-24.
- ~~1151-1148.~~ Yang IA, Holz O, Jorres RA, Magnussen H, Barton SJ, Rodriguez S, et al. 2005. Association of tumor necrosis factor-alpha polymorphisms and ozone-induced change in lung function. *Am J Respir Crit Care Med* 171:171-6.
- ~~1152-1149.~~ Ying RL, Gross KB, Terzo TS, Eschenbacher WL. 1990. Indomethacin does not inhibit the ozone-induced increase in bronchial responsiveness in human subjects. *Am Rev Respir Dis* 142:817-21.

- ~~1153~~-1150. Yokoyama E, Goto H, Kawai K, Kyono H. 1989. Mechanical properties of rabbit lung with edema caused by exposure to ozone. *J Environ Pathol Toxicol Oncol* 9:95-108.
- ~~1154~~-1151. Yokoyama E, Ichikawa I, Nambu Z, Kawai K, Kyono Y. 1984. Respiratory effects of intermittent exposure to ozone of rats. *Environ Res* 33:271-83.
- ~~1155~~-1152. Yokoyama E, Nambu Z, Ichikawa I, Uchiyama I, Arakawa H. 1987. Pulmonary response to exposure to ozone of emphysematous rats. *Environ Res* 42:114-20.
- ~~1156~~-1153. Yokoyama E. 1984. A simple method for measuring the respiratory uptake of carbon monoxide in unanesthetized rats: an application to rats acutely exposed to ozone. *Arch Environ Health* 39:375-8.
- ~~1157~~-1154. Yoshida. R. , Motomiya K, Saito H, Funabashi S 1974. *Clinical Implications of Air Pollution Research*. Acton, MA: Publishing Sciences Group, Inc.
- ~~1158~~-1155. Young WA, Shaw DB, Bates DV. 1964. Effect of low concentrations of ozone on pulmonary function in man. *J Appl Physiol* 19:765-8.
- ~~1159~~-1156. Yu M, Pinkerton KE, Witschi H. 2002. Short-term exposure to aged and diluted sidestream cigarette smoke enhances ozone-induced lung injury in B6C3F1 mice. *Toxicol Sci* 65:99-106.
- ~~1160~~-1157. Zeidberg LD, Prindle RA, Landau UE. 1961. The Nashville air pollution study. I. Sulfur dioxide and bronchial asthma. A preliminary report. *Am Rev Respir Dis* 84:489-503.
- ~~1161~~-1158. Zitnik LA, Schwartz LW, McQuillen NK, Zee YC, Osebold JW. 1978. Pulmonary changes induced by low-level ozone: morphological observations. *J Environ Pathol Toxicol* 1:365-76.

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- ~~1162~~-1159. Abbey DE, Nishino N, McDonnell WF, Burchette RJ, Knutsen SF, Lawrence Beeson W, Yang JX. 1999. Long-term inhalable particles and other air pollutants related to ~~death~~-mortality in nonsmokers. *Am J Respir Crit Care Med* 159:373-82.
- ~~1163~~-1160. Abbey DE, Petersen FF, Mills PK, Kittle L. 1993. Chronic respiratory disease associated with long-term ambient concentrations of sulfates and other air pollutants. *J Expo Anal Environ Epidemiol* 3 Suppl 1: 99-115.

- ~~1164-1161.~~ Anderson HOUR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. 2004. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (ozone). Report of a WHO task group. World Health Organization.
- ~~1165-1162.~~ Anderson HOUR, Ponce de Leon A, Bland JM, Bower JS, Emberlin J, Strachan DP. 1998. Air pollution, pollens, and daily admissions for asthma in London 1987-92. *Thorax* 53:842-8.
- ~~1166-1163.~~ Anderson HOUR, Ponce de Leon A, Bland JM, Bower JS, Strachan DP. 1996. Air pollution and daily ~~death-mortality~~ in London: 1987-92. *BMJ* 312:665-9.
- ~~1167-1164.~~ Anderson HOUR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Ponka A, Bacharova L, Schwartz J, Katsouyanni K. 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *Eur Respir J* 10:1064-71.
- ~~1168-1165.~~ Ballester F, Tenias JM, Perez-Hoyos S. 2001. Air pollution and emergency hospital admissions for cardiovascular diseases in Valencia, Spain. *J Epidemiol Community Health* 55:57-65-.
- ~~1169-1166.~~ Bates DV, Baker-Anderson M, Sizto R. 1990. Asthma attack periodicity: a study of hospital emergency visits in Vancouver. *Environ Res* 51 :51-70.~~Bell ML.~~
- ~~1170-1167.~~ Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. 2004. Ozone and short-term ~~death-mortality~~ in 95 US urban communities, 1987-2000. *JAMA*. 292(19):2372-8.
- ~~1171-1168.~~ Borja-Aburto VH, Castillejos M, Gold DR, Bierzwinski S, Loomis D. 1998. ~~Death-Mortality~~ and ambient fine particles in southwest Mexico City, 1993-1995. *Environ Health Perspect* 106:849-55.
- ~~1172-1169.~~ Borja-Aburto VH, Loomis DP, Bangdiwala SI, Shy CM, Rascon-Pacheco RA. 1997a. ozone, suspended particulates, and daily ~~death mortality~~ in Mexico City. 145. 145:258-68.
- ~~1173.~~~~Borja-Aburto VH, Loomis DP, Bangdiwala SI, Shy CM, Rascon-Pacheco RA. 1997b. Ozone, suspended particulates, and daily death in Mexico City. Am J Epidemiol 145:258-68.~~
- ~~1174-1170.~~ Brauer M, Blair J, Vedal S. 1996. Effect of ambient ozone exposure on lung function in farm workers. *Am J Respir Crit Care Med* 154:981-7.
- ~~1175-1171.~~ Bremner SA, Anderson HOUR, Atkinson RW, McMichael AJ, Strachan DP, Bland JM, Bower JS. 1999. Short-term associations between

outdoor air pollution and ~~death~~-mortality in London 1992-4. *Occup Environ Med* 56:237-44.

~~1176~~-1172. Buchdahl R, Parker A, Stebbings T, Babiker A. 1996. Association between air pollution and acute childhood wheezy episodes: prospective observational study. *BMJ* 312:661-5.

~~1177~~-1173. Buchdahl R, Willens C, Vander M, Babiker A. 2000. Associations between ambient ozone, hydrocarbons, and childhood wheezy episodes: a prospective observational study in southeast London. *57*. *57*:86-93.

~~1178~~-1174. Burnett RT, Brook J, Dann T, Delocla C, Philips O, Cakmak S, Vincent R, Goldberg MS, Krewski D. 2000. Association between particulate- and gas-phase components of urban air pollution and daily ~~death~~-mortality in eight Canadian cities. *Inhal Toxicol* 12 Suppl 4:15-39.

~~1179~~-1175. Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. 1997a. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environ Res* 72:24-31.

~~1180~~-1176. Burnett RT, Cakmak S, Brook JR, Krewski D. 1997b. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ Health Perspect* 105:614-20.

~~1181~~-1177. Burnett RT, Dales R, Krewski D, Vincent R, Dann T, Brook JR. 1995. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. *Am J Epidemiol* 142:15-22.

~~1182~~-1178. Burnett RT, Smith-Doiron M, Stieb D, Cakmak S, Brook JR. 1999. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. *Arch Environ Health* 54:130-9.

~~1183~~-1179. Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, Leech JA, Cakmak S, Krewski D. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 153:444-52.

~~1184~~-1180. Calderon-Garciduenas L, Osnaya N, Rodriguez-Alcaraz A, Villarreal-Calderon A. 1997. DNA damage in nasal respiratory epithelium from children exposed to urban pollution. *Environ Mol Mutagen* 30:11-20.

~~1185~~-1181. Calderon-Garciduenas L, Osorno-Velazquez A, Bravo-Alvarez H, Delgado-Chavez R, Barrios-Marquez R. 1992. Histopathologic changes of the nasal mucosa in southwest Metropolitan Mexico City inhabitants. *Am J Pathol* 140:225-32.

- ~~1186-1182.~~ Calderon-Garciduenas L, Rodriguez-Alcaraz A, Garcia R, Ramirez L, Barragan G. 1995. Nasal inflammatory responses in children exposed to a polluted urban atmosphere. *J Toxicol Environ Health* 45:427-37.
- ~~1187-1183.~~ Calderon-Garciduenas L, Wen-Wang L, Zhang YJ, Rodriguez-Alcaraz A, Osnaya N, Villarreal-Calderon A, Santella RM. 1999. 8-hydroxy-2'-deoxyguanosine, a major mutagenic oxidative DNA lesion, and DNA strand breaks in nasal respiratory epithelium of children exposed to urban pollution. *Environ Health Perspect* 107:469-74.
- ~~1188-1184.~~ Cassino C, Ito K, Bader I, Ciotoli C, Thurston G, Reibman J. 1999. Cigarette smoking and ozone-associated emergency department use for asthma by adults in New York City. *Am J Respir Crit Care Med* 159:1773-9.
- ~~1189-1185.~~ Castellsague J, Sunyer J, Saez M, Anto JM. 1995. Short-term association between air pollution and emergency room visits for asthma in Barcelona. *Thorax* 50:1051-6.
- ~~1190-1186.~~ Charpin D, Pascal L, Birnbaum J, Armengaud A, Sambuc R, Lanteaume A, Vervloet D. 1999. Gaseous air pollution and atopy. *Clin Exp Allergy* 29:1474-80.
- ~~1191-1187.~~ Chen L, Yang W, Jennison BL, Goodrich A, Omaye ST. 2002. Air pollution and birth weight in northern Nevada, 1991-1999. *Inhal Toxicol* 14:141-57.
- ~~1192-1188.~~ Chen PC, Lai YM, Chan CC, Hwang JS, Yang CY, Wang JD. 1999. Short-term effect of ozone on the pulmonary function of children in primary school. *Environ Health Perspect* 107:921-5.
- ~~1193-1189.~~ Chew FT, Goh DY, Ooi BC, Saharom R, Hui JK, Lee BW. 1999. Association of ambient air-pollution levels with acute asthma exacerbation among children in Singapore. *Allergy* 54:320-9.
- ~~1194-1190.~~ Chock DP, Winkler SL, Chen C. 2000. A study of the association between daily ~~death-mortality~~ and ambient air pollutant concentrations in Pittsburgh, Pennsylvania. *J Air Waste Manag Assoc* 50:1481-500.
- ~~1195-1191.~~ Dab W, Medina S, Quenel P, Le Moullec Y, Le Tertre A, Thelot B, Monteil C, Lameloise P, Pirard P, Momas I, Ferry R, Festy B. 1996. Short term respiratory health effects of ambient air pollution: results of the APHEA project in Paris. *J Epidemiol Community Health* 50 Suppl 1:s42-6.
- ~~1196-1192.~~ De Leon SF, Thurston GD, Ito K. 2003. Contribution of respiratory disease to nonrespiratory ~~death-mortality~~ associations with air pollution. *Am J Respir Crit Care Med* 167:1117-23.

- ~~1197-1193.~~ Delfino RJ, Coate BD, Zeiger RS, Seltzer JM, Street DH, Koutrakis P. 1996. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. *Am J Respir Crit Care Med* 154:633-41.
- ~~1198-1194.~~ Delfino RJ, Zeiger RS, Seltzer JM, Street DH. 1998a. Symptoms in pediatric asthmatics and air pollution: Differences in effects by symptom severity, anti-inflammatory medication use, and particulate averaging time. *Environ Health Perspect* 106: 751-61.
- ~~1199-1195.~~ Delfino RJ, Murphy-Moulton AM, Becklake MR. 1998b. Emergency room visits for respiratory illnesses among the elderly in Montreal: association with low level ozone exposure. *Environ Res* 76:67-77.
- ~~1200-1196.~~ Delfino RJ, Murphy-Moulton AM, Burnett RT, Brook JR, Becklake MR. 1997a. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. *Am J Respir Crit Care Med* 155:568-76.
- ~~1201-1197.~~ Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, Koutrakis P. 1997b. The effect of outdoor fungal spore concentrations on daily asthma severity. *Environ Health Perspect* 105:622-35.
- ~~1202-1198.~~ Delfino RJ, Gong H Jr, Linn WS, Hu Y, Pellizzari ED. 2003. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environ Health Perspect* 111:647-656.
- ~~1203-1199.~~ Delfino RJ, Quintana PJE, Floro J, Gastañaga VM, Samimi BS, Kleinman MT, Liu L-JS, Bufalino C, Wu C-F, McLaren CE. 2004. Association of FEV1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. *Environ Health Perspect* 112:932-41.
- ~~1204-1200.~~ Desqueyroux H, Pujet JC, Prosper M, Le Moullec Y, Momas I. 2002. Effects of air pollution on adults with chronic obstructive pulmonary disease. *Arch Environ Health* 57:554-60.
- ~~1205-1201.~~ Detels R, Tashkin DP, Sayre JW, Rokaw SN, Coulson AH, Massey FJ Jr, Wegman DH. 1987. The UCLA population studies of chronic obstructive respiratory disease. 9. Lung function changes associated with chronic exposure to photochemical oxidants; a cohort study among never-smokers. *Chest* 92:594-603.
- ~~1206-1202.~~ Detels R, Tashkin DP, Sayre JW, Rokaw SN, Massey FJ Jr, Coulson AH, Wegman DH. 1991. The UCLA population studies of CORD: X. A cohort study of changes in respiratory function associated with chronic exposure to SO_x, NO_x, and hydrocarbons. *Am J Public Health* 81:350-9.
- ~~1207-1203.~~ Devlin RB, McDonnell WF, Mann R, Becker S, House DE,

Schreinemachers D, Koren HS. 1991. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *Am J Respir Cell Mol Biol* 4:72-81.

~~1208-1204.~~ Diaz J, Garcia R, Ribera P, Alberdi JC, Hernandez E, Pajares MS, Otero A. 1999. Modeling of air pollution and its relationship with death mortality and disease-morbidity in Madrid, Spain. *Int Arch Occup Environ Health* 72:366-76.

~~1209-1205.~~ Dockery DW, Schwartz J, Spengler JD. 1992. Air pollution and daily death mortality: associations with particulates and acid aerosols. *Environ Res* 59:362-73.

~~1210-1206.~~ Dominici F, Daniels M, McDermott A, Zeger SL, Samet JM. 2003. Shape of the Exposure-Response Relation and Death—Mortality Displacement in the NMMAPS Database. Health Effects Institute Special Report. Health Effects Institute Special Report:91-6.

~~1211-1207.~~ Dominici F, McDermott A, Zeger SL, Samet JM. 2002. On the use of generalized additive models in time-series studies of air pollution and health. *Am J Epidemiol* 156:193-203.

~~1212-1208.~~ Euler GL, Abbey DE, Hodgkin JE, Magie AR. 1988. Chronic obstructive pulmonary disease symptom effects of long-term cumulative exposure to ambient levels of total oxidants and nitrogen dioxide in California Seventh-Day Adventist residents. *Arch Environ Health* 43:279-85.

~~1213-1209.~~ Fairley D. 1999. Daily death mortality and air pollution in Santa Clara County, California: 1989-1996. *Environ Health Perspect* 107: 637-41.

~~1214-1210.~~ Fairley D. 2003. Death—Mortality and Air Pollution for Santa Clara County, California, 1989-1996. Health Effects Institute Special Report. Health Effects Institute Special Report. 97-106.

~~1215-1211.~~ Friedman, M.S., Powell, K.E., Lori Hutwagner, LeRoy M. Graham, W. Gerald Teague. 2001. Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma. *JAMA* 285:897-905.

~~1216-1212.~~ Frischer T, Studnicka M, Gartner C, Tauber E, Horak F, Veiter A, Spengler J, Kuhr J, Urbanek R. 1999. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med* 160:390-6.

~~1217-1213.~~ Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ Health Perspect* 107:675-9.

- ~~1218-1214.~~ Gamble JF. 1998. PM_{2.5} and death-mortality in long-term prospective cohort studies: cause-effect or statistical associations? Environ Health Perspect 106:535-49.
- ~~1219-1215.~~ Garcia-Aymerich J, Tobias A, Anto JM, Sunyer J . 2000. Air pollution and death-mortality in a cohort of patients with chronic obstructive pulmonary disease: a time series analysis. J Epidemiol Community Health 54:73-4.
- ~~1220-1216.~~ Garty BZ, Kosman E, Ganor E, Berger V, Garty L, Wietzen T, Waisman Y, Mimouni M, Waisel Y. 1998. Emergency room visits of asthmatic children, relation to air pollution, weather, and airborne allergens. Ann Allergy Asthma Immunol 81:563-70.
- ~~1221-1217.~~ Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J. 2000. Association between air pollution and lung function growth in southern California children. Am J Respir Crit Care Med 162:1383-90.
- ~~1222-1218.~~ Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. 2004. The effect of air pollution on lung development from 10 to 18 years of age. N Engl J Med 351(11):1057-67
- ~~1223-1219.~~ Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, Leaderer BP. 2003. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. JAMA 290:1859-67.
- ~~1224-1220.~~ Gielen MH, van der Zee SC, van Wijnen JH, van Steen CJ, Brunekreef B. 1997. Acute effects of summer air pollution on respiratory health of asthmatic children. Am J Respir Crit Care Med 155:2105-8.
- ~~1225-1221.~~ Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. Epidemiology 12:43-54.
- ~~1226-1222.~~ Gold DR, Damokosh AI, Pope CA 3rd, Dockery DW, McDonnell WF, Serrano P, Retama A, Castillejos M. 1999. Particulate and ozone pollutant effects on the respiratory function of children in southwest Mexico City. Epidemiology 10:8-16.
- ~~1227-1223.~~ Goldberg MS, Burnett RT. 2003. Revised analysis of the Montreal time-series study. Health Effects Institute Special Report. Health Effects Institute Special Report:113-32.
- ~~1228-1224.~~ Goldberg MS, Burnett RT, Stieb D. 2003. A review of time-series studies used to evaluate the short-term effects of air pollution on human

health. Rev Environ Health 18:269-303.

- ~~1229-1225.~~ Gong H Jr, Simmons MS, Linn WS, McDonnell WF, Westerdahl D. 1998. Relationship between acute ozone responsiveness and chronic loss of lung function in residents of a high-ozone community. Arch Environ Health 53:313-9.
- ~~1230-1226.~~ Gouveia N, Fletcher T. 2000a. Respiratory diseases in children and outdoor air pollution in Sao Paulo, Brazil: a time series analysis. Occup Environ Med 57:477-83.
- ~~1231-1227.~~ Gouveia N, Fletcher T. 2000b. Time series analysis of air pollution and death mortality: effects by cause, age and socioeconomic status. J Epidemiol Community Health 54:750-5.
- ~~1232-1228.~~ Gryparis A, Forsberg B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Samoli E, Medina S, Anderson HOUR, Niciu EM, Wichmann HE, Kriz B, Kosnik M, Skorkovsky J, Vonk JM, Dortbudak Z. 2004. Acute effects of ozone on death mortality from the "air pollution and health: a European approach" project. Am J Respir Crit Care Med. 170:1080-7.
- ~~1233-1229.~~ Gwynn RC, Burnett RT, Thurston GD. 2000. A time-series analysis of acidic particulate matter and daily death mortality and disease morbidity in the Buffalo, New York, region. Environ Health Perspect 108:125-33.
- ~~1234-1230.~~ Hagen JA, Nafstad P, Skrandal A, Bjorkly S, Magnus P. 2000. Associations between outdoor air pollutants and hospitalization for respiratory diseases. Epidemiology 11:136-40.
- ~~1235-1231.~~ Hajat S, Anderson HOUR, Atkinson RW, Haines A. 2002. Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. Occup Environ Med 59:294-9.
- ~~1236-1232.~~ Hernandez-Garduno E, Perez-Neria J, Paccagnella AM, Pina-Garcia M, Munguia-Castro M, Catalan-Vazquez M, Rojas-Ramos M. 1997. Air pollution and respiratory health in Mexico City. J Occup Environ Med 39:299-307.
- ~~1237-1233.~~ Hiltermann TJ, Stolk J, van der Zee SC, Brunekreef B, de Bruijne CR, Fischer PH, Ameling CB, Sterk PJ, Hiemstra PS, van Bree L-. 1998. Asthma severity and susceptibility to air pollution. Eur Respir J 11:686-93.
- ~~1238-1234.~~ Hodgkin JE, Abbey DE, Euler GL, Magie AR. 1984. COPD prevalence in nonsmokers in high and low photochemical air pollution areas. Chest 86:830-8.
- ~~1239-1235.~~ Hoek G. 2003. Daily Death Mortality and Air Pollution in The Netherlands. Special Report, Health Effects Institute. Special Report, Health Effects Institute. 133-42.

