

I.

**Air Resources Board Staff Responses to Comments on
the Draft ETS Report Part A**

SUMMARY AND RESPONSES TO COMMENTS SUBMITTED ON THE ENVIRONMENTAL TOBACCO SMOKE DRAFT REPORT

Part A (Exposure Assessment)

Comments and the Air Resources Board's (ARB) staff responses on exposure assessment (Part A) of the "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant Draft Report, December 2003."

Coalition from the Natural Resources Defense Council, Breast Cancer Fund, San Francisco Bay Area Physicians for Social Responsibility, Los Angeles Physicians for Social Responsibility, March 29, 2004

**Alyonik Hrushow, Tobacco Free Project Director,
City and County of San Francisco, March 29, 2004**

**William V. Corr, National Center for Tobacco-Free Kids,
March 29, 2004**

**Susan Rappaport and Paul Knepprath,
American Lung Association, March 29, 2004**

**Robert T. Croyle, Ph.D., Director, Division of Cancer Control and
Population Sciences, National Cancer Institute – March 29, 2004**

1. Comment: In general, we support the conclusions of the draft report and ARB's action to identify ETS as a TAC.

Response: We appreciate your comment.

James Repace, March 5, 2004

1. Comment: As you know, there have been few measurements of ETS in outdoor microenvironments, and to the best of my knowledge, there are no data on outdoor carcinogen levels of ETS. I have collected indoor/outdoor particulate PAH data while on a cruise ship in the Caribbean. A preliminary report on this data is available.

Response: We agree. There are few studies done on the carcinogenic components of ETS in the outdoor air. We will incorporate the results of your study as soon as it is a published peer reviewed document.

R.J. Reynolds Tobacco Company, March 25, 2004

1. Comment: The current California Environmental Protection Agency 2003 Draft Report, "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant," does not support designation of environmental tobacco smoke (ETS) as a toxic air contaminant (TAC) in California. Specifically, Sections 39650-39674 of the California Health and Safety Code set forth several requirements that the Agency must meet before designating a substance as a TAC. For example, Section 39660 initially requires Cal/EPA generally to assess the exposure and health effects data for the substance and to specifically determine whether current California ETS exposures are responsible for adverse health effects, then to provide an estimate of the exposure level that may cause or contribute to adverse health effects in California.

Response: California Health and Safety Code Section 39660(a) states specifically that "Upon the request of the state board, the office, in consultation with and with the participation of the state board, shall evaluate the health effects of and prepare recommendations regarding substances, other than pesticides in their pesticidal use, which may be or are emitted into the ambient air of California and that may be determined to be toxic air contaminants." (underline is added for emphasis). A toxic air contaminant is defined in the Health and Safety Code, Section 39655 as "an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health." We believe there is sufficient evidence presented in the draft report (Parts A and B) to show that ETS is emitted into the ambient air in California and that there are various adverse health impacts associated with exposures to ETS.

Furthermore, Health and Safety Code section 39660(c) states that the evaluation shall also contain "an estimate of the levels of exposure that may cause or contribute (underline is added for emphasis) to adverse health effects in California." In Part A, Chapter V, a scenario approach was used to estimate possible ranges of public exposure to ETS. While we recognize that some of the public's exposure is very low, other people's exposures are higher as they go near the smoking public. Health and Safety Code section 39660.5 requires that ARB assess exposures in indoor environments as well as in ambient air.

Brian McGinn, Lorillard Tobacco Company, March 25, 2004

1. Comment: Personal monitoring studies provide the most reliable basis for measuring ETS exposure.

Response: As you are aware, fixed ambient monitoring is the basis of most outdoor air quality measurements. We believe our exposure assessment is representative of personal outdoor exposure for two reasons: 1) our ETS measurements were collected in the breathing zone on the edges of outdoor smoking areas where non-smokers also could have been exposed to ETS, and 2) our multiple exposure scenarios included periods of the day away from ETS exposure, as would be the case with personal exposure monitoring.

2. Comment: The ARB draft report largely ignores the findings of an Oak Ridge study of personal monitoring of ETS in 16 U.S. cities.

Response: The 16-city study (Jenkins *et al.*, 1996) was referenced in the biomarker section of Chapter V of the report, but not in our section on page V-6 about other air monitoring for ETS. Staff will add a reference to this study in the monitoring section on page V-6.

3. Comment: The field and trip spikes were prepared at only one level per study location (ranged from 10 to 400 micrograms of nicotine) and these levels were considerably higher than actual field samples, making these spikes inappropriate for evaluating the accuracy of measured air concentrations.

Response: The method detection limit for nicotine was based on lab spikes of 0.1 microgram of nicotine. Most field samples contained concentrations of nicotine above the method detection limit. The field and trip spikes were prepared at a higher concentration to ensure that there was no breakthrough in the sampling tubes. While it would have been interesting to have prepared field and trip spikes at more than one level (with one level closer to anticipated field concentrations), the monitoring budget was too limited. We do not believe that the lack of these data limit the use of the measured air concentrations.

4. Comment: Only a few, unrepresentative outdoor locations were used for monitoring, sites that appear to have been selected arbitrarily or to represent maximum potential exposures.

Response: Sites were selected to represent a variety of outdoor exposures near ETS. The results of the monitoring indicate a range in outdoor concentrations of ETS.

5. Comment: Monitoring was conducted only in, or immediately downwind and adjacent to, designated smoking areas, which can be readily avoided by non-smokers and, thus, are not representative of typical ETS exposures in the ambient air.

