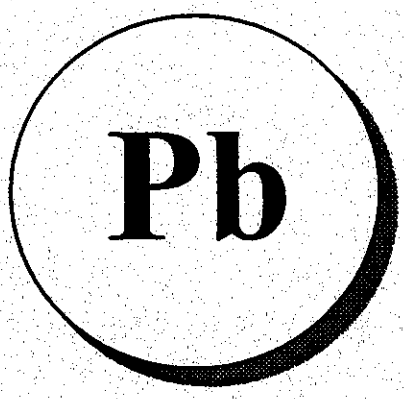
California Environmental Protection Agency

Air Resources Board

Proposed Identification of Inorganic Lead as a Toxic Air Contaminant



Part C
ARB/OEHHA
Staff Responses to Comments

Stationary Source Division
September 1996

PART C

PUBLIC COMMENTS AND ARB/OEHHA STAFF RESPONSES ON THE PROPOSED IDENTIFICATION OF INORGANIC LEAD AS A TOXIC AIR CONTAMINANT

Prepared by the staff of the Air Resources Board
and the Office of Environmental Health Hazard Assessment

March 1997

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PART C

Comment Letters Received on the Proposed Identification of Inorganic Lead as a Toxic Air Contaminant September 1996 Draft Version of the Executive Summary, Part A Exposure Assessment, and Part B Health Assessment

Gradient Corporation
AN ENI COMPANY

October 22, 1996

Dr. Joan Denton
Stationary Source Division – Air Resources Board
Attention: Inorganic Lead
2020 L St.
Sacramento, CA 95814

Dear Dr. Denton,

The enclosed text has been written to rebut California EPA (Cal EPA) responses to report comments submitted by Gradient Corporation on behalf of ARCO. The original Gradient comments were submitted in March of 1996. We request that these rebuttals be considered by the Scientific Review Panel at its public meeting scheduled for October 31, 1996.

While Cal EPA has written extensive responses to many of our comments, we still find the Agency's position unconvincing. We have therefore written comprehensive rebuttals to these responses. Our original comments, as well as these rebuttals, fall into four categories:

- The aggregate model overstates the impact of decreasing atmospheric lead concentrations on blood lead levels (Gradient Comment 1): Cal EPA has incorrectly used an aggregate model that implicitly assumes decreases in atmospheric lead levels will instantaneously decrease lead exposure both *via* inhalation and *via* ingestion of dust and soil contaminated by lead deposited from the air. We argue that decreasing atmospheric lead levels will not change soil and dust lead concentrations for an extended period, and hence, Cal EPA should have used the disaggregate model that quantifies the impact of atmospheric lead changes alone on blood lead levels. Use of the disaggregate model would reduce the magnitude of the predicted association between changes in atmospheric lead levels and blood lead levels by a factor of approximately 2.5.

- The IEUBK model overstates blood lead levels (Gradient Comments 2 and 3): Cal EPA may be correct that the IEUBK model better predicts blood lead levels for typical urban populations than it does for populations cited in our examples (*e.g.*, populations near mines). However, there is limited documentation to support this hypothesis. Moreover, use of the aggregate model, which is built into EPA's IEUBK model, will still overstate the impact of changes in atmospheric lead levels on blood lead levels, as described in our first comment. Finally, the differences between observed and IEUBK-predicted blood lead levels are often too large to be explained by "measurement error" affecting empirical values. Thus, Cal EPA's modeling has overstated the impact of decreases in atmospheric lead levels on blood lead levels.

- Cal EPA uses overly conservative parameter values to characterize blood lead levels in California (Gradient Comments 4-9): Our comments focus on the geometric mean (GM) and geometric standard deviation (GSD). The Agency's GM estimate reflects outdated NHANES III data collected between 1988 and 1991, a period prior to the complete phase-out of leaded automobile gasoline and the complete phase-out of lead solder used in the manufacture of food

44 Brattle Street, Cambridge, MA 02138 • (617) 576-1555 • Fax: (617) 864-8469
Email: info@gradcorp.com

cans. More recent studies indicate blood lead levels in California are lower than levels reported in NHANES III. By using too large a GM, Cal EPA overstates the impact of lowering lead exposure on the fraction of children with blood lead levels exceeding the CDC's concern threshold of 10 μg/dL. Cal EPA also overstates the likely range of blood lead levels (quantified by the GSD) in communities living near point sources (*e.g.*, smelters). By overstating the GSD, the Agency overstates the absolute fraction of children with blood lead levels exceeding the CDC's threshold of concern. This absolute fraction indicates how important the lead exposure problem is in the California population and hence should be taken into account when considering the proposed rule.

- Cal EPA overstates the association between lead exposure and hypertension (Gradient Comments 10-14): The literature on this association is far more controversial than Cal EPA portrays it to be. Moreover, Cal EPA uses a high-end estimate for the magnitude of this association. Overall, Cal EPA has provided an estimate of cardiovascular risk that is highly uncertain and a likely overestimate. Moreover, even the Agency's high-end risk estimates are not substantial. This risk assessment is therefore an inadequate basis for decision-making. Given the tenuous nature of the association between lead exposure and hypertension (and hence cardiovascular disease), these risks should not be given much consideration when evaluating the proposed rule.

Our rebuttal comments appear in the same order as they have been presented by Cal EPA in the Agency's September, 1996 response. We hope that our comments help the SRP to better evaluate the technical issues surrounding consideration of the proposed rule.

Sincerely yours,

GRADIENT CORPORATION

Barbara D. Beck

Barbara D. Beck, Ph.D., DABT
Principal

e-mail: bbeck@gradcorp.com

Rebuttal to Cal EPA Responses to Gradient Comments on the Proposed Cal EPA Rule Regarding Atmospheric Lead

Comment 1 (p 362)

The aggregate approach used by Cal EPA to estimate the change in blood lead levels associated with an incremental change in atmospheric lead concentrations overstates the magnitude of the air lead – blood lead slope. Specifically, Cal EPA should not assume that changes in atmospheric lead levels will result in changes in soil lead levels and dust lead levels as well since lead in soil and dust persists for a long period of time. The disaggregate approach isolates the impact of atmospheric lead level changes on blood lead levels and yields a slope factor that is smaller than Cal EPA's slope by a factor of approximately 2.5 (*i.e.*, the correct value should be approximately 1.7 μg/dL per $\mu g/m^3$ lead in air, rather than Cal EPA's recommended value of 4.2 μg/dL per $\mu g/m^3$ lead in air.

Cal EPA's Response

Cal EPA's response begins by acknowledging that the "aggregate slope model may overestimate the impact of certain exposure scenarios" (p 362). The Agency also admits that "A similar situation could also occur with the IEUBK model" (p 362), thus conceding potential problems with both approaches used by Cal EPA to calculate this association. Specifically, Cal EPA goes on to say that "when remediation of air risks are undertaken, the full reduction of risks predicted by the models and the supplemental equations may not occur due to the persistence of lead in soil" (p 362).

Cal EPA offers only three tentative lines of defense for the Agency's estimate of 4.2 μg/dL per $\mu g/m^3$ lead in air.

1. Cal EPA states that "soil in an urban environment is subject to several variables (wind, runoff, and mixing) and can vary significantly under different circumstances" (p 362). They then say that "When redistribution of lead occurs relatively fast, the models would not necessarily overstate risks from reductions in air lead, but would overstate risks if redistribution were very slow" (p 362).

2. Cal EPA refers to chamber studies that report a slope factor exceeding 1.7 μg/dL per $\mu g/m^3$ lead in air.

Rebuttal

In response to Cal EPA's first argument: The Agency correctly notes that if (hypothetically), redistributions of lead (in soil and dust) occur very quickly, the Agency's model and equations would not overstate risks. However, Cal EPA does not provide a compelling argument as to why this hypothetical phenomenon may occur (other than hinting at mechanisms that may be relevant in an urban environment). Nor does Cal EPA provide any evidence that such quick redistributions have ever been documented anywhere. Our original comments note three sites at which US EPA has insisted on cleanup of soil long after an atmospheric lead source has been eliminated. It therefore seems that the norm is for

soil lead levels to persist for extensive periods of time (or, at most, to decline relatively slowly) and hence Cal EPA's hypothetical scenario of a quick redistribution has no basis in reality.

In response to Cal EPA's second argument. Cal EPA does not cite the study or studies to which the Agency is referring. We therefore have no way to judge the applicability of these studies, their validity, or whether they are even representative of the literature. Moreover, Cal EPA does not even offer an alternative value for the slope factor of 1.7 μg/dL per $\mu g/m^3$ lead in air that we have suggested.

Since Cal EPA fails to refute our argument for a substantially weaker association between air lead levels and blood lead levels, and because all of the risks claimed by Cal EPA are expressed as a function of changes in blood lead levels, the conclusion that the magnitude of these risks must be reduced by at least a factor of 2.5 cannot be rejected.

Comment 2 (p 362-364)

We have claimed that the EPA's IEUBK model overstates estimated blood lead levels.

Cal EPA Response

Cal EPA cites recent validation exercises that claim to demonstrate that the model closely predicts the community distribution of blood lead levels. Moreover, Cal EPA claims that the examples we cite may not be applicable because the cause of the overprediction may be attributable to the failure to use a reduced bioavailability value in the model to reflect the lower bioavailability of lead typically found in mining areas.

Rebuttal

We note that there has been no extensive validation of the IEUBK model in typical urban settings that are a main focus of Cal EPA's risk assessment. Moreover, even if the model were to correctly predict blood lead levels in communities where lead levels in various media have reached equilibrium, it does not follow that the model correctly predicts the impact on blood lead levels associated with decreases in atmospheric lead levels when equilibrium has not been reached. In fact, the IBUEK model does overstate the impact of decreases in atmospheric lead levels on blood lead levels since the aggregate model challenged in Comment 1 above is built into the IEUBK software. That is, even concession of Comment 2 does not affect our contention under Comment 1 above.

Comment 3 (p 364)

Based on differences between the observed and IEUBK predicted blood lead levels observed at Bingham Creek, we claim that the IEUBK model overpredicts blood lead levels.

Cal EPA Response

The referenced differences between observed and IEUBK predicted blood lead levels is small and may reflect measurement error. Hence, this comparison does not, *per se*, demonstrate that the IEUBK model overpredicts blood lead levels.

Rebuttal

For the purpose of this proposed regulation only, we will not contest this point, except to say that the differences between observed and predicted blood lead levels may be real. For example, Table 1-1 in our original comments (see p 278 of Cal EPA's September, 1996 submission) documents substantial differences between observed and IEUBK-predicted blood lead levels. For example, in Leadville, CO, the model predicted 41% of the children would have blood lead levels exceeding 10 µg/dL; the observed fraction of children with blood lead levels exceeding this threshold was only 8.2% – approximately 1/5 the predicted value. In Butte, MT, the predicted fraction of children with blood lead levels above 10 µg/dL (16.8%) also greatly exceeded the observed fraction (5.1%).

We also note that it is not relevant that the difference between observed and IEUBK-predicted blood lead levels is small relative to the size of measurement error since these errors tend to cancel when averaged over a large population. Moreover, we agree, as Cal EPA notes in the Agency's response to comment 2, that any differences between predicted and observed blood lead levels may reflect use of parameter values for lead bioavailability that are inappropriate in the context of these communities.

In any case, even if the model does adequately predict blood lead levels in general, our point that it does not correctly predict the impact of a change in atmospheric lead concentrations on blood lead levels still stands, as explained in our rebuttal under comment 1.

Comment 4 (p 364-365)

Cal EPA claims that we have objected to three aspects of the Agency's characterization of the population blood lead distribution: 1) the concern threshold of 10 µg/dL; 2) the shape of the blood lead level distribution (*i.e.*, that it is lognormal); and 3) the magnitude of the geometric standard deviation, claimed by Cal EPA to be approximately 2.1, and by us to be no larger than 1.8.

Cal EPA Response

The concern threshold: Cal EPA responds that the 10 µg/dL concern threshold is consistent with a wide range of federal guidance.

The lognormality of the population distribution: Cal EPA offers two responses on this point. First, the Agency notes that "it is generally accepted that blood lead concentrations are lognormally distributed" (p 365). Second, the Cal EPA presents results of an alternative statistical test that shows that the log-transformed blood lead values with the cumulative normal distribution values corresponding to the rank of each value.

The magnitude of the geometric standard deviation: Cal EPA asserts that a geometric standard deviation of 2.14 (as reported by NHANES III) appears appropriate "Given the heterogeneous population in California as a whole and in the general urban environment..." Cal EPA does admit that this reasoning "should not preclude the use of another GSD for estimates pertaining to a local site or facility-specific scenario" Finally Cal EPA claims that changing the assumed value of the GSD does not substantially affect the predicted impact of changes in atmospheric lead levels on the fraction of children with blood lead levels above 10 µg/dL.

Rebuttal

The concern threshold: We do not contest the concern threshold in this document. The two statistics we were referring to were the geometric standard deviation and the geometric mean. We address the latter under Comment 6 below.

The lognormality of the population distribution: Although it is "generally accepted" that population blood lead level distributions are lognormal, that is not justification, *per se*, for using this distribution in much different settings. It must be recalled that our criticism of the use of the lognormal distribution pertained to its use to describe blood lead levels in a population "living near a point source" (see the title of our Section 1.2.2, located on page 281 of Sept 1996 Cal EPA documents). It is likely that such populations are more homogenous and hence that the distribution's skew is less, perhaps making the use of the lognormal inappropriate. Certainly, extrapolating from a national sample to a hypothetical community living near a point source is questionable.

Moreover, we still question whether even the national NHANES III blood lead data are lognormal, based on the statistical results quoted by Cal EPA. The original statistical test used by Cal EPA nearly rejected the hypothesis of lognormality ($p = 0.07$). That the Agency has found a statistical test that allegedly supports the hypothesis of lognormality does not eliminate the Agency's original finding. Choosing the second test's results and ignoring the results from the first test without explanation is arbitrary.

In any case, Cal EPA does not contest our argument that it is invalid to extrapolate the lognormality assumption from the nationally representative NHANES III dataset to a hypothetical community near a point source. To the extent that the lognormality assumption is unfounded, the risk estimates presented by Cal EPA for such communities are invalid.

The magnitude of the geometric standard deviation: We essentially do not object to Cal EPA's response to our comment on this issue. However, we believe that the Agency should further emphasize in its Health Assessment that since communities near point sources probably have lower GSDs (as Cal EPA admits in the Agency's response), the absolute fraction of individuals with blood lead levels exceeding the CDC concern threshold of 10 µg/dL is smaller than the fraction implied by use of the GSD of 2.14 inferred from the NHANES III dataset.

Comment 5 (p 365)

We claim that Cal EPA overstates the population blood lead levels and geometric standard deviations.

Cal EPA's Response

Cal EPA restates comments in our original report, namely that 1) OEHHA used data from NHANES III, and these data were collected between 1988 and , 1991; 2) Lead contamination has been reduced since that time (the completed phase out of leaded gasoline and the completed phase out of the use of lead in the manufacture of food cans); and therefore 3) Geometric mean blood lead levels may now be lower.

Rebuttal

Cal EPA appears to agree with us, so far as their response to this comment is concerned. However, see Comment 6, below.

Comment 6 (p 365-367)

We claim in our original report that the first phase of the NHANES III report, which reflects data collected between 1988 and 1991, is out of date and that Cal EPA should not use those data to establish a baseline blood lead level because lead exposures have decreased since that time. Specifically, the phase-out of leaded gasoline, and the phase-out of lead solder used in the manufacture of food cans, have both been completed. In its place, we identify a survey conducted between 1992 and 1993 of blood lead levels in Californian children receiving Medicaid benefits. Assuming a population geometric standard deviation of 2.1, the reported fraction of children with blood lead levels exceeding 10 μg/dL (2%) indicates that the geometric mean blood lead level 2.2 μg/dL. This geometric mean is substantially less than the NHANES III geometric mean blood lead level of 4.1 μg/dL for children.

We also claim the geometric standard deviation used by Cal EPA is too large. This issue was discussed earlier (see Comment 4).

Note: The last paragraph of Cal EPA's summary of this comment incorrectly uses the term "geometric standard deviation" where we have used the term "geometric mean."

Cal EPA Response

We restrict attention to the discussion of the geometric mean, since, as just noted, the geometric standard deviation was discussed earlier (see Comment 4).

Cal EPA objects to our use of the 1992-1993 Medicaid recipient survey of blood lead levels for the following reasons:

1. The study we cited did not report a geometric mean or geometric standard deviation;

2. The population discussed in our comment is the population of 1 to 6 year olds, a group less likely to have high blood lead levels than 1 to 2 year olds, the population that is the subject of Cal EPA's risk assessment.

3. The editors of the article that reports our survey note several possible reasons for the low blood lead levels found in the survey, including:

   (a) The study was conducted in the winter months when lead exposure is less severe.

   (b) The results may have reflected differences in study design.

4. Cal EPA claims that sensitivity analysis indicates that the Agency's findings are insensitive to the precise value of the geometric mean.

The remainder of the Cal EPA response addresses GSD issues that we have discussed under Comment 4.

Rebuttal

We address each of Cal EPA's responses, in turn.

1. While the study we cited did not report a geometric mean or geometric standard deviation, it did report the fraction of individuals with blood lead levels exceeding 10 μg/dL. Using Cal EPA's assumptions of lognormality and a GSD of 2.1, the only possible value for the geometric mean for population blood lead levels is 2.2 μg/dL. Cal EPA must therefore choose between our calculated geometric mean and its assumptions of lognormality and a GSD of 2.1. Giving up these assumptions undermines the Agency's entire risk assessment.

2. While the population discussed in the study we describe focuses on 1 to 6 year-old children, we note in our original text (see p 281 of Cal EPA's response document) that the fraction of 1 to 2 year-olds with elevated blood lead levels (1.7% for 1 year olds and 2.2% for 2 year olds) is at least as small as the fraction of 1 to 6 year-old children with elevated blood lead levels (2%). Hence, the data do not support Cal EPA's contention that, at least in this population, 1 to 2 year olds have higher blood lead levels than 1 to 6 year-olds.

3. (a) It is true that the study collected data during the winter months, a factor that would tend to decrease blood lead concentrations. However, it is unlikely that summer blood lead concentrations are high enough to increase the annual average geometric mean blood lead concentration to Cal EPA's 4.1 μg/dL. In order to have an annual average geometric mean of 4.1 μg/dL, the summer geometric mean would have to be approximately 6 μg/dL in order to balance the "winter" geometric mean of approximately 2 μg/dL.