- ~~1240~~.1236. Hoek G, Brunekreef B, Verhoeff A, van Wijnen J, Fischer P. 2000. Daily death-mortality and air pollution in The Netherlands. J Air Waste Manag Assoc 50:1380-9.
- ~~1241~~.1237. Hoek G, Fischer P, Van Den Brandt P, Goldbohm S, Brunekreef B. 2001. Estimation of long-term average exposure to outdoor air pollution for a cohort study on death mortality. J Expo Anal Environ Epidemiol 11:459-69.
- ~~1242~~.1238. Hoek G, Schwartz JD, Groot B, Eilers P. 1997. Effects of ambient particulate matter and ozone on daily death-mortality in Rotterdam, The Netherlands. Arch Environ Health 52:455-63.
- ~~1243~~.1239. Holmen A, Blomqvist J, Frindberg H, Johnelius Y, Eriksson NE, Henricson KA, Herrstrom P, Hogstedt B. 1997. Frequency of patients with acute asthma in relation to ozone, nitrogen dioxide, other pollutants of ambient air and meteorological observations. Int Arch Occup Environ Health 69:317-22.
- ~~1244~~.1240. Ito K. 2003. Associations of Particulate Matter Components with Daily Death-mortality and Disease-Morbidity in Detroit, Michigan. Health Effects Institute Special Report. Health Effects Institute Special Report, :143-56.
- ~~1245~~.1241. Ito K, Thurston GD. 1996. Daily PM10/death mortality associations: an investigation of at-risk subpopulations. J Expo Anal Environ Epidemiol 6:79-95.
- ~~1246~~.1242. Jaffe DH, Singer ME, Rimm AA. 2003. Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. Environ Res 91:21-8.
- ~~1247~~.1243. Jones GN, Sletten C, Mandry C, Brantley PJ. 1995. Ozone level effect on respiratory illness: an investigation of emergency department visits. South Med J 88:1049-56.
- ~~1248~~.1244. Kinney PL, Aggarwal M, Nikiforov SV, Nadas A. 1998. Long-term exposure to ozone: development of methods to estimate past exposures and health outcomes. ~~Methods development for epidemiologic investigations of the health effects of prolonged ozone exposure. Part III. An approach to retrospective estimation of lifetime ozone exposure using a questionnaire and ambient monitoring data (U.S. sites).~~ Res Rep Health Eff Inst. :79-108; discussion 109-21.
- ~~1249~~.1245. Kinney PL, Ito K, Thurston GD. 1995. A sensitivity analysis of death mortality/PM10 associations in Los Angeles. In: Phalen, RF, Bates DV, eds. Proceedings of the colloquium on particulate air pollution and human death and disease mortality and morbidity; January 1994, Irvine, CA. 7.

7:59-69.

~~1250~~.1246. Kinney PL, Lippmann M. 2000. Respiratory effects of seasonal exposures to ozone and particles. Arch Environ Health 55:210-6.

~~1251~~.1247. Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, El-Fawal H, Devlin RB, Rom WN. 1996. Biomarkers of lung inflammation in recreational joggers exposed to ozone. Am J Respir Crit Care Med 154:1430-5.

~~1252~~.1248. Kinney PL, Ozkaynak H. 1991. Associations of daily ~~death-mortality~~ and air pollution in Los Angeles County. Environ Res 54:99-120.

~~1253~~.1249. Klemm RJ, Mason RM Jr. 2000. Aerosol Research and Inhalation Epidemiological Study (ARIES): air quality and daily ~~death-mortality~~ statistical modeling--interim results. J Air Waste Manag Assoc 50:1433-9.

~~1254~~.1250. Kopp MV, Ulmer C, Ihorst G, Seydewitz HH, Frischer T, Forster J, Kuehr J. 1999. Upper airway inflammation in children exposed to ambient ozone and potential signs of adaptation. Eur Respir J 14:854-61.

~~1255~~.1251. Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. 1997. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen--results of a pilot study. Environ Res 72:8-23.

~~1256~~.1252. Kuo HW, Lai JS, Lee MC, Tai RC, Lee MC. 2002. Respiratory effects of air pollutants among asthmatics in central Taiwan. Arch Environ Health 57:194-200.

~~1257~~.1253. Lee JT, Schwartz J. 1999. Reanalysis of the effects of air pollution on daily ~~death-mortality~~ in Seoul, Korea: A case-crossover design. Environ Health Perspect 107:633-6.

~~1258~~.1254. Lee JT, Shin D, Chung Y. 1999. Air pollution and daily ~~death mortality~~ in Seoul and Ulsan, Korea. Environ Health Perspect 107:149-54.

~~1259~~.1255. Lierl MB, Hornung RW. 2003. Relationship of outdoor air quality to pediatric asthma exacerbations. Ann Allergy Asthma Immunol 90:28-33.

~~1260~~.1256. Lin CA, Martins MA, Farhat SC, Pope CA 3rd, Conceicao GM, Anastacio VM, Hatanaka M, Andrade WC, Hamaue WR, Bohm GM, Saldiva PH . 1999. Air pollution and respiratory illness of children in Sao Paulo, Brazil. Paediatr Perinat Epidemiol 13:475-88.

~~1261~~.1257. Lin M, Chen Y, Burnett RT, Villeneuve PJ, Krewski D. 2003. Effect of short-term exposure to gaseous pollution on asthma hospitalisation in children: a bi-directional case-crossover analysis. J Epidemiol Community Health_ 57:-:50-5.

- ~~1262~~.1258. Linn WS, Shamoo DA, Anderson KR, Peng RC, Avol EL, Hackney JD, Gong H Jr. 1996. Short-term air pollution exposures and responses in Los Angeles area schoolchildren. *J Expo Anal Environ Epidemiol* 6:449-72.
- ~~1263~~.1259. Linn WS, Szlachcic Y, Gong H Jr, Kinney PL, Berhane KT. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ Health Perspect* 108:427-34.
- ~~1264~~.1260. Lipfert FW, Morris SC, Wyzga RE. 2000. Daily ~~death~~mortality in the Philadelphia metropolitan area and size-classified particulate matter. *J Air Waste Manag Assoc* 50:1501-13.
- ~~1265~~.1261. Lippmann M, Ito K, Nadas A, Burnett RT. 2000. Association of particulate matter components with daily ~~death~~mortality and disease morbidity in urban populations. *Res Rep Health Eff Inst* :5-72, discussion 73-82.
- ~~1266~~.1262. Lipsett M, Hurley S, Ostro B. 1997. Air pollution and emergency room visits for asthma in Santa Clara County, California. *Environ Health Perspect* 105:216-22.
- ~~1267~~.1263. Mann JK, Tager IB, Lurmann F, Segal M, Quesenberry CP Jr, Lugg MM, Shan J, Van Den Eeden SK. 2002. Air pollution and hospital admissions for ischemic heart disease in persons with congestive heart failure or arrhythmia. *Environ Health Perspect* 110:1247-52.
- ~~1268~~.1264. Martins LC, Latorre Mdo R, Saldiva PH, Braga AL. 2002. Air pollution and emergency room visits due to chronic lower respiratory diseases in the elderly: an ecological time-series study in Sao Paulo, Brazil. *J Occup Environ Med* 44:622-7.
- ~~1269~~.1265. McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386-91.
- ~~1270~~.1266. McDonnell WF, Abbey DE, Nishino N, Lebowitz MD-. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG Study. *Environ Res* 80:110-21.
- ~~1271~~.1267. Moolgavkar SH, Luebeck EG, Anderson EL. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. *Epidemiology* 8:364-70.
- ~~1272~~.1268. Moolgavkar SH, Luebeck EG, Hall TA, Anderson EL. 1995. Air pollution and daily ~~death~~mortality in Philadelphia. *Epidemiology* 6: 476-84.
- ~~1273~~.1269. Moolgavkar, S. H. and Lubeck, E. G. 1996. A critical review of the evidence on particulate air pollution and ~~death~~mortality. *Epidemiology* 7,

420-428.

~~1274-1270.~~ Morgan G, Corbett S, Wlodarczyk J, Lewis P. 1998. Air pollution and daily ~~death-mortality~~ in Sydney, Australia, 1989 through 1993. Am J Public Health 88:759-64.

~~1275-1271.~~ Mortimer KM, Tager IB, Dockery DW, Neas LM, Redline S. 2000. The Effect of Ozone on Inner-City Children with Asthma. Identification of Susceptible Subgroups. Am J Respir Crit Care Med 162:1838-1845.

~~1276-1272.~~ Mullahy J, Portney PR. 1990. Air pollution, cigarette smoking, and the production of respiratory health. J Health Econ 9:193-205.

~~1277-1273.~~ Nauenberg E, Basu K. 1999. Effect of insurance coverage on the relationship between asthma hospitalizations and exposure to air pollution. Public Health Rep 114:135-48.

~~1278-1274.~~ Neas LM, Dockery DW, Koutrakis P, Speizer FE. 1999a. Fine particles and peak flow in children: acidity versus mass. Epidemiology 10:550-3.

~~1279-1275.~~ Neas LM, Schwartz J, Dockery D. 1999b. A case-crossover analysis of air pollution and ~~death-mortality~~ in Philadelphia. Environ Health Perspect 107:629-31.

~~1280-1276.~~ Nutman A, Solomon Y, Mendel S, Nutman J, Hines E, Topilsky M, Kivity S. 1998. The use of a neural network for studying the relationship between air pollution and asthma-related emergency room visits. Respir Med 92:1199-202.

~~1281-1277.~~ Ostro B. 1995. Fine particulate air pollution and ~~death-mortality~~ in two Southern California counties. Environ Res 70:98-104.

~~1282-1278.~~ Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. Epidemiology 12:200-8.

~~1283-1279.~~ Ostro B, Sanchez JM, Aranda C, Eskeland GS. 1996. Air pollution and ~~death-mortality~~: results from a study of Santiago, Chile. J Expo Anal Environ Epidemiol 6:97-114.

~~1284-1280.~~ Pereira LA, Loomis D, Conceicao GM, Braga AL, Arcas RM, Kishi HS, Singer JM, Bohm GM, Saldiva PH. 1998. Association between air pollution and intrauterine ~~death-mortality~~ in Sao Paulo, Brazil. Environ Health Perspect 106:325-9.

~~1285-1281.~~ Peters A, Skorkovsky J, Kotesovec F, Brynda J, Spix C, Wichmann HE, Heinrich J. 2000. Associations between ~~death-mortality~~ and air pollution

in central Europe. *Environ Health Perspect* 108:283-7.

~~1286-1282.~~ Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, Thomas DC. 1999a. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am J Respir Crit Care Med* 159:768-75.

~~1287-1283.~~ Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, Thomas DC. 1999b. A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory disease. *Am J Respir Crit Care Med* 159:760-7.

~~1288-1284.~~ Petroschevsky A, Simpson RW, Thalib L, Rutherford S. 2001. Associations between outdoor air pollution and hospital admissions in Brisbane, Australia. *Arch Environ Health* 56:37-52.

~~1289-1285.~~ Ponce de Leon A, Anderson HOUR, Bland JM, Strachan DP, Bower J. 1996. Effects of air pollution on daily hospital admissions for respiratory disease in London between 1987-88 and 1991-92. *J Epidemiol Community Health* 50 Suppl 1:s63-70.

~~1290-1286.~~ Ponka A, Savela M, Virtanen M. 1998. ~~Death~~Mortality and air pollution in Helsinki. *Arch Environ Health* 53:281-6.

1291. Ponka A, Virtanen M. 1996. Asthma and ambient air pollution in Helsinki. *J Epidemiol Community Health* 50 Suppl 1:s59-62.

1287.

~~1292-1288.~~ Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002. Lung cancer, cardiopulmonary ~~death~~ mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287:1132-41.

~~1293-1289.~~ Prescott GJ, Cohen GR, Elton RA, Fowkes FG, Agius RM. 1998. Urban air pollution and cardiopulmonary ill health: a 14.5 year time series study. *Occup Environ Med* 55:697-704.

~~1294-1290.~~ Ramadour M, Burel C, Lanteaume A, Vervloet D, Charpin D, Brisse F, Dutau H, Charpin D. 2000. Prevalence of asthma and rhinitis in relation to long-term exposure to gaseous air pollutants. *Allergy* 55:1163-9.

~~1295-1291.~~ Ritz B, Yu F, Chapa G, Fruin S. 2000. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology* 11:502-11.

~~1296-1292.~~ Roemer WH, van Wijnen JH. 2001. Daily ~~death~~mortality and air

pollution along busy streets in Amsterdam, 1987-1998. *Epidemiology* 12:649-53.

~~1297-1293.~~ Romieu I, Meneses F, Ruiz S, Huerta J, Siembra JJ, White M, Etzel R, Hernandez M. 1997. Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City-. *Arch Environ Health* 52:368-76.

~~1298-1294.~~ Romieu I, Meneses F, Ruiz S, Siembra JJ, Huerta J, White MC, Etzel RA. 1996. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *Am J Respir Crit Care Med* 154:300-7.

~~1299-1295.~~ Romieu I, Meneses F, Siembra-Monge JJ, Huerta J, Ruiz Velasco S, White MC, Etzel RA, Hernandez-Avila M. 1995. Effects of urban air pollutants on emergency visits for childhood asthma in Mexico City. *Am J Epidemiol* 141:546-53.

~~1300-1296.~~ Ross MA, Persky VW, Scheff PA, Chung J, Curtis L, Ramakrishnan V, Wadden RA, Hryhorczuk DO. 2002. Effect of ozone and aeroallergens on the respiratory health of asthmatics. *Arch Environ Health* 57:568-78.

~~1301-1297.~~ Saez M, Tobias A, Munoz P, Campbell MJ. 1999. A GEE moving average analysis of the relationship between air pollution and death mortality for asthma in Barcelona, Spain. *Stat Med* 18:2077-86.

~~1302-1298.~~ Saldiva PH, Lichtenfels AJ, Paiva PS, Barone IA, Martins MA, Massad E, Pereira JC, Xavier VP, Singer JM, Bohm GM. 1994. Association between air pollution and death-mortality due to respiratory diseases in children in Sao Paulo, Brazil: a preliminary report. *Environ Res* 65:218-25.

~~1303-1299.~~ Saldiva PH, Pope CA 3rd, Schwartz J, Dockery DW, Lichtenfels AJ, Salge JM, Barone I, Bohm GM. 1995. Air pollution and death-mortality in elderly people: a time-series study in Sao Paulo, Brazil. *Arch Environ Health* 50:159-63.

~~1304-1300.~~ Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. 2000. Fine particulate air pollution and death-mortality in 20 U.S. cities, 1987-1994. *N Engl J Med* 343:1742-9.

~~1305-1301.~~ Sartor F, Snacken R, Demuth C, Walckiers D. 1995. Temperature, ambient ozone levels, and death-mortality during summer 1994, in Belgium. *Environ Res* 70:105-13.

~~1306-1302.~~ Schmitzberger R, Rhomberg K, Buchele H, Puchegger R, Schmitzberger-Natzmer D, Kemmler G, Panosch B. 1993. Effects of air pollution on the respiratory tract of children. *Pediatr Pulmonol* 15:68-74.

~~1307-1303.~~ Schouten JP, Vonk JM, de Graaf A. 1996. Short term effects of air

pollution on emergency hospital admissions for respiratory disease: results of the APHEA project in two major cities in The Netherlands, 1977-89. *J Epidemiol Community Health* 50 Suppl 1:s22-9.

~~1308-1304.~~ Schwartz J. 1989. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. *Environ Res* 50:309-21.

~~1309-1305.~~ Schwartz J. 1991. Particulate air pollution and daily ~~death-mortality~~ in Detroit. *Environ Res* 56:204-13.

~~1310-1306.~~ Schwartz J. 1996. Air pollution and hospital admissions for respiratory disease. *Epidemiology* 7:20-8.

~~1311-1307.~~ Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 147:826-831.

~~1312-1308.~~ Schwartz J, Spix C, Touloumi G, Bacharova L, Barumamdzadeh T, le Tertre A, Piekarksi T, Ponce de Leon A, Ponka A, Rossi G, Saez M, Schouten JP. 1996. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Community Health* 50 Suppl 1:S3-11.

~~1313-1309.~~ Sheppard L, Levy D, Norris G, Larson TV, Koenig JQ. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology* 10:23-30.

~~1314-1310.~~ Simpson RW, Williams G, Petroeschovsky A, Morgan G, Rutherford S. 1997. Associations between outdoor air pollution and daily ~~death-mortality~~ in Brisbane, Australia. *Arch Environ Health* 52:442-54.

~~1315-1311.~~ Stern B, Jones L, Raizenne M, Burnett R, Meranger JC, Franklin CA. 1989. Respiratory health effects associated with ambient sulfates and ozone in two rural Canadian communities. *Environ Res* 49:20-39.

~~1316-1312.~~ Stern BR, Raizenne ME, Burnett RT, Jones L, Kearney J, Franklin CA. 1994. Air pollution and childhood respiratory health: exposure to sulfate and ozone in 10 Canadian rural communities. *Environ Res* 66: 125-42.

~~1317-1313.~~ Stieb DM, Burnett RT, Beveridge RC, Brook JR. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect* 104:1354-60.

~~1318-1314.~~ Sunyer J, Basagana X. 2001. Particles, and not gases, are associated with the risk of death in patients with chronic obstructive pulmonary disease. *Int J Epidemiol* 30:1138-40.

~~1319-1315.~~ Sunyer J, Castellsague J, Saez M, Tobias A, Anto JM. 1996. Air

pollution and ~~death~~-mortality in Barcelona. J Epidemiol Community Health 50 Suppl 1:s76-80.

~~1320~~-1316. Tager IB, Kunzli N, Ngo L, Balmes J. 1998. Methods development for epidemiologic investigations of the health effects of prolonged ozone exposure. Part I: Variability of pulmonary function measures. Res Rep Health Eff Inst :1-25; discussion 109-21.

~~1321~~-1317. Tellez-Rojo MM, Romieu I, Ruiz-Velasco S, Lezana MA, Hernandez-Avila MM. 2000. Daily respiratory ~~death~~-mortality and PM10 pollution in Mexico City: importance of considering place of death. Eur Respir J 16: 391-6.

~~1322~~-1318. Tenias JM, Ballester F, Perez-Hoyos S, Rivera ML. 2002. Air pollution and hospital emergency room admissions for chronic obstructive pulmonary disease in Valencia, Spain. Arch Environ Health 57:41-7.