Response: While it is true that monitoring was conducted adjacent to designated smoking areas, we do not agree that non-smokers could always avoid these exposures. Following is a summary of the exposures at the locations

where monitoring was conducted: 1) At the airport smoking area where monitoring was conducted, the only exit from the baggage claim area passed through the outdoor smoking area. Arriving passengers were witnessed standing near the smoking area while they waited to be picked up. 2) At the community college where monitoring was conducted, smoking was allowed at an eating area outside of the cafeteria. If a student or faculty member chose to eat outdoors, they could be exposed to ETS. 3) At the two office buildings where monitoring was conducted, smoking was allowed outdoors. Upon entering or leaving the building, sitting outside for a break or lunch, or using an ATM machine, there was potential for exposure to ETS. 4) At the amusement park smoking area where monitoring was conducted, the designated area was centrally located near main walkways. Some parents brought their children into the smoking area, as witnessed by our monitoring staff.

6. Comment: The ARB study was an area monitoring study that did not measure exposure duration or the level of exposure to particular individuals. Personal monitoring data is preferred over area sampling.

Response: The purpose of the monitoring study was only to gather ambient data. The study was not an individual exposure assessment. See response to comment #5 above.

7. Comment: The ARB study used nicotine as the marker for ETS exposure. There are shortcomings with the use of nicotine as an ETS marker. The dilution of ETS emitted in outdoor air, combined with possible absorption to outdoor surfaces in proximity of smokers, renders risk estimation of outdoor exposures based upon nicotine problematic. The report mischaracterized a paper by LaKind *et al.* regarding 3-EP as a marker and should rephrase this section.

Response: The ARB monitoring used nicotine as a marker for ETS because, based on information we reviewed, we believed there would be less adsorption to outdoor surfaces than indoor environments, where adsorption has been documented as a problem with using nicotine as a marker. We agree that dilution of ETS emitted in outdoor air, especially on windy days as were experienced at three of the monitored locations, may have resulted in lower ETS concentrations than would have been measured with less dilution. However, these measurements were representative of realistic exposure. Nicotine was also chosen as a marker because of its relative ease with regard to sampling and analysis. We did not intend to mischaracterize the LaKind *et al.* paper's discussion of the value of 3-EP as a marker for nicotine. We will delete the sentence in question that inaccurately refers to 3-EP on page V-6 of the report.

8. Comment: The ARB air monitoring study has not been published in a peer-reviewed scientific journal.

Response: The ARB has not typically published results of an air monitoring study for a candidate TAC, prior to identification of the candidate as a TAC. Peer review of the report, which includes the details of the air monitoring study, is provided by the Scientific Review Panel on Toxic Air Contaminants. In addition, comments are received from other agencies and the public. Many of the individuals that submitted comments are experts in their respective fields (e.g., exposure assessment).

9. Comment: Under the Tanner Act, passed in 1983, the ARB has authority to identify and adopt control measures for “toxic air contaminants.” The ARB is limited to regulate based on ambient or outdoor air and has no authority to regulate indoor air or to rely upon indoor air exposure levels as a basis for regulation of outdoor air.

Response: California’s air toxics law, Assembly Bill 1807 (sponsored by Tanner) established ARB’s authority to identify and control toxic air contaminants in California. The law requires the ARB to first establish if a substance is toxic and to what extent. This step is called the risk assessment or identification phase of the process. In this process, the ARB is required to evaluate the exposures in indoor environments as well as in ambient air conditions (Health and Safety Code section 39660.5). Once a substance is determined to be a toxic air contaminant by the ARB, it enters into the next step of the program. This step is called the risk management or control phase of the process. In this phase, the ARB is required to evaluate the possibilities of reducing exposures to TACs in consideration of costs and risk as well as a number of other factors (Health and Safety Code sections 39665 and 39666).

The evaluation of ETS as a TAC falls under the first step, risk assessment. This rulemaking effort is a proposal to identify ETS as a TAC in California. Therefore, no control measures are being proposed as part of the risk assessment process at this time to reduce public exposure to ETS.

See response to comment #1 by R.J. Reynolds Tobacco Company, which is incorporated by reference here, for a discussion of authority to identify substances as TACs.

10. Comment: The draft exposure assessment does not demonstrate a meaningful level of outdoor ETS exposure. In view of the limited data on outdoor ETS exposures and the localized nature of such exposures, the ARB lacks a reliable scientific basis to conclude that ETS exposures in the outdoor environment in California are of sufficient intensity, duration or scope to justify listing ETS as a TAC.

Response: Under State law, the ARB is to identify a substance as a toxic air contaminant if it determines the substance is “an air pollutant which may cause or contribute to an increase in mortality or increase in serious illness, or which

may pose a present or potential hazard to human health.” Under this same law, an air pollutant may include groups of substances such as soot, gases, particulate matter, smoke, or any combination (Health and Safety Code section 39013).

Under State law, the ARB must show that Californians are exposed to ETS and that exposures to ETS may cause or contribute to adverse health effects (Health and Safety Code section 39657, 39660 et seq.). The Part A (exposure assessment) document shows that the public is exposed to ETS in California outdoors and the OEHHA’s Part B (health assessment) document shows that exposures to ETS at different levels results in several different adverse health effects. See responses to comments #1 by R.J. Reynolds and comment #9, which are incorporated by reference here.

11. Comment: The ARB’s ETS exposure assessment is inconsistent with the U.S. EPA’s Final Guidelines for Exposure Assessment (U.S. EPA, 1992)

Response: The ARB is required by law to evaluate exposures to and emissions of potential toxic air contaminants. The State is not required to follow U.S. EPA’s Guidelines for Exposure Assessment (see Health and Safety Code Section 39656). The two programs are separate and are different both in scope and purpose.