   There are at least two reasons to believe this is not the case. First, seasonal changes in lead exposure do not fluctuate to this extent (*i.e.*, by a factor of 3). Results from a study of children living in Port Pirie, Australia (Baghurst *et al.*, 1985) revealed maximum seasonal changes of approximately 13%. These changes were observed in children aged 15 months. Seasonal changes in other age groups were substantially smaller (6% for 6 month olds, and 1.4% for 24 month olds). In a cross-sectional study in New York City entailing 170,000 blood lead measurements over a 6-year period among children of the same age, Billick *et al.* (1979) report that summer blood lead levels exceeded winter blood lead levels by 10-15%. Another cross-sectional study of 8,000 children in New Haven, Connecticut conducted over a 2.5 year period (Marrero *et al.*, 1983) identified peak blood lead levels in both summer and later winter that exceeded late spring and fall values by 10% to 30%. More recently, Johnson *et al.* (1996) report that blood lead levels in a population living in Syracuse, New York fluctuated from a geometric mean of 7.20 to a geometric mean of 9.01. This difference amounts to a 25% fluctuation. However, it must be noted that the Johnson *et al.* cohort was not randomly selected. Instead, the children in this study were largely from high risk areas where children were known to have high blood lead levels. It is possible that these children

were disproportionately exposed to lead in soil and dust, and hence more susceptible to seasonal fluctuations. In any case, none of the seasonal fluctuations observed in any of these studies is sufficiently large to explain the difference between the low geometric mean blood lead level reported in the MWR study cited in our original comments.

Second, we would expect seasonal variation in California to be even less than it is in other parts of the country since the year-round temperate climate in California ensures that exposure to lead in soil is not interrupted during the winter months. In order to discount the data from this study using the seasonal variation argument, Cal EPA must be required to demonstrate that seasonal exposure fluctuations (especially in California) are large enough to make plausible the claim that the NHANES III geometric mean of 4.1 μg/dL is still valid.

There are several other relevant points here. First, Cal EPA's new text in Section 5.1.D notes that geometric mean blood lead level for the Western Region of the U.S. reported by NHANES III is 2.9 μg/dL, somewhat lower than the 3.6 μg/dL value for the nation as a whole. The fraction of children with blood lead levels exceeding 10 μg/dL in the Western Region is only 3.5%, compared to 8.9% for the entire country. Cal EPA notes that blood lead levels in California may be closer to the national average than blood lead levels in other states in the Western Region that are more rural (*e.g.*, Colorado). However, the degree of urbanization (or lack thereof) in the Western Region of the U.S. cannot explain the lower blood lead levels observed in that region. While we cannot find documentation specifying which states belong to each of the four NHANES III defined geographic regions, data published by the U.S. Bureau of the Census (1994, Table 44) indicate that the Western region (comprised of MT, ID, WY, CO, NM, AZ, UT, NV, WA, OR, CA, AK, and HI) is more urbanized (86.3% of the population) than any other region (78.9% for the Northeast, 71.7% for the Midwest, and 68.6% for the South). Hence, if fraction of the population living in urban regions had a strong influence on blood lead levels, we would expect the geometric mean for the Western Region to be greater than the geometric mean for other regions or the nation as a whole. The fact that the Western Region has a lower geometric mean than these other areas casts doubt on Agency's hypothesis that urbanization in California would substantially inflate blood lead levels in that state relative to other states in the NHANES III Western Region. The Agency also notes that blood lead levels in the Western and Southern Regions were collected during the winter, which could explain why the Western region results are lower than results from other parts of the country that were surveyed during the summer. However, as noted above, seasonality should not make as great a difference in California (and the South) as it does in colder climates where winter weather does limit access and hence exposure to lead in soil.

Section 5.1.D goes on to describe several other studies of childhood blood lead levels in California. Cal EPA states that "they are not necessarily representative of the state's population" (p 5-6). Nonetheless, the studies do support the contention that the 1988-1991 NHANES III blood lead data overstates contemporary blood lead levels. For example, despite the aforementioned qualifications to the 1992-1993 study of children attending a Medicaid clinic, Cal EPA notes that the results "may be indicative of a continuing downward trend in children's blood lead levels observed between NHANES II and NHANES III and in the Los Angeles air basin from 1991 through 1994 (Williams

et al. 1996). The complete phase-out of lead from automotive gasoline and other measures taken by California to remove lead from the environment, drinking water, tableware, and food containers are all likely to result in lowering blood lead levels in the near future. Based on these findings, we believe Cal EPA should add to its conclusions that it is likely that blood lead levels are lower now than suggested by the NHANES III results, and the Agency should quantitatively describe how changes to the assumed geometric mean blood lead concentration affect its predicted risk estimates.

(b) Cal EPA does not explain how "differences in study design" would yield a downward bias of any magnitude, and the Agency certainly does not explain the substantial difference between the NHANES III geometric mean of 4.1 μg/dL and the geometric mean inferred from the Medicaid study data of 2.2 μg/dL.

4. We have quantified the extent to which changes in the geometric mean, along with changes to other unsupported assumptions, affect the predicted risks. Cal EPA should present its own estimates of how changes to the geometric mean affect predicted risks. Qualitative statements in the Agency's response that claim sensitivity analysis shows the predicted risks are not sensitive to changes in the geometric mean are not sufficient.

Comment 7 (p 367)

We present a recalculation of the fraction of individuals with blood lead levels exceeding 10 μg/dL. Our results differ somewhat from those presented by Cal EPA.

Cal EPA Response

The differences reported in the comment are attributable to a rounding error. The Agency used the actual GSD reported by NHANES III of 2.14, rather than 2.1, as suggested by the original text. A footnote will clarify this point.

Rebuttal

This refinement is acceptable to us. However, we note that our comment, brought up again by Cal EPA that, "the use of alternative GSDs does not affect the change in the relative fraction of children that will move above 10 μg/dL due to change in air lead" (p 367) should not be taken out of context. Our comment is intended to convey that changing this parameter alone has a relatively small effect on the predicted risk. However, the impact of alterations to all uncertain or unsupported assumptions (including the value of the geometric mean and the relationship between air lead levels and blood lead levels) must be considered simultaneously. Moreover, the assumed magnitude of GSD affects estimate of absolute risk, meaning the baseline fraction of the population with blood lead concentrations exceeding 10 μg/dL. We return to this point under Comment 8.

Comment 8 (p. 367-368)

The absolute fraction of individuals in the population with blood lead levels exceeding the threshold of concern is of public health interest. By using an inflated estimate of the geometric standard deviation, Cal EPA incorrectly overstates this fraction.

Cal EPA Response

Cal EPA states that the baseline "severity" of lead exposure in California is uncertain, with the fraction of children with elevated blood lead levels (*i.e.*, above 10 μg/dL) ranging from 2.5% to 10.9%. This range is based on the set of baseline blood lead values projected by three models in Figure 5-3 of the revised draft (the Aggregate model, the IEUBK model calibrated using data from East Helena, and Aggregate IEUBK model). Because the baseline level is uncertain, Cal EPA chooses to focus on the relative contribution of atmospheric lead to blood lead levels.

Rebuttal

First, Cal EPA overstates the uncertainty in the baseline "severity" of lead in exposure. Although the three models used by the Agency are uncertain, empirical data have been collected that demonstrate that the fraction of children with blood lead levels exceeding 10 μg/dL is likely to be very small (less than 5%). Here, we again appeal to the Medicaid clinic study discussed in Comment 6 (approximately 2% of children have blood lead levels exceeding 10 μg/dL), or even to the NHANES III Western Regional results (approximately 3.6% of the children had blood lead levels exceeding 10 μg/dL). Federal EPA guidance indicates that when the fraction of children with blood lead levels above 10 μg/dL is so small (less than 5%), lead exposure is acceptable. Cal EPA should acknowledge this so that the reader can understand the appropriate level of priority that should be placed on this public health issue.

Comment 9 (p 368)

We point out that the three models used by Cal EPA to predict the impact of changes in atmospheric lead levels on the fraction of individuals with blood lead levels exceeding 10 μg/dL differ substantially. Hence, Cal EPA cannot claim that different models yield essentially the same results and hence that the risk assessment results are robust.

Cal EPA Response

Cal EPA claims that we chose a worst case comparison by comparing the models' predicted change in the fraction of individuals with blood lead levels exceeding 10 μg/dL associated with a 1 $\mu g/m^3$ change in atmospheric lead levels. The differences are much smaller when comparing the models' predictions for smaller changes in atmospheric lead levels – *e.g.*, 0.06 $\mu g/m^3$, the ambient level in the state.

Rebuttal

In absolute terms, Cal EPA is right: the differences among the models' predictions are smaller when assessing the impact of smaller changes in atmospheric lead levels. However, the relative differences are at least as large. Figure 5-3 in the revised Cal EPA draft suggests the Aggregate IEUBK model predicts an increase in atmospheric lead levels increases the fraction of the population with blood lead levels above 10 μg/dL by approximately 3% to 4%, whereas the other two models predict such a change in atmospheric lead levels would increase this fraction by 10% to 11%. The relative differences among the model predictions span a factor of as much as approximately 4. These differences can hardly be considered small when the results of the risk assessment depend in a linear manner on the magnitude

of these results. That is, the predicted risks may span a factor of 4 depending on which model is selected. This level of uncertainty suggests Cal EPA's risk assessment is far from robust.

Comment 10 (p 368 - 369)

This comment addresses the alleged existence of an association between blood lead concentrations and diastolic blood pressure. We focus on diastolic blood pressure rather than systolic blood pressure since Cal EPA's risk assessment quantifies an association between diastolic blood pressure and adverse cardiac events.

Our original comment points out that at least one major study of the association between blood lead levels and blood pressure (Dolenc *et al.*, 1993) found a negative correlation between these two quantities (*i.e.*, a protective effect associated with blood lead levels). We therefore conclude that the existence of such an association is uncertain at best.

Cal EPA Response

Cal EPA claims that "the weight of evidence suggests an association between lead exposure and blood pressure" (p 368). The Agency cites the conclusion of the National Research Council, as well as the animal studies, mechanistic results, and "the moderate concordance of effect size..." (p 368) (see our discussion on this point in Comment 11 below). Finally, Cal EPA points out that the Cadmibel studies that we cite report contradictory results, inconsistencies that may reflect different model specifications, or the presence of highly correlated variables. Since no other studies report a "protective" association between blood lead levels and blood pressure, Cal EPA argues that the existence of an effect cannot be dismissed.

Rebuttal

The argument over the existence of an adverse association between increased blood lead levels and blood pressure is, by its nature, qualitative. We believe that Cal EPA has overstated the case for the existence of such an association, casting the Agency's conclusions in terms that are too strong.

While the Dolenc *et al.* (1993) study is the only investigation to report a negative association between blood lead levels and blood pressure, Cal EPA does not adequately highlight the fact that numerous studies have failed to find any association between blood pressure and, especially, diastolic blood pressure. The following table indicates that even among the studies cited by Cal EPA, a substantial portion fail to identify a statistically significant association.

Cal EPA Section 3.2 Human Studies of the Association Between Blood Lead Levels and (Diastolic) Blood Pressure

Study	Findings: The Association Between Blood Lead Levels and Diastolic Blood Pressure[a]	Data Set and Comments
Harlan, 1985	S	NHANES II
Schwartz, 1985a,b; 1986a,b; 1988	S	Reanalysis of Pirkle *et al.* (1985), which was based on NHANES II
Landis and Flegal, 1988	S	NHANES II
Gartside, 1988	NS	NHANES II
Coate and Foles, 1989	S	NHANES II
Sorel *et al.*, 1991	S	NHANES II.
Schwartz, 1991	S	NHANES II
Pocock *et al.*, 1988	S	British Regional Heart Study
Neri *et al.*, 1988	NS	Canada Health Survey; Significant association reported for model without all potential confounders.
Moller and Kristensen, 1992	NS	Population from Copenhagen, Denmark. Significance reported for female subjects
Hense *et al.*, 1993	NS	Augsburg, Germany
Orssaud *et al.*, 1985	NR[c]	Paris, France
Moreau *et al.*, 1988	NS	129 adult males
Kromhout, 1985; 1988	NS	152 males, Zutphen, the Netherlands
Morris, 1990	S	251 males in a clinical trial
Apostoli *et al.*, 1992	S	254 Italian males and 271 Italian females
Menditto *et al.*, 1994	S	1,800 men from Rome, Italy
Sharp *et al.*, 1988	S	342 San Francisco Bus Drivers
Weiss, 1988	NS	89 Boston policemen followed over time
Kort, 1987	NR[c]	Occupational exposure
Weiss *et al.*, 1988	NS	Occupational exposure
Egeland *et al.*, 1992	NS[d]	Occupational exposure
Rabinowitz, 1987	NA[b]	Pregnant women
Staessen *et al.*, 1991; 1993; 1996	NS	Two locations in Belgium
Hu, 1996	S	US Armed Forces Veterans

Notes:

(a) *S indicates statistical significance;*
NS indicates not statistically significant;
Results are reported for the final model only.

(b) *NA indicates that the study did not evaluate the relationship between blood lead levels and diastolic blood pressure for men.*

Where results are reported for men and women, we report results for men only since it is this population that is the subject of Cal EPA's risk assessment.

(c) *NR – Relevant results could not be ascertained from the Cal EPA report (not reported); nor could the article be located.*

(d) *The Egeland et al. study found a significant association between blood lead levels and diastolic blood pressure only in study subjects occupationally exposed to Carbon disulfide, and even in this group statistical significance was marginal ($p=0.06$). We judged this finding to be week support for the hypothesis of an association and hence judged the association to be statistically not significant.*

Of the 22 studies in the preceding table for which we could determine the statistical significance of the association between blood lead levels and diastolic blood pressure in men, 10 report a non-significant association. Moreover, as Cal EPA notes on p 3-21 of the Agency's revised report, blood lead levels are correlated with age, a well known predictor of blood pressure. Hence, the lack of negative associations reported by these studies (with the exception of the Dolenc *et al.* (1993) investigation) may reflect the fact that age is not properly controlled. Specifically, if the functional relationship between blood pressure and age is positive, but is best expressed by a function that is difficult to express, than some of the impact of age on blood pressure will be incorrectly attributed to blood lead levels, which are also associated with age.

Cal EPA concludes its discussion of this issue summarizing a literature review (Hertz-Picciotto and Croft, 1993) and two meta-analyses (Schwartz, 1995; and Staessen, 1995). The Staessen (1995) failed to find a statistically significant association between blood lead levels and diastolic blood pressure, although Cal EPA contends that proper analysis of the studies reviewed by Staessen would review a statistically significant relationship. Cal EPA also notes that Schwartz reports a statistically significant association between blood lead levels and diastolic blood pressure. Hertz-Picciotto and Croft (1993) come down in the middle, stating (according to Cal EPA on p 3-25 of the Agency's revised report) that, "the literature is strongly suggestive, but not definitive, of an association."

In conclusion, Cal EPA's claim that an association between blood lead levels and blood pressure is incontrovertible is not supported by the literature, or by the reviews and meta-analyses of this literature.

Comment 11 (p 369)

Cal EPA uses a high-end estimate to quantify the relationship between blood lead levels and diastolic blood pressure (1.9 mm Hg for every doubling of blood lead levels). The Agency should use a more central estimate. For example, the Staessen meta-analysis (1994) of 23 studies with a total sample size of 33,141 subjects, reports that the magnitude of the aforementioned association is only 0.6 mm Hg per µg/dL blood lead.

Cal EPA Response

The Staessen meta-analysis is inappropriate because it includes populations not relevant to the risk assessment (*i.e.*, individuals who are <u>not</u> males between the ages of 40 and 59). The Schwartz (1986a,b) reanalysis of the NHANES II data analyzed by Pirkle *et al.* (1985) reports a central estimate for this association; specifically, a doubling of blood lead levels increases diastolic blood pressure by 1.9 mm Hg.

Rebuttal

Cal EPA's conclusion is based on a very selective review of the available data. The following examples offer alternative values:

- On page 3-20 of the Agency's revised report, Cal EPA states that "across these studies using data from NHANES II, a doubling of blood lead corresponded to approximately a 2 mm Hg increase in systolic blood pressure and *a 1 mm Hg increase in diastolic blood pressure* [emphasis added]."

- On page 3-20 to 3-21, Cal EPA notes that Pocock *et al.* (1988) studied men between the ages of 40 and 49. For this group, there was "an estimated mean increase of 1.2 to 1.4 , Hg in both systolic and diastolic blood pressure ... for every doubling of blood lead concentration..."

- The Sharp *et al.* (1988) study of San Francisco bus drivers (p 3-22 of Cal EPA's revised risk assessment) reported an increase in diastolic blood pressure of 1.83 to 2.45 mm Hg for each 1 unit change in the natural log of blood lead levels. This range corresponds to an increase in blood pressure of between 1.26 and 1.69 mm Hg for each doubling of blood lead levels.

- Cal EPA's modifications to the Staessen *et al.* meta-analysis (1995) (see p 3-34 to 3-25 in the Agency's revised risk assessment) increases the magnitude of the association between blood pressure and blood lead levels to approximately 1.2 mm Hg diastolic blood pressure per doubling of blood lead levels.

Based on these results, we take issue with Cal EPA's conclusion that "There is reasonable agreement about the size of the effect of blood lead on blood pressure" (p 3-25). Moreover, we do not understand how the Agency's estimate of an increase in diastolic blood pressure of 1.9 mm Hg per doubling of blood lead levels can be considered a central estimate. In the very same paragraph in which Cal EPA concludes there is reasonable agreement between studies on this issue, the Agency states that "the U.S. EPA's external Science Advisory Board, and the National Research Council (1993) have concluded that a doubling of blood lead is associated with a 1 to 2 mm Hg increase in *systolic* blood pressure [emphasis added]." Since the reported impact of lead on diastolic blood pressure is often smaller than the impact of lead on systolic blood pressure, these statements cast a great deal of doubt on the Agency's use of 1.9 mm Hg as the central estimate for the magnitude of this effect.

We conclude that Cal EPA should use a range of values to represent the central estimate of the association between blood lead levels and diastolic blood pressure. The Agency should then consider stochastic uncertainty (see p 6-3 of Cal EPA's revised report) in addition to the uncertainty in the central estimate introduced by the range of results reported in the literature. The set of plausible central estimates for this relationship should probably range from approximately 0.6 mm Hg per doubling of blood lead levels (the magnitude reported by Staessen, 1995) to approximately 1.9 mm Hg (the result now used by Cal EPA). Use of this range will place more weight on relatively small predictions of risk for CHD and CHD mortality in the population than Cal EPA currently reports.

Comment 12 (p 369-370)

Cal EPA uses a supra-linear dose-response relationship that is an artifact of statistical considerations, but which is at odds with toxicological principals. A threshold dose-response relationship, or a sublinear dose-response relationship is more likely and would imply a smaller change in diastolic blood pressure at the low blood lead levels reported for contemporary populations.

Response

Cal EPA states that it uses a logistic relationship to model the probability of hypertension (diastolic blood pressure exceeding 90 mm Hg) as a function of blood lead levels, and a semi-log function to model diastolic blood pressure as a function of blood lead levels.

The Agency also argues that a supralinear relationship is not unreasonable, and has been observed for other lead-related biological phenomena, including the relationship between lead intake (*e.g.*, from food, water, or air) and blood lead levels.

Finally, Cal EPA states that researchers have found that a semi-log model provides the best fit to the data. For example, Cal EPA quotes Schwartz (1988) as stating that "the natural log of blood lead was more normally distributed, more significant, and gave a higher R^2 than untransformed blood lead... All of the results reported here are for the natural log of blood lead, but regressions for untransformed lead gave very similar results" (p 370 of the Response to Comments).