~~1323~~-1319. Tenias JM, Ballester F, Rivera ML. 1998. Association between hospital emergency visits for asthma and air pollution in Valencia, Spain. Occup Environ Med 55:541-7.

~~1324~~-1320. Thompson AJ, Shields MD, Patterson CC. 2001. Acute asthma exacerbations and air pollutants in children living in Belfast, Northern Ireland. Arch Environ Health_ 56:234-41.

~~1325~~-1321. Tobias A, Campbell MJ, Saez M. 1999. Modelling asthma epidemics on the relationship between air pollution and asthma emergency visits in Barcelona, Spain. Eur J Epidemiol 15:799-803.

~~1326~~-1322. Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, White MC. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. Am J Epidemiol 151:798-810.

~~1327~~-1323. Touloumi G, Katsouyanni K, Zmirou D, Schwartz J, Spix C, de Leon AP, Tobias A, Quennel P, Rabczenko D, Bacharova L, Bisanti L, Vonk JM, Ponka A. 1997. Short-term effects of ambient oxidant exposure on ~~death~~ mortality: a combined analysis within the APHEA project. Air Pollution and Health: a European Approach. Am J Epidemiol 146:177-85.

~~1328~~-1324. US EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Vol. 3.

~~1329~~-1325. Vedal S, Brauer M, White R, Petkau J. 2003. Air pollution and daily ~~death~~-mortality in a city with low levels of pollution. Environ Health Perspect 111:45-52.

~~1330~~-1326. Verhoeff AP, Hoek G, Schwartz J, van Wijnen JH-. 1996. Air

pollution and daily ~~death~~-mortality in Amsterdam. *Epidemiology* 7:225-30.

~~1331-1327.~~ Weisel CP, Cody RP, Georgopoulos PG, Purushothaman V, Weiss SH, Bielory L, Gregory P, Stern AH. 2002. Concepts in developing health-based indicators for ozone. *Int Arch Occup Environ Health* 75:415-22.

~~1332-1328.~~ Weisel CP, Cody RP, Lioy PJ. 1995. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. *Environ Health Perspect* 103 Suppl 2:97-102.

~~1333-1329.~~ White MC, Etzel RA, Wilcox WD, Lloyd C. 1994. Exacerbations of childhood asthma and ozone pollution in Atlanta. *Environ Res* 65:56-68.

~~1334-1330.~~ Wong TW, Lau TS, Yu TS, Neller A, Wong SL, Tam W, Pang SW. 1999. Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong. *Occup Environ Med* 56:679-83.

~~1335-1331.~~ Zmirou D, Barumandzadeh T, Balducci F, Ritter P, Laham G, Ghilardi JP. 1996. Short term effects of air pollution on ~~death~~-mortality in the city of Lyon, France, 1985-90. *J Epidemiol Community Health* 50 Suppl 1:S30-5.

~~1336-1332.~~ Zmirou D, Schwartz J, Saez M, Zanobetti A, Wojtyniak B, Touloumi G, Spix C, Ponce de Leon A, Le Moullec Y, Bacharova L, Schouten J, Ponka A, Katsouyanni K. 1998. Time-series analysis of air pollution and cause-specific-~~death~~ mortality. *Epidemiology* 9:495-503.

~~1337-1333.~~ Zwick H, Popp W, Wagner C, Reiser K, Schmogger J, Bock A, Herkner K, Radunsky K. 1991. Effects of ozone on the respiratory health, allergic sensitization, and cellular immune system in children. *Am Rev Respir Dis* 144:1075-9.

Chapter 11

~~1338-1334.~~ Abbey DE, Nishino N, McDonnell WF, Burchette RJ, Knutsen SF, Lawrence Beeson W, Yang JX. 1999. Long-term inhalable particles and other air pollutants related to ~~death~~-mortality in nonsmokers. *Am J Respir Crit Care Med* 159:373-82.

~~1339-1335.~~ Adams WC. 1998. Dose-response effects of varied equivalent minute ventilation rates on pulmonary function responses during exposure to ozone. Final Report to the American Petroleum Institute. Washington, D. C.

- ~~1340~~-1336. Adams WC. 2002. Comparison of chamber and face-mask 6.6-hour exposures to ozone on pulmonary function and symptoms responses. *Inhal Toxicol* 14:745-64.
- ~~1341~~-1337. Adams WC. 2003. Comparison of chamber and face mask 6.6-hour exposure to 0.08 ppm ozone via square-wave and triangular profiles on pulmonary responses. *Inhal Toxicol* 15:265-81.
- ~~1342~~-1338. American Thoracic Society. 1985. Guidelines as to what constitutes an adverse respiratory health effect, with special reference to epidemiologic studies of air pollution. 131. *131:666-8*.
- ~~1343~~-1339. American Thoracic Society. 2000. What constitutes an adverse health effect of air pollution? 161. *161:665-73*.
- ~~1344~~-1340. Anderson HOUR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Ponka A, Bacharova L, Schwartz J, Katsouyanni K. 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *Eur Respir J* 10:1064-71.
- ~~1345~~-1341. Bates DV (1992) Health indices of the adverse effects of air pollution: the question of coherence. *Environ Res* 43:217-31.
- ~~1346~~-1342. Bell M, McDermott A, Zeger S, Samet J, Dominici F. 2004. Ozone and short-term ~~death~~-mortality in 95 US urban communities, 1987-2000. *JAMA* 292, 19:2372-2378.
- ~~1347~~-1343. Brauer M, Blair J, Vedal S. 1996. Effect of ambient ozone exposure on lung function in farm workers. *Am J Respir Crit Care Med* 154:981-7.
- ~~1348~~-1344. Burnett RT, Cakmak S, Brook JR, Krewski D. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ Health Perspect* 105:614-20.
- ~~1349~~-1345. California Department of Health Services. 1987. Recommendation for an Ambient Air Quality Standard for Ozone.
- ~~1350~~-1346. Catalano PJ, Rogus J, Ryan LM. 1995. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part X: Robust composite scores based on median polish analysis. *Res Rep Health Eff Inst* :1-57; discussion 59-64.
- ~~1351~~-1347. Dominici F. 2003. Shape of the Exposure-Response Relation and ~~Death~~-Mortality Displacement in the NMMAPS Database. Health Effects Institute Special Report. Health Effects Institute Special Report. 91-6.

- ~~1352~~.1348. Drechsler-Parks DM, Bedi JF, Horvath SM. 1987. Pulmonary function responses of older men and women to ozone exposure. *Exp Gerontol* 22:91-101.
- ~~1353~~.1349. Drechsler-Parks DM, Bedi JF, Horvath SM. 1989. Pulmonary function responses of young and older adults to mixtures of O₃, NO₂ and PAN-. *Toxicol Ind Health* 5:505-17.
- ~~1354~~.1350. Emmons K, Foster WM. 1991. Smoking cessation and acute airway response to ozone. *Arch Environ Health* 46:288-95.
- ~~1355~~.1351. Evans MJ, Fanucchi MV, Baker GL, Van Winkle LS, Pantle LM, Nishio SJ, Schelegle ES, Gershwin LJ, Miller LA, Hyde DM, Sannes PL, Plopper CG. 2003. Atypical development of the tracheal basement membrane zone of infant rhesus monkeys exposed to ozone and allergen. *Am J Physiol Lung Cell Mol Physiol* 285:L931-9.
- ~~1356~~.1352. Folinsbee LJ, Horstman DH, Kehrl HOUR, McDonnell WF, Gerrity TR, Seal. E. , Larson R, Hazucha MJ, Abdul-Salaam S, Faucette B, Ives PJ. 1991. Effects of single and repeated prolonged low-level ozone exposure in man. Presented at the annual meeting of the Society for Occupational and Environmental Health, March, Washington, DC.
- ~~1357~~.1353. Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ Health Perspect* 107:675-9.
- ~~1358~~.1354. Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J. 2000. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med* 162:1383-90.
- ~~1359~~.1355. Gauderman WJ, Gilliland GF, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. 2002. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med* 166:76-84.
- ~~1360~~.1356. Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, Leaderer BP. 2003. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* 290:1859-67.
- ~~1361~~.1357. Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 12:43-54.
- ~~1362~~.1358. Goldberg MS, Burnett RT, Valois MF, Flegel K, Bailar JC 3rd,

Brook J, Vincent R, Radon K. 2003. Associations between ambient air pollution and daily ~~death~~-mortality among persons with congestive heart failure. Environ Res 91 :8-20.

~~1363~~-1359. Goldberg MS, Burnett RT. 2003. Revised analysis of the Montreal time-series study. Health Effects Institute Special Report. Health Effects Institute Special Report 113-32.

~~1364~~-1360. Gong H Jr, Bradley PW, Simmons MS, Tashkin DP. 1986. Impaired exercise performance and pulmonary function in elite cyclists during low-level ozone exposure in a hot environment. Am Rev Respir Dis 134:726-33.

~~1365~~-1361. Gong H Jr, Wong R, Sarma RJ, Linn WS, Sullivan ED, Shamoo DA, Anderson KR, Prasad SB. 1998 . Cardiovascular effects of ozone exposure in human volunteers. Am J Respir Crit Care Med 158:538-46.

~~1366~~-1362. Gryparis A, Forsberg B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Samoli E, Medina S, Anderson HOUR, Niciu EM, Wichmann HE, Kriz B, Kosnik M, Skorkovsky J, Vonk JM, Dortbudak Z. 2004. Acute effects of ozone on ~~death~~-mortality from the "air pollution and health: a European approach" project. Am J Respir Crit Care Med. 170:1080-7.

~~1367~~-1363. Harkema JR, Plopper CG, Hyde DM, St George JA, Wilson DW, Dungworth DL. 1993. Response of macaque bronchiolar epithelium to ambient concentrations of ozone. Am J Pathol 143:857-66.

~~1368~~-1364. Hazucha MJ, Folinsbee LJ, Seal E Jr. 1992. Effects of steady-state and variable ozone concentration profiles on pulmonary function. Am Rev Respir Dis 146:1487-93.

~~1369~~-1365. Hoek G, Brunekreef B, Verhoeff A, van Wijnen J, Fischer P. 2000. Daily ~~death~~-mortality and air pollution in The Netherlands. J Air Waste Manag Assoc 50:1380-9.

~~1370~~-1366. Horstman DH, Folinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF. 1990. Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. Am Rev Respir Dis 142:1158-63.

~~1371~~-1367. Kinney PL, Lippmann M. 2000. Respiratory effects of seasonal exposures to ozone and particles. Arch Environ Health 55:210-6.

~~1372~~-1368. Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, El-Fawal H, Devlin RB, Rom WN. 1996. Biomarkers of lung inflammation in recreational joggers exposed to ozone. Am J Respir Crit Care Med 154:1430-5.

~~1373~~-1369. Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. 1997.

Association between lifetime ambient ozone exposure and pulmonary function in college freshmen--results of a pilot study. *Environ Res* 72:8-23.

~~1374~~.1370. Larson SD, Schelegle ES, Walby WF, Gershwin LJ, Fanuccihi MV, Evans MJ, Joad JP, Tarkington BK, Hyde DM, Plopper CG. 2004. Postnatal remodeling of the neural components of the epithelial-mesenchymal trophic unit in the proximal airways of infant rhesus monkeys exposed to ozone and allergen. *Toxicol Appl Pharmacol* 194:211-20.

~~1375~~.1371. Last JA, Gelzleichter TR, Harkema J, Hawk S. 1994. Consequences of prolonged inhalation of ozone on Fischer-344/N rats: collaborative studies. Part I: Content and cross-linking of lung collagen. *Res Rep Health Eff Inst* 1:1-29; discussion 31-40.

~~1376~~.1372. Levy JI, Carrothers TJ, Tuomisto JT, Hammitt JK, Evans JS. 2001. Assessing the public health benefits of reduced ozone concentrations. *Environ Health Perspect* 109:1215-26.

~~1377~~.1373. Linn WS, Shamoo DA, Anderson KR, Peng R-C, Avol EL, Hackney JD, Gong H Jr. 1996. Short-term air pollution exposure and responses in Los Angeles area schoolchildren. *J Exp Anal Environ Epidemiol* 6:449-471.

~~1378~~.1374. McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386-91.

~~1379~~.1375. McDonnell WF, Horstman DH, Hazucha MJ, Seal E Jr, Haak ED, Salaam SA, House DE. 1983. Pulmonary effects of ozone exposure during exercise: dose-response characteristics. *J Appl Physiol* 54:1345-52.

~~1380~~.1376. McDonnell WF, Kehrl HOUR, Abdul-Salaam S, Ives PJ, Folinsbee LJ, Devlin RB, O'Neil JJ, Horstman DH. 1991. Respiratory response of humans exposed to low levels of ozone for 6.6 hours. *Arch Environ Health* 46: 145-50.

~~1381~~.1377. McDonnell WF 3rd, Chapman RS, Leigh MW, Strobe GL, Collier AM. 1985a. Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am Rev Respir Dis* 132:875-9.

~~1382~~.1378. McDonnell WF 3rd, Horstman DH, Abdul-Salaam S, House DE. 1985b. Reproducibility of individual responses to ozone exposure. *Am Rev Respir Dis* 131:36-40.

~~1383~~.1379. Moolgavkar SH, Luebeck EG, Anderson EL. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. *Epidemiology* 8:364-70.

~~1384~~.1380. National Asthma Education and Prevention Program . 2002.

National Asthma Education and Prevention Program Expert Panel Report: guidelines for the diagnosis and management of asthma update on selected topics. 110. 110((5 pt 2)): S141-219.

~~1385~~-1381. Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, Thomas DC. 1999. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am J Respir Crit Care Med* 159:768-75.

~~1386~~-1382. Pinkerton KE, Menache MG, Plopper CG. 1995. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part IX: Changes in the tracheobronchial epithelium, pulmonary acinus, and lung antioxidant enzyme activity. *Res Rep Health Eff Inst* :41-98; discussion 99-110.

~~1387~~-1383. Pinkerton KE, Weller BL, Menache MG, Plopper CG. 1998. Consequences of prolonged inhalation of ozone on F344/N rats: collaborative studies. Part XIII. A comparison of changes in the tracheobronchial epithelium and pulmonary acinus in male rats at 3 and 20 months. *Res Rep Health Eff Inst* :1-32; discussion 33-7.

~~1388~~-1384. Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002a. Lung cancer, cardiopulmonary ~~death~~ mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287:1132-41.

~~1389~~-1385. Reiser KM, Tyler WS, Hennessy SM, Dominguez JJ, Last JA. 1987. Long-term consequences of exposure to ozone. II. Structural alterations in lung collagen of monkeys. *Toxicol Appl Pharmacol* 89:314-22.

~~1390~~-1386. Sarnat JA, Schwartz J, Catalano PJ, Suh HH. 2001. Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? *Environ Health Perspect* 109:1053-61.

~~1391~~-1387. Schelegle ES, Miller LA, Gershwin LJ, Fanucchi MV, Van Winkle LS, Gerriets JE, Walby WF, Mitchell V, Tarkington BK, Wong VJ, Baker GL, Pantle LM, Joad JP, Pinkerton KE, Wu R, Evans MJ, Hyde DM, Plopper CG. 2003a. Repeated episodes of ozone inhalation amplifies the effects of allergen sensitization and inhalation on airway immune and structural development in Rhesus monkeys. *Toxicol Appl Pharmacol* 191:74-85.

~~1392~~-1388. Seal E Jr, McDonnell WF, House DE, Salaam SA, Dewitt PJ, Butler SO, Green J, Raggio L. 1993. The pulmonary response of white and black adults to six concentrations of ozone. *Am Rev Respir Dis* 147:804-10.

~~1393~~-1389. Sherwin R. 1983. What is an adverse health effect? *Environ Health Perspect* 52:177-182.

~~1394-1390.~~ Simpson RW, Williams G, Petroeschovsky A, Morgan G, Rutherford S. 1997. Associations between outdoor air pollution and daily death-mortality in Brisbane, Australia. Arch Environ Health 52:442-54.

~~1395-1391.~~ Szarek JL, Stewart NL, Zhang JZ, Webb JA, Valentovic MA, Catalano P. 1995. Contractile responses and structure of small bronchi isolated from rats after 20 months' exposure to ozone. Fundam Appl Toxicol 28:199-208.

~~1396-1392.~~ Tager IB, Kunzli N, Lurmann F, Ngo L, Segal M, Balmes J. 1998. Methods development for epidemiologic investigations of the health effects of prolonged ozone exposure. Part II. An approach to retrospective estimation of lifetime ozone exposure using a questionnaire and ambient monitoring data (California sites). Res Rep Health Eff Inst 27:78; discussion 109-21.

~~1397-1393.~~ Thurston GD, Ito K. 2001. Epidemiological studies of acute ozone exposures and death-mortality. J Expo Anal Environ Epidemiol 11: 286-94.

~~1398-1394.~~ Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, White MC. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. Am J Epidemiol 151:798-810.

~~1399-1395.~~ Tyler WS, Tyler NK, Last JA, Gillespie MJ, Barstow TJ. 1988. Comparison of daily and seasonal exposures of young monkeys to ozone. Toxicology 50:131-44.

~~1400-1396.~~ Vedal S, Brauer M, White R, Petkau J. 2003. Air pollution and daily death-mortality in a city with low levels of pollution. Environ Health Perspect 111:45-52.

~~1401-1397.~~ Weisel CP, Cody RP, Liroy PJ. 1995. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. Environ Health Perspect 103 Suppl 2:97-102.

Appendix B

~~1402-1398.~~ ARB (2004) Aerometric Data Analysis and Management System (ADAM) <http://www.arb.ca.gov/adam/welcome.html>

~~1403-1399.~~ Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. 2004. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (~~-ozone~~ O_3). Report of a WHO task group. World Health Organization. (<http://www.euro.who.int/document/e82792.pdf>)

- ~~1404~~.1400. Anderson HR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Ponka A, Bacharova L, Schwartz J, Katsouyanni K. 1997. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *Eur Respir J* 10:1064-71.
- ~~1405~~.Bates, DV. 2005. ~~Personal statement made at Air Quality Advisory Committee Meeting, January 12, 2005.~~
- ~~1406~~.1401. Bell M, McDermott A, Zeger S, Samet J, Dominici F. 2004. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *JAMA* 292, 19:2372-2378.
- ~~1407~~.1402. Burnett RT, Brook JR, Yung WT, Dales RE, Krewski D. 1997. Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environ Res* 72:24-31.
- ~~1408~~.1403. Burnett RT, Dales RE, Raizenne ME, Krewski D, Summers PW, Roberts GR, Raad-Young M, Dann T, Brook J. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. *Environ Res* 65:172-94.
- ~~1409~~.1404. Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, Leech JA, Cakmak S, Krewski D. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 153:444-52.
- ~~1410~~.1405. Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG. 2001. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA* 285:897-905.
- ~~1411~~.1406. Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, London SJ, Margolis HG, McConnell R, Islam KT, Peters JM. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 12:43-54.
- ~~1412~~.1407. Gryparis A, Forsberg, B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, Somoli, E, Medina S, Anderson R, Niciu, E, Wichmann H, Kriz B, Kosnik M, Skordovsky J, Vonk J, Dorbudak Z. 2004. Acute effects of ozone on mortality from the "Air Pollution and Health: A European Approach" project. *Am J Respir Crit Care Med* 170: 1080-1087.
- ~~1413~~.1408. Hall JV, Brajer V, Lurmann FW. 2003. Economic valuation of ozone-related school absences in the south coast air basin of California.

Contemporary Economic Policy 21:407-417.