12. Comment: The Rogge *et al.*, (1994) study referred to in Chapter V of the exposure assessment is outdated and fundamentally flawed. Smoking rates have declined and smoking patterns have changed since the original study in 1982.

Response: We agree that the information presented in the Rogge study is outdated. Smoking rates have declined since the date of the Rogge *et al.*, 1994 study. We state this clearly in Chapter II (pages II-2), Chapter IV (pages IV -4 and IV -5, IV-9 and IV -10) and Chapter V (pages V-4, V-11, and V-31 and V-32). This study, along with others, was used for comparison purposes only and presented a source apportionment approach of estimating outdoor concentrations of ETS. In addition, the Rogge study was included to address our requirement to consider all available data when identifying a substance as a TAC.

13. Comment: The outdoor exposure levels calculated in the Exposure Chapter are based exclusively on a 2003 ARB air monitoring study.

Response: Chapter V of the Part A report, includes studies by Rogge *et al.*, 1994, Eisner *et al.*, 2001 and Schauer *et al.*, 1996 (see Chapter V, pages V-6 through V-13). Since there are relatively few data on outdoor ambient concentrations, the ARB ambient monitoring results from its ETS study were used, in part, as the outdoor ambient concentration input to the exposure

scenarios (see Chapter V pages V-34 through V-47). The scenario-based approach to estimate a person's daily exposure to ETS uses several estimates of exposure from different microenvironments. One of these includes an estimate of outdoor levels of ETS.

14. Comment: In almost all previous TAC exposure assessments, the ARB relied upon California population-weighted exposures to outdoor average ambient concentrations of the candidate substances. By contrast, the ARB has relied exclusively upon localized short-term exposures, in or immediately downwind and adjacent to, designated smoking areas, data that have no relevance to general long-term ETS exposure in the ambient air in California.

Response: As stated in Chapter V, page V-1, A scenario-based approach was used to characterize the range of the public's exposure to ETS in this report. This approach differs from previous TAC exposure assessments, which were based on California population-weighted exposures to outdoor average ambient concentrations. That approach was appropriate for TACs emitted from area-wide or region-wide sources such as motor vehicles and industrial plants. However, cigarettes and cigars, the primary sources of ETS, are smaller sources that emit pollutants near people, and ETS is not monitored at ambient monitoring stations like most other previously identified TACs (See Chapter V-5 for reasons why ETS as a whole cannot be measured). Staff did include an estimate of an urban background level to Chapter V of the draft report for illustration purposes. The text was included in subchapter C, section 5. A more detailed discussion was newly included to the draft report as appendix D.

This is not the first time the ARB has taken this approach. For example, there is no population weighted exposure assessment for vinyl chloride. Exposures, in this case, occur near localized specific sources and such "hot spots" data was used in the TAC exposure assessment. Also, there is interest in short-term exposures to ETS as well as long-term exposures. There are adverse health effects associated with both durations of exposures.

15. Comment: The ARB's scenario-based approach is an inadequate basis to demonstrate outdoor exposure to ETS.

Response: As stated in Chapter V of the report, the scenario-based exposure method uses the results from ARB's ETS air monitoring study, available indoor ETS concentration data, and scenario-based activity patterns to estimate exposures under different conditions. ARB's scenario-based approach is intended to provide a "snapshot" of what some subpopulations ETS exposure might be. We believe this approach provides the best estimated range of exposures a person, adult or child, may experience each day. See also response to comment #13. In addition, staff estimated a statewide outdoor urban background level of ETS as mentioned in response to previous comment #14.

See response to comment #1 by R.J. Reynolds Tobacco Company for a discussion on Health and Safety Code requirements for evaluating exposures to potential TACs.

16. Comment: All prior TAC listings have been based on more extensive and reliable exposure data than that available for ETS. The draft report does not identify the number of people exposed to ETS in the ambient air in California, or the duration or level of such exposure.

Response: We disagree that all prior listings have been based on more extensive and reliable exposure data. In this report, we present several measurements of ETS concentration data as well as smoking prevalence data (see Chapter V). In the scenarios, we provide estimates of duration and level of exposure (Chapter V-34 through V-48). Refer to response to comment #14 with regard to why we did not feel a population-weighted exposure assessment was appropriate for ETS.

17. Comment: ARB has failed to characterize the intensity, duration or frequency of ETS exposure in outdoor air, and failed properly to characterize the exposed population.

Response: See response to comments #14, #15 and #16 above.

Roger A. Jenkins, March 16, 2004

1. Comment: The report ignores key available data that is California-specific. The report relies on modeling studies of exposure rather than relying on direct measurement of exposure.

Response: We have included California-specific data in Chapter V of the Part A report. In addition, our scenario-based exposure approach uses measured concentration results from several studies, including California-specific studies, along with California-specific activity patterns to estimate a range of possible daily public exposures to ETS. The purpose of our personal exposure estimate was to provide a more realistic estimate of public exposure under various scenarios.

Data from direct measurements of exposure are found in Chapter V, page V-48, Biological Markers of Exposure to ETS. Likewise, California-specific data was included in this section. The commenter did not submit key data on either ETS exposure modeling or measurement studies for our consideration.

2. Comment: Criticism, either direct or thinly veiled, is leveled at some but not all of the studies. This provides an unnecessarily advocative tone to the Report, which seriously diminishes its credibility. If the authors believe that an analysis of

the strengths and limitations of studies are useful to the discussion, then such an analysis must be performed on all of the studies considered for discussion.