Rebuttal

We respond to each of these arguments in turn. First, the use of the logistic function to model the probability of hypertension as a function of blood lead levels is irrelevant since the risk assessment calculates the risk of probability of CHD as a function of diastolic blood pressure, not as a function of whether an individual is hypertensive.

Second, the assertion that the supralinear dose-response relationship is reasonable is not supported by the examples provided by Cal EPA. Each of those relationships, properly speaking, is not a dose-response relationship, but is instead an "exposure-dose relationship," where "exposure" is the amount of lead taken into the body in food, water, or air, and "dose" is the amount of lead that reaches some tissue; in the case of blood, the amount of lead reaching the tissue is measured in µg/dL lead in blood. In the case of an exposure-dose relationship, a supralinear function would be expected since at higher exposure levels, active transport mechanisms become saturated and the incremental amount of lead that enters the body's circulatory system for each incremental increase in exposure is depressed. On the other hand, this saturation principal supports the hypothesis that dose-response relationships tend to be sub-linear. At low dose levels (*i.e.*, low blood lead levels), mechanisms to protect the body's organs from toxicity can safely handle the challenge presented by the offending substance. Hence, the incremental toxicity (response) associated with each incremental increase in blood lead levels (dose) will be smaller at low blood lead levels than at high blood lead levels. This sub-linear dose-response hypothesis is at odds with the semi-log relationship advanced by Cal EPA since the semi-log relationship is steeper at low blood lead levels than at high blood lead levels. For example, the semi-log relationship implies that a doubling of blood lead levels from 5 to 10 µg/dL has the same incremental toxicity as a doubling of blood lead levels from 10 to 20 µg/dL.

Third, the rationale that Cal EPA quotes from Schwartz (1988) is not compelling. The fact that the log-transformed blood lead data are more normally distributed than the untransformed data means that ordinary least squares (OLS) regression techniques are not valid if the data are left untransformed. However, this fact does not mean that the shape of the dose-response relationship should be dictated regressing blood pressure against the log of the blood lead levels (*i.e.*, by the semi-log regression). Likewise, the fact that the semi-log regression yielded a higher R2 and was more significant than the untransformed regression does not mean that it better describes the shape of the relationship. With skewed data (in this case, the blood lead data), one would expect failure to transform the data (*e.g.*, by taking the logs) would yield a model that does not fit as well because transformation of the data eliminates "outliers" that degrade the model fit. Finally, it is not clear what Schwartz meant when he stated that "regressions for untransformed lead gave very similar results" as the transformed regressions. We are interested in the strength of the association between blood lead levels and blood pressure at the particularly low blood lead levels that are relevant to contemporary populations. The extracted quote from Schwartz does not show that a linear (*i.e.*, untransformed) dose-response relationship yields the same predictions in this low range of blood lead levels as the semi-log relationship used by Cal EPA.

Comment 13 (p 370)

The dose-response data relied upon by Cal EPA for the relationship between blood lead levels and blood pressure does not describe the behavior of this relationship below 7 μg/dL, a level that exceeds the blood lead levels of most adults now living in California. Since the dose-response relationship may be sublinear, or perhaps even exhibit threshold properties, the existence – or at least the magnitude – of a relationship between blood lead levels and blood pressure is highly uncertain for most adults in California.

Cal EPA Response

Cal EPA admits that the studies using NHANES II data that investigate the relationship between blood lead levels and blood pressure establish such a relationship only at blood lead levels exceeding 7 μg/dL. However, Cal EPA notes that other studies (*e.g.*, Harlan *et al.*, 1985) establish the existence of a relationship at even lower blood lead levels (*e.g.*, 4 μg/dL). The Agency adds that no threshold for this relationship has been identified, and conclude by noting that Pirkle failed to identify a threshold using "segmented regression analysis."

Rebuttal

While it is true that no threshold has been identified for the relationship between blood lead levels and blood pressure, it is also true that the vast majority of studies have focused on populations with substantially higher blood lead levels than the levels typical of contemporary Californian adults. This problem applies to the Pirkle segmented analysis since the NHANES II dataset that Pirkle analyzed establishes any type of relationship between blood lead levels and blood pressure only down to a level of 7 μg/dL, still well above contemporary adult blood lead levels in California. It is also difficult to see how Harlan (1985) could have conclusively demonstrated the lack of a threshold down to a level of 4 μg/dL since Harlan also relied on the NHANES II dataset. Even so, the fact that the geometric mean blood lead level for adults in California is 3 μg/dL indicates that more than half of this population has blood lead levels below 4 μg/dL. That is, there is no evidence supporting the existence of a dose-response relationship between blood lead levels and blood pressure for at least half the population, and certainly no evidence quantifying the magnitude of the relationship in this region of the dose-response

relationship. This lack of evidence makes the risk assessment results presented by Cal EPA even more uncertain.

Comment 14 (p 370-371)

Cal EPA has not identified any data directly documenting an association between blood lead levels and adverse cardiac events.

Cal EPA's response

Although there is no direct data linking elevated blood levels to adverse cardiac events, the Agency has identified evidence linking elevated blood lead levels to increased blood pressure, and the association between increased blood pressure and adverse cardiac events is very strong. The reason why no data have directly linked elevated blood lead levels to adverse cardiac events is that the relative risk of an adverse event is small and to date, no study has had sufficient power to detect such an association.

Rebuttal

The fact that a study as large as NHANES II lacks the power to detect the association hypothesized by Cal EPA again emphasizes how small the alleged risk must be.

Even so, we believe that the lack of such evidence leaves the existence and magnitude of the alleged association highly uncertain. Moreover, we believe that Cal EPA has not fully characterized this uncertainty in the Agency's revised report. The only uncertainty reflected in the Agency's risk assessment is the stochastic uncertainty in the one study used by Cal EPA to quantify the strength of the association between elevated blood lead levels and blood pressure, and the stochastic uncertainty in the association between elevated blood pressure and adverse cardiac events. We believe in particular that Cal EPA has neglected many sources of uncertainty underlying our understanding of the first of these two causal links. A full characterization of uncertainty should address the following additional issues:

- Whether there exists a relationship between blood lead levels and diastolic blood pressure (see Comment 10);
- The magnitude of the central estimate of the relationship between an incremental increase in blood lead levels and the associated change in diastolic blood pressure (see Comment 11)
- The shape of the dose-response relationship (see Comment 12) and whether there may be a threshold at levels relevant to the current population of adults living in California (see Comment 13).

It is our opinion that taking all these factors into account would lower the bottom end of the range of plausible adverse cardiac mortalities to per year to zero, and would place much less probabilistic weight at the current upper end of the this range. Without addressing these issues, Cal EPA has failed to reflect the current state of knowledge in its risk assessment and has therefore not produced a valid characterization of the range of possible risks associated with slightly elevated atmospheric lead levels.

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Comments of

Lead Industries Association, Inc.
Battery Council International
GNB Technologies, Inc.

submitted to the

Air Resources Board
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

on the

Proposed Identification of
Inorganic Lead as a Toxic Air Contaminant
(Draft SRP Version)

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LIA - 295 Madison Avenue, 19th Floor, New York, New York 10017 (212) 578-4750
BCI - 401 N. Michigan Avenue, Chicago, Illinois 60611 (312) 644-6610
GNB - 2700 South Indiana, Los Angeles, California (213) 262-1101

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I. INTRODUCTION AND SUMMARY

These comments are filed by Lead Industries Association, Inc. (LIA), Battery Council International (BCI), and GNB Technologies, Inc. (GNB) in response to the draft Scientific Review Panel (SRP) Version of the report consisting of Parts A, B and C and Executive Summary (hereinafter "report") released in September 1996 by the staff of the Air Resources Board (ARB) and the Office of Environmental Health Hazard Assessment (OEHHA) as part of the rulemaking on the Proposed Identification of Inorganic Lead as a Toxic Air Contaminant (TAC).

The new draft report represents a substantial improvement over earlier versions, particularly in its revision of current emission levels for stationary lead sources in California from 150 to 6 tons a year, a 95 percent drop from the report's previous estimates. Moreover, the new report now states that the previously reported statewide ambient air lead level (0.06 $\mu g/m^3$) has also declined dramatically -- by two thirds, according to still more recent information communicated orally by Air Resources Board staff -- and even before taking that drop into account, Part B of the report concludes that air lead is a "minor contributor" to children's overall lead exposures. Other positive changes include the use of updated NHANES III data in certain calculations, inclusion of more recent studies, improved characterization of the uncertainty in health effect estimates and insertion of language in some places warning against misuse of the conclusions of the report.

Despite these advances, critical problems remain. These are described in detail in these comments, but in overview they are as follows:

- The report fails to carry forward into the Executive Summary -- the section most likely to be widely distributed and read by the public -- key acknowledgments that appear in other sections of the multi-volume report, including the statement above that air lead is a "minor contributor" to children's exposures, and others, such as the conclusion that neurodevelopmental health effects associated with prenatal exposures disappear after age two in the absence of independent exposures, that "the impact of lead on the incidence of cardiovascular disease is small," and that OEHHA's use of an aggregate air lead to blood lead slope factor "may overestimate the impact of certain exposure scenarios." The report's failure to incorporate these important points into the Executive Summary will mislead those who do not read the full report.

- In fact, notwithstanding that the worst the report can say is that "ambient and near source exposures may *still* present *a potential* public health concern," the report gives the impression that ambient air lead levels are responsible for causing: (a) a one percent increase in the number of children whose IQ level is below 80, (b) an additional 26,000 cases of hypertension, and (c) 49 or more deaths in California every year.

Elsewhere, the report makes clear that ambient exposures are not a public health threat, and accordingly, they should not be used in risk assessment calculations. There is no legitimate reason to make a numerical risk estimate for a threat that is not real.

- While the most recent revision to the draft report includes references to studies that disagree with OEHHA's conclusions, there are major problems with the treatment of these studies. In one case, OEHHA rejects the authors' (Staessen et al.) contrary conclusions and extracts one isolated data subset from the study to support its own views, and in others OEHHA steadfastly refuses to acknowledge the existence of widespread, legitimate controversy in the scientific literature about the asserted health effects. At most, OEHHA deals with these concerns by sprinkling statements in the Executive Summary with a host of qualifiers, using words such as "may," "might," "could," and "potential" rather than fairly recognizing that fundamental questions remain about the validity of these conclusions. Indeed, we have attached as Attachment 1 a major new review of the neurodevelopmental studies by one of the world's foremost experts on IQ test design and interpretation, Dr. Alan Kaufman, who concludes that OEHHA's views are not supported by the best available scientific evidence. Even if OEHHA is determined to reject all the evidence contrary to its position on neurodevelopmental and blood pressure-related health effects, at a minimum the Executive Summary should acknowledge that there is continuing disagreement in the scientific community regarding the impact, if any, of lead on these health endpoints.

- The report fails to reconcile the revised emission estimates with the prioritization criteria found in the California Health and Safety Code as set forth in the Executive Summary, which would result in the ranking of lead as a low priority for evaluation as a possible TAC. ARB and OEHHA must, at a minimum, adjust the priority of listing inorganic lead as a TAC in light of the five criteria set forth in the statute.

II. MAJOR FLAWS IN THE REPORT'S ANALYSIS AND CONCLUSIONS

As noted above, while some improvements have been made to the report, it remains seriously flawed in several critical respects. These are discussed in turn below.

A. Acknowledgment of Undisputed Points in the Executive Summary

Though Parts A and B, as we have stated, acknowledge various points that LIA, BCI, and GNB have long urged, these acknowledgments are not carried forward into the

2. Insignificance of lead's impact on cardiovascular disease. In Part C, at page 371, OEHHA "acknowledge[s] that the impact of lead on the incidence of cardiovascular disease is small." As noted in section II.C.2. below, a substantial body of scientific evidence -- indeed, many would say the prevailing weight of the evidence -- places in question whether any casual link at all exists between lead and blood pressure increases, but at worst OEHHA can characterize the effect as no more than "small" and suggest that "given the pervasiveness of exposure, it *may* have public health significance" (*id.*, emphasis added).

In contrast, the Executive Summary claims that ambient air lead exposures of 0.6 $\mu g/m^3$ in the state of California are responsible for 26,000 cases of hypertension, 48 additional fatal and non-fatal heart attacks and coronary heart disease deaths, as well as 49 additional deaths per year. Ex. Summ. at p. 10. These numbers do not appear to be "small." And in view of the major controversy that surrounds this issue in the scientific community, as well as OEHHA's own admission that the calculations are subject to "considerable uncertainty," the inclusion of these numbers is speculative and inflammatory, particularly when they are in direct conflict with the more balanced statements about the size and likelihood of a public health impact that are buried some 300 pages deep in Part C of the report. As discussed below, this entire subject is so unsettled in the scientific literature that it should not be used for this kind of risk assessment. Suggested rewording of the statements on page 8 of the Executive Summary is as follows:

> Lead in the environment, including the occupational setting, has also been correlated with increased blood pressure and related cardiovascular effects in adults, although there is disagreement in the scientific community regarding whether causality has been established or whether the observed effects are the result of confounding factors. Several large population-based studies have examined the relationship between blood lead and either systolic or diastolic blood pressure. A relationship between systolic or diastolic blood pressure and blood lead has been reported over a wide range of blood lead levels for middle-aged Caucasian men, with some studies reporting evidence of effects in women, other races, and other age groups. In those studies that find an effect, the impact of lead on the incidence of cardiovascular disease is small.

3. Overestimation resulting from use of an aggregate slope. In response to comments from LIA and others, OEHHA has "acknowledge[d] the point that the aggregate slope model may overestimate the impact of certain exposure scenarios." Part C, p. 362; accord, p. 377. As OEHHA explains, "[t]his is because soil and household dust levels do not, in the shorter term, change to the degree predicted by the model. A similar situation can also occur with the IEUBK model when U.S. EPA's supplemental equations are used to predict soil and housedust lead levels." *Id.*, p. 377.

Executive Summary -- the part of the report most likely to be widely distributed and read. The Executive Summary must be revised to incorporate these points as follows:

1. Lack of persistence of neurodevelopmental health effects from prenatal exposure. At Part C, pp. 373-74, OEHHA acknowledges that recent major analyses such as the ACGIH review (1995) and Pocock et al. meta-analysis (*British Medical Journal* 1994) "did not find an association [between persistent neurodevelopmental deficits and] prenatal exposures," but appears to suggest that the distinction between prenatal and postnatal exposures is unimportant "[b]ecause OEHHA's estimate for risk assessment is based on postnatal blood lead, [and therefore] the failure of Pocock et al. to find consistent antenatal and neonatal associations does not alter OEHHA's conclusions." *Id.* at 374. Regardless of whether OEHHA's quantitative calculations are affected, however, there is no justification for failing to make this distinction clear in OEHHA's qualitative conclusions and in the Executive Summary, which continues to state that "several carefully conducted prospective human epidemiological studies have shown an association between general measures of intelligence and both pre- or post-natal blood lead concentrations." Ex. Summ. at p. 8.

This statement is at odds with OEHHA's own Part B analysis. OEHHA notes that "only 3 of the studies exhibit an association [with *either* prenatal or postnatal exposures] after adjustment for covariates. In addition, significance is not found for every age at which exposure or intelligence was measured." Part B. p. 3-10. Further, OEHHA recognizes that "[i]n those studies which do show an effect of prenatal blood lead level on intelligence after adjustment, the effect seems to decline by the time the child is 2 to 4 years old." *Id.* Accordingly, it is disingenuous for the Executive Summary to continue to state that "several carefully conducted prospective human epidemiological studies" show an association between "both pre- or post-natal blood lead concentrations" without noting at least the lack of persistence of the prenatal effects. As discussed below, the debate on the question of low level health effects is intensifying, but even on the basis of OEHHA's own acknowledgments, we submit that the statement in the Executive Summary should be revised and either the entire reference to prenatal effects dropped or, at a minimum, language along the following lines substituted.

> At low blood lead concentrations, several carefully conducted prospective human epidemiological studies have shown an association between general measures of intelligence and ~~both~~ either pre- or post-natal blood lead concentrations, or both, although some of the associations disappeared after adjustment for covariates. While controversy remains regarding the lower blood lead ranges, and particularly with respect to the persistence of pre-natal exposure effects, based on these studies, a blood lead level of 10 micrograms per deciliter has been identified as the level of concern for children.

These important caveats, however, appear nowhere in the unequivocal statements about the blood lead to air lead slope in the Executive Summary (p. 9). There, in contrast, the reader is informed that "[e]xisting studies indicate a consistent association between ambient concentrations of lead in the air and subsequently measured blood lead levels in children and adults," that OEHHA used these studies as the basis for an aggregate model "which quantitatively relates exposures from ambient air lead concentrations to blood lead levels" and allows risks to be calculated "that relate different levels of ambient air lead to potential adverse neurodevelopmental outcomes." Slope factors of apparent scientific precision, 1.8 μg/dl per 1 $\mu g/m^3$ for adults and 4.2 μg/dl per $\mu g/m^3$ for children, are set forth, and the discussion concludes with the pronouncement that these slopes "are assumed to be linear within the range of blood and air lead concentrations currently experienced in California."

Here again, OEHHA's acknowledgment of this important point does not appear until almost 400 pages into the report. It should be brought forward into the Executive Summary, so that readers can be made aware, at the same time the model's results are summarized, that there are conditions under which this model would overstate the impact of air lead exposures. The following language should be added to the discussion at page 9:

> Existing studies indicate an association between ambient concentrations of lead in the air and subsequently measured blood lead levels in children and adults. The OEHHA used these studies as the basis for an "aggregate" model which quantitatively relates exposures from ambient air lead concentrations to blood lead levels, both directly through inhalation and indirectly through other media impacted by airborne lead, such as soil and household dust. Although the aggregate model may overstate the impact of certain exposure scenarios, particularly those involving short-term effects, with this aggregate model, risks could be estimated that relate different levels of ambient air lead to potential adverse neurodevelopmental outcomes.

B. Inappropriate Focus on Ambient Exposures

Although the report at Part B, p. 1-1 makes clear that ambient exposures are a "minor contributor" to human lead exposure at the levels cited in the report (0.06 $\mu g/m^3$) -- much less at the new levels the ARB staff expects will be documented in the near future -- nevertheless it would appear from a reading of the Executive Summary and even other sections that current ambient exposures are thought by OEHHA to increase by one percent the number of children in the state with IQ levels below 80 and to cause 26,000 excess cases of "hypertension" and nearly 100 deaths every year in California (Ex. Summ. at p. 10).