- ~~1414-1409.~~ Hubbell, BJ, Halberg A, McCubbin, DR, Post, E. 2005. Health-related benefits of attaining the 8-hr ozone standard. *Environ Health Perspect* 113:73-82.
- ~~1415-1410.~~ Jaffe DH, Singer ME, Rimm AA. 2003. Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. *Environ Res* 91:21-8.
- ~~1416-1411.~~ Levy JI, Carrothers TJ, Tuomisto JT, Hammitt JK, Evans JS. 2001. Assessing the public health benefits of reduced ozone concentrations. *Environ Health Perspect* 109:1215-26.
- ~~1417-1412.~~ National Research Council. 2002. Estimating the public health benefits of proposed air pollution regulations. Washington, D.C.: National Academy Press.
- ~~1418-1413.~~ Ostro BD, Rothschild S. 1989. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. *Environ Res* 50: 238-47.
- ~~1419-1414.~~ Romieu I, Meneses F, Sienna-Monge JJ, Huerta J, Ruiz Velasco S, White MC, Etzel RA, Hernandez-Avila M. 1995. Effects of urban air pollutants on emergency visits for childhood asthma in Mexico City. *Am J Epidemiol* 141:546-53.
- ~~1420-1415.~~ Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW *et al.* (2000). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. *Health Effects- Institute* (94 Pt 2).
- ~~1421-1416.~~ Schwartz J. 1997 Health effects of air pollution from traffic: ozone and particulate matter. In: Fletcher T and McMichael AJ, eds. Health at the crossroads: transport policy and urban health. New York: John Wiley. Chapter 6.
- ~~1422-1417.~~ Stieb DM, Burnett RT, Beveridge RC, Brook JR. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect* 104:1354-60.
- ~~1423-1418.~~ Stieb DM, Judek S, Burnett RT. 2003. Meta-analysis of time-series studies of air pollution and mortality: update in relation to the use of generalized additive models. *J Air Waste Manag Assoc* 53:258-261.
- ~~1424-1419.~~ Thurston, G.T. and Ito, K. 1999. Epidemiologic studies of ozone exposure effects. In: Holgate ST, Samet JM, Koren HS, Maynard RL, eds. Air Pollution and Health. (Holgate ST, Samet JM, Koren Hs, Maynard RL,

eds.) London: Academic Press.

- ~~1425-1420.~~ Thurston GD, Ito K. 2001. Epidemiological studies of acute ozone exposures and mortality. *J Expo Anal Environ Epidemiol* 11: 286-94.
- ~~1426-1421.~~ Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res* 65: 271-90.
- ~~1427-1422.~~ Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine OJ, Carlin BP, Klein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, White MC. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *Am J Epidemiol* 151:798-810.
- ~~1428-~~U.S. Environmental Protection Agency. 1999. The benefits and costs of the clean air act 1990 to 2010: EPA report to Congress. Washington, D.C.: Office of
- ~~1429-1423.~~ Air and Radiation and Office of Policy. Report No.: EPA-410-R-99-001, November. (<http://www.epa.gov/air/sect812/copy99.html>).
- ~~1430-~~U.S. Environmental Protection Agency. 2004. Advisory on plans for health effects analysis in the analytical plan for EPA's second prospective analysis benefits and costs of the clean air act, 1990-2020; advisory by the health effects subcommittee of the advisory council on clean air compliance analysis. Washington, D.C. Report No.: EPA-SAB-COUNCIL-ADV-04-002 Environmental.
- ~~1431-1424.~~ (http://www.epa.gov/sab/pdf/council_adv_04002.pdf)

Appendix G

- ~~1432-1425.~~ Abraham WM, Januszkiewicz AJ, Mingle M, Welker M, Wanner A, Sackner MA (1980 May). Sensitivity of bronchoprovocation and tracheal mucous velocity in detecting airway responses to O₃. *J Appl Physiol* 48(5):789-93.
- ~~1433-1426.~~ Abraham WM, Sielczak MW, Delehunt JC, Marchette B, Wanner A (1986 Feb). Impairment of tracheal mucociliary clearance but not ciliary beat frequency by a combination of low level ozone and sulfur dioxide in sheep. *Eur J Respir Dis* 68(2):114-20.
- ~~1434-1427.~~ Adamson IY, Vincent R, Bjarnason SG (1999 May). Cell injury and interstitial inflammation in rat lung after inhalation of ozone and urban particulates. *Am J Respir Cell Mol Biol* 20(5):1067-72.
- ~~1435-1428.~~ Allegra L, Abraham WMCGA, Wanner A (1983). Targets of allergic airway challenge and tracheobronchial irritation with ozone in an animal model (sheep). *European Journal of Respiratory Diseases* 64 (suppl

- 126):45-52.
- ~~1436~~.1429. Aranyi C, Vana SC, Thomas PT, Bradof JN, Fenters JD, Graham JA, et al. (1983 Jul). Effects of subchronic exposure to a mixture of O₃, SO₂, and (NH₄)₂SO₄ on host defenses of mice. *J Toxicol Environ Health* 12(1):55-71.
- ~~1437~~.1430. ARB (1987). Effects of Ozone on Health. Technical Support Document. Prepared by the California Air Resources Board. September.
- ~~1438~~.1431. Arito H, Uchiyama I, Arakawa H, Yokoyama E (1990 Jul). Ozone-induced bradycardia and arrhythmia and their relation to sleep- wakefulness in rats. *Toxicol Lett* 52(2):169-78.
- ~~1439~~.1432. Arito H, Uchiyama I, Yokoyama E (1992). Acute effects of ozone on EEG activity, sleep-wakefulness and heart rate in rats. *Ind Health* 30(1):23-34.
- ~~1440~~.1433. Armstrong LC, Watkins K, Pinkerton KE, Last JA (1994 Jul). Collagen mRNA content and distribution in the lungs of rats exposed to ozone. *Am J Respir Cell Mol Biol* 11(1):25-34.
- ~~1441~~.1434. Barry BE, Mercer RR, Miller FJ, Crapo JD (1988). Effects of inhalation of 0.25 ppm ozone on the terminal bronchioles of juvenile and adult rats. *Exp Lung Res* 14(2):225-45.
- ~~1442~~.1435. Barry BE, Miller FJ, Crapo JD (1985 Dec). Effects of inhalation of 0.12 and 0.25 parts per million ozone on the proximal alveolar region of juvenile and adult rats. *Lab Invest* 53(6):692-704.
- ~~1443~~.1436. Bassett DJ, Bowen-Kelly E, Elbon CL, Reichenbaugh SS (1988). Rat lung recovery from 3 days of continuous exposure to 0.75 ppm ozone. *J Toxicol Environ Health* 25(3):329-47.
- ~~1444~~.1437. Becker S, Quay J, Koren HS (1991 Nov). Effect of ozone on immunoglobulin production by human B cells in vitro. *J Toxicol Environ Health* 34(3):353-66.
- ~~1445~~.1438. Ben-Jebria A, Hu SC, Kitzmiller EL, Ultman JS (1991 Dec). Ozone absorption into excised porcine and sheep tracheae by a bolus- response method. *Environ Res* 56(2):144-57.
- ~~1446~~.1439. Bermudez E, Ferng SF, Castro CE, Mustafa MG (1999 Jul). DNA strand breaks caused by exposure to ozone and nitrogen dioxide. *Environ Res* 81(1):72-80.
- ~~1447~~.1440. Bhalla DK (1996 Dec). Alteration of alveolar macrophage chemotaxis, cell adhesion, and cell adhesion molecules following ozone exposure of rats. *J Cell Physiol* 169(3):429-38.
- ~~1448~~.1441. Bhalla DK (1999 Jan-1999 Mar). Ozone-induced lung inflammation and mucosal barrier disruption: toxicology, mechanisms, and implications. *J Toxicol Environ Health B Crit Rev* 2(1):31-86.

- 1449-1442. Bhalla DK, Crocker TT (1986 Sep). Tracheal permeability in rats exposed to ozone. An electron microscopic and autoradiographic analysis of the transport pathway. *Am Rev Respir Dis* 134(3):572-9.
- 1450-1443. Bhalla DK, Crocker TT (1987). Pulmonary epithelial permeability in rats exposed to O₃. *J Toxicol Environ Health* 21(1-2):73-87.
- 1451-1444. Bhalla DK, Hoffman L (1997). Time course of airway epithelial and inflammatory changes in rats exposed to moderate levels of ozone. *Inhalation Toxicology* 9:829-42.
- 1452-1445. Bhalla DK, Mannix RC, Kleinman MT, Crocker TT (1986). Relative permeability of nasal, tracheal, and bronchoalveolar mucosa to macromolecules in rats exposed to ozone. *J Toxicol Environ Health* 17(2-3):269-83.
- 1453-1446. Bhalla DK, Mannix RC, Lavan SM, Phalen RF, Kleinman MT, Crocker TT (1987). Tracheal and bronchoalveolar permeability changes in rats inhaling oxidant atmospheres during rest or exercise. *J Toxicol Environ Health* 22(4):417-37.
- 1454-1447. Bhalla DK, Rasmussen RE, Daniels DS (1993 Dec). Adhesion and motility of polymorphonuclear leukocytes isolated from the blood of rats exposed to ozone: potential biomarkers of toxicity. *Toxicol Appl Pharmacol* 123(2):177-86.
- 1455-1448. Bignami G (1996 Apr). Economical test methods for developmental neurobehavioral toxicity. *Environ Health Perspect* 104 Suppl 2:285-98.
- 1456-1449. Bignami G (1996 Apr). Economical test methods for developmental neurobehavioral toxicity. *Environ Health Perspect* 104 Suppl 2:285-98.
- 1457-1450. Bignami G, Musi B, Dell'Omo G, Laviola G, Alleva E (1994 Dec). Limited effects of ozone exposure during pregnancy on physical and neurobehavioral development of CD-1 mice. *Toxicol Appl Pharmacol* 129(2):264-71.
- 1458-1451. Bleavins MR, Dziedzic D (1990 Aug). An immunofluorescence study of T and B lymphocytes in ozone-induced pulmonary lesions in the mouse. *Toxicol Appl Pharmacol* 105(1):93-102.
- 1459-1452. Boehme DS, Hotchkiss JA, Henderson RF (1992 Feb). Glutathione and GSH-dependent enzymes in bronchoalveolar lavage fluid cells in response to ozone. *Exp Mol Pathol* 56(1):37-48.
- 1460-1453. Bolarin DM, Bhalla DK, Kleinman MT (1997). Effects of repeated exposures of geriatric rats to ozone and particle-containing atmospheres: an analysis of bronchoalveolar lavage and plasma proteins. *Inhalation Toxicology* 9:423-34.
- 1461-1454. Boorman GA, Hailey R, Grumbein S, Chou BJ, Herbert RA, Goehl T, et al. (1994 Sep-1994 Oct). Toxicology and carcinogenesis studies of ozone and ozone 4-(N-nitrosomethylamino)-1-(3-pyridyl)-1-butanone in

- Fischer-344/N rats. *Toxicol Pathol* 22(5):545-54.
- ~~1462-1455.~~ Boorman GA, Sills RC, Grumbein S, Hailey R, Miller RA, Herbert RA (1995 Dec). Long-term toxicity studies of ozone in F344/N rats and B6C3F1 mice. *Toxicol Lett* 82-83:301-6.
- ~~1463-1456.~~ Borek C, Ong A, Zaider M (1989 Aug). Ozone activates transforming genes in vitro and acts as a synergistic co-carcinogen with gamma-rays only if delivered after radiation. *Carcinogenesis* 10(8):1549-51.
- ~~1464-1457.~~ Borek C, Zaider M, Ong A, Mason H, Witz G (1986 Sep). Ozone acts alone and synergistically with ionizing radiation to induce in vitro neoplastic transformation. *Carcinogenesis* 7(9):1611-3.
- ~~1465-1458.~~ Bornholdt J, Dybdahl M, Vogel U, Hansen M, Loft S, Wallin H (2002 Sep). Inhalation of ozone induces DNA strand breaks and inflammation in mice. *Mutat Res* 520(1-2):63-71.
- ~~1466-1459.~~ Bouthillier L, Vincent R, Goegan P, Adamson IY, Bjarnason S, Stewart M, et al. (1998 Dec). Acute effects of inhaled urban particles and ozone: lung morphology, macrophage activity, and plasma endothelin-1. *Am J Pathol* 153(6):1873-84.
- ~~1467-1460.~~ Burleson GR, Keyes LL, Stutzman JD (1989). Immunosuppression of pulmonary natural killer activity by exposure to ozone. *Immunopharmacol Immunotoxicol* 11(4):715-35.
- ~~1468-1461.~~ Calderon-Garciduenas L, Gambling TM, Acuna H, Garcia R, Osnaya N, Monroy S, et al. (2001b Jun). Canines as sentinel species for assessing chronic exposures to air pollutants: part 2. Cardiac pathology. *Toxicol Sci* 61(2):356-67.
- ~~1469-1462.~~ Calderon-Garciduenas L, Mora-Tiscareno A, Fordham LA, Chung CJ, Garcia R, Osnaya N, et al. (2001a Jun). Canines as sentinel species for assessing chronic exposures to air pollutants: part 1. Respiratory pathology. *Toxicol Sci* 61(2):342-55.
- ~~1470-1463.~~ Campen MJ, Norwood J, McKee JL, Mebane R, Hatch GE, Watkinson WP (2000). Ozone-induced hypothermia and bradycardia in rats and guinea pigs in nose-only or whole-body inhalation systems. *Journal of Thermal Biology* 25:81-9.
- ~~1471-1464.~~ Canning BJ, Hmieleski RR, Spannhake EW, Jakab GJ (1991 Oct). Ozone reduces murine alveolar and peritoneal macrophage phagocytosis: the role of prostanoids. *Am J Physiol* 261(4 Pt 1):L277-82.
- ~~1472-1465.~~ Cassee FR, Boere AJ, Bos J, Fokkens PH, Dormans JA, van Loveren H (2002 Jul). Effects of diesel exhaust enriched concentrated PM2.5 in ozone preexposed or monocrotaline-treated rats. *Inhal Toxicol* 14(7):721-43.
- ~~1473-1466.~~ Chang LY, Huang Y, Stockstill BL, Graham JA, Grose EC, Menache MG, et al. (1992 Aug). Epithelial injury and interstitial fibrosis in

- the proximal alveolar regions of rats chronically exposed to a simulated pattern of urban ambient ozone. *Toxicol Appl Pharmacol* 115(2):241-52.
- ~~1474-1467.~~ Chang MM, Wu R, Plopper CG, Hyde DM (1998 Sep). IL-8 is one of the major chemokines produced by monkey airway epithelium after ozone-induced injury. *Am J Physiol* 275(3 Pt 1):L524-32.
- ~~1475-1468.~~ Cheek JM, Buckpitt AR, Li C, Tarkington BK, Plopper CG (1994 Mar). Ozone injury to alveolar epithelium in vitro does not reflect loss of antioxidant defenses. *Toxicol Appl Pharmacol* 125(1):59-69.
- ~~1476-1469.~~ Chen LC, Miller PD, Lam HF, Guty J, Amdur MO (1991 Nov). Sulfuric acid-layered ultrafine particles potentiate ozone-induced airway injury. *J Toxicol Environ Health* 34(3):337-52.
- ~~1477-1470.~~ Chen LC, Qu Q, Amdur MO, Schlesinger RB (1995 Jan-1995 Feb). Alteration of pulmonary macrophage intracellular pH following inhalation exposure to sulfuric acid/ozone mixtures. *Exp Lung Res* 21(1):113-28.
- ~~1478-1471.~~ Cheng PW, Boat TF, Shaikh S, Wang OL, Hu PC, Costa DL (1995 May-1995 Jun). Differential effects of ozone on lung epithelial lining fluid volume and protein content. *Exp Lung Res* 21(3):351-65.
- ~~1479-1472.~~ Chow CK, Kaneko JJ (1979 Jun). Influence of dietary vitamin E on the red cells of ozone-exposed rats. *Environ Res* 19(1):49-55.
- ~~1480-1473.~~ Christman CA, Schwartz LW (1982 Aug). Enhanced phagocytosis by alveolar macrophages induced by short-term ozone insult. *Environ Res* 28(2):241-50.
- ~~1481-1474.~~ Churg A, Brauer M, Keeling B (1996 Apr). Ozone enhances the uptake of mineral particles by tracheobronchial epithelial cells in organ culture. *Am J Respir Crit Care Med* 153(4 Pt 1):1230-3.
- ~~1482-1475.~~ Cohen Hubal EA, Kimbell JS, Fedkiw PS (1996). Incorporation of nasal-lining mass-transfer resistance into a CFD model for prediction of ozone dosimetry in the upper respiratory tract. *Inhalation Toxicology* 8:831-57.
- ~~1483-1476.~~ Cohen MD, Sisco M, Baker K, Bowser D, Chen LC, Schlesinger RB (2003 Jan). Impact of coexposure to ozone on the carcinogenic potential of inhaled chromium. 1. effects on retention and on extra- and intracellular distribution. *J Toxicol Environ Health A* 66(1):39-55.
- ~~1484-1477.~~ Cohen MD, Sisco M, Baker K, Li Y, Lawrence D, van Loveren H, et al. (2002 Jun). Effects of inhaled ozone on pulmonary immune cells critical to antibacterial responses in situ. *Inhal Toxicol* 14(6):599-619.
- ~~1485-1478.~~ Cohen MD, Sisco M, Li Y, Zelikoff JT, Schlesinger RB (2001 Mar). Ozone-induced modulation of cell-mediated immune responses in the lungs. *Toxicol Appl Pharmacol* 171(2):71-84.
- ~~1486-1479.~~ Cohen MD, Zelikoff JT, Chen L-C, Schlesinger RB (1997). Pulmonary retention and distribution of inhaled chromium: effects of particle

- solubility and coexposure to ozone. *Inhalation Toxicology* 9:843-65.
- ~~1487-1480.~~ Cohen MD, Zelikoff JT, Chen LC, Schlesinger RB (1998 Sep). Immunotoxicologic effects of inhaled chromium: role of particle solubility and co-exposure to ozone. *Toxicol Appl Pharmacol* 152(1):30-40.
- ~~1488-1481.~~ Creutzenberg O, Bellmann B, Klingebiel R, Heinrich U, Muhle H (1995 May). Phagocytosis and chemotaxis of rat alveolar macrophages after a combined or separate exposure to ozone and ARB on black. *Exp Toxicol Pathol* 47(2-3):202-6.
- ~~1489-1482.~~ Dell'Omo G, Fiore M, Petruzzi S, Alleva E, Bignami G (1995a). Neurobehavioral development of CD-1 mice after combined gestational and postnatal exposure to ozone. *Arch Toxicol* 69(9):608-16.
- ~~1490-1483.~~ Dell'Omo G, Wolfer D, Alleva E, Lipp HP (1995b Nov). Developmental exposure to ozone induces subtle changes in swimming navigation of adult mice. *Toxicol Lett* 81(2-3):91-9.
- ~~1491-1484.~~ DeLucia AJ, Adams WC (1977 Jul). Effects of O₃ inhalation during exercise on pulmonary function and blood biochemistry. *J Appl Physiol* 43(1):75-81.
- ~~1492-1485.~~ Devlin RB, Folinsbee LJ, Biscardi F, Hatch G, Becker S, Madden MC, et al. (1997). Inflammation and cell damage induced by repeated exposure of humans to ozone. *Inhalation Toxicology* 9:211-35.
- ~~1493-1486.~~ Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D, et al. (1991 Jan). Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *Am J Respir Cell Mol Biol* 4(1):72-81.
- ~~1494-1487.~~ Dillon D, Combes R, McConville M, Zeiger E (1992). Ozone is mutagenic in *Salmonella*. *Environ Mol Mutagen* 19(4):331-7.
- ~~1495-1488.~~ Donaldson K, Brown GM, Brown DM, Slight J, Maclaren W, Davis JMG (1993). Characteristics of bronchoalveolar leucocytes from the lungs of rats inhaling 0.2-0.8 ppm of ozone. *Inhalation Toxicology* 5:149-64.
- ~~1496-1489.~~ Donaldson K, Brown GM, Brown DM, Slight J, Maclaren WM, Davis JM (1991 Oct). Leukocyte-mediated epithelial injury in ozone-exposed rat lung. *Res Rep Health Eff Inst* (44):1-27.
- ~~1497-1490.~~ Dormans JA, Rombout PJ, van Loveren H (1990 Sep). Surface morphology and morphometry of rat alveolar macrophages after ozone exposure. *J Toxicol Environ Health* 31(1):53-70.
- ~~1498-1491.~~ Dormans JA, van Bree L, Boere AJ, Marra M, Rombout PJ (1999 Apr). Interspecies differences in time course of pulmonary toxicity following repeated exposure to ozone. *Inhal Toxicol* 11(4):309-29.
- ~~1499-1492.~~ Dormans JAMA, Boere AJF, van Loveren H, Rombout PJA, Marra M, van Bree L (1996). Age-related toxicity in rat lungs following acute and repeated ozone exposure. *Inhalation Toxicology* 8:903-25.