Response: We believe we have presented a balanced assessment of the studies used in our report.

3. Comment: No analysis was performed on the only California-specific data set available for personal exposure to nicotine and salivary cotinine levels, despite the fact that such data has been publicly available for years.

Response: The commenter is not specific as to what data set was referenced in the peer-reviewed literature. To the extent that they are based on the design used for the rest of the 16 Cities Study, we have the same concerns about the data and potential bias mentioned in the response to comment #5 below.

4. Comment: There is discussion of biomarker levels in smoking mothers, but no effort is made to rationalize its connection with the topic of section: biomarkers and ETS exposure.

Response: See responses to comment #14 below.

5. Comment: There are no substantive conclusions for this section with regard to the stated objective (page V-50) to examine “the utility of biomarkers to assess the extent of exposure to ETS.” The “conclusion,” that cotinine in body fluids can be used to distinguish smokers from ETS exposed individuals, is hardly a quantitative assessment, and ignores key scientific findings in the area. These are a) overall indicators of exposure (number of cigarettes observed to have been smoked near subjects, smoking/non-smoking home/workplace classification groupings, etc, show proportional increases in cotinine levels for increasing nicotine exposure when data from individuals is composited into larger groupings. (This may be due to dampening of individual differences in metabolism.); b) individual cotinine levels, while having statistically significant correlations with nicotine exposure, appear to have little *quantitative* predictive capability (in other words, one cannot use cotinine level to quantitatively predict an individual’s exposure to within a factor of 2, or even 5); c) models based on metabolism of nicotine by smokers appear to be unable to quantitatively estimate the magnitude of inhaled dose of nicotine; and d) other biomarkers of tobacco specific constituents, such as tobacco specific nitrosamines, may ultimately be useful for qualitative or even semi-quantitative indicators of inhaled ETS dose. However, the analytical challenges of measuring extremely trace quantities of these markers in biological fluids are preclude their applicability to broad studies of ETS dose at this time.

Response: We agree that many of the biomarkers examined in this section are not particularly useful for measuring ETS exposure at this time for reasons given in the comment and in the text of the document. However, the commenter’s

objections notwithstanding, at this time nicotine and cotinine represent reasonable markers of tobacco smoke exposure. For this reason we have concentrated on measurements of nicotine and cotinine as the best currently available measures of ETS exposure.

6. Comment: On Page V-54. The 16 Cities Study was not performed by LaKind *et al.*. The 1999 manuscript is a further analysis of the data reported first (and conducted by) Jenkins *et al.*, 1996. If it is important to provide the reader with funding sponsorship or affiliation of authors, then full disclosure should be made for all authors cited: eg. Smith et al., 2005 well-recognized anti-smoking advocates, reported Frankly, if the data have been reported in the peer reviewed literature, sponsorship or the personal preferences of the authors should not be considered in the analysis. Period. Also, Dietrich Hoffmann's name is incorrectly spelled at the bottom of the page.

Response: The text has been re-worded to eliminate references to funding source. We have also corrected the spelling of Hoffmann and clarified LaKind's role regarding analysis of data from the 16 Cities Study.

7. Comment: On page V-55. The statement that the EPA had raised a multitude of concerns (unspecified) regarding the 16 Cities Study in some post hearing commentary in February of 1996, when the peer-reviewed manuscript was not even published until December 1996, suggests that the authors are bending over backward to appear as advocates, rather than dispassionate, unbiased assessors of the scientific data.

Response: Although not specified in the comment, the post-hearing commentary to which the commenter refers is probably Repace's invited analysis of comments to the OSHA docket regarding an indoor air rulemaking concerning ETS. This analysis raised questions regarding the credibility of the reported workplace nicotine levels presented as data collected in the 16 Cities Study. Specifically, it suggests that the reported values are far lower than would be expected for an office workplace, and are lower than would be predicted based on the study's associated salivary cotinine levels.

Our own examination of the published report also led to concerns about how representative the data are. For example, compared to the general population, the study population is disproportionately female (68% vs 53%), better educated (79% had at least some college education vs 47% in the general population), of higher socioeconomic status (70% had income = \$30,000/year vs 50% in the general population), and biased towards professional employment (only 12% were in the categories of service, production and crafts, operators, laborers and fabricators, or other compared to 42% for the general population). These are all characteristics associated with that portion of the population that tends to have lower exposure to tobacco smoke. It appears that in the study group, only 13% had actual ETS exposure. These characteristics of the study group would tend

to bias the results towards no effect. Our concerns regarding the validity of the data, not an advocacy position, are the reasons we chose not to include the 16 Cities Study in this update.

8. Comment: Also, it should be noted that the 16 Cities Study reported personal exposures, and the work described in Hammond *et al.*, 1999 are area concentrations of ETS nicotine. As such, the two data sets are not comparable.

Response: The text has been amended to show that Hammond's measurements are of area concentrations. However, as shown in Figures 2 and 3 of Jenkins and Counts (1999), there appears to be a reasonably linear relationship between area and personal monitoring for nicotine, at least among restaurant servers and bartenders. It is likely that a similar relationship exists for office measurements as well.

9. Comment: Finally, the statement is made that personal exposure nicotine concentrations reported by Phillips *et al.*, 1998 in Prague are lower than in comparable studies. The reference to comparable studies is unclear. Do the author's mean compared to Phillips' other studies (most of which have, inexplicably, not been even cited by the report). Do the author's mean lower than the US 16 Cities Study? Whatever studies that are considered truly comparable to the Phillips work (large number of subjects, careful segregation of exposure types, breathing zone personal monitoring) need to be specifically cited here.