OEHHA's theoretical quantitation of purported health effects from air lead exposure has been carried out with a precision far greater than is scientifically justified. Furthermore, its projections of health impacts have been extrapolated downward to ambient

exposure levels so small that their potential health impacts are completely lacking in credibility and cannot possibly be verified. For example, OEHHA's assertion that the number of children with IQs below 80 is increased by 1 percent as a consequence of exposure to the estimated statewide average air lead level (0.06 $\mu g/m^3$) is based on a projected differential of only 0.1 percent (an increase from 10.56 to 10.66 percent) in the lower tail percentage of the "standard" IQ distribution. There is far too much uncertainty, with regard to both causality and quantitation, inherent in the empirical relationships OEHHA has employed to link air lead to blood lead and then IQ to justify the reporting of such minuscule differences. Indeed, the NHANES III distribution of blood lead levels assumed by OEHHA to apply to California children is by itself far more uncertain than would be required to justify scientifically such extraordinarily precise quantitation. For example, the 95 percent confidence interval for the mean blood lead level employed by OEHHA runs from 3.7 to 4.5 μg/dl. In the face of such uncertainty, it is inconceivable that a 0.1 percent shift in the percentage of children with IQs below 80 could be attributed with any confidence to a 0.06 $\mu g/m^3$ differential in air lead levels. The same concerns apply to OEHHA's projections of increased cases of hypertension and related cardiovascular effects purportedly arising from exposure to background air lead levels in California.

OEHHA should abandon its attempts to quantify health impacts due to statewide ambient air lead levels. These levels are far too small, and the linkages between exposure and effect are far too uncertain, to permit meaningful, scientifically justifiable quantitation. Further, OEHHA should acknowledge clearly and explicitly in the Executive Summary and elsewhere in the report that at worst, air lead may pose health risks only in extreme near source situations.

C. Flaws in the Health Effects Analysis and Risk Calculations

1. Neurological effects. Controversy continues to surround the entire question of linkages between low blood lead levels and neurodevelopmental health effects. As noted above, there appears to be an emerging consensus that prenatal health effects dissipate after age two in the absence of independent exposures to the child, a point that OEHHA's Part B analysis acknowledges. Part B, p. 3-10. The issue of postnatal effects is more complicated, but more and more scientists in the field are questioning the strength of the evidence to support conclusions like those OEHHA seeks to draw. As LIA pointed out in its March 1996 comments, Dr. Sergio Piomelli, one of the original pioneers in the effort to prevent lead poisoning in children, wrote as a member of the CDC Committee on Environmental Health Hazard and Health Effects in September 1995 (see Attachment 1) that the evidence on IQ losses claimed to be associated with low-level lead exposure reflects effects that are "trivial and unmeasurable in the individual children" even at levels of 15 to 20 μg/dl. As to the claim that such negligible impact "across a population may have important effects," (Part C, p. 375), Dr. Piomelli takes a view completely contrary to OEHHA's, concluding that "the effect that an average loss of [as much as] 2.6 IQ points may have on the frequencies of children with an IQ $\leq$ 80 points or $\geq$ 125 points, appears insignificant, when calculated statistically." Att. 1, p. 2.

In a new examination of the existing studies (Attachment 2), Dr. Alan Kaufman, a leading scientist in the field of assessment of intelligence and psychological research design -- indeed, the author of or contributor to many of the tests used in the studies OEHHA relies upon -- comes to the same conclusion. Dr. Kaufman reviewed the 26 studies that were included in one or more of the three meta-analyses dealing with the issue of whether there is a causal relationship between moderate to low blood lead levels and IQ. Dr. Kaufman applied to each of the studies seven criteria to determine whether the authors have made unfounded assumptions or failed to correct for confounders that, in his judgment, are critical in determining causal impacts on IQ. Dr. Kaufman's conclusions concerning the seven criteria are as follows:

- Many of the studies infer causality from correlational data, an inference Dr. Kaufman considers inappropriate for reasons spelled out in his report.

- Potential confounding variables have not been appropriately controlled in any of the studies, placing the entire asserted relationship in question, since "it is quite possible if not highly probable that much or all of the so-called IQ loss due to lead level is due to these other confounding variables." Att. 2, p. 8.

- A number of researchers conducted multiple comparison analyses and failed to correct for chance error that occurs in this kind of "shotgun approach." Att. 2, p. 11. An example is the frequently cited Bellinger Needleman study, upon which OEHHA principally relies, which made simultaneous comparisons, only two of which were statistically significant. Statistical techniques that account for the simultaneous testing of multiple comparisons to control for chance error indicate that none of the comparisons in this study are statistically significant and strongly suggest that OEHHA's reliance on this study is misplaced.

- The remaining three criteria involved the appropriateness of comparing "extreme" lead groups, whether a satisfactory measure of IQ was employed, and the extent to which verbal, rather than performance, IQ was affected (which suggests confounding by socio-economic status rather than a true relationship with lead exposure).

Dr. Kaufman's report also examines OEHHA's assumption that there is a linear relationship between blood lead and IQ down to very low levels, a point also questioned by Dr. Piomelli (Att. 1, p. 2). Dr. Kaufman points out that the data that are said to support a linear relationship clearly do not do so at lower blood lead levels. He concludes that "[t]here is no evidence whatsoever to support the notion that small amounts of lead in the blood have any impact on a child's IQ," (Att 2, p. 19). Further, like Dr. Piomelli, Dr. Kaufman takes issue with OEHHA's view that there are significant societal impacts from such small IQ losses, even if they are meaningless for an individual child. Att. 2, pp. 32-38, 39-40.

Finally, Dr. Kaufman's report refers to the many instances in which OEHHA's analysis fractionalizes IQ points, and states that fractionalizing an IQ point "is asking an IQ test to do something that it is not equipped to do." Att. 2, p. 20. After explaining the inherent constraints of IQ test design and administration, Dr. Kaufman's ultimate conclusion on this issue is that "it is irresponsible to fragment an IQ point for any type of interpretative result; no meaningful interpretation is possible." *Id.*, p. 22.

In 1991 EPA acknowledged that "[t]here remains uncertainty in the global scientific community about the causal relationship between low level lead exposure and certain health effects in children," and that the issue as to neurological effects is "controversial."[1] That controversy is, if anything, intensifying, as both Dr. Piomelli's and Dr. Kaufman's statements make abundantly clear. If OEHHA is unwilling to withdraw or temper its conclusions despite the mounting questions on this issue, it should nonetheless at a minimum amend the Executive Summary discussion to acknowledge the existence of responsible contrary scientific opinion that differs with OEHHA's analysis and conclusions.

2. Blood pressure. In its March 1996 comments, LIA pointed out that OEHHA's conclusions on lead and blood pressure were problematic, as revealed by the fact that they could only be made by stringing together the words "appears," "possibly," and "some evidence" so that the Executive Summary statement read: "A relationship between systolic and diastolic blood pressure and blood lead **appears** to exist for a wide range of blood lead levels, **possibly** extending down to as low as 7 micrograms per deciliter for middle-aged Caucasian men, with **some evidence** of effects in women, other races, and other age groups as well." Jan. 1996 version, Ex. Summ., p. 8 (emphasis added).

OEHHA's September 1996 response to this comment was that each of these qualifiers indicated a different element of uncertainty in the blood lead-blood pressure relationship (Part C, p. 379). Rather than acknowledging that the combined effect of these multiple uncertainties illustrates one reason why there is unabated controversy in the scientific literature on whether or not a causal relationship exists at all, OEHHA simply revised its Executive Summary language, presumably to make the wording appear less tentative, and for good measure extended the range of effects even lower, to 4 μg/dl.

While it may technically be true that "a relationship between systolic and diastolic blood pressure and blood lead has been reported over a wide range of blood lead levels, as low as 4 micrograms per deciliter for middle-aged Caucasian men," OEHHA's discussion makes it clear that these statements strain at the outer edges of reported findings -- the support for them consists of "one study" that found an effect at 7 μg/dl (Part C, p. 379), and a vague reference in the Part C document to the fact that "in the analysis of males age 20 to 74 in the NHANES II study, the minimum level was around 4.0 μg/dl," *id.* The analysis in Part B does not appear to

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[1] Report to the Organisation for Economic Co-operation and Development, "Cooperation on Existing Chemicals: Risk Reduction Lead Country Report" (May 1991) p. 54.

refer to any study showing effects at 4.0 μg/dl, and the lowest effect level asserted occurs in a sentence referring to a study showing a relationship between diastolic blood pressure and "blood lead values extending possibly down to 5 μg/dl for middle-aged men." Part B, p. 3-21.

The questionable scientific validity of making health interpretations upon such a tenuous health endpoint is further complicated by application of this speculative effect to low blood lead levels. Assessments of the incidence of hypertension evaluate the impact of 0.06 $\mu g/m^3$ air lead upon a baseline of 3 μg/dl for adults. More significant cardiovascular events are based upon air lead impacts at baseline levels in adult males of 4.4 μg/dl. Both values are significantly below the blood lead ranges which characterize the majority of the studies conducted to date.

Moreover, estimates of blood lead's impact upon blood pressure are relative, as opposed to absolute, lead measures. Doublings of blood lead are used as a matter of statistical convenience to define blood lead-blood pressure relationships in most studies. Extrapolation to exposure levels lower than those which were studied can produce significant inflation of estimates of health impact. Thus, an 0.06 $\mu g/m^3$ air lead yields a presumed 0.108 μg/dl increase in blood lead. Use of a baseline of 3 μg/dl yields hypertension impacts 3.3-fold greater than that which would be predicted to occur at a baseline of 10 μg/dl, and five-fold greater than would be calculated at an average of 15 μg/dl.

The present OEHHA analysis suggests that estimates of lead's impact are relatively independent of baseline blood lead values. This statement is difficult to reconcile with the preceding analysis, but one is not able to evaluate the range of baseline values considered in this portion of OEHHA's analysis. The report refers to recent extrapolations of Schwartz (1995) that estimated impacts of blood lead reductions from 10 to 5 μg/dl (Part B, p. 6-1). If OEHHA has determined that calculated health benefits from reductions at baselines of 3-4.4 μg/dl are not materially different from those at 5 μg/dl, this is probably correct. However, it is important to recognize that the calculations made by Schwartz represent an extrapolation of existing studies to blood lead levels lower than those actually studied. The presence of an effect in the blood lead range of 5-10 μg/dl largely rests upon the results of a single study (Hense, 1993). Even in that study, the suggestion of an effect is largely contingent upon data that are not corrected for confounders most investigators feel should be included in an analysis. Confounder correction largely eliminates the suggestion of an effect at low blood lead levels.

It is difficult to understand why the report is so determined to stake out the most extreme ground on this issue and to do so only in the direction of finding an effect. If the report's apparent one-study prerequisite were applied fairly, for example, the Executive Summary ought to note the findings by Staessen et al. (1996) of an inverse (protective) effect from lead exposure in males.

LIA noted in its March 1996 comments the highly unsettled status of this entire issue in the scientific community and identified a host of studies, literature reviews, and meta-analyses that disagree with OEHHA's conclusions on whether causality can be shown at all, *id.*,

pp. 8-12. An independent commenter, Dr. Jan Staessen, filed comments detailing the research findings in his studies and the scientific literature, and concluding that the evidence does not establish causality. The Part C response rejects all contrary evidence, even going so far as to extract from Staessen's meta-analysis one data subset (40 to 59 year old males) and use it to support a conclusion opposite to that reached by the authors (Part C, p. 385).[2]

The SRP previously concluded at its October 21, 1993 meeting that OEHHA's projected blood pressure increases at current California air lead levels would be "minuscule" (Tr. p. 113), and urged OEHHA to incorporate in its analysis the possibility of zero effects if some of the studies considered "credible" by OEHHA included that outcome within the confidence intervals of their findings. *Id.*, pp. 115-16. OEHHA's September 1996 response states that the data set that it considers most representative for the current population of California -- NHANES II, which dates back to 1976 -- does not include zero in its confidence intervals and OEHHA declines to do so in its analysis (Part C, p. 383). We submit that several highly credible studies, including more recent ones, find zero within the range of effects and this possible outcome should be included in the "body count" projections shown in the Executive Summary.

We continue to object to the treatment of this entire issue, and particularly to the one-sided, extreme conclusions that appear in the Executive Summary, including the quantitative risk projections. Legitimate, serious debate continues in the scientific community over the question of causal inferences between blood lead and blood pressure. At a minimum, the Executive Summary should be amended to acknowledge that the question of causality remains an issue, as the amended language suggested above for page 8 of the Executive Summary would make clear. In addition, the risk assessment on page 10 of the Executive Summary should be modified to include zero in the range of possible effects. Updated ambient air values should be used in these calculations as well.

3. Air lead-blood lead slope issues. OEHHA's aggregate slope factor and IEUBK analyses continue to overstate significantly the effects of air lead changes on blood lead. OEHHA acknowledges that its aggregate slope factor and IEUBK analyses include indirect exposure pathways, such as soil and dust, whose equilibrium with air lead levels requires periods on the order of decades or more to achieve. Thus, OEHHA has greatly overestimated the direct incremental benefits or risks that might arise from changes in air lead levels. OEHHA should state explicitly how long one would have to wait for its calculations to be appropriate. As we have pointed out before, OEHHA should include calculations for shorter term exposures for which a direct inhalation slope factor on the order of 1 $\mu g/dl$ per $\mu g/m^3$ would be far more

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[2] The report incorrectly states that the International Conference on Lead and Blood Pressure (Victery 1988) supports a finding that there is "overwhelming evidence for the causality of the association," (Part C, p. 380). In fact, the opposite is true. No collective conclusions were produced at this conference; instead each panelist summarized his or her own view of the evidence. Of the seven, only one took the position that the evidence on causality was sufficient and even that panelist, Dr. Joel Schwartz, found it only "probably although not definitively established" (Discussion Report, p. 155).

appropriate, as is borne out in some of OEHHA's worked examples of IEUBK-projected effects on blood lead levels of changes in air lead.

Even results from controlled laboratory studies indicating a *direct* inhalation slope factor of 1.7 overstate the blood lead/air lead slope factor that would be appropriate for typical exposure conditions because the latter involve variable concentrations and intermittent exposure periods. Table IV-8 of Part A shows that the average indoor concentration of airborne lead in California is about 0.03 $\mu g/m^3$, about half the ambient average level of 0.06 $\mu g/m^3$ used by OEHHA in its calculations. This table further shows that both children and adults spend about 86 percent of their time indoors. They are thus not exposed to a background level of 0.06 $\mu g/m^3$ air lead on a continuous basis. OEHHA's projections of health effects attributable to ambient levels of 0.06 $\mu g/m^3$ accordingly appear to be greatly exaggerated.

OEHHA has also failed to make adjustments for the differential proximity of individuals to sources and the intermittency of their exposures, both of which would serve to reduce the effective blood lead-air lead slope factor from its controlled laboratory study value. No one is exposed continuously to maximum receptor site concentrations all day, every day, yet OEHHA's calculations have assumed exactly that.

4. Other failings in the risk calculations.

a. Neurodevelopmental Effects

OEHHA's estimates of adverse neurodevelopmental effects rely on NHANES III national data regarding geometric mean blood levels for 1-2 year old children and their geometric standard deviation (GSD). Both the mean and the GSD have been assumed implicitly by OEHHA to be known without error. However, the original publications of NHANES III findings reported that the 4.1 µg/dl geometric mean has a 95 percent confidence interval of 3.7 to 4.5 µg/dl. This represents a range of uncertainty of nearly 20 percent, a range far greater than any effect that estimated air lead levels would have on blood lead. A similar and perhaps even greater degree of uncertainty applies to OEHHA's estimated GSD of 2.1. OEHHA needs to take into explicit account the substantial uncertainties in these estimates. How is it possible that OEHHA can confidently project a 0.6 percent change in the proportion of children with blood leads above 10 µg/dl (10.9 to 11.5 percent) from an air lead increment of 0.06 $\mu g/m^3$ when the distributional parameters that go into its calculation are so much more uncertain? Similar concerns apply to OEHHA's calculations of blood lead levels and changes arising from various air lead differentials for sensitive subpopulations.

In addition, OEHHA has assumed that the distribution of blood lead levels is long-normal in form. While this theoretical curve may provide a reasonable rough approximation to the overall shape of the true blood lead distribution, it is not necessarily valid for particular narrow regions of that distribution, such as for blood lead levels near 10 µg/dl, or for still higher levels. For example, in the NHANES III data there is a readily apparent cluster of children with blood lead levels in the 20 to 26 µg/dl range that constitutes a far greater percentage of the total

than would be predicated by any log-normal curve fitted to the overall blood lead distribution (cf., Figure 2 in Pirkle et al., 1994). This is simply an indication that the general population distribution is in reality a mixture of distinct distributions that apply to individual subpopulations of children. The resulting distribution for the overall mixture could not possibly be log-normal, even if each and every one of the individual subpopulation distributions were log-normal.

b. Blood Pressure and Cardiovascular Disease

In developing its estimate of increased probability of hypertension, OEHHA has employed NHANES III data for adults (males and females) aged 20 to 74 in combination with an empirical relationship derived from analyses of NHANES II data. It has similarly employed NHANES III data, but this time only for adult males aged 20 to 69, in three race/ethnicity categories, in combination with another empirical relationship derived from analyses of NHANES II data in developing its estimate of the average increase in diastolic blood pressure among men aged 40 to 59 that would be attributable to the ambient air lead level of 0.06 $\mu g/m^3$.

OEHHA also employed blood pressure data for 1979 California white males (age category unspecified) together with an empirical relationship derived from analyses of Pooling Project data in developing its estimate of the 10 year probability of coronary heart disease. Finally, OEHHA employed the same blood pressure data together with another empirical relationship derived from analyses of the Framingham study data to estimate the 12 year probability of death.

While OEHHA made an effort to characterize some of the uncertainty inherent in its combinations of data and relationships from the various studies, it did not go far enough in this effort. The only factors that were considered explicitly as uncertain by OEHHA were the regression coefficients, either for the logarithm of blood lead or for diastolic blood pressure, in each of the empirical relationships it employed. The other important factors in the relationships, namely, the means of the many covariates that are in fact far more significant explanatory variables than is blood lead, and the mean blood lead or blood pressure levels, were apparently presumed to be known without error.

OEHHA must adequately characterize these additional sources of uncertainty. It needs also to address explicitly the critical question of model uncertainty, since it is entirely inappropriate to simply assume that (1) the mathematical form of the true dose-response relationship (if there is one) is exactly and correctly specified, and (2) the relationship extends smoothly all the way down to zero air lead levels. Satisfactory consideration of these additional sources of uncertainty should widen substantially the confidence intervals that surround OEHHA's estimates of potential adverse health effects among adults, and would likely lead to the conclusion that no such effects are confidently attributable to levels as small as or smaller than the near source or statewide ambient air lead levels of interest to OEHHA.

D. Exposure Scenario

A number of significant problems remain with the exposure scenario described in the Part A document. Those problems lead to an overestimate of risk and an inaccurate portrayal of the extent and impact of lead emissions.

OEHHA has estimated an average "near source" air lead contribution of 0.20 $\mu g/m^3$ and the number of individuals supposedly exposed at that level is estimated at 2500 (Part A, p. 58). This latter estimate needs to be reduced markedly. ARB's own example of the measurements taken around the Vernon facility demonstrates how far beyond reality OEHHA's estimated number of individuals exposed really is.