- ~~1500-1493.~~ Driscoll KE, Vollmuth TA, Schlesinger RB (1987). Acute and subchronic ozone inhalation in the rabbit: response of alveolar macrophages. *J Toxicol Environ Health* 21(1-2):27-43.
- ~~1501-1494.~~ Duan X, Buckpitt AR, Pinkerton KE, Ji C, Plopper CG (1996 Jan). Ozone-induced alterations in glutathione in lung subcompartments of rats and monkeys. *Am J Respir Cell Mol Biol* 14(1):70-5.
- ~~1502-1495.~~ Dubick MA, Keen CL (1983 Jul). Tissue trace elements and lung superoxide dismutase activity in mice exposed to ozone. *Toxicol Lett* 17(3-4):355-60.
- ~~1503-1496.~~ Dziedzic D, White HJ (1986a Dec). T-cell activation in pulmonary lymph nodes of mice exposed to ozone. *Environ Res* 41(2):610-22.
- ~~1504-1497.~~ Dziedzic D, White HJ (1986b Dec). Thymus and pulmonary lymph node response to acute and subchronic ozone inhalation in the mouse. *Environ Res* 41(2):598-609.
- ~~1505-1498.~~ Dziedzic D, Wright ES, Sargent NE (1990 Apr). Pulmonary response to ozone: reaction of bronchus-associated lymphoid tissue and lymph node lymphocytes in the rat. *Environ Res* 51(2):194-208.
- ~~1506-1499.~~ el-Fawal HA, McGovern T, Schlesinger RB (1995 Jan-1995 Feb). Nonspecific bronchial responsiveness assessed in vitro following acute inhalation exposure to ozone and ozone/sulfuric acid mixtures. *Exp Lung Res* 21(1):129-39.
- ~~1507-1500.~~ el-Fawal HA, Schlesinger RB (1994 Mar). Nonspecific airway hyperresponsiveness induced by inhalation exposure to sulfuric acid aerosol: an in vitro assessment. *Toxicol Appl Pharmacol* 125(1):70-6.
- ~~1508-1501.~~ Elsayed NM, Kass R, Mustafa MG, Hacker AD, Ospital JJ, Chow CK, et al. (1988). Effect of dietary vitamin E level on the biochemical response of rat lung to ozone inhalation. *Drug Nutr Interact* 5(4):373-86.
- ~~1509-1502.~~ Eskew ML, Scheuchenzuber WJ, Scholz RW, Reddy CC, Zarkower A (1986 Aug). The effects of ozone inhalation on the immunological response of selenium- and vitamin E-deprived rats. *Environ Res* 40(2):274-84.
- ~~1510-1503.~~ Evans MJ, Fanucchi MV, Baker GL, Van Winkle LS, Pantle LM, Nishio SJ, et al. (2003 Oct). Atypical development of the tracheal basement membrane zone of infant rhesus monkeys exposed to ozone and allergen. *Am J Physiol Lung Cell Mol Physiol* 285(4):L931-9.
- ~~1511-1504.~~ Farman CA, Watkins K, van Hoozen B, Last JA, Witschi H, Pinkerton KE (1999 Feb). Centriacinar remodeling and sustained procollagen gene expression after exposure to ozone and nitrogen dioxide. *Am J Respir Cell Mol Biol* 20(2):303-11.
- ~~1512-1505.~~ Ferg SF, Castro CE, Afifi AA, Bermudez E, Mustafa MG (1997 Jul). Ozone-induced DNA strand breaks in guinea pig tracheobronchial

- epithelial cells. *J Toxicol Environ Health* 51(4):353-67.
- ~~1513-1506.~~ Foster WM, Freed AN (1999 Feb). Regional clearance of solute from peripheral airway epithelia: recovery after sublobar exposure to ozone. *J Appl Physiol* 86(2):641-6.
- ~~1514-1507.~~ Freed AN, Cueto R, Pryor WA (1999 Nov). Antioxidant transport modulates peripheral airway reactivity and inflammation during ozone exposure. *J Appl Physiol* 87(5):1595-603.
- ~~1515-1508.~~ Fujimaki H (1989 Apr). Impairment of humoral immune responses in mice exposed to nitrogen dioxide and ozone mixtures. *Environ Res* 48(2):211-7.
- ~~1516-1509.~~ Fujimaki H, Ozawa M, Imai T, Shimizu F (1984 Dec). Effect of short-term exposure to O₃ on antibody response in mice. *Environ Res* 35(2):490-6.
- ~~1517-1510.~~ Fujimaki H, Shiraishi F, Ashikawa T, Murakami M (1987 Jun). Changes in delayed hypersensitivity reaction in mice exposed to O₃. *Environ Res* 43(1):186-90.
- ~~1518-1511.~~ Gelzleichter TR, Witschi H, Last JA (1992a Jan). Concentration-response relationships of rat lungs to exposure to oxidant air pollutants: a critical test of Haber's Law for ozone and nitrogen dioxide. *Toxicol Appl Pharmacol* 112(1):73-80.
- ~~1519-1512.~~ Gelzleichter TR, Witschi H, Last JA (1992b Sep). Synergistic interaction of nitrogen dioxide and ozone on rat lungs: acute responses. *Toxicol Appl Pharmacol* 116(1):1-9.
- ~~1520-1513.~~ Gilmour MI, Hmieleski RR, Stafford EA, Jakab GJ (1991 May-1991 Jun). Suppression and recovery of the alveolar macrophage phagocytic system during continuous exposure to 0.5 ppm ozone. *Exp Lung Res* 17(3):547-58.
- ~~1521-1514.~~ Gilmour MI, Jakab GJ (1991). Modulation of immune function in mice exposed to 0.8 ppm ozone. *Inhalation Toxicology* 3:293-308.
- ~~1522-1515.~~ Gilmour MI, Park P, Doerfler D, Selgrade MK (1993b May-1993b Jun). Factors that influence the suppression of pulmonary antibacterial defenses in mice exposed to ozone. *Exp Lung Res* 19(3):299-314.
- ~~1523-1516.~~ Gilmour MI, Park P, Selgrade MK (1993a Mar). Ozone-enhanced pulmonary infection with *Streptococcus zooepidemicus* in mice. The role of alveolar macrophage function and capsular virulence factors. *Am Rev Respir Dis* 147(3):753-60.
- ~~1524-1517.~~ Gilmour MI, Selgrade MK (1993 Dec). A comparison of the pulmonary defenses against streptococcal infection in rats and mice following O₃ exposure: differences in disease susceptibility and neutrophil recruitment. *Toxicol Appl Pharmacol* 123(2):211-8.

- ~~1525-1518.~~ Goldsmith CA, Ning Y, Qin G, Imrich A, Lawrence J, Murthy GG, et al. (2002 Apr). Combined air pollution particle and ozone exposure increases airway responsiveness in mice. *Inhal Toxicol* 14(4):325-47.
- ~~1526-1519.~~ Goodman JW, Peter-Fizaine FE, Shinpock SG, Hall EA, Fahmie DJ (1989 May-1989 Jun). Immunologic and hematologic consequences in mice of exposure to ozone. *J Environ Pathol Toxicol Oncol* 9(3):243-52.
- ~~1527-1520.~~ Graham JA, Gardner DE, Blommer EJ, House DE, Menache MG, Miller FJ (1987). Influence of exposure patterns of nitrogen dioxide and modifications by ozone on susceptibility to bacterial infectious disease in mice. *J Toxicol Environ Health* 21(1-2):113-25.
- ~~1528-1521.~~ Grose EC, Stevens MA, Hatch GE, Jaskot RH, Selgrade MJK, Stead AG, et al. (1989). The impact of a 12-month exposure to a diurnal pattern of ozone on pulmonary function, antioxidant biochemistry and immunology. In: Schneider, T. //Lee, S. D. //Wolters, G. J. R. //Grant, L. D. eds. *Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium; May 1988; Nijmegen, The Netherlands, Elsevier Science Publishers B. V., Amsterdam, The Netherlands.* pp. 535-543 (Studies in environmental science 35).
- ~~1529-1522.~~ Gross KB, White HJ (1987). Functional and pathologic consequences of a 52-week exposure to 0.5 PPM ozone followed by a clean air recovery period. *Lung* 165(5):283-95.
- ~~1530-1523.~~ Grotberg JB, Sheth BV, Mockros LF (1990 May). An analysis of pollutant gas transport and absorption in pulmonary airways. *J Biomech Eng* 112(2):168-76.
- ~~1531-1524.~~ Gunnison AF, Weideman PA, Sobo M, Koenig KL, Chen LC (1992 Apr). Age-dependence of responses to acute ozone exposure in rats. *Fundam Appl Toxicol* 18(3):360-9.
- ~~1532-1525.~~ Guth DJ, Warren DL, Last JA (1986 Aug). Comparative sensitivity of measurements of lung damage made by bronchoalveolar lavage after short-term exposure of rats to ozone. *Toxicology* 40(2):131-43.
- ~~1533-1526.~~ Hackney JD, Linn WS, Buckley RD, Pedersen EE, Karuza SK, Law DC, et al. (1975 Aug). Experimental studies on human health effects of air pollutants: I. Design considerations. *Arch Environ Health* 30(8):373-8.
- ~~1534-1527.~~ Haney JT Jr, Connor TH, Li L (1999 Apr). Detection of ozone-induced DNA single strand breaks in murine bronchoalveolar lavage cells acutely exposed in vivo. *Inhal Toxicol* 11(4):331-41.
- ~~1535-1528.~~ Hanna LM, Frank R, Scherer PW (1989). Absorption of soluble gases and vapors in the respiratory system. In: Chang, H. K.; Paiva, M., eds. *Respiratory physiology: an analytical approach.* New York: Marcel Dekker, Inc.; pp. 277-316.
- ~~1536-1529.~~ Haro R, Paz C (1993 Dec). Effects of ozone exposure during pregnancy on ontogeny of sleep in rats. *Neurosci Lett* 164(1-2):67-70.

- 1537-1530. Hassett C, Mustafa MG, Coulson WF, Elashoff RM (1985a Aug). Splenomegaly in mice following exposure to ambient levels of ozone. *Toxicol Lett* 26(2-3):139-44.
- 1538-1531. Hassett C, Mustafa MG, Coulson WF, Elashoff RM (1985b Oct). Murine lung carcinogenesis following exposure to ambient ozone concentrations. *J Natl Cancer Inst* 75(4):771-7.
- 1539-1532. Hatch GE, Slade R, Harris LP, McDonnell WF, Devlin RB, Koren HS, et al. (1994 Sep). Ozone dose and effect in humans and rats. A comparison using oxygen-18 labeling and bronchoalveolar lavage. *Am J Respir Crit Care Med* 150(3):676-83.
- 1540-1533. Hatch GE, Slade R, Stead AG, Graham JA (1986). Species comparison of acute inhalation toxicity of ozone and phosgene. *J Toxicol Environ Health* 19(1):43-53.
- 1541-1534. Hatch GE, Wiester MJ, Overton JHJr, Aissa M (1989). Respiratory tract dosimetry of [18]O-labeled ozone in rats: Implications for a rat-human extrapolation of ozone dose. In: Schneider, T. //Lee, S. D. //Wolters, G. J. R. //Grant, L. D. eds. *Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium; May 1989; Nijmegen, The Netherlands, Elsevier Science Publishers B. V., Amsterdam, The Netherlands. pp. 553-560 (Studies in environmental science 35).*
- 1542-1535. Herbert RA, Hailey JR, Grumbein S, Chou BJ, Sills RC, Haseman JK, et al. (1996 Sep-1996 Oct). Two-year and lifetime toxicity and carcinogenicity studies of ozone in B6C3F1 mice. *Toxicol Pathol* 24(5):539-48.
- 1543-1536. Hicks JJ, Medina-Navarro R, Guzman-Grenfell A, Wachter N, Lifshitz A (1996 Summer). Possible effect of air pollutants (Mexico City) on superoxide dismutase activity and serum lipoperoxides in the human adult. *Arch Med Res* 27(2):145-9.
- 1544-1537. Highfill JW, Hatch GE, Slade R, Crissman KM, Norwood J, Devlin RB, et al. (1992). Concentration-time models for the effects of ozone on bronchoalveolar lavage fluid protein from rats and guinea pigs. *Inhalation Toxicology* 4:1-16.
- 1545-1538. Hoffer E, Baum Y, Tabak A, Frevert C (1999 Sep). Adhesion molecules of blood polymorphonuclear leukocytes and alveolar macrophages in rats: modulation by exposure to ozone. *Hum Exp Toxicol* 18(9):547-51.
- 1546-1539. Hornof WJ, Schelegle E, Kammerman M, Gunther RA, Fisher PE, Cross CE (1989 Sep). Ozone-induced accelerated lung clearance of 99mTc-DTPA aerosol in conscious sheep. *Respir Physiol* 77(3):277-90.
- 1547-1540. Horstman DH, Folinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF (1990 Nov). Ozone concentration and pulmonary response

relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. *Am Rev Respir Dis* 142(5):1158-63.

~~1548-1541.~~ Hotchkiss JA, Harkema JR, Kirkpatrick DT, Henderson RF (1989a). Response of rat alveolar macrophages to ozone: quantitative assessment of population size, morphology, and proliferation following acute exposure. *Exp Lung Res* 15(1):1-16.

~~1549-1542.~~ Hotchkiss JA, Harkema JR, Sun JD, Henderson RF (1989b Apr). Comparison of acute ozone-induced nasal and pulmonary inflammatory responses in rats. *Toxicol Appl Pharmacol* 98(2):289-302.

~~1550-1543.~~ Hu SC, Ben-Jebria A, Ultman JS (1992 Oct). Longitudinal distribution of ozone absorption in the lung: quiet respiration in healthy subjects. *J Appl Physiol* 73(4):1655-61.

~~1551-1544.~~ Hyde DM, Hubbard WC, Wong V, Wu R, Pinkerton K, Plopper CG (1992 May). Ozone-induced acute tracheobronchial epithelial injury: relationship to granulocyte emigration in the lung. *Am J Respir Cell Mol Biol* 6(5):481-97.

~~1552.~~ Ichinose T, Sagai M (1989 Dec). Biochemical effects of combined gases of nitrogen dioxide and ozone. III. Synergistic effects on lipid peroxidation and antioxidative protective systems in the lungs of rats and guinea pigs. *Toxicology* 59(3):259-70.

~~1545.~~

~~1553-1546.~~ Ichinose T, Sagai M (1992 Sep). Combined exposure to NO₂, O₃ and H₂SO₄-aerosol and lung tumor formation in rats. *Toxicology* 74(2-3):173-84.

~~1554-1547.~~ Ishii Y, Hirano K, Morishima Y, Masuyama K, Goto Y, Nomura A, et al. (2000-Sep). Early molecular and cellular events of oxidant-induced pulmonary fibrosis in rats. *Toxicol Appl Pharmacol* 167(3):173-81.

~~1555-1548.~~ Iwasaki T, Takahashi M, Saito H, Arito H (1998 Jan). Adaptation of extrapulmonary responses to ozone exposure in conscious rats. *Ind Health* 36(1):57-60.

~~1556-1549.~~ Jackson RM, Frank L (1984 Mar). Ozone-induced tolerance to hyperoxia in rats. *Am Rev Respir Dis* 129(3):425-9.

~~1557-1550.~~ Jakab GJ, Bassett DJ (1990 May). Influenza virus infection, ozone exposure, and fibrogenesis. *Am Rev Respir Dis* 141(5 Pt 1):1307-15.

~~1558-1551.~~ Jakab GJ, Hmieleski RR (1988). Reduction of influenza virus pathogenesis by exposure to 0.5 ppm ozone. *J Toxicol Environ Health* 23(4):455-72.

~~1559-1552.~~ Joad JP, Bric JM, Pino MV, Hyde DM, McDonald RJ (1993 Jun). Effects of ozone and neutrophils on function and morphology of the isolated rat lung. *Am Rev Respir Dis* 147(6 Pt 1):1578-84.

- 1560-1553. Kavlock R, Daston G, Grabowski CT (1979 Mar). Studies on the developmental toxicity of ozone. I. Prenatal effects. *Toxicol Appl Pharmacol* 48(1 Pt 1):19-28.
- 1561-1554. Kavlock RJ, Meyer E, Grabowski CT (1980 Jan). Studies on the developmental toxicity of ozone: postnatal effects. *Toxicol Lett* 5(1):3-9.
- 1562-1555. Kimmel TA, Chen LC, Bosland MC, Nadziejko C (1997 Jun). Influence of acid aerosol droplet size on structural changes in the rat lung caused by acute exposure to sulfuric acid and ozone. *Toxicol Appl Pharmacol* 144(2):348-55.
- 1563-1556. Kirschvink N, Fievez L, Bureau F, Degand G, Maghuin-Rogister G, Smith N, et al. (2002 Jan). Adaptation to multiday ozone exposure is associated with a sustained increase of bronchoalveolar uric acid. *Free Radic Res* 36(1):23-32.
- 1564-1557. Kleeberger SR, Levitt RC, Zhang LY (1993 Jan). Susceptibility to ozone-induced inflammation. I. Genetic control of the response to subacute exposure. *Am J Physiol* 264(1 Pt 1):L15-20.
- 1565-1558. Kleeberger SR, Levitt RC, Zhang LY, Longphre M, Harkema J, Jedlicka A, et al. (1997 Dec). Linkage analysis of susceptibility to ozone-induced lung inflammation in inbred mice. *Nat Genet* 17(4):475-8.
- 1566-1559. Kleeberger SR, Longphre M, Tankersley CG (1999 Apr). Mechanisms of response to ozone exposure: the role of mast cells in mice. *Res Rep Health Eff Inst* (85):1-30; discussion 31-6.
- 1567-1560. Kleinman MT, Bhalla DK, Ziegler B, Bucher-Evans S, McClure T (1993). Effects of inhaled fine particles and ozone on pulmonary macrophages and epithelia. *Inhalation Toxicology* 5:371-88.
- 1568-1561. Kleinman MT, Bufalino C, Rasmussen R, Hyde D, Bhalla DK, Mautz WJ (2000 Sep-2000 Oct). Toxicity of chemical components of ambient fine particulate matter (PM 2.5) inhaled by aged rats. *J Appl Toxicol* 20(5):357-64.
- 1569-1562. Kleinman MT, Mautz WJ, Bjarnason S (1999 Mar). Adaptive and non-adaptive responses in rats exposed to ozone, alone and in mixtures, with acidic aerosols. *Inhal Toxicol* 11(3):249-64.
- 1570-1563. Kleinman MT, Phalen RF, Mautz WJ, Mannix RC, McClure TR, Crocker TT (1989 Feb). Health effects of acid aerosols formed by atmospheric mixtures. *Environ Health Perspect* 79:137-45.
- 1571-1564. Kobayashi T, Todoroki T, Sato H (1987). Enhancement of pulmonary metastasis of murine fibrosarcoma NR-FS by ozone exposure. *J Toxicol Environ Health* 20(1-2):135-45.
- 1572-1565. Kodavanti UP, Costa DL, Dreher KL, Crissman K, Hatch GE (1995b Jul). Ozone-induced tissue injury and changes in antioxidant homeostasis in normal and ascorbate-deficient guinea pigs. *Biochem Pharmacol*

50(2):243-51.