Response: The workplace nicotine data reported by Phillips *et al.*, 1998 for Prague are lower than those reported by Phillips and Bentley (2001) for Bremen (arithmetic mean 1.1 $\mu\text{g}/\text{m}^3$ versus 1.9-2.4) using comparable techniques. They are also lower than the range of workplace measurements (2-8 $\mu\text{g}/\text{m}^3$) reported in Table V-9 of the document that includes area measurements by Hammond (1999). Although personal breathing space and area monitoring are not strictly comparable, as mentioned in the response to comment #8, the two measures appear to be reasonably linearly correlated. The text has been modified to identify studies to which Phillips *et al.*, 1998 is compared.

10. Comment: On page V-58. "The validity of workplace nicotine levels has been challenged..." Which workplace nicotine levels? Those reported by Phillips for Prague? If the authors want to critique individual studies, then the criticism needs to be spelled out and it needs to be done for all studies that are included in the data analysis. My suspicion is that the authors are referring to a criticism of the 16 Cities Study (Jenkins *et al.*, 1996) published many months prior to the publication of the peer-reviewed manuscript. To include such comments without specifying the criticism gives a tone of apparent bias to the entire Report. Also, despite the fact that the data from the 16 Cities Study for Fresno (nicotine exposure and salivary cotinine levels that could have been analyzed) has been available for years (see the last page of Graves *et al.*, 2000,

or http://www.ornl.gov/sci/csd/Research_areas/ecms_rd_etsce_16cities.html), the authors of the Report did not analyze that data.

Response: As the commentor suspects, the workplace nicotine levels to which the sentence refers are those in the 16 Cities Study presented on the OSHA docket regarding an indoor air rulemaking in 1996. These are described in the responses to comment #7 above.

11. Comment: Finally, the analysis by LaKind *et al.*, 1999 of salivary cotinine levels from the 16 Cities Study shows median salivary cotinine levels for subjects only exposed in the workplace (Cell 3, Table V-15) of 0.347 ng/mL. When corrected for typical differences between saliva and serum cotinine levels, the levels reported by Pirkle *et al.*, 1996 for subjects exposed only in the workplace would be 0.40 ng/mL. To report a criticism of the 16 Cities Study by EPA regarding workplace nicotine levels, and then have the actual cotinine values reported by two independent groups be nearly indistinguishable makes no sense. This sort of biased data presentation jeopardizes the credibility of the Report, and calls other conclusions by the authors of the Report into question.

Response: The cotinine levels presented in LaKind *et al.*, 1999 reportedly represent the average of two measurements, one taken the evening before a 24-hour workplace measurement period (approximately one-half day), and the second, 24 hours after the workplace measurement period. As the authors recognize, a substantial fraction of the cotinine derived from workplace ETS exposure may have been excreted prior to the second measurement. The implication is that the actual workplace nicotine exposures may have been larger than suggested by the cotinine measurements. For individuals with ETS exposure in the workplace but not at home, whether or not the first cotinine sample was taken after a workday or after a weekend day could substantially alter the measured cotinine levels. It is thus unclear how well the median value of 0.347 ng/ml reported by LaKind reflects work exposure, and how this compares with Pirkle's geometric mean value of 0.318 ng/ml. Our concerns regarding the nicotine measurements remain.

12. Comment: On page V-59. The original data analysis of salivary cotinine and nicotine exposure from the US 16 Cities Study (Jenkins and Counts, 1999b) is not even cited in the references for the chapter. Also, the presentation of the cotinine data from NHANES III, reported in Pirkle, (1996), even though it is segregated such that it would be directly comparable to that reported by LaKind *et al.*, 1999 is missing from this analysis.

Response: The reasons for not including the 16 Cities study are addressed above in responses to comments #7 and #11.

13. Comment: In addition, the whole body of Phillips' work (eg, Phillips *et al.*, 1998, etc) is not referenced or discussed in the Report. This one page affords

several examples of inadequate literature review, reporting and analysis of the applicable scientific literature for this Report. It would be easy for the reader to draw the conclusion that if *these* key studies are not considered, *other* key investigations in other parts of the report have been ignored.

Response: Contrary to this commenters assertion, Phillips' work is cited or referred to several times, on pages V-55 thru V-58.

14. Comment: On page V-65. The authors need to clarify the relevance of maternal smoking biomarkers to the topic being discussed in the Report. Such is not evident on this page.

Response: Prior to the section in question, the report discusses various compounds, their utility as biomarkers of exposure, and their relative levels in adults. Arguably the discussion of maternal exposure to smoking could have followed at the end of section 3: Analytical methods for nicotine/cotinine. However, inasmuch as the exposure to smoke components in utero represent a more complex exposure scenario compared to that of an adult, it was decided that a separate section following the discussion of biomarkers in adults was appropriate.

15. Comment: In Chapter V of the report, there is a discussion as to “exposure to smokers” by considering the time spent around smokers. However, no data is presented to support the contention that time spent around smokers, or the detection by the human that they have been exposed to ETS, results in exposures that are relevant from a clinical or health standpoint. To mention exposure without detailing the effects of such exposure is irrelevant. To simply say that a person is exposed provides no useful information, because no perspective on the degree of exposure is provided.

Response: The ARB and OEHHA are required by Health and Safety Code Sections 39660 *et seq.* to evaluate the health effects of and prepare recommendations regarding substances which may be emitted into the ambient air in California. The draft report as a whole (Parts A and B) clearly shows that there are exposures to ETS in California and that there are adverse health effects associated with ETS exposures.