The near source above background level of 0.20 $\mu g/m^3$ represents a maximal annual average at a distance of 210 meters from the stack centroid in the example described in Part A, not population-weighted annual average levels, which the report estimates to be on the order of 0.0001 $\mu g/m^3$, *i.e.*, hundreds of times smaller (Part A, Tables IV-3 and IV-4). Yet OEHHA has assumed that 2500 persons would be exposed at the 0.20 $\mu g/m^3$ level. Note that only 2,011 of the nearly 3.5 million people considered to be near the local source, *i.e.*, within about 15 kilometers, were exposed at concentrations above 0.03 $\mu g/m^3$, and none were exposed to levels as high as 0.20 $\mu g/m^3$. Yet OEHHA persists in using the estimate of 0.20 $\mu g/m^3$ as if it were the average exposure level for 2,500 people. OEHHA has also failed to make adjustments for the differential proximity of individuals to sources and the intermittency of their exposures, both of which would reduce the exposure scenario.

Another factor that has not been taken into account in the exposure scenario is the high prevalence of indoor exposure. As described above, the average indoor concentration of airborne lead in California is about half the ambient average and both children and adults spend about 86-87 percent of their time indoors. Were indoor exposure to be taken into account, the estimates provided in the exposure scenario would drop by approximately one-half.

We noted in previous comments that OEHHA had failed to honor its original commitment to limit the applicability of air lead controls to residential settings (Part C, p. 414, comment 5). OEHHA has not responded to this comment except to state that "the appropriate degree of controls" will be determined at the next stage of this proceeding. We reiterate our position that the modeled health impacts provided in the Part B document are all based on potential exposures which could only take place in a residential setting, and OEHHA should confirm that the exposures that it has modeled take place in a residential, *i.e.*, non-industrial, non-commercial setting.

We continue to object to the characterization in the Part A document, p. 14, that near source monitoring results conducted near the two secondary lead recycling facilities in Southern California showed "significant concentrations near the facilities" without qualification as to the meaning of the term "significant." Relying on emissions data from 6-7 years before the implementation of South Coast Air Quality Management District Rule 1420 is inappropriate.

Similarly, we would also object to the characterization that "[s]tationary area sources release small quantities of pollutants from any closely-located sites over a relatively large geographic area" (Part A, p. 25). Not only is this representation confusing, but ARB has provided no evidence that "pollutants" would be released over a "large" area.

A particularly troubling issue is the continued reference in the Part A document and in the Executive Summary to ambient lead concentrations measured at the Vernon facility "as much as 52-fold higher" than the mean and annual South Coast Air Basin ambient concentration of 0.07 $\mu g/m^3$ (Part A, p. 57, Ex. Summ, p. 5). Despite the fact that we have repeatedly indicated that this number is unrepresentative, ARB staff state that they cannot conclude that it is an "aberrational" value because they do not have sufficient prior data (Part C, pp. 414-15). All references to the 52-fold value should be deleted from the report.

E. Lack of an Air Lead Problem

When this rulemaking first began, we voiced concerns about whether there was a need for further regulation of point sources of lead in California. ARB and OEHHA have presented information in this most recent report from which one must conclude that there is simply not an air lead problem and the ARB should reevaluate the priority it has assigned to inorganic lead. The best evidence of the need for reevaluation is the reduction in the estimate of emissions. As compared to the previous draft report, the total statewide lead emissions estimate has dropped from 345-465 tons to 175-82 tons (Ex. Summ., p. 3). Only a tiny fraction of this overall emissions estimate is attributable to stationary sources. Statewide emission inventory results from AB 2588 reporting and from local air district lead-specific rules have dramatically improved the state of knowledge of lead emissions. The estimate of total emissions from stationary facilities has dropped from a prior estimate of 150 tons to 6 tons, a reduction of 95 percent. Given that emissions from stationary sources now represent somewhere between 3 to 4 percent of total lead emissions (and perhaps even less) and the other air lead sources identified in the report, reentrainment[3] and airplane fuel, are ambient sources that are likely to decrease, we question whether there is any further need for pursuing this rulemaking.

The factors that ARB must consider in setting the priority for "evaluation and regulation" of toxic air contaminants are set forth in Health & Safety Code section 39660(f): (1) risk of harm to public health, (2) amount or potential amount of emission, (3) manner of, exposure to and usage of the substance in California, (4) persistence in the atmosphere, and (5) ambient concentrations in the community. A review of these factors suggests that the priority for the evaluation and regulation of inorganic lead is extremely low.

[3] The report offers no substantiation for the surprisingly high estimate of 390 tons of lead from reentrainment (Part A, p. 7).

First, as described elsewhere in these comments and as set forth in the report, the risk of harm to public health from current levels of ambient and near source emissions of inorganic lead ranges somewhere between nonexistent and "minor." Each iteration of this report has led to a lowering of risk estimates. Second, both the amount and potential amount of emissions of inorganic lead as described in the above section are much lower than initially estimated and are continuing to shrink. Not only are the actual near source emissions from stationary sources now conceded to be 5 percent of the original ARB estimate, but the reentrainment of lead dust will likely continue to diminish as reflected in the revised ambient emissions data. Third, the primary exposures to lead in the state of California are not occurring in the air lead context. These exposures may result from improperly conducted abatement activities, improperly fired ceramics, home remedies, and other non-air mechanisms. This is confirmed by the reports estimating that the contribution of average ambient air lead exposure is somewhere between zero and three percent. Fourth, lead's persistence in the atmosphere has steadily decreased, as reflected by the significant decline in ambient air lead concentrations. Moreover, these trends towards lower near source emissions and the continuing reduction in ambient levels are expected to continue. The ambient levels are so low and the near source exposure so limited that ARB and OEHHA should reduce the priority for regulating inorganic lead as a toxic air contaminant. In fact, this adjustment is required by law.

III. CONCLUSION

For the foregoing reasons, we urge ARB and OEHHA to review the draft report in accordance with these comments.

Respectfully submitted,

Lead Industries Association, Inc.
Battery Council International
GNB Technologies, Inc.

ATTACHMENT 1

College of Physicians & Surgeons of Columbia University | New York, N.Y. 10032
630 West 168th Street
Telephone: (212) 305-5808
Facsimile: (212) 305-5848

DIVISION OF PEDIATRIC HEMATOLOGY-ONCOLOGY
Sergio Piomelli M.D., James A. Wolff Professor of Pediatrics, Director

September 7, 1995

Henry Falk, M.D.
Environmental Hazard & Health Effects (NCEH)
Mail Stop F28
4770 Buford Hwy NE
Atlanta, Georgia 30341-3724

Dear Dr. Falk:

I have read the draft version of the revision to the CDC document on childhood lead poisoning and I am submitting the following written comments, since, as you know, I cannot be at the September 12th meeting, because of previous commitments outside the USA.

As a Committee member, I request that these comments be distributed and discussed at the meeting.

My comments will address exclusively two crucial issues:

1. The definition of the threshold for *"lead poisoning"* at 10 µg/dL
2. The results of the NHANES III that clearly support targeted screening.

1. The definition of the threshold for "lead poisoning" at 10 µg/dL

The draft ignores the recent conclusions about the effect of lead on children's intelligence and remains anchored to a definition of *"lead poisoning"* that is unscientific, alarmist, arbitrary, and not supported by the current evidence[4,5].

- Recently two separate meta-analyses have quantitated the effects of lead on children's intelligence[7,8]. Despite the different approaches, both came to very similar conclusions. The reduction of IQ as the blood Pb increases from 10 µg/dL to 20 µg/dL was assessed by one meta-analysis at between 1 and 2 IQ points, and by the other at 2.6 IQ points. We have therefore now a solid quantitative estimate of the effects of lead on children's intelligence on which to base a rational definition.
- Even using the highest meta-analyses estimate, a child with a blood Pb level of 11 µg/dL is at *"risk"* of losing 0.26 IQ point, an effect unmeasurable in the individual child. Everybody would agree that such an effect is trivial and insignificant. Yet, the CDC guidelines recommend that parents of a child with such a blood Pb level be told *"your child has lead poisoning,"* an assessment that creates panic, anguish and confusion to thousands of families.
- Almost anybody would agree that the potential loss of 0.26 to 1.3 IQ points, as may be observed in a cohort of children with blood Pb level 10-14 µg/dL, is trivial.

- Many would agree that even a potential loss of 1.3 to 2.6 IQ points, as may be observed in a cohort of children with blood Pb level 15-20 µg/dL, is trivial and unmeasurable in the individual child. The effect that an average loss of 2.6 IQ points may have on the frequencies of children with an IQ ≤80 points or ≥125 points (see page-11 of the 1991 statement), appears insignificant, when calculated statistically.
- Although we know the magnitude of the IQ loss as the blood Pb increases from 10 µg/dL to 20 µg/dL, we do not know whether this effect is linear over that entire range. It appears more likely that the rise is exponential, hence the effect is probably even more negligible in the lower part of the 10 to 20 µg/dL range.
- Moreover, at a blood Pb level of 10 µg/dL, there is considerable background noise, provided both by the analytical inaccuracy at that level (±2 µg/dL in the best laboratories) and by day-by-day variation[5]. Thus, many blood lead levels initially reported between 10 and 12 µg/dL prove, on repeat testing, to be below the magical 10 µg/dL. Yet, the CDC recommends that the parents of such a child be told "*your child has lead poisoning, and should be retested in 3 months*". This is irresponsible and lacks respect and understanding for the involved families.

It is evident that children with blood Pb level 10-19 µg/dL cannot be classified as cases of "lead poisoning," as this definition implies significant adverse effects, not supported by the present evidence. This all-inclusive definition of lead poisoning is unacceptable. Its consequences have been far reaching. Many have taken 10 µg/dL as "*the level of concern*" or "*the level of intervention*"[1]. States have passed legislation based on it. All this is unjustified.

It is the responsibility of the CDC to provide guidelines to the nation that are based on a careful assessment of the risks involved[5]. The average blood Pb level of U.S. children is today 3.7 µg/dL, with a log-normal distribution and a standard deviation of 0.642 (antilog = 1.9 µg/dL). Thus, children with blood Pb levels = 10-13.5 µg/dL are not even outside two standard deviations from the mean. This, and other evidence mentioned above that has accumulated since this definition was hastily reached in 1991, indicate that setting a threshold for "*lead poisoning*" at 10 µg/dL is poor public health policy and it fails the duty of the CDC to provide guidelines judiciously derived from the scientific evidence. The CDC should have the courage to recognize and rectify this error.

Children with blood Pb level of 14-20 µg/dL are outside two standard deviations from the mean and cannot be classified as "*average.*" Several appropriate definitions could be applied to these children, such as:

- "*modestly increased lead burden*".
- "*minimally elevated blood lead*", etc.

Any definition including the word "*poisoning*" would not reflect accurately the current evidence, and would be unnecessarily alarmist.

2. The results of the NHANES III clearly support targeted screening.

The opening statement of the draft: "*Childhood lead poisoning remains a major environmental health problem in the United States. Despite progress in addressing lead poisoning, around 1.7 million children in the U.S. under the age of six continue to have elevated blood Pb level*" is alarmist, inaccurate, and exaggerated.

This statement fails to properly acknowledge the incredible decrease in blood Pb level of the US children from 1980 to 1991 (e.g., the percentage of children with blood Pb level ≥ 10 µg/dL decreased from 88.2% to 8.9%; the percent of those with blood Pb level ≥ 20 µg/dL, even more impressively from 24.7% to 1.1%)[6].

I am very proud to have been among those who contributed to this magnificent public health achievement through active presentation of evidence at the EPA hearings that led to the removal of lead from gasoline[3].

It can be easily seen from the NHANES III phase 1 report that of the 1.7 million children referred to above[1],

- 1,156,000 have a blood Pb level 10 to 14 µg/dL
- 298,000 have a blood Pb level 14 to 19 µg/dL

These children only have elevated blood Pb levels because of the 1991 CDC definition, but have no meaningful health effect. (Is the wording "*epidemic by edict*"[7] really inappropriate?)

The NHANES III phase 1 report indicates that only 93,000 US children have blood Pb level above 25 µg/dL. This is the level when significant and severe lead poisoning occurs, and the IQ loss clearly rises well above 2.6 points.

The NHANES III phase 1 report also indicated that, now more than ever, childhood Pb poisoning today is not "*a major environmental health problem in the United States*", but it remains a disease of the poor and underprivileged. Of the 93,000 US children estimated to have a blood Pb level ≥25 µg/dL, at least 57,000 (61%) belong to the Afro-American and Mexican-American minorities. And, even among the remaining 36,000 white-Americans, the majority are estimated to be from the urban poor. (Similar proportions are applicable to the 205,000 children with blood lead level above 20 µg/dL and to the 42,000 children with blood lead level above 30 µg/dL).

The data from the NHANES III most cogently argue that screening of children should be targeted to the poor minority children and to the children of the urban poor. The present recommendation that suggests inclusion in the screening of millions of non-Hispanic white American non-poor children diverts precious funds from the poor to the rich children. Funds that should be used for the Afro-American children, the Mexican-American children and the poor non-Hispanic white-American children, who are still at the greatest risk, are wasted to test the least poor non-Hispanic white-American children, who are at minimal risk. This policy is unfair, and both socially and racially biased.

I urge the CDC to modify the draft, by including and discussing objectively:

- the implications of the two recent meta-analyses;
- the implications of the NHANES III study.

I request that the draft be modified

1. by redefining *"lead poisoning"* as blood Pb level ≥20 µg/dL, and classifying children with blood lead level between 14 and 19 µg/dL by a more appropriate and not alarmist definition, that should not include the word *"poisoning"*, since these children have no or only minimal significant health effect.
2. by renouncing the wasteful and unnecessary universal screening, and recommending instead intense targeted screening of poor and minority children, particularly in the urban areas;
3. by recommending that funds presently directed at screening children at no risk be instead used to delead the housing stock, as suggested in the CDC *"strategy"* document in 1991[2].

As an investigator and a physician who fought against childhood lead poisoning for many years, I cannot, in conscience, subscribe to conclusions that I consider unscientific and unjustifiable. Unless the draft is rectified to include the above modifications, I shall exercise my right, as a Committee member, to attach to the final document a minority report.

Respectfully submitted.

Sergio Piomelli, MD
James A. Wolff Professor of Pediatrics

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ATTACHMENT 2

IQ, LEAD LEVEL, AND INFERENCES FROM RESEARCH STUDIES

Comments Addressing The Underlying Science Forming The Basis Of OEHHA's Analysis

by

Alan S. Kaufman, Ph.D.*

Senior Research Scientist
Psychological Assessment Resources, Inc. (PAR)
Odessa, Florida

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* Under a consulting agreement, Ethyl Corporation asked for my help to better understand the use of intelligence testing in studies on low levels of blood lead and IQ. Work done under this agreement prompted these comments. The conclusions and views expressed are my own, and are based upon my understanding of the scientific literature.

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I. Introduction

When research results form the basis for regulatory policies flowing from analyses such as the Health Assessment provided by California's Office of Environmental Health Hazard Assessment (OEHHA), it is in the interest of everyone that these research results be rooted in excellence and conform to the highest standards of science. Since neuropsychological research into the relationship of low levels of blood lead to intelligence is the principal basis for OEHHA's recommendations, I believe it is important for me to comment in light of my particular background in these areas. My Ph.D. is from Columbia University in the area of Psychology with a Major in Measurement, Research, and Evaluation. Along with my wife, Nadeen, I co-authored the Kaufman Assessment Battery for Children (K-ABC), the Kaufman Test of Educational Achievement (K-TEA), the Kaufman Adolescent and Adult Intelligence Test (KAIT), the Kaufman Short Neuropsychological Assessment Procedure (K-SNAP), and four other neuropsychological tests. I worked closely with David Wechsler on the revision of the Wechsler Intelligence Scale for Children (WISC) and supervised the standardization of the WISC-R. My 1979 book, Intelligent Testing with The WISC-R, became the standard for WISC-R interpretation; similarly, my 1990 book, Assessing Adolescent and Adult Intelligence, became the standard for WAIS-R interpretation and my 1994 book, Intelligent Testing with the WISC-III, has followed suit for the latest revision of Wechsler's children's test. Also, I worked closely with Dorothea McCarthy in the development and standardization of the McCarthy Scales of Children's Abilities, and I co-authored, with Nadeen, the 1977 book that became the standard for McCarthy interpretation (Clinical Evaluation of Young Children with the McCarthy Scales). In addition to these and other books, I have authored more than 100 chapters and professional journal articles on the subject of intelligence testing and neuropsychological assessment, and am a Fellow of both the American Psychological Association and the American Psychological Society, co-editor of the journal Research in the Schools and have served on the Editorial Boards of 12 professional journals (currently, I serve on 8 Boards). I have won four awards for outstanding research, including the Mensa Education and Research Foundation Award for Excellence in 1989.

My comments are aimed at the manner in which the results of neuropsychological research are used in OEHHA's analysis and, in particular, the treatment of IQ in OEHHA's quantitative risk assessment. There has been a tacit acceptance of the results of research studies that have related blood lead levels to children's IQs. OEHHA professionals have concluded that elevated blood lead levels in young children, even relatively slight elevations, are causally related to the loss of several IQ points. Further, OEHHA professionals have inferred that it is possible to relate lead level to IQ loss in a linear fashion, leading to statements such as "several prospective cohort studies of neurodevelopment suggest a 0.33 (± 0.01) decrease in IQ points, based on the WISC-R full scale IQ test (FSIQ), per ug/dL increase in blood lead." (Part B, p.5-10.) This use of the research results and the inference of a linear relationship between lead level and IQ loss are open to serious challenge, as I have delineated in the pages that follow.

II. There Are Flaws In The Research Design Of Studies That Have Related Lead Level To IQ.

The initial research studies relating lead level to IQ were filled with blatant flaws, such as failure to control for socioeconomic status. Subsequent studies employed improved methodology but even these so-called "better" studies continued to have serious flaws in their design and execution. For the purpose of this paper, I will define as the "better" studies the 26 investigations that were included in one or more of three meta-analyses: Needleman & Gatsonis (JAMA, 1990); Schwartz (Environmental Research, 1994); and Pocock, Smith, & Baghurst (BMJ, 1994). Each of these studies, whether they implicate lead level as an IQ-depressing variable or whether they indicate that lead level is an insignificant contributor to intellectual functioning, includes several of the flaws indicated here. To provide an overview of the 26 studies, I have prepared Table 1, which lists each study and then denotes with an "X" the various flaws that characterize each investigation. The seven flaws listed in the table correspond to the flaws that are discussed at length in the pages that follow.

1. **Causality cannot be inferred simply from correlational data.**

Nearly all of the 26 studies analyzed correlations between lead level, and IQ as well as between a wide variety of potentially confounding variables (e.g., socioeconomic status) and IQ. When the correlation between lead level and IQ remains statistically significant after controlling for the potentially confounding variables, the researchers have concluded that the elevated lead level in the blood caused the lowered IQ. No such inference is possible from correlational analysis. Significant correlations indicate a meaningful relationship between two variables, but one cannot be inferred to cause the other. If low lead levels are associated with lower IQs, then it is just as feasible that the lowered level of intelligence made it more likely that the young child ate paint chips or dirt. The child with the lower IQ or poorer memory is more likely than the smarter child to forget the parents' rules about not putting paint or dirt in their mouths, or to understand that such behavior is wrong or dangerous. Similarly, elevated lead level is said by some researchers to be significantly associated with Attention-Deficit Hyperactivity Disorder (ADHD) and with concomitant negative behaviors such as impulsiveness, hyperactivity, or poor attention span. It is known that children who are diagnosed with ADHD had to have demonstrated characteristics of this disorder at young ages. Again, if elevated lead level is significantly associated with ADHD or with behaviors such as impulsiveness and inattention, such a correlation only attests to a meaningful relationship between the two sets of variables. Causality simply cannot be inferred. Elevated lead level may cause hyperactive behavior. Or, just as feasibly, ADHD may cause elevated lead level: Young children who demonstrate hyperactive, impulsive, inattentive behavior are much more likely to ingest lead and to fail to heed their parents' warnings and teachings than are their calmer, more reflective, attentive peers.