- ~~1573-1566.~~ Kodavanti UP, Costa DL, Richards J, Crissman KM, Slade R, Hatch GE (1996 Jul-1996 Aug). Antioxidants in bronchoalveolar lavage fluid cells isolated from ozone-- exposed normal and ascorbate-deficient guinea pigs. *Exp Lung Res* 22(4):435-48.
- ~~1574-1567.~~ Kodavanti UP, Hatch GE, Starcher B, Giri SN, Winsett D, Costa DL (1995a Feb). Ozone-induced pulmonary functional, pathological, and biochemical changes in normal and vitamin C-deficient guinea pigs. *Fundam Appl Toxicol* 24(2):154-64.
- ~~1575-1568.~~ Koike E, Kobayashi T, Nelson DJ, McWilliam AS, Holt PG (1998 Feb). Effect of ozone exposure on alveolar macrophage-mediated immunosuppressive activity in rats. *Toxicol Sci* 41(2):217-23.
- ~~1576-1569.~~ Koren HS, Devlin RB, Becker S, Perez R, McDonnell WF (1991). Time-dependent changes of markers associated with inflammation in the lungs of humans exposed to ambient levels of ozone. *Toxicol Pathol* 19(4 Pt 1):406-11.
- ~~1577-1570.~~ Koren HS, Devlin RB, Graham DE, Mann R, McDonnell WF (1989a). The inflammatory response in human lung exposed to ambient levels of ozone. In: Schneider, T. //Lee, S. D. //Wolters, G. J. R. //Grant, L. D. eds. *Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium; May 1988; Nijmegen, The Netherlands, Elsevier Science Publishers B. V., Amsterdam, The Netherlands. pp. 745-753 (Studies in environmental science 35).*
- ~~1578-1571.~~ Koren HS, Devlin RB, Graham DE, Mann R, McGee MP, Horstman DH, et al. (1989b Feb). Ozone-induced inflammation in the lower airways of human subjects. *Am Rev Respir Dis* 139(2):407-15.
- ~~1579-1572.~~ Larson SD, Schelegle ES, Walby WF, Gershwin LJ, Fanuccihi MV, Evans MJ, et al. (2004 Feb). Postnatal remodeling of the neural components of the epithelial-mesenchymal trophic unit in the proximal airways of infant rhesus monkeys exposed to ozone and allergen. *Toxicol Appl Pharmacol* 194(3):211-20.
- ~~1580-1573.~~ Laskin DL, Heck DE, Laskin JD (1998 Dec). Role of inflammatory cytokines and nitric oxide in hepatic and pulmonary toxicity. *Toxicol Lett* 102-103:289-93.
- ~~1581-1574.~~ Laskin DL, Pendino KJ, Punjabi CJ, Rodriguez del Valle M, Laskin JD (1994 Dec). Pulmonary and hepatic effects of inhaled ozone in rats. *Environ Health Perspect* 102 Suppl 10:61-4.
- ~~1582-1575.~~ Last JA, Gelzleichter T, Harkema J, Parks WC, Mellick P (1993b Nov). Effects of 20 months of ozone exposure on lung collagen in Fischer 344 rats. *Toxicology* 84(1-3):83-102.
- ~~1583-1576.~~ Last JA, Gelzleichter TR, Pinkerton KE, Walker RM, Witschi H (1993a Aug). A new model of progressive pulmonary fibrosis in rats. *Am*

Rev Respir Dis 148(2):487-94.

- ~~1584-1577.~~ Last JA, Pinkerton KE (1997 Jan). Chronic exposure of rats to ozone and sulfuric acid aerosol: biochemical and structural responses. *Toxicology* 116(1-3):133-46.
- ~~1585-1578.~~ Last JA, Warren DL, Pecquet-Goad E, Witschi H (1987 Jan). Modification by ozone of lung tumor development in mice. *J Natl Cancer Inst* 78(1):149-54.
- ~~1586-1579.~~ Lee JS, Mustafa MG, Afifi AA (1990). Effects of short-term, single and combined exposure to low-level NO₂ and O₃ on lung tissue enzyme activities in rats. *J Toxicol Environ Health* 29(3):293-305.
- ~~1587-1580.~~ Lee S-L, Afifi AA, Mustafa MG (1989). Effects of short-term, single and combined exposure of rats to NO₂ and O₃ on lung tissue enzyme activities. *Inhalation Toxicology* 1:21-35.
- ~~1588-1581.~~ Li AF, Richters A (1991a Jan-1991a Feb). Ambient level ozone effects on subpopulations of thymocytes and spleen T lymphocytes. *Arch Environ Health* 46(1):57-63.
- ~~1589-1582.~~ Li AFY, Richters A (1991b). Effects of 0.7 ppm ozone exposure on thymocytes: in vivo and in vitro studies. *Inhalation Toxicology* 3:61-71.
- ~~1590-1583.~~ Long NC, Suh J, Morrow JD, Schiestl RH, Murthy GG, Brain JD, et al. (2001 Oct). Ozone causes lipid peroxidation but little antioxidant depletion in exercising and nonexercising hamsters. *J Appl Physiol* 91(4):1694-700.
- ~~1591-1584.~~ Madden MC, Richards JH, Dailey LA, Hatch GE, Ghio AJ (2000 Oct). Effect of ozone on diesel exhaust particle toxicity in rat lung. *Toxicol Appl Pharmacol* 168(2):140-8.
- ~~1592-1585.~~ Mariassy AT, Abraham WM, Phipps RJ, Sielczak MW, Wanner A (1990 Jun). Effect of ozone on the postnatal development of lamb mucociliary apparatus. *J Appl Physiol* 68(6):2504-10.
- ~~1593-1586.~~ Mariassy AT, Sielczak MW, McCray MN, Abraham WM, Wanner A (1989 Nov). Effects of ozone on lamb tracheal mucosa. Quantitative glycoconjugate histochemistry. *Am J Pathol* 135(5):871-9.
- ~~1594-1587.~~ Mautz WB, Nadziejko C (2000). California Air Resources Board. Effects of ozone on proteases and protease inhibitors of the human and rat lung. Sacramento, CA: Research Division; 2000. Contract No. A033-175.
- ~~1595-1588.~~ Mautz WJ, Bufalino C (1989 Apr). Breathing pattern and metabolic rate responses of rats exposed to ozone. *Respir Physiol* 76(1):69-77.
- ~~1596-1589.~~ Mautz WJ, Finlayson-Pitts BJ, Messer K, Kleinman MT, Norgren MB, Quirion J (1991). Effects of ozone combined with components of acid fogs on breathing pattern, metabolic rate, pulmonary surfactant composition, and lung injury in rats. *Inhalation Toxicology* 3:1-25.

- ~~1597-1590.~~ Mautz WJ, Kleinman MT, Bhalla DK, Phalen RF (2001 Jun). Respiratory tract responses to repeated inhalation of an oxidant and acid gas-particle air pollutant mixture. *Toxicol Sci* 61(2):331-41.
- ~~1598-1591.~~ Mautz WJ, Kleinman MT, Phalen RF, Crocker TT (1988). Effects of exercise exposure on toxic interactions between inhaled oxidant and aldehyde air pollutants. *J Toxicol Environ Health* 25(2):165-77.
- ~~1599-1592.~~ McBride RK, Oberdoerster G, Marin MG (1991 Jun). Effects of ozone on the cholinergic secretory responsiveness of ferret tracheal glands. *Environ Res* 55(1):79-90.
- ~~1600-1593.~~ Mercer RR, Anjilvel S, Miller FJ, Crapo JD (1991 May). Inhomogeneity of ventilatory unit volume and its effects on reactive gas uptake. *J Appl Physiol* 70(5):2193-205.
- ~~1601-1594.~~ Miller FJ, Conolly RB (1995). Uncertainties in health risk assessments: commentary on selected issues and research needs. In: Lee, S. D. //Schneider, T. eds. *Comparative risk analysis and priority setting for air pollution issue: proceedings of the 4th US-Dutch international symposium*; June 1993; Keystone, CO, Air and Waste Management Association, Pittsburg, PA. pp. 76-91.
- ~~1602-1595.~~ Miller FJ, Illing JW, Gardner DE (1978a). Effect of urban ozone levels on laboratory-induced respiratory infections. *Toxicology Letters* 2:163-9.
- ~~1603-1596.~~ Miller FJ, Menzel DB, Coffin DL (1978b Aug). Similarity between man and laboratory animals in regional pulmonary deposition of ozone. *Environ Res* 17(1):84-101.
- ~~1604-1597.~~ Miller FJ, Overton JH Jr, Jaskot RH, Menzel DB (1985 Jun). A model of the regional uptake of gaseous pollutants in the lung. I. The sensitivity of the uptake of ozone in the human lung to lower respiratory tract secretions and exercise. *Toxicol Appl Pharmacol* 79(1):11-27.
- ~~1605-1598.~~ Miller FJ, Overton JH, Gerrity TR, Graham RC (1988). Interspecies dosimetry of reactive gases. In: Mohr U, Dungworth D, Kimmerle G, Lewkowski J, McClellan R, Stober W, editors. *Inhalation toxicology: the design and interpretation of inhalation studies and their use in risk assessment*. New York: Springer-Verlag; pp. 139-155.
- ~~1606-1599.~~ Miller PD, Gordon T, Warnick M, Amdur MO (1986). Effect of ozone and histamine on airway permeability to horseradish peroxidase in guinea pigs. *J Toxicol Environ Health* 18(1):121-32.
- ~~1607-1600.~~ Mills PC, Roberts CA, Smith NC (1996 Sep). Effects of ozone and airway inflammation on glutathione status and iron homeostasis in the lungs of horses. *Am J Vet Res* 57(9):1359-63.
- ~~1608-1601.~~ Mochitate K, Ishida K, Ohsumi T, Miura T (1992 Apr). Long-term effects of ozone and nitrogen dioxide on the metabolism and population of alveolar macrophages. *J Toxicol Environ Health* 35(4):247-60.

- ~~1609~~.1602. Mole ML Jr, Stead AG, Gardner DE, Miller FJ, Graham JA (1985 Sep). Effect of ozone on serum lipids and lipoproteins in the rat. *Toxicol Appl Pharmacol* 80(3):367-76.
- ~~1610~~.1603. Musi B, Dell'Omo G, Ricceri L, Santucci D, Laviola G, Bignami G, et al. (1994 Winter). Effects of acute and continuous ozone (O₃) exposure on activity/exploration and social behavior of CD-1 mice. *Neurotoxicology* 15(4):827-35.
- ~~1611~~.1604. Mustafa MG, Hassett CM, Newell GW, Schrauzer GN (1988). Pulmonary carcinogenic effects of ozone. *Ann N Y Acad Sci* 534:714-23.
- ~~1612~~.1605. Nikula KJ, Wilson DW, Giri SN, Plopper CG, Dungworth DL (1988 May). The response of the rat tracheal epithelium to ozone exposure. Injury, adaptation, and repair. *Am J Pathol* 131(2):373-84.
- ~~1613~~.1606. Oosting RS, van Golde LM, Verhoef J, Van Bree L (1991 Aug). Species differences in impairment and recovery of alveolar macrophage functions following single and repeated ozone exposures. *Toxicol Appl Pharmacol* 110(1):170-8.
- ~~1614~~.1607. Overton JH, Barnett AE, Graham RC (1989a). Significances of the variability of tracheobronchial airway paths and their air flow rates to dosimetry model predictions of the absorption of gases. In: Crapo JD; Smolko ED; Miller FJ; Graham JA; Hayes AW; eds. *Extrapolation of dosimetric relationships for inhaled particles and gases*. San Diego: Academic Press, Inc.; pp. 273-291.
- ~~1615~~.1608. Overton JH, Graham RC (1989b). Predictions of ozone absorption in human lungs from newborn to adult. *Health Phys* 57 Suppl 1:29-36.
- ~~1616~~.1609. Overton JH, Graham RC, Miller FJ (1987 May). A model of the regional uptake of gaseous pollutants in the lung. II. The sensitivity of ozone uptake in laboratory animal lungs to anatomical and ventilatory parameters. *Toxicol Appl Pharmacol* 88(3):418-32.
- ~~1617~~.1610. Paz C (1997 Summer). Some consequences of ozone exposure on health. *Arch Med Res* 28(2):163-70.
- ~~1618~~.1611. Paz C, Bazan-Perkins B (1992 Jun). Sleep-wake disorganization in cats exposed to ozone. *Neurosci Lett* 140(2):270-2.
- ~~1619~~.1612. Paz C, Huitron-Resendiz S (1996 Feb). The effects of ozone exposure on the sleep-wake cycle and serotonin contents in the pons of the rat. *Neurosci Lett* 204(1-2):49-52.
- ~~1620~~.1613. Pearson AC, Bhalla DK (1997 Feb). Effects of ozone on macrophage adhesion in vitro and epithelial and inflammatory responses in vivo: the role of cytokines. *J Toxicol Environ Health* 50(2):143-57.
- ~~1621~~.1614. Petruzzi S, De Acetis L, Chiarotti F, Sorace A, Alleva E (1999). Limited changes in handedness and morphine reactivity in CD-1 mice after pre- and postnatal ozone exposure. *Acta Neurobiol Exp (Warsz)* 59(2):115-

22.

- ~~1622~~.1615. Petruzzi S, Fiore M, Dell'Omo G, -Alleva E (1995a). Exposure to ozone inhibits isolation-induced aggressive behavior of adult CD-1 male mice. *Aggressive Behavior* 21:387-96.
- ~~1623~~.1616. Petruzzi S, Fiore M, Dell'Omo G, Bignami G, Alleva E (1995 Jul-1995 Aug). Medium and long-term behavioral effects in mice of extended gestational exposure to ozone. *Neurotoxicol Teratol* 17(4):463-70.
- ~~1624~~.1617. Phalen RF, Crocker TT, McClure TR, Tyler NK (1986). Effect of ozone on mean linear intercept in the lung of young beagles. *J Toxicol Environ Health* 17(2-3):285-96.
- ~~1625~~.1618. Phipps RJ, Denas SM, Sielczak MW, Wanner A (1986 Mar). Effects of 0.5 ppm ozone on glycoprotein secretion, ion and water fluxes in sheep trachea. *J Appl Physiol* 60(3):918-27.
- ~~1626~~.1619. Pickrell JA, Gregory RE, Cole DJ, Hahn FF, Henderson RF (1987b Apr). Effect of acute ozone exposure on the proteinase-antiproteinase balance in the rat lung. *Exp Mol Pathol* 46(2):168-79.
- ~~1627~~.1620. Pickrell JA, Hahn FF, Rebar AH, Horoda RA, Henderson RF (1987a Apr). Changes in collagen metabolism and proteinolysis after repeated inhalation exposure to ozone. *Exp Mol Pathol* 46(2):159-67.
- ~~1628~~.1621. Pinkerton KE, Brody AR, Miller FJ, Crapo JD (1989 Oct). Exposure to low levels of ozone results in enhanced pulmonary retention of inhaled asbestos fibers. *Am Rev Respir Dis* 140(4):1075-81.
- ~~1629~~.1622. Pinkerton KE, Joad JP (2000 Jun). The mammalian respiratory system and critical windows of exposure for children's health. *Environ Health Perspect* 108 Suppl 3:457-62.
- ~~1630~~.1623. Pino MV, Levin JR, Stovall MY, Hyde DM (1992a Jan). Pulmonary inflammation and epithelial injury in response to acute ozone exposure in the rat. *Toxicol Appl Pharmacol* 112(1):64-72.
- ~~1631~~.1624. Pino MV, Stovall MY, Levin JR, Devlin RB, Koren HS, Hyde DM (1992b Jun). Acute ozone-induced lung injury in neutrophil-depleted rats. *Toxicol Appl Pharmacol* 114(2):268-76.
- ~~1632~~.1625. Plopper CG, Duan X, Buckpitt AR, Pinkerton KE (1994 Jul). Dose-dependent tolerance to ozone. IV. Site-specific elevation in antioxidant enzymes in the lungs of rats exposed for 90 days or 20 months. *Toxicol Appl Pharmacol* 127(1):124-31.
- ~~1633~~.1626. Plopper CG, Hatch GE, Wong V, Duan X, Weir AJ, Tarkington BK, et al. (1998 Sep). Relationship of inhaled ozone concentration to acute tracheobronchial epithelial injury, site-specific ozone dose, and glutathione depletion in rhesus monkeys. *Am J Respir Cell Mol Biol* 19(3):387-99.
- ~~1634~~.1627. Postlethwait EM, Langford SD, Bidani A (1994 Mar). Determinants of inhaled ozone absorption in isolated rat lungs. *Toxicol Appl Pharmacol*

- 125(1):77-89.
- ~~1635-1628.~~ Prasad SB, Rao VS, Mannix RC, Phalen RF (1988). Effects of pollutant atmospheres on surface receptors of pulmonary macrophages. *J Toxicol Environ Health* 24(3):385-402.
- ~~1636-1629.~~ Pryor WA (1992). How far does ozone penetrate into the pulmonary air/tissue boundary before it reacts? *Free Radic Biol Med* 12(1):83-8.
- ~~1637-1630.~~ Pryor WA, Das B, Church DF (1991 May-1991 Jun). The ozonation of unsaturated fatty acids: aldehydes and hydrogen peroxide as products and possible mediators of ozone toxicity. *Chem Res Toxicol* 4(3):341-8.
- ~~1638-1631.~~ Rahman I, Clerch LB, Massaro D (1991 Jun). Rat lung antioxidant enzyme induction by ozone. *Am J Physiol* 260(6 Pt 1):L412-8.
- ~~1639-1632.~~ Rahman I, Massaro GD, Massaro D (1992). Exposure of rats to ozone: evidence of damage to heart and brain. *Free Radic Biol Med* 12(4):323-6.
- ~~1640-1633.~~ Rajini P, Gelzleichter TR, Last JA, Witschi H (1993 Aug). Alveolar and airway cell kinetics in the lungs of rats exposed to nitrogen dioxide, ozone, and a combination of the two gases. *Toxicol Appl Pharmacol* 121(2):186-92.
- ~~1641-1634.~~ Raub JA, Miller FJ, Graham JA (1983). Effects of low-level ozone exposure on pulmonary function in adult and neonatal rats. *Advances in Modern Environmental Toxicology* 5:363-7.
- ~~1642-1635.~~ Reinhart PG, Bassett DJ, Bhalla DK (1998 May). The influence of polymorphonuclear leukocytes on altered pulmonary epithelial permeability during ozone exposure. *Toxicology* 127(1-3):17-28.
- ~~1643-1636.~~ Reiser KM, Tyler WS, Hennessy SM, Dominguez JJ, Last JA (1987 Jul). Long-term consequences of exposure to ozone. II. Structural alterations in lung collagen of monkeys. *Toxicol Appl Pharmacol* 89(3):314-22.
- ~~1644-1637.~~ Richters A (1988). Effects of nitrogen dioxide and ozone on blood-borne cancer cell colonization of the lungs. *J Toxicol Environ Health* 25(3):383-90.
- ~~1645-1638.~~ Rietjens IM, Van Bree L, Marra M, Poelen MC, Rombout PJ, Alink GM (1985 Dec). Glutathione pathway enzyme activities and the ozone sensitivity of lung cell populations derived from ozone exposed rats. *Toxicology* 37(3-4):205-14.
- ~~1646-1639.~~ Rivas-Arancibia S, Vazquez-Sandoval R, Gonzalez-Kladiano D, Schneider-Rivas S, Lechuga-Guerrero A (1998 Jan). Effects of ozone exposure in rats on memory and levels of brain and pulmonary superoxide dismutase. *Environ Res* 76(1):33-9.
- ~~1647-1640.~~ Rivas-Manzano P, Paz C (1999 Nov). Cerebellar morphological alterations in rats induced by prenatal ozone exposure. *Neurosci Lett*

276(1):37-40.