16. Comment: The comment is made that solanesol can not be a good marker for ETS outdoors because it degrades in sunlight is misleading since many other ETS constituents do as well. Based on National Academy of Sciences criteria for good markers, it would sound like solanesol would do a good job tracking those constituents that degrade in sunlight. The report should also consider that under standard protocols for analysis of nicotine and 3-EP, 4-EP eludes at essentially the same time and has been used by several laboratories for a standard.

Response: Our purpose was to show what markers have been used and what researchers have said about those markers. We did not use these markers in our analysis of exposure.

17. Comment: In a study by Djordjevic *et al.*, 2000 it is unclear how a discussion of carbon monoxide (CO) in mainstream cigarette smoke relates to ETS emissions.

Response: The Djordjevic study compared mainstream smoke from a Federal Trade Commission (FTC) machine-smoking test method to mainstream smoke generated by an actual smoker. Although ETS consists of thousands of compounds, the Djordjevic study focused on the mainstream smoke content of CO, nicotine, and tar from the FTC machine-smoking test method and actual smokers. The results presented in our report indicate that the results from both the machine-tested mainstream smoke (nicotine, tar, and CO) and the actual smoker are similar, although slightly lower for the FTC machine-smoking test method. We believe that these three compounds are a good comparison to what might be seen overall in ETS emissions.

18. Comment: There is a lack of data comparing ETS emissions with other sources regarding CO, nicotine, and RSP.

Response: The report has been revised to provide perspective on the contribution to ETS emissions on the statewide emission inventories.

19. Comment: It is unclear how ambient ETS emissions were calculated since all cigarettes are assumed to be smoked outside.

Response: Ambient ETS emissions are primarily based on California's cigarette distribution and emission factors (see Appendix B, Part A). Because no studies exist to quantify ETS emissions, ARB staff opted to estimate an indoor and outdoor upper limit. However, the report has been revised to further clarify the relative difference between indoor and outdoor ETS emissions.

20. Comment: Evidence is provided in the report to indicate that the constituents of ETS begin to react and decompose within short periods of time following their emission into the ambient environment. Clearly, ETS in ambient air in sunlight for any important length of time is no longer ETS. And yet the report, provides no justification or rationale as to why the use of existing regulations that establish safe concentrations of many of the compounds of interest in ETS is not an appropriate approach.

Response: In the report, we characterize ETS as a mixture of several thousands of compounds and recognize that complex chemical reactions take place immediately upon formation of ETS. However, it is the exposure to the entire mix that has been related to adverse health outcomes in many

epidemiological studies. From an exposure perspective, it seems clear that the public is exposed to the “mixture” of ETS. So, it is reasonable to consider ETS as a whole and not on the basis of individual effects from individual ETS compounds, as suggested by the commenter.

21. Comment: Page III-2: The statement: “...With few exceptions (e.g. hydrogen cyanide and organic acids), sidestream smoke contains greater mass emissions as compared to mainstream smoke (Jenkins *et al.*, 2000) on a per cigarette basis...” requires some additional explanation. The reason why SS smoke contains more material typically is because greater mass of tobacco is consumed during smoldering, compared with active puffing.

Response: Staff agrees and has revised the draft report to show that more sidestream emissions occur due to greater tobacco mass consumption during smoldering.

22. Comment: Page III-3: In the top paragraph (Page III-3), the text fails to make clear that most of the mainstream smoke that contributes to ETS is exhaled mainstream, that has been diluted in the lungs of the smoker, aged, and scrubbed of some of its more soluble gas components.

Response: We agree with the commenter. The report has been revised to add clarity.

23. Comment: Page III-4, last paragraph: The monograph to which the citation Jenkins *et al.*, 2000 refers did not involve any new experimental work. No measurements were made.

Response: The commenter is correct that no new data was generated in the referenced work. Staff have revised the report to reflect this fact.

24. Comment: Page III-5, first paragraph: The statement “...In general, highly concentrated mainstream smoke has constituents preferentially distributed in the particle phase region (Jenkins *et al.*, 2000). Smaller sidestream smoke particles in the ambient air can be inhaled deeply into the lower respiratory tract, where they can have a deleterious health effect...” Suggests a nearly binary distribution of tobacco smoke droplets (particles) between SS and MS smoke. However, given the huge breadth of the distribution, the distribution of both smokes should be considered as continuums. Also, the suggestion that somehow the slightly smaller particle size distribution of SS may result in more deleterious health effects is not supported in the scientific literature. While there are many differences that are statistically different in the distribution parameters, such as the mass median aerodynamic diameter, it is not altogether clear that there is a true functional difference in the two distributions. If there is evidence of this, then the authors need to cite such.

Response: In developing the citation above, the staff recognized that ETS has a broad particle size distribution. While some scientific literature suggests that a continuum exists between mainstream and sidestream smoke, other researchers have found some differences with particle distribution. In figure III-3, staff show data from Morowska *et al.*, 1997 indicating that there exists an apparent difference in the number of ETS particles, which fall either into the submicron level, or the supermicron level indicating a binary distribution among these two aerodynamic sizes.

The second part of the comment takes issue with the statement indicating that smaller particles in sidestream smoke have more deleterious health effects. In general, it is well known that inhalation of fine particulate matter (i.e. PM10 and smaller) is more harmful than larger particles as the fine PM reaches deeper down in the lung.

25. Comment: "...there is little attempt to discuss the rationale of using outdoor air markers (such as the iso-alkanes or ante-isoalkanes) as long term markers for ETS in ambient air when many of the components of ETS have relatively short half lives outdoors. This apparent inconsistency needs to be addressed.