The warnings given in a standard statistical text (Hopkins & Glass, 1978, Basic Statistics for the Behavioral Sciences) are worth noting:

> The presence of a correlation between two variables does not necessarily mean there exists a causal link between them. Even though concomitance (correlation) between events can be useful in identifying causal relationships when coupled with other methodological approaches, *it is a dangerous and potentially misleading test for causation when used alone* .... [T]he relationships that exist among variables in behavioral and social sciences are almost always too complex to be explained in terms of a single cause .... Failure to recognize that correlation may not mean causation is a widespread logical error" (pp. 144-145, italics added).

A glance at Table 1 makes it clear that an abundance of the lead-IQ investigators made this widespread error.

2. With such a small effect attributed to lead, confounding variables cloud conclusions drawn from even the best studies.

Experimenters have certainly improved in attempting to control potential confounds, but there is still a long way to go in this area. The variables that are undoubtedly most related to a young child's ingestion of lead are the hardest to assess accurately. Such variables involve parenting skills, parenting styles of child rearing, parental time spent with the child, the skills and styles of key caretakers other than the parents, and so forth. These variables have not been measured accurately in any of the 26 studies included in the meta-analyses. Many of the 26 studies have measured socioeconomic status (SES) in a very global way (for example, parents' education, father's occupation, a combination of education and occupation), as shown in Table 1. It is commendable to control for SES, indeed essential, but a global control is not enough. One must attempt to control for specific SES variables concerning the specific subjects in a given study. A few studies have attempted to do just this by administering the HOME inventory (Home Observation for Measurement of the Environment), which requires an observer to go to the child's home and observe the interaction between the parent and child, ask questions of the parent, and observe the environment (e.g., the number of books in the

home). The use of the HOME inventory is a good thing, but there are several flaws with the procedure: (a) there is no way to check on the parents' veracity in responding to questions about parent-child interactions; (b) the HOME inventory is intended as a screening test, based on a relatively brief visit, and is not comprehensive; (c) normative data are inadequate, based on small samples that are not representative of the nation as a whole; (d) there is little or no evidence that the HOME data are consistent from observer to observer or from time to time; and (e) usually the HOME observation takes place at the time of the research study, when the child is older, and may not generalize to the earlier ages when the child ingested lead as an infant or toddler.

In all, 14 of the 26 studies made an effort to obtain some type of specific information about parenting: 9 of the 26 studies administered the HOME inventory and an additional 5 studies interviewed the parent in an attempt to obtain information about parent-child interactions, parenting styles, and so forth. Unfortunately, the other 12 studies, nearly half of the best studies conducted on the lead-IQ relationship relied on global indexes of SES and made no attempt to measure the specific kinds of socioeconomic and interactional variables that are most likely to relate directly to a young child's lead intake.

It would be one thing if the 13 studies that obtained specific parenting information and other data about the child's home could be said truly to have controlled for a large portion of the confounding variance in a child's IQ. But, that is just not the case. Flaws in the HOME instrument have already been delineated, but whatever those flaws are, at least the HOME inventory is a psychometric test that was constructed with care. The parenting questionnaires and interviews used in the 5 studies have typically been made up by the experimenters, vary from study to study, have unknown reliability or validity and, therefore, may yield data that are wholly or partially inaccurate.

But, that isn't even the biggest problem. There are other variables that are known to affect IQ substantially that are rarely considered in the lead-IQ studies. A good case in point

is otitis media. When infants or toddlers have several incidents of ear infections, research has demonstrated that they are likely to have Verbal IQs that are lowered by several points when they reach school age. Yet only 2 of the 26 studies specifically controlled for persistent otitis media or illnesses affecting sensory function (Ernhart et al., 1989; Hatzakis et al., 1989). In the Hatzakis study, the "illness" variable proved to be a significant confound in the lead-IQ relationship; in the Ernhart study, a "Medical Problems" score correlated significantly with IQ. Further, in view of the known strong relationships between a mother's substance abuse or poor nutrition during pregnancy and the child's cognitive ability and behaviors, it is astonishing how relatively few lead-IQ studies systematically adjusted for confounds associated with prenatal factors. Of the 26 studies, only 8 either specifically controlled for pregnancy risk factors or otherwise controlled for maternal smoking in general.

The failure of most researchers to control known key confounding variables such as children's medical problems or prenatal care presents a huge challenge to the validity of the results of lead-IQ studies. Even more of a threat, however, are the potentially confounding variables associated with intelligence that are either unknown or unmeasurable. There is a great deal that we do not know regarding the relationship of environmental variables to intellectual development. A good illustration is the bulk of research that has shown unequivocally that children and adults are getting smarter from one generation to the next at the steady rate of 3 points per decade within the United States (the rate is higher - sometimes twice as high - in many other developed nations in the world). When the research was first published in the late 1970s by the Australian psychologist John Flynn, the data were based on individuals tested between the 1930s and 1970s. The explanation that was widely given to what has now become known as the "Flynn effect" seemed simple enough: In the 1930s and 1940s, there was far less environmental stimulation than in the 1960s and 1970s owing to technology (television and other forms of mass media) and to changes in parenting styles (increased awareness of the importance of providing cognitive stimulation in infancy). But, as the research has continued to accumulate, it has become clear that the 3 points per decade gain did not stop in the 1970s. Rather, it has continued unabated, and was just as constant a gain

from 1985 to 1995 as it was from 1947 to 1957. Quite clearly, the explanation cannot simply be greater exposure to mass media. The precise variables that are responsible for the steady gain in human intelligence from generation to generation are unknown. Some theorists have speculated that improved nutrition is largely responsible, and others give alternate explanations, but current research has yet to answer the question empirically. But one thing is clear: The explanations involve environmental variables because, as at least one geneticist has exclaimed, the generational gains "are rather faster than the gene can travel!"

Why have I dwelled so much on an issue concerning improved IQ when the lead research suggests loss of IQ? For a few reasons. First, whatever environmental variables are responsible for the steady gain are likely to lead to loss of intellectual functioning when they are diminished or withdrawn. Next, the generational research demonstrates quite clearly that sometimes changes of a few points in IQ occur in the absence of a known cause; yet even though people were so sure that the IQ gain was due to the introduction of television and the escalation of mass media, hypotheses are sometimes totally wrong. In this instance, the gain per decade of 3 points is quite similar in magnitude to the size of the effect that lead is purported to have, which makes my analogy even more pertinent.

Ultimately, though the point goes beyond any one topic I have raised here, my overall argument is that most studies of lead and IQ even when limited to the "best" available studies have failed to control for important parenting variables, subtle socioeconomic variables, and medical variables, and they have been unable to control for confounds due to what are undoubtedly a plethora of unknown but potentially potent unknown variables. The net result is to make the results of the lead studies inconclusive and uninterpretable. This point is made even clearer by understanding one significant limitation of the multiple regression procedure that has been used in nearly all of the 26 studies that have been included in the meta-analyses. The procedure requires that one must account for as much of the variability in IQ as possible by first entering the potential confounding variables into the regression equation. Then, lead level is added to the equation to see if it contributes a significant amount of variance to the

prediction of IQ, over and above the prediction that is obtainable from the confounds alone. When lead level adds significantly to the equation, researchers conclude that lead level leads to IQ loss. However, this conclusion is not accurate.

The correct statement is that the significant increase in prediction is due not only to lead level, but also to all other potential confounds - known or unknown - that were not controlled in the study. As the history of lead-IQ research has shown us, the more that pertinent confounding variables are identified and controlled, the smaller the relationship between lead level and alleged IQ loss. Yet, since so many variables that are usually uncontrolled in the lead research bear obvious potential relationship to the ingestion of lead, such as parental supervision of infants and toddlers (and since so many environmental variables associated with IQ are unknown - witness the increase in IQ across generations), it is quite possible if not highly probable that much or all of the so-called IQ loss due to lead level is due to these other confounding variables.

3. Even in the best studies, parental IQ - a key variable affecting children's IQ - was either measured poorly or not at all.

Lead researchers have become aware that one of the strongest correlates both of IQ and lead level in a young child is the child's parental IQ, and that this potential confound must be controlled in lead-IQ studies. Parental IQ is related to SES and to genetic factors; controlling for it as a confound, even when SES is otherwise controlled, is absolutely essential for a competent research design. Although most lead researchers realize this necessity, it is nonetheless true that 8 of the 26 studies failed to measure parental IQ. Furthermore, despite the rigorous attempts that most experimenters made to use state-of-the-art IQ tests for the children in the studies, a similar rigor was not followed when assessing their parents. Only two studies administered the accepted criterion of adult intelligence, the Wechsler Adult Intelligence Scale-Revised or WAIS-R (Baghurst et al., 1992; McMichael et al., 1994 - both reporting on the Port Pirie study). An additional 5 studies used a two subtest short form of the

WAIS or WAIS-R (Vocabulary and Block Design) as the estimate of parental intelligence. In view of the fact that the WAIS and WAIS-R include 11 subtests, the elimination of 9 subtests forces the researchers to estimate the parents' IQs from only a small portion of the complete battery. Even a good short form such as the one used in these studies makes substantial errors when estimating the Full Scale IQ that would have been obtained had the complete battery of 11 subtests been administered. Standard errors of estimate for the Vocabulary-Block Design short form average about 6 points, which means that each parent's obtained IQ on the short form has a two out of three chance to be within 6 points (in either direction) of the IQ the parent would have earned on the complete test battery. That band of error does not represent very good accuracy in view of the importance of controlling for parents' IQs in studies of lead level.

Even worse than a WAIS-R short form, however, is the use of the Peabody Picture Vocabulary Test-Revised (PPVT-R) as the measure of parents' intelligence. This is a one subtest measure that is not an intelligence test. Dunn and Dunn, authors of the 1981 PPVT-R, state in the manual that, "The PPVT-R is designed primarily to measure a subject's receptive (hearing vocabulary .... It is not, however, a comprehensive test of general intelligence" (p. 2). It yields "standard score equivalents," not IQs, and is misused as a measure of general intelligence. Nevertheless, this quick-and-easy test (or its predecessor, the PPVT) has been used as the sole measure of parents' intelligence in 5 studies, while other brief tests or two subtest combinations have been used in a total of 6 studies.

Overall, parental IQ was not assessed in 8 studies and was assessed inadequately (i. e., by a picture vocabulary test-PPVT, PPVT-R, Quick Test or a group-administered test) in 8 more studies. An additional 8 studies administered a 2 subtest WAIS or WAIS-R short form or other brief measures of intelligence (e.g., Raven's Matrices plus Mill Hill Vocabulary Test). Even though short forms have the built-in error described previously, and are not nearly optimal measures in studies that affect public policy - I have not listed the 8 "brief test"

studies as "flawed" in Table 1, because at least these short measures assess intelligence validly.

Also, despite the desirability of controlling for both parents' IQs in lead studies, only a single study systematically tested both fathers and mothers (Lansdown, et al., 1986, who administered the WAIS short form to both parents). With the exception of a few studies that tested the caretaker of the child (typically the mother), virtually all attempts to control for the potential confounding of maternal IQ. It is true that there are practical issues involved in getting cooperation from parents for research studies (as Ernhart et al., 1989, pointed out), but that does not excuse using a test that is not intended as an IQ test (PPVT-R) or virtually eliminating fathers from consideration. And it certainly does not excuse Bellinger, et. al. (1992), for using the original PPVT, which was standardized on an all-white sample from Nashville, TN, in 1959, as the measure of maternal IQ. Not only isn't picture vocabulary a good way of estimating IQ, but the test's norms were 30 years out of date when Bellinger and Needleman collected their data.

The net conclusion is that even though researchers have claimed to control for the key potentially confounding variable of parents' IQs, nearly all studies have done an unimpressive job of it. In the previous section, it was pointed out that whenever lead level is found to add significantly to a regression equation, the proper interpretation is that the significant increase in prediction is due not only to lead level, but also to all other potential confounds - known or unknown - that were not controlled in the study. That statement now requires elaboration to include those confounding variables, like parents' IQ that were controlled, but inadequately.

4. It is inappropriate for researchers to conduct many analyses at once, and then choose to interpret only the ones that support their position.

Competent experimental research requires that the investigators hypothesize the results they are anticipating before they examine the data, not after they examine the results of the study. It is wrong to conduct "multiple analyses" and then interpret only the ones that give the

answers they were seeking. The type of research that involves conducting many analyses at once and then picking and choosing the analyses the experimenters like best, is known informally as a "shotgun approach," or "seek-and-ye-shall-find" research. Whenever many analyses are conducted simultaneously, the odds of finding one with significant results increases dramatically, simply because the shotgun approach allows experimenters to take advantage of chance error. The more analyses that are conducted, the greater the likelihood of finding a significant relationship. Because it is considered wrong and unscientific to take advantage of chance error, correct research methodology requires experimenters to control for the chance errors that necessarily creep in when conducting simultaneous multiple comparisons. One popular such correction is known as the Bonferroni procedure, although other techniques are applied as well.

Unfortunately, several of the 26 lead-IQ studies made this precise kind of error: The researchers used a shotgun approach, and then honed in on the specific significant results that were consistent with their a priori hypotheses about the importance of lead level on a child's intellectual functioning; the researchers in these studies made no attempt to control for the multiple comparisons. The most notable studies that made this error in experimental design include the following: Bellinger, et al., 1992; Bergomi, et al., 1989; Fulton, et al., 1989; Hansen, et al., 1989; and Winneke, et al., 1983. The 1992 Bellinger-Needleman study serves as a good illustration of this type of error in experimental design. These investigators related blood level at 7 points in time (cord, 6 mo., 12 mo., 18 mo., 24 mo., 57 mo., and 10 yr.) to WISC-R Verbal, Performance, and Full Scale IQ (Table 2 of their article). Thus, they made 21 simultaneous comparisons of which only 2 were statistically significant (WISC-R Verbal and Full Scale IQ at age 24 mos.). When the obtained probabilities are corrected by the Bonferroni procedure (which Bellinger-Needleman did not do), neither of the two WISC-R relationships in Table 2 remain significant at the .05 level. The authors, likewise, made multiple comparisons with achievement scores from the Kaufman Test of Educational Achievement that I developed with my wife (Table 3 of their article), but failed to correct for chance error. It is true that the Bellinger- Needleman team offers possible explanations for the

significant results at only one of the 7 ages in the Discussion section of their article (although they never mention the obvious multiple-comparison/chance-error explanation), and they advise that the significant findings "should be interpreted cautiously until confirmed by other studies" (p. 859). However, the authors do not heed their own advice. In the Abstract to the article, they feature the significant findings at age 24 mo. without once advising caution, and little caution has been followed when the results at age 24 mo. have been the only results interpreted in the meta-analyses.

5. It is inappropriate to compare the IQs of the two extreme "lead-level" groups when several additional groups are included in the study.

An error in experimental procedure that is similar to the shotgun type of errors described in the preceding section concerns the comparison of mean IQs earned by those children who have the lowest lead levels with the mean IQs of those children who have the highest lead levels. Again, such comparisons are not warranted when multiple regression methodology is applied. The regression procedure involves relationships among predictor variables (lead level + the potential confounds) and the criterion variable (typically IQ) for the total sample of children in the study. This technique permits interpretation of data for the total sample. It does not allow the experimenter to pluck out a portion of the sample - such as the two groups with extreme lead levels - and eliminate the rest (usually the bulk) of the sample.

Even though the IQs for the extreme groups that are compared have been adjusted for the potential confounding variables, those adjustments were based on the large total sample. In order to properly compare the two extreme groups (which, together, now comprise a new "total" sample that is considerably smaller than the original one), a new set of adjustments must be made for the confounding variables. Otherwise, there is no way of knowing that the "high lead" and "low lead" groups are properly matched on SES, maternal IQ, and so forth. Without the assurance that the two extreme groups are matched with each other on the confounding variables, the most probable explanation of a mean IQ difference between them is

one or more of the confounding variables. Unfortunately, none of the researchers who have compared the mean IQs of the extreme groups have conducted the pertinent additional multiple regression analyses based only on the two extreme groups.

As with the problem of multiple comparisons, the effect of chance errors looms very large when only data from the extreme groups are analyzed to determine the supposed effect of lead level on IQ. It is possible to compare groups who differ in lead level based on the reanalysis of data described in the previous paragraph, but the most appropriate technique for group comparison is analysis-of-variance (ANOVA) methodology, as employed in the study conducted by Smith et al., 1983. In that study, three groups were specified in advance, they were matched on pertinent variables, the relevant statistical procedure was applied, the comparisons among pairs of groups were specified in advance, and corrections for simultaneous multiple comparisons were built into the procedure.

In contrast, several teams of investigators used multiple regression methodology inappropriately when they compared the two extreme "lead-level" groups. Examples of these studies are Dietrich, et al., 1993; Fulton, et al., 1987 (who divided their sample into 10 subgroups and then compared the two extremes); Hatzakis, et al., 1989; and Needleman, et al., 1979. Whenever mean differences are obtained from multiple regression studies in the manner described here, such mean differences are uninterpretable and cannot be meaningfully used to reach inferences on the relationship between lead level and IQ.

6. The central outcome measure used in even the best studies - children's IQ - was inadequately assessed in a few studies.

The problems with short forms were mentioned previously regarding the assessment of parents' intelligence. Those problems loom even bigger when a short form is used to measure the lead-exposed children's intelligence. Three studies administered a four-subtest short form of the WISC (Winneke, et al., 1985; Winneke, et al., 1990) or WISC-R (Bergomi, et al.,

1989), and a fourth study administered an eight-subtest WISC-R short form, eliminating two-subtests - Picture Arrangement and Comprehension (Silva, et al., 1988). For the four-subtest short forms, the standard error of estimate for predicting the IQ the child would have earned on the complete test battery is ± 5 points. And even though the error of estimate is smaller for the eight-subtest short form (about ± 3 points), the two particular subtests eliminated are the two best measures of real-life, common-sense social intelligence in the Wechsler scales (the precise kind of tasks that one would most want to include if the goal is to translate IQ scores to future functioning in society). Note that the errors of estimate are of about the same general order of magnitude as the amount of IQ loss that is purported to be associated with elevated lead level. It is not correct to infer a loss of intellectual functioning of 3, 4, or 5 points if that is the size of the error in the estimation of each child's IQ. Yet, 4 of the 26 studies (15%) did just that in their investigations of lead level and IQ.

7. Research suggests that differences due to lead level should be greater on Wechsler's Performance IQ than Verbal IQ, but the opposite pattern is found more often - Why?