- ~~1648~~.1641. Rombout PJA, van Bree L, Heisterkamp SH, Marra M (1989). The need for an eight hour ozone standard. In: Schneider, T.; Lee, S. D.; Wolters, G. J. R.; Grant, L. D. eds. Atmospheric ozone research and its policy implications: proceedings of the 3rd US-Dutch international symposium; May 1988; Nijmegen, The Netherlands, Elsevier Science Publishers B. V., Amsterdam, The Netherlands. pp. 701-710 (Studies in Environmental Science 35).
- ~~1649~~.1642. Romero-Velazquez RM, Alfaro-Rodriguez A, Gonzalez-Pina R, Gonzalez-Maciel A (2002). Effect of ozone prenatal exposure on postnatal development of cerebellum. *Proc West Pharmacol Soc* 45:65-7.
- ~~1650~~.1643. Ross BK, Hlastala MP, Frank R (1979 May-1979 Jun). Lack of ozone effects on oxygen hemoglobin affinity. *Arch Environ Health* 34(3):161-3.
- ~~1651~~.1644. Ryer-Powder JE, Amoruso MA, Czerniecki B, Witz G, Goldstein BD (1988 Nov). Inhalation of ozone produces a decrease in superoxide anion radical production in mouse alveolar macrophages. *Am Rev Respir Dis* 138(5):1129-33.
- ~~1652~~.1645. Sagai M, Ichinose T (1991 Feb). Biochemical effects of combined gases of nitrogen dioxide and ozone. IV. Changes of lipid peroxidation and antioxidative protective systems in rat lungs upon life span exposure. *Toxicology* 66(2):121-32.
- ~~1653~~.1646. Saldiva PH, King M, Delmonte VL, Macchione M, Parada MA, Daliberto ML, et al. (1992 Feb). Respiratory alterations due to urban air pollution: an experimental study in rats. *Environ Res* 57(1):19-33.
- ~~1654~~.1647. Sarangapani R, Gentry PR, Covington TR, Teeguarden JG, Clewell HJ 3rd (2003 Sep). Evaluation of the potential impact of age- and gender-specific lung morphology and ventilation rate on the dosimetry of vapors. *Inhal Toxicol* 15(10):987-1016.
- ~~1655~~.1648. Schelegle ES, Miller LA, Gershwin LJ, Fanucchi MV, Van Winkle LS, Gerriets JE, et al. (2003a Aug). Repeated episodes of ozone inhalation amplifies the effects of allergen sensitization and inhalation on airway immune and structural development in Rhesus monkeys. *Toxicol Appl Pharmacol* 191(1):74-85.
- ~~1656~~.1649. Schelegle ES, Walby WF, Alfaro MF, Wong VJ, Putney L, Stovall MY, et al. (2003b Feb). Repeated episodes of ozone inhalation attenuates airway injury/repair and release of substance P, but not adaptation. *Toxicol Appl Pharmacol* 186(3):127-42.
- ~~1657~~.1650. Schlesinger RB, Gorczynski JE, Dennison J, Richards L, Kinney PL, Bosland MC (1992a Jul-1992a Aug). Long-term intermittent exposure to sulfuric acid aerosol, ozone, and their combination: alterations in tracheobronchial mucociliary clearance and epithelial secretory cells. *Exp*

Lung Res 18(4):505-34.

- ~~1658~~.1651. Schlesinger RB, Zelikoff JT, Chen LC, Kinney PL (1992b Aug). Assessment of toxicologic interactions resulting from acute inhalation exposure to sulfuric acid and ozone mixtures. *Toxicol Appl Pharmacol* 115(2):183-90.
- ~~1659~~.1652. Selgrade MK, Cooper KD, Devlin RB, van Loveren H, Biagini RE, Luster MI (1995 Jan). Immunotoxicity--bridging the gap between animal research and human health effects. *Fundam Appl Toxicol* 24(1):13-21.
- ~~1660~~.1653. Selgrade MK, Daniels MJ, Grose EC (1990). Acute, subchronic, and chronic exposure to a simulated urban profile of ozone: effects on extrapulmonary natural killer cell activity and lymphocyte mitogenic responses. *Inhalation Toxicology* 2:375-89.
- ~~1661~~.1654. Selgrade MK, Illing JW, Starnes DM, Stead AG, Menache MG, Stevens MA (1988 Jul). Evaluation of effects of ozone exposure on influenza infection in mice using several indicators of susceptibility. *Fundam Appl Toxicol* 11(1):169-80.
- ~~1662~~.1655. Shepson PB, Kleindienst TE, Edney EO, Namie GR, Pittman JH, Cupitt LT, et al. (1985). The mutagenic activity of irradiated toluene/NO_x/H₂O/air mixtures. *Environ Sci Technol* 19:249-55.
- ~~1663~~.1656. Sherwin RP, Richters V (1985). Effect of 0.3 ppm ozone exposure on type II cells and alveolar walls of newborn mice: an image-analysis quantitation. *J Toxicol Environ Health* 16(3-4):535-46.
- ~~1664~~.1657. Sherwood RL, Lippert WE, Goldstein E (1986 Dec). Effect of 0.64 ppm ozone on alveolar macrophage lysozyme levels in rats with chronic pulmonary bacterial infection. *Environ Res* 41(2):378-87.
- ~~1665~~.1658. Shore SA, Johnston RA, Schwartzman IN, Chism D, Krishna Murthy GG (2002 Mar). Ozone-induced airway hyperresponsiveness is reduced in immature mice. *J Appl Physiol* 92(3):1019-28.
- ~~1666~~.1659. Sielczak MW, Denas SM, Abraham WM (1983 Apr-1983 Jun). Airway cell changes in tracheal lavage of sheep after ozone exposure. *J Toxicol Environ Health* 11(4-6):545-53.
- ~~1667~~.1660. Sills RC, Hong HL, Greenwell A, Herbert RA, Boorman GA, Devereux TR (1995 Jul). Increased frequency of K-ras mutations in lung neoplasms from female B6C3F1 mice exposed to ozone for 24 or 30 months. *Carcinogenesis* 16(7):1623-8.
- ~~1668~~.1661. Slade R, Crissman K, Norwood J, Hatch G (1993 Jul-1993 Aug). Comparison of antioxidant substances in bronchoalveolar lavage cells and fluid from humans, guinea pigs, and rats. *Exp Lung Res* 19(4):469-84.
- ~~1669~~.1662. Slade R, Highfill JW, Hatch GE (1989). Effects of depletion of ascorbic acid or nonprotein sulfhydryls on the acute inhalation toxicity of nitrogen dioxide, ozone, and phosgene. *Inhalation Toxicology* 1:261-71.

- ~~1670-1663.~~ Slade R, Watkinson WP, Hatch GE (1997 Jan). Mouse strain differences in ozone dosimetry and body temperature changes. *Am J Physiol* 272(1 Pt 1):L73-7.
- ~~1671-1664.~~ Sorace A, de Acetis L, Alleva E, Santucci D (2001 Feb). Prolonged exposure to low doses of ozone: short- and long-term changes in behavioral performance in mice. *Environ Res* 85(2):122-34.
- ~~1672-1665.~~ Sterner-Kock A, Kock M, Braun R, Hyde DM (2000 Sep). Ozone-induced epithelial injury in the ferret is similar to nonhuman primates. *Am J Respir Crit Care Med* 162(3 Pt 1):1152-6.
- ~~1673-1666.~~ Tepper JS, Costa DL, Lehmann JR, Weber MF, Hatch GE (1989 Aug). Unattenuated structural and biochemical alterations in the rat lung during functional adaptation to ozone. *Am Rev Respir Dis* 140(2):493-501.
- ~~1674-1667.~~ Tepper JS, Wiester MJ, Weber MF, Menache MG (1990 Feb). Measurements of cardiopulmonary response in awake rats during acute exposure to near-ambient concentrations of ozone. *J Appl Toxicol* 10(1):7-15.
- ~~1675-1668.~~ Thomassen DG, Harkema JR, Stephens ND, Griffith WC (1991 Jun). Preneoplastic transformation of rat tracheal epithelial cells by ozone. *Toxicol Appl Pharmacol* 109(1):137-48.
- ~~1676-1669.~~ Tyler WS, Tyler NK, Last JA, Gillespie MJ, Barstow TJ (1988 Jul). Comparison of daily and seasonal exposures of young monkeys to ozone. *Toxicology* 50(2):131-44.
- ~~1677-1670.~~ Tyler WS, Tyler NK, Magliano DJ, Hinds DM, Tarkington B, Julian MD, et al. (1991). Effects of ozone inhalation on lungs of juvenile monkeys. Morphometry after a 12 month exposure and following a 6 month post-exposure period. In: Berglund RL, Lawson DR, McKee DJ, eds. *Tropospheric Ozone and the Environment: Papers from an International Conference, March 1990, Los Angeles, CA, Pittsburgh, PA: Air and Waste Management Association*, p. 151-160.
- ~~1678-1671.~~ Tyson CA, Lunan KD, Stephens RJ (1982 May-1982 Jun). Age-related differences in GSH-shuttle enzymes in NO₂- or O₃-exposed rat lungs. *Arch Environ Health* 37(3):167-76.
1672. U.S. Environmental Protection Agency (1996). *Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume III. Chapter 8. Extrapolation of Animal Toxicological Data to Humans.* EPA/600/P-93/004a-cF p. 8-1 to 8-101. Can be obtained online at: <http://cfpub.epa.gov/ncea/cfm/ozone.cfm?ActType=default>.
- ~~1680-1673.~~ Uchiyama I, Simomura Y, Yokoyama E (1986 Dec). Effects of acute exposure to ozone on heart rate and blood pressure of the conscious rat. *Environ Res* 41(2):529-37.
- ~~1681-1674.~~ Uchiyama I, Yokoyama E (1989 Feb). Effects of short- and long-term exposure to ozone on heart rate and blood pressure of

- emphysematous rats. *Environ Res* 48(1):76-86.
- ~~1682-1675.~~ Ulrich MM, Alink GM, Kumarathasan P, Vincent R, Boere AJ, Cassee FR (2002 Oct). Health effects and time course of particulate matter on the cardiopulmonary system in rats with lung inflammation. *J Toxicol Environ Health A* 65(20):1571-95.
- ~~1683-1676.~~ Umezu T, Shimojo N, Tsubone H, Suzuki AK, Kubota K, Shimizu A (1987 Jan-1987 Feb). Effect of ozone toxicity in the drinking behavior of rats. *Arch Environ Health* 42(1):58-62.
- ~~1684-1677.~~ van Bree L, Dormans JA, Boere AJ, Rombout PJ (2001 Aug). Time study on development and repair of lung injury following ozone exposure in rats. *Inhal Toxicol* 13(8):703-18.
- ~~1685-1678.~~ van Bree L, Dormans JA, Koren HS, Devlin RB, Rombout PJ (2002 Aug). Attenuation and recovery of pulmonary injury in rats following short-term, repeated daily exposure to ozone. *Inhal Toxicol* 14(8):883-900.
- ~~1686-1679.~~ Van Loveren H, Krajnc EI, Rombout PJ, Blommaert FA, Vos JG (1990 Jan). Effects of ozone, hexachlorobenzene, and bis(tri-n-butyltin)oxide on natural killer activity in the rat lung. *Toxicol Appl Pharmacol* 102(1):21-33.
- ~~1687-1680.~~ Van Loveren H, Rombout PJ, Wagenaar SS, Walvoort HC, Vos JG (1988 Jul). Effects of ozone on the defense to a respiratory *Listeria monocytogenes* infection in the rat. Suppression of macrophage function and cellular immunity and aggravation of histopathology in lung and liver during infection. *Toxicol Appl Pharmacol* 94(3):374-93.
- ~~1688-1681.~~ Vaughan WJ, Adamson GL, Lindgren FT, Schooley JC (1984 Jul). Serum lipid and lipoprotein concentrations following exposure to ozone. *J Environ Pathol Toxicol Oncol* 5(4-5):165-73.
- ~~1689-1682.~~ Victorin K (1996). Genotoxicity and carcinogenicity of ozone. *Scand J Work Environ Health* 22 Suppl 3:42-51.
- ~~1690-1683.~~ Victorin K, Stahlberg M (1988). A method for studying the mutagenicity of some gaseous compounds in *Salmonella typhimurium*. *Environ Mol Mutagen* 11(1):65-77.
- ~~1691-1684.~~ Vincent R, Bjarnason SG, Adamson IY, Hedgecock C, Kumarathasan P, Guenette J, et al. (1997 Dec). Acute pulmonary toxicity of urban particulate matter and ozone. *Am J Pathol* 151(6):1563-70.
- ~~1692-1685.~~ Warren DL, Guth DJ, Last JA (1986 Jul). Synergistic interaction of ozone and respirable aerosols on rat lungs. II. Synergy between ammonium sulfate aerosol and various concentrations of ozone. *Toxicol Appl Pharmacol* 84(3):470-9.
- ~~1693-1686.~~ Warren DL, Last JA (1987 Apr). Synergistic interaction of ozone and respirable aerosols on rat lungs. III. Ozone and sulfuric acid aerosol. *Toxicol Appl Pharmacol* 88(2):203-16.

- ~~1694-1687.~~ Watkinson WP, Aileru AA, Dowd SM, Doerfler DL, Tepper JS, Costa DL (1993). Acute effects of ozone on heart rate and body temperature in the unanesthetized, unrestrained rat maintained at different ambient temperatures. *Inhalation Toxicology* 5:129-47.
- ~~1695-1688.~~ Watkinson WP, Campen MJ, Nolan JP, Costa DL (2001 Aug). Cardiovascular and systemic responses to inhaled pollutants in rodents: effects of ozone and particulate matter. *Environ Health Perspect* 109 Suppl 4:539-46.
- ~~1696-1689.~~ Watkinson WP, Wiester MJ, Highfill JW (1995 Mar). Ozone toxicity in the rat. I. Effect of changes in ambient temperature on extrapulmonary physiological parameters. *J Appl Physiol* 78(3):1108-20.
- ~~1697-1690.~~ Weller BL, Crapo JD, Slot J, Posthuma G, Plopper CG, Pinkerton KE (1997 Nov). Site- and cell-specific alteration of lung copper/zinc and manganese superoxide dismutases by chronic ozone exposure. *Am J Respir Cell Mol Biol* 17(5):552-60.
- ~~1698-1691.~~ Wiester MJ, Tepper JS, King ME, Menache MG, Costa DL (1988 Oct). Comparative study of ozone (O₃) uptake in three strains of rats and in the guinea pig. *Toxicol Appl Pharmacol* 96(1):140-6.
- ~~1699-1692.~~ Wiester MJ, Tepper JS, Winsett DW, Crissman KM, Richards JH, Costa DL (1996a May). Adaptation to ozone in rats and its association with ascorbic acid in the lung. *Fundam Appl Toxicol* 31(1):56-64.
- ~~1700-1693.~~ Wiester MJ, Watkinson WP, Costa DL, Crissman KM, Richards JH, Winsett DW, et al. (1996b Oct). Ozone toxicity in the rat. III. Effect of changes in ambient temperature on pulmonary parameters. *J Appl Physiol* 81(4):1691-700.
- ~~1701-1694.~~ Wiester MJ, Williams TB, King ME, Menache MG, Miller FJ (1987 Jul). Ozone uptake in awake Sprague-Dawley rats. *Toxicol Appl Pharmacol* 89(3):429-37.
- ~~1702-1695.~~ Wiester MJ, Winsett DW, Richards JH, Jackson MC, Crissman KM, Costa DL (2000 Jul). Ozone adaptation in mice and its association with ascorbic acid in the lung. *Inhal Toxicol* 12(7):577-90.
- ~~1703-1696.~~ Witschi H (1988 Jan). Ozone, nitrogen dioxide and lung cancer: a review of some recent issues and problems. *Toxicology* 48(1):1-20.
- ~~1704-1697.~~ Witschi H (1991 Mar-1991 Apr). Effects of oxygen and ozone on mouse lung tumorigenesis. *Exp Lung Res* 17(2):473-83.
- ~~1705-1698.~~ Witschi H, Espiritu I, Pinkerton KE, Murphy K, Maronpot RR (1999 Dec). Ozone carcinogenesis revisited. *Toxicol Sci* 52(2):162-7.
- ~~1706-1699.~~ Witschi H, Wilson DW, Plopper CG (1993 Jan). Modulation of N-nitrosodiethylamine-induced hamster lung tumors by ozone. *Toxicology* 77(1-2):193-202.
- ~~1707-1700.~~ Wong CG, Bonakdar M, Mautz WJ, Kleinman MT (1996 Feb).

Chronic inhalation exposure to ozone and nitric acid elevates stress-inducible heat shock protein 70 in the rat lung. *Toxicology* 107(2):111-9.

~~1708-1701.~~ Wright ES, Kehrer JP, White DM, Smiler KL (1988 Mar). Effects of chronic exposure to ozone on collagen in rat lung. *Toxicol Appl Pharmacol* 92(3):445-52.

~~1709-1702.~~ Yokoyama E, Frank R (1972 Aug). Respiratory uptake of ozone in dogs. *Arch Environ Health* 25(2):132-8.

~~1710-1703.~~ Young C, Bhalla DK (1992 Feb). Time course of permeability changes and PMN flux in rat trachea following O₃ exposure. *Fundam Appl Toxicol* 18(2):175-80.

~~1711-1704.~~ Yu M, Pinkerton KE, Witschi H (2002 Jan). Short-term exposure to aged and diluted sidestream cigarette smoke enhances ozone-induced lung injury in B6C3F1 mice. *Toxicol Sci* 65(1):99-106.

~~1712-1705.~~ Zelikoff JT, Kraemer GL, Vogel MC, Schlesinger RB (1991 Dec). Immunomodulating effects of ozone on macrophage functions important for tumor surveillance and host defense. *J Toxicol Environ Health* 34(4):449-67.