Response: Staff included a discussion of iso- and ante-iso alkanes (pg. V-6) as potential ETS markers. Staff noted that these markers are more stable in outdoor air and have characteristic concentration patterns associated with tobacco leaf combustion. In this section of the report, staff fully recognizes that there are several ETS markers that have been used by researchers, each with their own pros and cons.

26. Comment: Page VI-1: The statement ".....Alternatively, as ETS ages, semi-volatile constituents of ETS, such as nicotine, may shift from particulate phase to gaseous phase...." seems to be incongruent with the latest scientific evidence regarding the state of nicotine in ETS. Most nicotine in fact is in the vapor phase of ETS (mainly emanating from sidestream smoke) as the ETS begins to form. A much better example of the shift from particle phase to vapor phase would be neophytadiene or n-C₂₇H₅₆.

Response: The scientific literature supports the notion that particulate phase nicotine converts to gaseous nicotine. See response to comment #5. Staff recognizes that other ETS particulate components also convert to gaseous components and will also include neophytadiene as an example of this chemical phenomenon.

27. Comment: Page VI-2: The data reported in Table VI-1 presents a large range of atmospheric lifetimes for known constituents of ETS. The reported range is from 5 minutes to 12 days. Given this data, and the likely reactivity of many of the other constituents of interest, it seems very hard to make a case that what we refer to as "environmental tobacco smoke" is likely to maintain much of

its character after a few tens of minutes in the outdoor air. Given such, one would have expected for the Report to provide some rationale as to why it is reasonable to consider ETS wholistically as a toxic air contaminant....Without a clear, strong justification as to why we should consider as some sort of single entity, when it is clearly not such, it would seem that the pollution which results from ETS best be considered on a constituent by constituent basis. Many of the compounds of interest are already regulated under a variety of regulations. No compelling evidence is provided for the case that ETS survives as an entity and should be considered as such.

Response: It is reasonable to consider ETS wholistically as a toxic air contaminant as it is emitted from a common source. The ARB has used this approach in the past when evaluating diesel exhaust as a toxic air contaminant. Diesel exhaust is also an example of a complex pollutant comprised of many individual compounds. Staff included data on the atmospheric persistence of individual ETS compounds because it is important to point out that the chemical nature of ETS has a temporal effect.

28. Comment: Data on indoor air from the 16 Cities Study (Jenkins *et al.*, 1996) should be included in the report. In particular, data from Fresno, California should be included.

Response: Published data from the 16 Cities Study has been added to the report. However, neither the Jenkins *et al.* nor the Graves *et al.* published papers provide results specific for Fresno, California, and ARB does not have the staff available to obtain the data set and separate out the Fresno data. Because of the sample bias and lack of representativeness of the Jenkins *et al.* sample (discussed further below), particularly relative to California exposures as discussed in the Report, we do not believe such an effort to be worthwhile.

29. Comment: A) The commentor questions citation of Graves *et al.* instead of Jenkins *et al.*, and also questions the statement that "...results are somewhat low relative to other similar studies...". B) Criticism of the demographic information presented in the Jenkins *et al.*, 1996 report is unjustified. The report fails to cite similar personal exposure studies and does not discuss the skewed demographics of other studies, such as Leaderer and Hammond (1991).

Response: A. Additional information from Jenkins *et al.*, 1996 has been added to the report. The results were viewed as somewhat low relative to other studies based on inspection of results of other studies of home nicotine measurements reported in Tables V-5 and V-6. For example, Guerin *et al.*, 1992 found that means across studies ranged from 1.6-21 $\mu\text{g}/\text{m}^3$ for homes with smoking, compared to a mean of 1.41 $\mu\text{g}/\text{m}^3$ in Jenkins *et al.*, for individuals exposed away from work, but not at work.), and in Table V-6, Hammond (1999) showed a range of 1.5 to 5.8 $\mu\text{g}/\text{m}^3$, and Glasgow *et al.*, 1998 a mean of 6.3 $\mu\text{g}/\text{m}^3$ in homes with smoking during the monitoring period.

B. While the Jenkins *et al.* study is unique in obtaining a sample from cities across the United States, the representativeness and utility of that sample was compromised by the multiple selection criteria for participants reported in the Jenkins *et al.* paper. For example, several groups in several broad employment categories were excluded, and a criteria for 75% of time spent in their personal workspace was included; these and other restrictions on those selected for participation resulted in a study population that over-represented white females and white-collar workers, and under-represented blue collar workers, minorities, and some other groups, because of the nature of their jobs. Such extensive exclusion criteria are generally not found in scientific studies without serious reason. Most importantly, it is unclear how to apply the results of the study to the California population, because of our substantial non-Caucasian minority populations. Regarding the lack of similar discussion for other studies such as Leaderer and Hammond, as indicated throughout the report, we do not specifically discuss individual studies conducted prior to 1996 because those have been discussed previously in the documents cited in the report. We agree with the commentor that we do not cite any studies of ETS exposure that achieved a truly demographically representative sample of the U.S. population, because to our knowledge no such study has been conducted.

30. Comment: ARB failed to incorporate several important studies of nicotine and PM concentrations in smoking environments in Tables V-6 and V-8 (p. V-17, Table V-6, p. V-24). A list of citations was provided.

Response: Staff reviewed the studies cited in the comments. The 14 studies by Keith Phillips are international studies from Europe (e.g., Britain, Germany, Spain), Asia (e.g., Hong Kong, Beijing, Kuala Lumpur), and the Pacific Islands (e.g., Australia). In these countries smoking behavior, cigarette formulation, housing characteristics, and non-smoker behavior may be very different than those in the United States and therefore would not be relevant to California indoor concentrations. Thus, they were not included in the report. Some of the Phillips work is used in the section on biomarkers.