If elevated lead level in the blood is associated with decreased intellectual functioning, as is alleged by many researchers and OEHHA professionals, then the implication is that the presence of lead causes some type of neurological dysfunction or damage in the child that impairs cognitive ability. A large body of research on brain-behavior relationships has indicated that it is Wechsler's Performance Scale - more so than his Verbal Scale - that is particularly vulnerable to the effects of brain damage or dysfunction. In fact, Performance IQ is usually affected by any kind of brain damage, to the left hemisphere, right hemisphere, or both hemispheres. There is something about the nonverbal, visual-spatial problems on Wechsler's Performance Scale that is especially sensitive to brain damage.

Whereas it is the Performance IQ that is most affected by brain damage or brain dysfunction of any sort, it is Verbal IQ that is most related to SES variables. Therefore, if lead level in the blood causes lowered IQs because of some type of brain damage (as is alleged by many lead researchers), then it should be Performance IQ, and not Verbal IQ, that is most affected by high lead level. In fact the opposite has occurred. Many studies show similar IQ losses (or no losses at all) for both the Verbal and Performance IQs. But, when different results emerge for the two IQs, it has typically been the Verbal IQ that has been most affected by the lead. There are occasional exceptions, such as the Cincinnati Lead Study (Dietrich, et al., 1993), but more often than not it is the Verbal IQ that has been affected rather than the Performance IQ (e.g., Baghurst, et al., 1992; Bellinger, et al. 1992; Hansen, et al., 1989). This finding of greater losses in Verbal than Performance IQ was noted in the earlier lead-IQ studies, as well as the more recent, better-designed studies, leading Smith to state in her 1985 review of studies (Journal of the American Academy of Child Psychiatry), "In most, but not all studies ..., differences between lead groups in IQ scores are predominantly in the verbal IQ, and less so in performance IQ .... This is inconsistent with clinical experience which shows that verbal IQ is more sensitive to socioeconomic factors, while performance IQ is more vulnerable to neurotoxic insult such as excessive alcohol intake" (p. 31). But, it is not just clinical experience that suggests that verbal abilities are more vulnerable to SES factors and nonverbal abilities are more vulnerable to neurological insult; a wide variety of research on brain damage and alcoholism supports these clinical observations.

The fact that Verbal IQ has generally decreased more than Performance IQ in studies of lead level increases the possibility that the decrease is due more to unknown or uncontrolled socioeconomic variables than to lead ingestion.

Conclusions Regarding Flaws in the Research Design

The preceding seven points taken together represent a strong argument for interpreting all of the lead-IQ research, both the positive and the 13 negative findings regarding the impact

of low levels of lead on IQ, with a huge amount of caution. The results of these studies, whether they are evaluated one at a time or in meta-analyses, are merely speculative of the possible role of lead level on IQ functioning. They are not conclusive, and should not be interpreted as fact. There is so much that we do not know about the normal and abnormal development of children's intelligence particularly regarding advantageous or deleterious environmental variables, that it is highly probable that unknown socioeconomic or child-rearing factors are responsible for the alleged IQ loss that is often attributed to low levels of blood lead. At the same time, there is much that we do know from decades of research on brain dysfunction, medical problems, prenatal and perinatal factors, and so forth, that makes it quite evident that important, known environmental variables were not controlled in virtually all of the "best" lead-IQ investigations. Again, the same conclusion is inescapable: IQ loss purportedly due to slightly elevated lead level is likely to have alternate explanations because too many known confounds were not controlled.

One might look carefully at the study that probably controlled more important SES and family-related variables than any other study - the 1992 Bellinger-Needleman study. In addition to the HOME inventory, that team of investigators administered to parents the FACES test (Family Adaptability and Cohesive Evaluation Scale), Social Readjustment Rating Scale, Parenting Stress Index, Children's Life-Events Inventory-Revised, and Social Support Network. All of these measures help identify the relationship of many potential child-related and parent-related variables to the child's IQ. In that regard, these experimenters went well beyond the norm established in lead-IQ studies for the control of key potential confounds, and did an excellent job of controlling for variables that have been largely ignored by other lead researchers.

Yet despite these bold efforts, the study has many serious pitfalls. As noted, the essential variable of paternal intelligence was measured only by testing the mother, and using a test that was not intended as a measure of IQ. Indeed, the authors apparently did not give the revised version of the picture vocabulary test (PPVT-R), but gave the 30-year-old original

version, the PPVT. In addition, these authors fell prey to the shotgun approach, choosing to interpret the only statistically significant IQ results that they obtained out of 21 analyses (2 of the 3 at age 24 mos.). As indicated, when proper correction is made for the multiple comparisons, neither of the apparently significant findings remains significant.

But, there are other problems as well. The authors used the WISC-R, which ordinarily would be a good choice of IQ test in view of its stature and validity. But the data were collected around 1990-1991. The experimenters cannot be faulted for not using the WISC-III (it did not get published until 1991), but they can be faulted for using a test that was normed in 1972 - 18 years prior to the data collection. Instead, the authors would have been wise to use a newer measure of intelligence, such as the 1983 Kaufman Assessment Battery for Children (K-ABC) on the 1989 Woodcock-Johnson-Revised Tests of Cognitive Ability. Because of the "Flynn effect," mentioned previously, norms of U.S. tests of intelligence get out of date at the rate of 3 points every 10 years. In 1990, the WISC-R was yielding IQs that averaged about 5 points too high; that is to say, the 1972 norms were 5 points out of date.

In addition, the study included children (95% of whom were white) from relatively higher SES environments. Their average IQs of 116 - 119 reflect "High Average" functioning, even when the IQs are corrected for the 5-point spuriousness due to outdated norms. The technique of multiple regression is very sensitive to the specific sample tested, and the results for one sample are not readily generalizable to samples that differ in meaningful ways from the original sample. Therefore, the results of the Bellinger-Needleman study, even if they truly had been significant (after the Bonferroni correction for multiple comparisons), would only have generalized to similar samples of high SES white children, not to minorities or to whites from lower social classes. Generalizations from multiple regression studies conducted in other countries are likewise not generalizable to children living in the U.S. In view of the fact that the meta-analyses include studies conducted in countries such as Greece, Germany, Great Britain, Denmark, China, Australia, Scotland, and New Zealand, any generalizations to American children from the merged data sets are tenuous at best.

III. There Is No Linear Relationship Between Lead Level And IQ.

Obviously, it is my judgment that the scientific evidence militates against reaching any conclusions about the so-called deleterious effects of slightly elevated blood lead levels. The studies on which such conclusions have been reached are far too flawed to permit such an inference. If my judgment, based on the points I have just presented, is accepted, then there would be no reason for me to proceed further. But, because of the realities of how the results of the lead-IQ studies have been used by OEHHA, I must proceed with a new set of arguments. For the moment, I will assume that the results of the studies are valid, namely, that small amounts of blood lead can cause a few points of IQ loss. Then another issue must be faced: Is it reasonable to assume that the relationship between blood lead and IQ is a linear one that can be extrapolated down to a minute amount of lead in the blood, and extended upward to accommodate large amounts of blood lead? The answer is "No," and that answer is supported by data.

In order to determine whether the relationship is linear, the authors of the various studies would have needed to present adjusted IQs for children at each portion of the lead-level continuum. Most researchers have not done that. Those who have, have presented the data in scatter plots or bar graphs, and these pictorial representations do not seem to reflect linear relationships (even if the authors chose to interpret the relationships as linear).

Dietrich, et al. (1993), presented a line graph (Fig. 2 on p. 42) that shows the mean adjusted and unadjusted Performance IQs for four lead-level groups (0-10 ug/dL, > 10-15 ug/dL, > 15-20 ug/dL, and > 20 ug/dL). Only the adjusted values are interpretable, and these show no meaningful difference among the first three groups (each averaging a Performance IQ of 90 ± 2). Only the most extreme lead group deviated from the other three (averaging about 85 on Performance IQ), suggesting a threshold effect (at about 20 ug/dL) rather than a linear relationship.

In the 1992 Bellinger-Needleman study, a bar graph is shown (page 858) that presents adjusted WISC-R Full Scale IQs and K-TEA Battery Composite standard scores for the following groups: 0-4.9 ug/dL, 5.0-9.9 ug/dL, 10.0-14.9 ug/dL, and ≥ 15.0 ug/dL. The two groups with the lowest lead levels were indistinguishable from each other, averaging IQs of 118 - 120 and standard scores of about 119 - 122. Similarly, the two groups with the highest lead levels were indistinguishable from each other, each earning mean IQs of about 112 and mean standard scores of about 110. Again, a threshold effect (this time at about 10 - 15 ug/dL) is a more realistic explanation of the relationship than is a linear one.

Hatzakis, et al. (1989) presented a line graph (Fig. 5, p. 220) that shows unadjusted and adjusted WISC-R Full Scale IQs for the following groups: ≤ 14.9 ug/dL, 15.0-24.9 ug/dL, 25.0-34.9 ug/dL, 35.0-44.9 ug/dL, and ≥ 45.0 ug/dL. The lowest two groups did not differ meaningfully from each other, averaging adjusted IQs of 90 + 1. Likewise, the highest three groups earned similar mean IQs of 85 ± 2. Once more, there was an apparent threshold, this time at about 25.0 - 34.9 ug/dL. Although the graph suggested linearity for the four groups with lead levels of 15 and above (adjusted mean IQs of about 91, 86, 84, and 83 with increasing lead level), there was decidedly no linearity for children with lead levels below 15 ug/dL.

Fulton, et al. (1987) presented a scatterplot for 10 lead-level groups (Fig. 1, p. 1223) that are defined by the log blood lead. The mean adjusted British Ability Scales (BASC) score difference from the school mean is presented for each group. Although the authors draw a line of best fit through the points, visual inspection suggests no meaningful difference in the means for any of the samples; the values for nine samples (all but the lowest lead-level group) seem virtually identical to each other. These data suggest neither a threshold effect nor a linear relationship.

The graphs shown by the authors of the aforementioned studies indicate quite dramatically that if lead level truly affects IQ negatively (a big if, as I have explained) then

there is likely a threshold effect to explain the relationship, but there does not exist a linear relationship between lead level and adjusted IQ. Or if such a linear relationship exists, then it does so only at the higher levels of blood lead. There is no evidence whatsoever to support the notion that small amounts of lead in the blood have an impact at all on a child's IQ.

IV. Fractions of an IQ Point Have No Meaning for Public Policy.

The OEHHA documents are filled with statements about 0.33 IQ point or 0.08 IQ point, but such statements have no meaning at all. Fractionating an IQ point is asking an IQ test to do something that it is just not equipped to do. It is a bit like stepping on a $30 bathroom scale and expecting it to give your weight to the nearest hundredth of a pound, or measuring your daughter's height with a 6-inch ruler and trying to record the result to the nearest tenth of a millimeter. The main difference between fractionating an IQ point and these other examples, is that it is even more ridiculous to try to interpret a fraction of an IQ point than to anticipate incredible accuracy from a bathroom scale or ruler.

When Francis Galton first "invented" the IQ test, he defined intelligence as an amalgam of sensory-motor functions such as reaction time, strength of pull, visual and auditory acuity, and so forth. He was able to measure these functions with amazing reliability and accuracy, sometimes to the nearest tenth or hundredth. The problem was that his so-called IQ test, though reliable, was subsequently shown to have no validity as a measure of intelligence. It took Alfred Binet to convince the scientific world that in order to measure something as complex as human intelligence, the measurements had to be complex, such as tests of reasoning, memory, and judgment. And whenever complex measures are used, the inevitable side effect is to have measurement error. Binet's willingness to accept a certain amount of error in order to achieve validity may have been his most important contribution to science.

Even the best IQ tests, such as Wechsler's scales, have a band of error of about ± 3 points that surrounds each person's obtained IQ. And that band of error only affords about 68% accuracy. To be 90% or 95% certain that you have captured a person's "true" IQ within the band of error, that confidence interval must be expanded to ± 5 or ± 6 points. These errors occur for a variety of reasons, such as boredom, fatigue, luck (good or bad), rapport with the examiner, and so forth, and are a built-in aspect of every IQ assessment. Sometimes errors occur in the scoring of tests due to carelessness (a pervasive, serious problem, even among experienced examiners), and sometimes scoring errors occur that cannot be helped. Three of the five WISC-R or WISC-III Verbal subtests that contribute to a child's Verbal IQ (Similarities, Comprehension, Vocabulary) have scoring systems that are very subjective and that require the examiner to distinguish between scores of 0, 1, and 2 for each item. The scoring systems are illustrative, not exhaustive, and some responses are extremely hard to classify, especially the fine distinctions between a score of 0 and 1 or between a score of 1 and 2. Similarly, many of the child's subjective responses to these three subtests need to be queried by the examiner if they are incomplete or ambiguous. Unfortunately, the guidelines for querying are incomplete and ambiguous, leading to considerable examiner differences in exactly which responses they question and how often, in general, they tend to query a child. Experienced scorers will differ from one another both in querying responses and in evaluating each response's merit; consequently, the Verbal IQs earned by a child will vary from examiner to examiner - sometimes by several points - just based on the administration and scoring decisions made on subjective items. I developed the scoring systems with David Wechsler for the subjective WISC-R subtests, and we often disagreed on when to query and how to score certain responses.

Furthermore, the Performance Scale has a similar kind of problem. Three of the five subtests that make up Performance IQ (Picture Arrangement, Block Design, Object Assembly) give up to 3 bonus points for quick, perfect performance on most items. However, the time a child takes to solve an item varies considerably from examiner to examiner. It is often unclear when a child has finished solving an item. Many children will not tell the examiner when they

have finished, even though they are instructed to do so. It is a subjective decision when to stop the stopwatch. Some children may seem like they have stopped solving a puzzle, but they are actually thinking quietly and will suddenly rearrange all of the pieces. It is common to turn off the stopwatch and then have the child continue to complete a nonverbal item. Again, there is great variability in the speed of an examiner's "trigger" with the stopwatch, which affects the number of bonus points earned and can affect substantially the Performance IQ earned by the child.

Since the Full Scale IQ is composed of the Verbal IQ and Performance IQ in equal parts, it is evident that errors on either IQ will add error to the Full Scale IQ. These errors - or differences in administration and scoring decisions - are an unavoidable aspect of IQ test administration, even with highly trained and experienced examiners. When the examiners are less experienced, then the differences from examiner to examiner can be dramatic, as evidenced in a lead-IQ study conducted by Gregory, Lehman, & Mohan (1976, Intelligence scores for children with and without undue lead absorption, In G. Wegner (Ed.), Shoshone, Lead Health Project, pp. 120-150, Boise: Idaho Department of Health and Welfare). These investigators, by happenstance, discovered that the examiner's attitude and demeanor had a greater impact on IQ than the lead level in the child's blood. They noticed that one of the five graduate student examiners came up with relatively low scores for the children tested (average IQ of 90), and another consistently produced inflated scores (average IQ of 104). The first examiner "was very formal, precise, cold, and hurried," while the second offered "support and encouragement that bordered on leading the subjects to the correct answer." (R. J. Gregow, 1987, Adult Intellectual Assessment, Boston, MA: Allyn & Bacon, p. 154).

Unfortunately, inexperienced examiners are commonly used to collect research data, making it reasonable that examiner errors such as the one just described (even if not as extreme) occurred in a number of the 26 "best" lead-IQ studies. Yet, the quality of the IQ test administrations and the concomitant validity of the obtained IQs is rarely assessed thoroughly or systematically (the study by Winneke, et al., 1990, is a notable exception). In all, in view

of the errors that are built into any IQ administration and the other errors that creep in, it is irresponsible to fragment an IQ point for any type of interpretive analysis; no meaningful interpretation is possible.

V. Elevated Blood Lead Level Does Not Affect All Children To The Same Extent.

Implicit in the formulas that are developed by OEHHA to relate lead level to IQ is the notion that each child will respond to about the same degree to similar levels of blood lead. That notion simply is not true, not for lead level or for any number of variables that are known or believed to affect intelligence. For the purpose of the present discussion, let us suppose that lead is ultimately found to be responsible for a loss of 3 IQ points, after removing every conceivable confound. Would such a finding be able to be applied in clinical or other societal settings for a given individual? The answer is a resounding "NO!"

IQ data obtained on groups is reliable, but data on individuals within that group (or within society as a whole) is not. With research studies, it is possible to control for confounding variables, with individuals it is not. Research studies allow us to match a group with high or moderate lead levels to a comparable group with a low lead level. That comparison group, ideally, could match the lead-exposed group on a myriad number of background variables to permit potentially meaningful interpretation of any mean IQ difference observed between the groups. That type of matching is not possible on an individual basis. Even if you found a perfect match for a lead-exposed child, the errors of measurement for individuals are too large to permit a meaningful comparison of the IQs of the two children.

As I've indicated, a reasonable band of error around any individual's IQ score is not less than $\pm$ 5 or $\pm$ 6 points which is larger than the alleged IQ loss due to lead. That reason alone makes it ludicrous to try to interpret an effect that -- even if it is subtracted from a child's IQ score -- leaves an IQ that is still within the band of error.

Also important is the fact that a group difference is an average. It says nothing about the specific effect for a specific child. If children in general are shown to "lose" 3 IQ points due to slightly elevated blood lead level, then any individual child may lose more or less than that value. Some might lose 3 points, some 5 points, some 1 point, and some 0 points.

A huge trauma to the brain, such as stroke or a tumor may cause a large IQ loss in one patient and no loss whatsoever in another patient. For reasons we cannot explain, individuals react differently to the same stimulus. We know based on research data from over 2,000 neurological patients (spanning about 40 studies) that patients with known, localized damage to the right hemisphere will lose about 9 IQ points on Wechsler's Performance Scale. This is a reasonable result because the right cerebral hemisphere controls visual/spatial functions, such as the abilities required to construct abstract designs with blocks, identify the missing part in incomplete pictures, or assemble cut-up picture puzzles (all subtests on Wechsler's Performance Scale). Yet the 9-point loss is merely the average for groups of patients. Some patients will lose 12 points, and others will lose 5 points; some will lose no cognitive function whatever, despite the trauma to an area of the brain that is known to control spatial reasoning. For reasons that are not easily understood, males lose about twice as many Performance IQ points as females even when the trauma is identical.

And the same is true for other variables with known relationships to IQ test scores. As indicated, SES is strongly related to IQ test scores. The differences in mean IQs for extremes of SES are huge. Among adults, for example, the average WAIS-R IQ for college graduates (115) is 33 points higher than the average IQ for adults with 0 - 7 years of schooling. Similarly, mean IQs for professional and technical workers (112) are 25 points higher than mean IQs for unskilled workers (87). These numbers are for Full Scale IQ, but the results are similar for both the school-related Verbal IQ and the "novel problem solving" Performance IQ. The differences are indeed a bit larger for Verbal IQ, since verbal skills improve as a direct result of education, but they are nonetheless quite substantial for Performance IQ as

well. For example, the mean difference between the IQs of college graduates and those with 0 - 7 years of schooling is 33.5 points for Verbal IQ and 26.5 points for Performance IQ.