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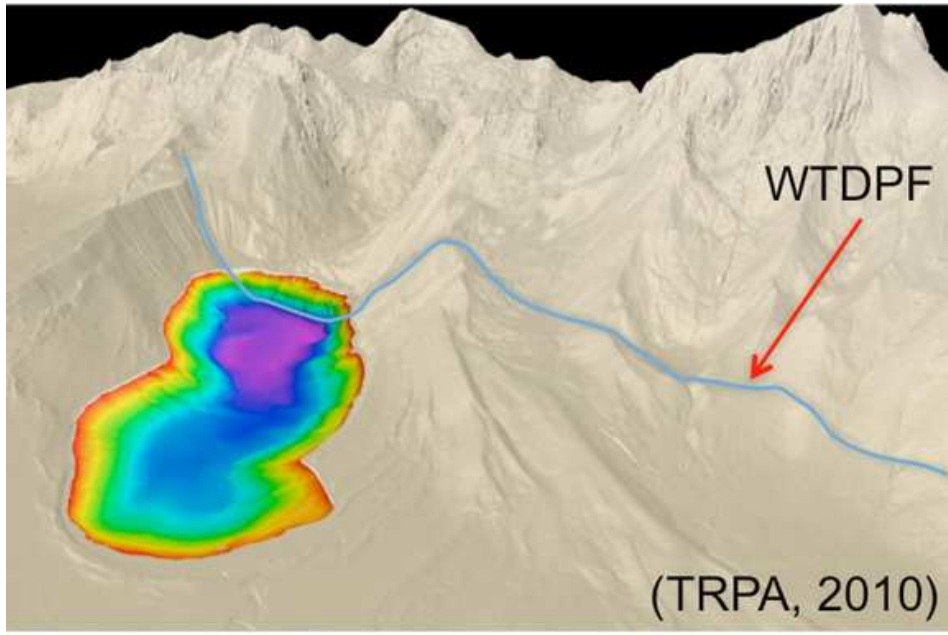
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Tsunamis, Earthquakes Overdue in Lake Tahoe

Becky Oskin, OurAmazingPlanet Staff Writer | December 06, 2012 10:58am ET



SAN FRANCISCO — A tsunami-producing fault in Lake Tahoe is overdue for another earthquake, scientists said here



yesterday (Dec. 4) at the annual meeting of the American Geophysical Union.

The West Tahoe Fault is capable of producing a magnitude-7.3 earthquake and tsunamis up to 30 feet (10 meters) high in the clear blue lake, where million-dollar homes line the shore, researchers said.

[Earthquakes strike](#) every 3,000 to 4,000 years on the fault, and the most recent shaker was 4,500 years ago, indicating th fault is overdue for another earthquake, said Jillian Maloney, a graduate student a the Scripps Institution of Oceanography in San Diego.

A lidar image of Fallen Leaf Lake in the Lake Tahoe Basin. The blue line is the West Tahoe Fault. The rainbow hues reflect the depth of the lake.

Credit: Jillian Maloney, Scripps. [View full size image](#)



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The West Tahoe fault defines the west shore of the lake, coming on shore at Baldwin Beach, passing through the southern third of Fallen Leaf Lake, and then descending into Christmas Valley near Echo Summit.

Underwater tracking

To trace the fault's history, Maloney and her colleagues examined data from a CHIRP seismic imaging system, which details underwater sediment layers at very high resolution. (CHIRP stands for compressed high intensity radar pulse.) The researchers correlated landslide deposits, which could be related to past earthquakes, throughout western Lake Tahoe and in small lakes immediately to the south with radiocarbon dates from the sediments.

The West Tahoe Fault has a complicated history, the analysis reveals. The fault appears to alternate between breaking all at once, in a 31-mile long (50 kilometer) fracture, and in smaller, shorter segments. The discovery has implications for the [Tahoe's seismic hazard](#), because the size of an earthquake relates to the length of a fault rupture, Maloney said. The biggest earthquakes come from the longest fault fractures.

The correlations, while still at an early stage, indicate the last time the fault's entire length ruptured was 7,800 years ago, Maloney told OurAmazingPlanet. More recent quakes occurred on individual segments, she said.

Tsunami risk

Because the fault crosses the lake, scientists worry a future earthquake will cause a tsunami in Lake Tahoe. The [monster waves](#) could form in two ways: by the fault displacing ground under the lake, similar to Japan's Tohoku tsunami, or by causing [landslides that displace the water](#). A combination of both could also create an even bigger wave.

Layers of sediment preserved in and around Lake Tahoe record evidence of past tsunamis, said Graham Kent, director of the

Nevada Seismological Laboratory in Reno.

However, having smaller earthquakes on the West Tahoe Fault would be better for the ski town. "If it breaks up into multiple segments, it might not be as great a tsunami risk," Kent told OurAmazingPlanet.

The most recent earthquake in the Tahoe region was about 575 years ago, on the Incline Fault, which becomes active about every 10,000 to 15,000 years. Scientists estimate its earthquake size potential at magnitude 7.

At more than 1,645 feet (501 meters) deep, Lake Tahoe, which straddles the California and Nevada border in the [seismically active Sierra Nevada region](#), is one of the world's deepest freshwater lakes.

This story was provided by [OurAmazingPlanet](#), a sister site to LiveScience. Reach Becky Oskin at boskin@techmedianetwork.com. Follow her on Twitter [@beckyoskin](#). Follow OurAmazingPlanet on Twitter [@OAPlanet](#). We're also on [Facebook](#) and [Google+](#).

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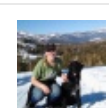
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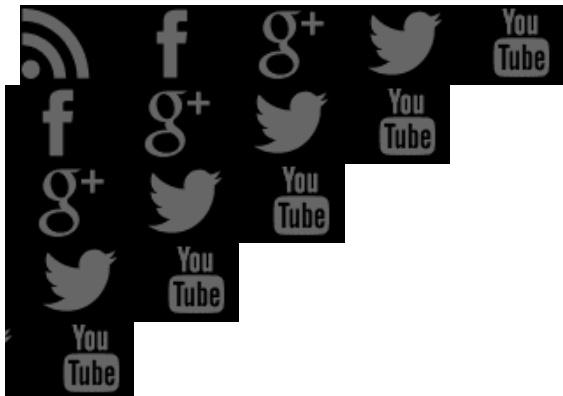
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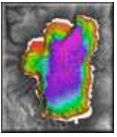
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Three Faults Under Lake Tahoe

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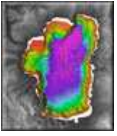
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Lake Tahoe is located in one of the most seismically active regions of the United States. We are wise to always be ready to shake, rattle, and roll.

The Tahoe Basin wasn't scoured out by glaciers or extruded by volcanic eruption. Over the course of 10 million years, powerful mountain-building processes slowly uplifted the Sierra range from a shallow sea.

Lake Formation

The Tahoe Basin itself formed when the seismic thrusting finally triggered a dramatic land collapse on the eastern (Nevada) side of the Sierra uplift. Next, lava from the Mt. Pluto volcano in the Northstar ski area sealed off the basin's northern end. Eventually the deep crevasse filled with water... and became what we know as Lake Tahoe. It is the tenth deepest lake in the world at 1645 ft.

Tahoe's mountainous topography was not uplifted as a whole mass, but heaved and wrenched along rock fractures known as fault lines. The geologic history of the Sierra is measured in millions of years, yet it is still a growing mountain range that can generate earthquakes in swarms. Since pioneers settled the Tahoe basin, most temblors have been too weak too cause much damage. But scientists warn that a catastrophic earthquake is only a matter of time.

The Tahoe Basin is bounded by faults on the northern and western sides. Traces of these faults, submerged on the lake bottom and hidden by eroded soil, rock, and glacial deposits, have been revealed by new imaging technologies. John Anderson, director of the Nevada Seismological Laboratory in Reno, recently stated: "Geologists here believe that the **underwater faults** have the capability to cause a large earthquake, but *any* of the faults in the area have that potential."

Three Main Faults

There are three potentially dangerous fault lines that run deep under Lake Tahoe. The northern portion of Tahoe appears to be the most tectonically active. Near Stateline Point (location of Cal-Neva), one fault cuts directly through the area, while an extension of the fault trends northeast through Incline Village.

Scientists believe that these two North Shore faults are part of one system and may tend to rupture together.

Another prominent fault zone is the north-south trending West Tahoe-Dollar Point fault zone. Submerged from Emerald bay to McKinney Bay (near Tahoma/Chambers Landing), the West Tahoe fault continues on to become the Dollar Point Fault. Geologists suspect that both of these faults may also rupture together.

Geologists warn that the faults beneath Lake Tahoe are capable of generating a 7.1-magnitude quake and enough movement to produce tsunami waves exceeding 30 feet in height.

Low Risk

However, don't panic and sell your lakefront home! Scientists estimate the risk of a magnitude-7 quake under Lake Tahoe in the next 50 years to be between 3% and 4%, far less than the perennial dangers of forest fires and flood in the region. Experts do suggest that if you are near Tahoe's shoreline and feel a severe tremor that lasts for more than 10 seconds, "first duck and cover, then sprint upwards 30 feet in elevation," --hopefully to safety.

Mark McLaughlin is a weather/historian who lives at Lake Tahoe.

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West Tahoe Fault: Iconic area could see earthquake, 'seiche wave' tsunami

Tuesday, July 23, 2013 at 9:44AM



Photo of Lake Tahoe: Sascha Brück via Wikipedia

Lake Tahoe is familiar to most of us as a place to go have fun—skiing, water sports and hiking are a few options in this beautiful U.S. landmark. Tahoe, however, has physical attributes that are just now being explored because of developments in technology.

Experts are concerned about the potential for an earthquake and tsunami. But if the most active fault, West Tahoe, is the source, the consequences would be cataclysmic, producing a type of tsunami most never heard of.

The Las Vegas Sun said the West Tahoe fault is “the most active of three major faults in the lake.”

The fault wasn't put on the map until 1998. The last big quake was approximately 4,000 years ago.

Experts told the paper it wouldn't be surprising “if it happened tomorrow,” because they believe such events could occur every 2,500-4,000 years.

The California Geological Survey is studying the Lake and the team is using an unmanned submarine normally used to explore ice shelves in Antarctica.

Should a quake occur, there would be a phenomenon many of us have never considered. Engineer Gordon Seitz deemed the situation “urgent enough” that the study needed to be done:

“Unlike ocean tsunamis, whose massive waves break and then disappear, a tsunami in Lake Tahoe

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would go back and forth for hours, hitting one side of the lake and then the other, again and again, Seitz said.”

The California Geological Survey **explained the phenomenon:**

There are several faults thought capable of generating large earthquakes in the region that encompasses Lake Tahoe, Truckee and Carson City, Nev., including the Genoa, Antelope Valley, Incline Village and West Tahoe faults. The latter fault runs under part of Lake Tahoe and may be able to generate damaging seiche waves (essentially a tsunami in an enclosed body of water).

Lake Tahoe is on the border between California and Nevada.

(Filed by [Kay B. Day](#)/July 23, 2013)

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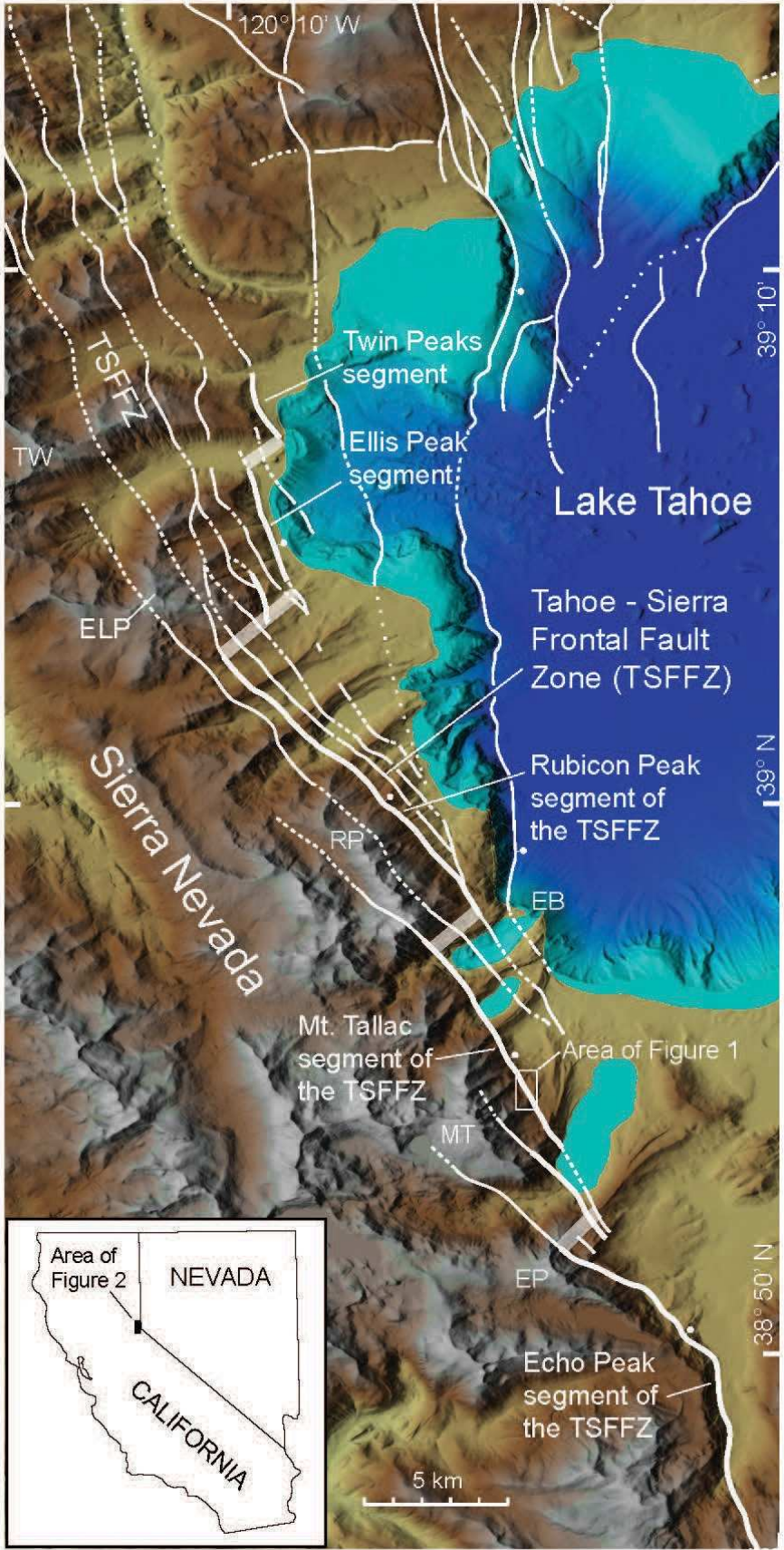
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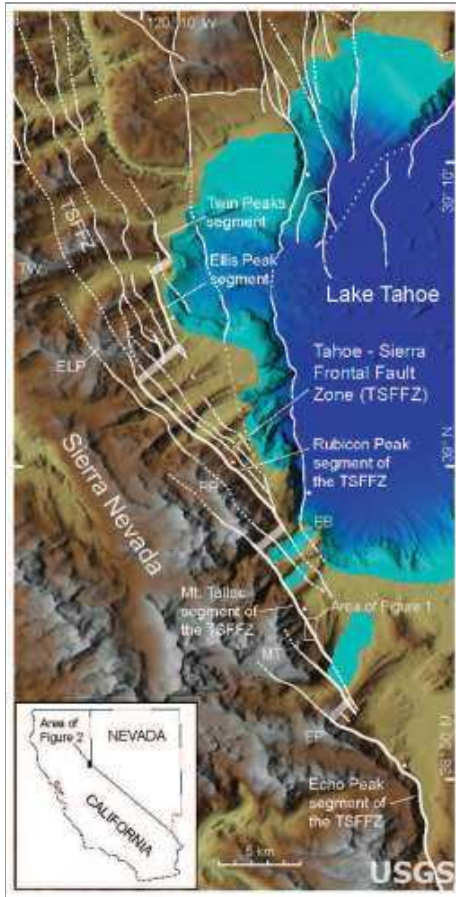
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Lake Tahoe Faults, Shaded Relief Map



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Description:

Shaded relief map of western part of the Lake Tahoe basin, California. Faults lines are dashed where approximately located, dotted where concealed, bar and ball on downthrown side. Heavier line weight shows principal range-front fault strands of the Tahoe-Sierra frontal fault zone (TSFFZ). Opaque white boxes indicate approximate segment boundaries and right steps in range front separating principal fault strands. EB—Emerald Bay; ELP—Ellis Peak; EP—Echo Peak; MT—Mt. Tallac; RP—Rubicon Peak; TW—Twin Peaks

Location: Lake Tahoe, CA, USA

Date Taken: May 2012

Photographer: [James Howle](#), U.S. Geological Survey

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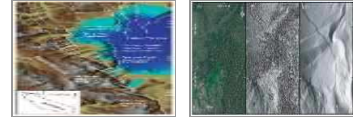
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U.S. Geological Survey

LiDAR Technology Reveals Faults Near Lake Tahoe

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CARNELIAN BAY, Calif. — Results of a new U.S. Geological Survey study conclude that faults west of Lake Tahoe, Calif., referred to as the Tahoe-Sierra frontal fault zone, pose a substantial increase in the seismic hazard assessment for the Lake Tahoe region of California and Nevada, and could potentially generate earthquakes with magnitudes ranging from 6.3 to 6.9. A close association of landslide deposits and active faults also suggests that there is an earthquake-induced landslide hazard along the steep fault-formed range front west of Lake Tahoe.

Using a new high-resolution imaging technology, known as bare-earth airborne LiDAR (Light Detection And Ranging), combined with field observations and modern geochronology, USGS scientists, and their colleagues from the University of Nevada, Reno; the University of California, Berkeley; and the U.S. Army Corps of Engineers, have confirmed the existence of previously suspected faults. LiDAR imagery allows scientists to "see" through dense forest cover and recognize earthquake faults that are not detectable with conventional aerial photography.

"This study is yet one more stunning example of how the availability of LiDAR information to precisely and accurately map the shape of the solid Earth surface beneath vegetation is revolutionizing the geosciences," said USGS Director Marcia McNutt. "From investigations of geologic hazards to calculations of carbon stored in the forest canopy to simply making the most accurate maps possible, LiDAR returns its investment many times over."

Motion on the faults has offset linear moraines (the boulders, cobbles, gravel, and sand deposited by an advancing glacier) providing a record of tectonic deformation since the moraines were deposited. The authors developed new three-dimensional techniques to measure the amount of tectonic displacement of moraine crests caused by repeated earthquakes. Dating of the moraines from the last two glaciations in the Tahoe basin, around 21 thousand and 70 thousand years ago, allowed the study authors to calculate the rates of tectonic displacement.

"Although the Tahoe-Sierra frontal fault zone has long been recognized as forming the tectonic boundary between the Sierra Nevada to the west, and the Basin and Range Province to the east, its level of activity and hence seismic hazard was not fully recognized because dense vegetation obscured the surface expressions of the faults," said USGS scientist and lead author, James Howle. "Using the new LiDAR technology has improved and clarified previous field mapping, has provided visualization of the surface expressions of the faults, and has allowed for accurate measurement of the amount of motion that has occurred on the faults. The results of the study demonstrate that the Tahoe-Sierra frontal fault zone is an important seismic source for the region."

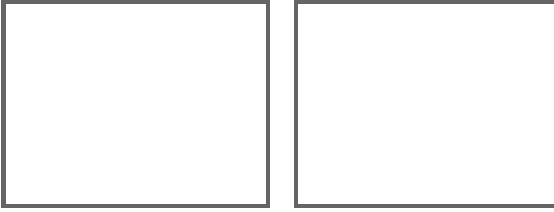
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