Studies by Sterling *et al.*, 1996 and Jenkins *et al.*, 2001 discuss smoking exposure in one or two office buildings in the eastern U.S. where smoking is prevalent and unrestricted. These and a number of other studies were not specifically included in the report because of the limited new information provided and the desire to focus on information most relevant to exposures in California, where unrestricted office building smoking is no longer permitted.

Two of the listed studies, Trout *et al.*, 1998 and Maskarinec *et al.*, 2000 discuss employee exposures to ETS in casinos, restaurants, and taverns. These results may be relevant to workers in California casinos, and so have been added to the report.

31. Comment: (p. V-23, and others) Unpublished data was cited from Repace (2003). There is concern that the method used over-reports the RSP concentration by a factor of 2.

Response: The work by Repace was published in September 2004 in the *Journal of Occupational and Environmental Medicine*. The appropriate citation has been added. In his paper, Repace discusses the fact that humidity and particle size effects oppose each other when measuring RSP with the MIE personal Data-RAM (pDR-1200: Thermo Electron Corporation). He also provides a figure to show comparability of his method with a model 3511 piezobalance.

A. Judson Wells, February 10, 2004

1. Comment: Page III-4 and 5: There has been too little attention paid in the U.S. to the work of Pritchard *et al.*, *Environ Technol Lett* 1988; 9:545-552 ...on what happens to aged, diluted ETS. They... found that, during aging and dilution, 70% of the particulate ETS tar evaporates into the vapor phase. Vapor phase tar, like other organic vapors (Bond *et al.* *Toxicol Appl Pharmacol* 1985;78:259-267) would deposit quantitatively in the lung and the lung has no clearance mechanism for vapor phase deposits, whereas only about 15% of the particulates deposit on the lung, the remainder being exhaled. This phenomenon may explain why the passive risk is so similar to the active risk in non-contact sites like the heart and breast. It appears that the tar compounds that would evaporate would have molecular weights in the 100 to 200 range which would include quinoline, ethyl quinoline, benzoquinoline, phenanthridene, nor nicotine, beta-naphthyl amine, nitroso pyrrolidine, nitroso nor nicotine, pyrene, fluoranthrene, phenol, the cresols, 2,4-dimethyl phenol, catechol, and the methyl catechols, all of which have some carcinogenic activity.

Response: Staff agrees with the commenter and have revised the report to include the findings of Pritchard *et al.*, 1988.

Maurice E. LeVois, Ph.D., March 25, 2004

1. Comment: The draft report presents in Part A, Appendix A List of Known ETS constituents, a list of constituents of mainstream and sidestream smoke rather than constituents of ETS. This is a misleading title that should be corrected. Table III-1 and Table III-2 list constituents that have actually at least been qualitatively measured in ETS. The draft report also notes that some chemical constituents of sidestream smoke are produced in higher concentrations than in mainstream smoke. This is true, but it is no basis for concluding that risk estimates based upon spousal smoking associations are plausible when compared to active smoking risk estimates. That "cigarette equivalent" exposure comparison should be based upon a comparison of actual

mainstream smoke and ETS exposure levels, not upon a comparison of constituent levels in mainstream smoke with levels in fresh, distilled and concentrated sidestream smoke. Environmental tobacco smoke is aged, diluted and dissipated in natural environments and is not the same as sidestream smoke. Most sidestream smoke constituents are transformed to such low concentrations that they are no longer quantifiable in ETS.

Response: As indicated by the references at the end of Appendix A, the list of known ETS constituents was taken from several studies which identified numerous compounds in ETS. The purpose of the list is to compile a list of known constituents that could be generated as tobacco products (i.e. cigars and cigarettes) are consumed. The staff did not present the list as all-inclusive and does not agree that the title is misleading. Furthermore, the compounds that are listed in both Table III-1 and Table III-2 represent those ETS components for which known health effects have been determined. The tables are shown to illustrate that several ETS constituents have been found to be harmful as individual compounds. However, the health effect evaluation conducted by OEHHA in Part B of the staff report do not distinguish between the health effects of individual compounds, but rather the effect of the total “mix” of compounds that make up ETS.

Robert F. Phalen, University of California, Irvine, March 1, 2004

1. Comment: Identification of ETS as a TAC will ultimately lead to more violence in bars and other establishments.

Response: If ETS meets the criteria for designation as a TAC, then it is the Board’s responsibility to determine if it should be identified as one. This will occur only after a full public process which provides a full scientific debate of the issues. Furthermore, authoritative reviews over the past two decades have presented scientific evidence linking ETS exposures to a number of adverse health outcomes. These reviews were endorsed by organizations/agencies such as the U.S. Environmental Protection Agency, U.S. Department of Human Health Services, National Research Council, and the International Agency for Research on Cancer.

Further, no control measures are being proposed in this report. If a substance is identified as a TAC by the State ARB, it will enter into the control phase of the program. Any consideration of control measures will be made only after a thorough public process including public workshops, meetings with affected parties, and local air pollution control districts.

**Peter N. Lee, P.N. Lee Statistics and Computing LTD.,
March 11, 2004**

1. Comment: My paper is cited as "P.N. Lee, 1999" when all the other references in the Draft do not give initials on page V-61. The reference on page V-78 is also not in its correct alphabetical order.

Response: We have corrected the citation to read Lee, 1999 and have put the reference in the correct order on page V-78.