SES data for children are a bit smaller in magnitude than for adults, but still rather extreme. I have access to data on parents' education for the WISC-III, and parents' occupation data for the WISC-R. The extreme education levels differ by 20 IQ points and the extreme occupational groups differ by 21 IQ points. Again, differences are a little larger for Verbal IQ than Performance IQ, but they are large for both. It is important to note that these substantial SES differences should not be thought of as solely reflecting the influence of environment on IQ tests. Genetics is also an important consideration in determining how far a person goes in formal schooling and in what occupation the person chooses (obviously occupational choice is largely determined by one's educational attainment). Therefore, the huge impact of SES on IQ scores should be thought of primarily as a statement of fact, with no causality either suggested or implied. A good illustration of this lack of causality is the strong correlation between years of formal schooling (a common marker of SES) and IQ. Surely those with higher education will learn more and score higher on IQ tests. But, at the same time, those with low IQs will not be able to complete high school or go on to college. There is simply no way to untangle cause from effect in the IQ-SES relationship.

Yet, despite large average IQ differences between individuals from high or low SES backgrounds, one cannot predict any given person's IQ simply based on SES. There is huge variability in the IQs for children and adults at every socioeconomic strata. Gifted people emerge from low SES backgrounds and low functioning children and adults come from the most advantaged homes. And this substantial variability applies to any environmental variable, even those that have a relatively small impact on IQ. For example, 3 to 5 points of IQ loss are associated with anoxia, malnutrition during infancy, and the effects of birth order (first-borns earn higher IQs than later-borns). A similar magnitude of loss in Verbal IQ has been attributed to middle ear infections (otitis media) if several episodes occur during the first 2 years of life, thereby affecting language development (a point I raised earlier). Again, 3 - 5

points is the average IQ loss attributed to these variables. But, children will differ vastly in whether or not they are affected by malnutrition or the degree to which they are affected, just as radical differences will occur for those suffering from anoxia during the birth process or multiple ear infections during infancy; some might lose 7 IQ points and others will suffer no IQ loss at all.

Thus, even if lead does cause an IQ loss for a group of children, there is simply no way to determine which children will have their IQs affected and which children will be - in effect - immune to lead. In a sense, it is no different from some people being naturally immune to certain viruses. It happens, but we don't know why. The contrast between interpreting group differences versus individual differences is really the contrast between experimental psychology and clinical psychology. The former group attempts to rely on science by conducting careful experiments to uncover significant and meaningful relationships - to discover the "truth." The latter group often attempts to apply these truths to individuals, but that translation from laboratory to real world is fraught with difficulties and often requires as much "art" as "science." Children are complex; once they are no longer subjects in a laboratory experiment but are individuals living in their own particular environment, the various findings from research may or not be helpful for them. A therapeutic method that is shown to produce highly significant improvement for individuals suffering from phobias, for example, may be decidedly unsuccessful for a specific phobic. The same is true for lead level. Even if slight elevations in blood lead are ever shown conclusively to lower IQ for carefully matched groups of children, such elevations may have no effect at all on a given child's IQ.

VI. IQ Is A Relative Concept, Not An Absolute Concept.

In his various writings, Needleman has claimed that the loss of a few IQ points will affect greatly those who are functioning at the low end of the IQ spectrum, creating a substantial increase in the numbers of individuals with low IQs. This contention simply is not true, and may reflect Needleman's lack of appreciation of the true meaning of "low IQ." This

concept is not an absolute; it is not determined by a specific set of skills that indicate that a person is deficient in this or that type of mental functioning. Rather, low IQs are relative concepts. What defines an IQ of, say, 75 changes over time. As I indicated in the discussion of generational changes in IQ, Americans are getting smarter at the rate of about 3 points per decade. That means that the same exact test performance that merited an IQ of 75 in 1960 (e.g., answering 5 questions of general information, solving 3 block designs, defining 6 vocabulary words, etc.) would only merit an IQ of 72 in 1970 and would now (in 1996) merit an IQ of only 64!

The yardstick for defining low, average, and high IQ is constantly changing. Test makers have to continue to determine what level of performance corresponds to different levels of IQ. Some variables may tend to lower a society's IQ (such as rate of unemployment), while others will counteract those variables and raise society's average IQ (such as the amount of information that can be accessed from a personal computer). But the end result is that the same percentage of people will wind up in each "tail" of the bell curve. Why? Because the test makers determine in advance the percentage of people who will obtain every possible IQ. Every time an IQ test is re-standardized, new norms are developed. Those norms reflect the contemporary "yardstick" for equating test performance to IQ level, and the IQ distributions are made to fit the bell curve.

Imagine that scientists discovered a pill that would increase everyone's IQ by 20 points. All of a sudden, the percentage of people in the "tail" at the high end of the curve would increase dramatically and there would be almost no one left in the tail at the low end. The mean would shift from 100 to 120. But, all that would be temporary. The test developers would simply say, "Whoops, our norms are now very wrong. Let's go out and get a new standardization sample." They would do so, and then everything would be back to "normal." The tail at the high end would still produce about 2% who score above 130; except now, these are the people who would have scored 150 on the old norms. And the tail at the low end would still produce about 2% who earn IQs below 70. These people are now in the "mentally

retarded" range of IQ and will perhaps need special placement. It makes no difference that before the magic pill they would have earned IQs as high as 90. Now they are retarded by the new current definition of mental retardation.

IQs are relative concepts. Every time the norms get out of date, the publishers simply re-standardize to get them back in line. It makes no difference whether variables are lowering or raising the IQs of its citizens. New bell curves are always being formed to reflect updated norms, and the percentages of individuals at different IQ levels can never really rise or fall; it will always be returned to the percents that define the normal bell curve. Needleman's argument simply holds no water. IQ is a relative concept that is constantly in flux; there is nothing absolute about it.

VII. IQ Is Only One Aspect Of Human Intelligence.

The OEHHA documents make extreme statements such as, "while a 4-point IQ loss might not have much impact on an individual child, this decrease could have a *significant* public health impact in a community." (Part B, p.3-17.) What about the notion that loss of a few IQ points can have important societal implications? Is that a valid contention? To address this question, I examine the purpose for which IQ tests were developed, the theories on which these tests are based, and alternate theories of intelligence that might be more pertinent to the issue of societal implications.

1. For what purposes are IQ tests designed, and for what purposes are they not designed?

IQ tests originated with Alfred Binet's quest back in the late 1800s and early 1900s in Paris, to identify school children who were likely to do poorly in school. His tests were largely verbal measures of memory, comprehension, judgmental and verbal expression. Nonverbal measures were added to the mix during World War I when new methods were

needed to assess non-English speaking immigrants for service in the military. In the mid-1930s, David Wechsler blended the verbal approach of Binet with the nonverbal emphasis derived from the first world War, and the modern notion of intelligence - as measured by IQ tests - was born.

Contemporary IQ tests, therefore, trace their direct roots to Binet's work in France about a century ago and to the need to develop nonverbal tests to assess members of the Armed Forces in World War I. Binet's goal was to predict school achievement. The World War I psychologists were trying to evaluate the intelligence of people who did not speak English well, and importantly, to detect malingerers. Those were the original goals for constructing the tasks that remain popular for assessing the IQ of children and adults.

Note that these tests were developed from a practical, not a theoretical, perspective. Will school children do well in a regular classroom? Should particular recruits be inducted into the Army? The tasks developed between a half-century and a century ago are the same tasks used today in Wechsler's current series of tests for children, adolescents, and adults. For sure, other tests have been developed from theory during the past 15 years, including the ones I have developed with my wife, but the Wechsler scales reign as the most used instruments in schools and clinics, and are the primary instrument used in lead studies. Therefore, it is reasonable to say that theory has played a limited role in the development of the tasks that constitute Wechsler's tests. Similarly, the results of neuropsychological research based on individuals with brain damage have not influenced the choice of tasks for Wechsler's scales.

As a whole, the prediction of school-related ability is still a main goal of all current individually-administered IQ tests; they have important uses for identifying mentally retarded, learning disabled, and gifted children; adults with mental retardation, dyslexia, Alzheimer's disease; and adolescents and adults who need vocational or scholastic guidance. IQ tests are designed to measure cognitive problem-solving abilities and brain functioning, and are intended for use with a variety of people with known or suspected neurological problems,

emotional or behavioral problems, learning or memory problems, attention-deficit disorders, and the like.

For all of these uses, IQ tests are decidedly not intended to be used alone. Decisions are always supposed to be based on the use of multiple tests. An IQ does not classify a person as mentally retarded unless that person has also scored in the retarded range on a separate measure of adaptive behavior (playing appropriate social roles and functioning competently in society), administered to an informant such as a parent or teacher. A child is diagnosed learning disabled based on an IQ test, an individual achievement test, additional tests of special abilities, and other measures as well. An adult is provided vocational guidance based on the results of an IQ test along with other measures that assess personality variables and vocational interests. A person is diagnosed with neurological impairment based on the results of an IQ test, plus a thorough neuropsychological test battery, and neurological tests such as EEGs. And so forth.

IQ tests measure a limited aspect of human functioning. They are not intended to be used as the sole criterion for making any decisions that have educational, vocational, neurological, or societal implications. They are too narrow in scope and in design. They are not intended to measure interpersonal skills (social intelligence), creativity, special talents, or any of a number of qualities that are commonly associated with intelligent people. When they are used within schools, IQ tests perform reasonably well. Scores on IQ tests typically correlate about .50 to .70 with various criteria of school success. But even coefficients of that magnitude explain about 25 to 50 percent of the variability in achievement scores. That means that variables other than IQ account for one-half to three-quarters of the variability in school success. And once you get out of the school environment and into the work place, the coefficients are even lower. IQ tests predict job success to the tune of correlations that range from about .20 to .40. Such coefficients are often statistically significant, but that does not necessarily mean that they are meaningful in a practical sense. Such values explain only about 5 to 15 percent of the variability in job success, meaning that other variables are responsible

for 85 to 95 percent of the variance associated with performing well on a job.

And when you move out of the school and the work place, correlations are even lower. IQ tests do not typically correlate significantly with any known measures of "life success." IQ and income, for example, correlate very low, with IQ explaining less than 1 percent of the variance (usually much less than 1 percent of the variance) in earned income.

2. What theories form the foundation of popular IQ tests?

There are a great many theories of intelligence, but relatively few have led to the development of individual IQ tests. As noted, neither Wechsler's tests nor the original Binet scale are theory-based, but evolved from practical considerations. Similarly, the British Abilities Scales that has been used in several lead-IQ studies is deliberately non-theoretical. Really only three theories have been very influential in affecting today's tests: (a) Luria's theory of mental processing, (b) Sperry's cerebral specialization theory, and (c) the Horn-Cattell theory of fluid and crystallized intelligence (and Horn's expansion and elaboration of that theory). The first two are neuropsychological in orientation, whereas the third stems from cognitive psychology. Luria's theory forms the foundation (with Sperry's theory) of the K-ABC, and it also is the theoretical model for a new test called the Das-Naglieri Cognitive Assessment System. The Horn-Cattell theory underlies the 4th edition of the Stanford-Binet and the KAIT, while Horn's expanded theory forms the structure of the revised Woodcock-Johnson Tests of Cognitive Ability.

These theories offer a limited view of the world. They all focus on one main area: How people solve problems. Although the KAIT makes an attempt to measure authentic, real-life problems (one of the subtests, Auditory Comprehension, requires understanding of a mock news broadcast), all of the IQ tests taken together do not have a practical focus. The subtests more closely resemble laboratory tasks than the kinds of real-life problem solving that people are confronted with in their daily life. Or they resemble school-like tasks (answering

general information, arithmetic, and vocabulary items), or they are game-like (putting together picture puzzles). But the kinds of things we are asked to do on intelligence tests do not resemble everyday life very closely. The subtests were constructed straight-forward.

Real life is complex and intricate, and is not very conducive to such structure. It involves creative thought, social interactions, and much more than conventional IQ tests were ever intended to measure. IQ tests were developed with a confined purpose. The theories they were built on are few in number and limited in scope, and are concerned primarily with the ability to reason abstractly in artificial situations. IQ tests resulting from such theories, or from no theories at all like the Wechsler scales or BASC, cannot - and were never intended to - have societal implications in and of themselves. They were intended to measure a limited aspect of mental functioning, and to do it well. That's it.

3. What theories are the most valid for answering questions about societal implications of intelligence?

Psychologist Robert Sternberg of Yale University has come up with a highly respected "triarchic" view of intelligence - a three-pronged theory that stresses analytic abilities, practical or adaptive skills, and insightful-thinking skills as components of human intelligence. He criticizes conventional IQ tests for measuring only one of the three essential prongs - analytical abilities (R. J. Sternberg, 1985, Beyond IQ: A Triarchic Theory of Human Intelligence. Cambridge: Cambridge University Press). Another well-respected theorist is Howard Gardner of Harvard University who emphasizes what he refers to as seven kinds of intelligence (H. Gardner, 1983, Frames of Mind. New York: Basic Books).

Both of these theorists have structured a view of intelligence that is multi-faceted, that encompasses a divergent set of intellectual skills, that is very much real-life oriented, and that - in its totality - might reasonably be related to societal impact. Of the two theories, I will focus a bit on Sternberg's triarchic theory because it has been better researched and it has a test

(group administered) known as the Sternberg Triarchic Abilities Test (STAT) which is as yet unpublished.

Basically, Sternberg has broken down cognitive problem solving into a series of components that he has researched fairly extensively. His triarchic notion of intelligence involves applying these components to (a) abstract and academic problems (analytic thinking), (b) novel and unfamiliar problems (creative thinking), and (c) concrete and familiar everyday problems (practical thinking). The analytic component of his theory, and of the STAT, involves tasks such as number series (e.g., 12 16 20 24 _; or 2 8 3 27 4 64 5 _). The creative component includes tasks such as creative analogies. (You are given a "pretend" statement, and then you have to solve the analogy, as if the statement is true. Example: The pretend statement is "Money falls off trees." Then you have to solve the analogy, snow is to shovel as dollar is to: A. bill, B. rake, C. bank, D. green. The correct answer is B. rake.) The practical component includes tasks like route planning (reading maps and finding the shortest route from one place to another) and everyday verbal reasoning. Questions of the latter type require solving problems such as: How can a would-be college student who needs $1,000 per year to supplement her scholarship obtain money yet remain financially independent? How can a teen-age boy who just moved from Arizona to Iowa, who has had a hard time making friends, and who enjoys writing stories, best solve his problem?

IQ tests measure analytic thinking, but not the other two types of intelligence. Creative thinking is required for many aspects of successful functioning in society, both in school, on the job, and in dealing with people. Practical thinking is associated in Sternberg's theory with "tacit knowledge," or the kinds of information and skills that people need to succeed in a variety of situations, for example, getting into the college of your choice, having a successful job interview, maintaining the respect of colleagues who work under you, and so forth. Taken together, the three types of intelligence, though not all-inclusive, would have many more societal implications than any one of the three in isolation.

In Sternberg's research with the STAT, he has shown that the three components of intelligence correlate modestly with each other. Coefficients obtained for 224 high school students in grades 9-12 ranged from .14 to .23. These values indicate a small overlap of only about 2 to 5 percent among the components. The three aspects of intelligence, therefore, are pretty independent of each other and measure different aspects of mental functioning. A person who has low analytic ability (akin to a low IQ on a conventional IQ test) is just about as likely as a person with a high IQ to perform well on creative and practical tasks. In regression analyses intended to predict performance in high school courses, Sternberg showed that the creative and practical components consistently improved prediction significantly over and above the prediction that was obtained from the analytic component. In other words, even for academic courses, there was more to being successful than just having a high IQ. In summary, IQ is too narrow a concept to have societal implications even if exposure to very low levels of lead should be shown to lower IQ by as much as 5 points, much less a fraction of a point. Theories of intelligence that are used to develop IQ tests are much too limited in scope to affect a society as a whole. IQ tasks are not sufficiently real-world-oriented and do not tap an adequate breadth of mental abilities to encompass the kinds of activities that are necessary to maintain and advance a society. When other more comprehensive, theories are applied (in this case, Sternberg's) it is clear that being intelligent within society has multiple components. Merely losing a small amount of ability in one's analytic abilities does not mean that one would also lose the ability to demonstrate practical or creative intelligence. I believe that there are no societal implications whatsoever from any alleged loss in IQ points due to lead level. If people have diminished analytic abilities due to lead, then they can compensate for that small loss quite well by relying on their creative and practical capacities.

VIII. Summary

The preceding portion of this document has focused on the diverse and extensive scientific, conceptual, and rational reasons for challenging OEHHA's treatment of lead and

IQ. These points, taken together, assert that the existing body of research that relates lead level to IQ is replete with major flaws that preclude making any inferences whatever about the alleged loss of IQ points due to slightly elevated blood lead levels. The points raised further challenge the sense of manipulating IQ risk calculations the way OEHHA does.

To summarize the essential points:

1. Causality cannot be inferred simply from correlational data.

Nearly all of the major studies that related lead level to IQ have used the statistical procedures of correlational and multiple regression analysis. Such techniques do not permit any inferences about causality. Significant coefficients denote a meaningful relationship between two variables. From a statistical perspective, however, it is just as likely that low IQ caused slightly elevated levels of blood lead as it is that lead level caused lowered IQs. As noted by the statisticians Hopkins and Glass (1978), "Failure to recognize that correlation may not mean causation is a widespread logical error."

2. With such a small effect attributed to lead, confounding variables cloud conclusions drawn from even the best studies.

No matter how many confounding variables are controlled in the lead-IQ research studies, there are many additional variables - pertaining to subtle aspects of socioeconomic status, childhood diseases, parenting skills, and even unknown influences on IQ - that remain uncontrolled in every lead-IQ study. Whenever lead level is found to be a significant predictor of IQ, after first controlling for several confounds, researchers are quick to conclude that it is lead level, and lead level alone, that accounts for the significant loss of a few IQ points. That conclusion is erroneous and incomplete. The correct statement is that any significant increase in prediction is due not only to lead level, but also to all other potential confounds - known or unknown - that were not controlled in the study. Because many of the uncontrolled factors

bear a powerful relationship to children's IQ, any conclusions about lead level are premature and unfounded.

3. **Even in the best studies, parental IQ - a key variable affecting children's IQ - was either measured poorly or not at all.**

Parents' IQs relate to a variety of genetic and environmental factors that contribute substantially to their children's IQs. This crucial variable has been recognized by many lead researchers, but the measurement of parents' IQs has been done poorly. Such researchers have commonly used very brief tests, such as short forms of Wechsler's scales or short tests of picture vocabulary. The short forms do not provide very good estimates of parents' Full Scale IQs (producing errors of about 6 points) and the picture vocabulary tests were not even intended as measures of intelligence. Furthermore, maternal IQ is the only variable assessed in these studies; fathers are almost universally ignored.

4. **It is inappropriate for researchers to conduct many analyses at once, and then choose to interpret only the ones that support their position.**

Experimenters sometimes use what is known as a "shotgun" approach. They conduct a great number of analyses at once in an attempt to find at least one significant finding. Whenever this approach is used, chance error assumes a large role in the results (the more analyses that are conducted, the greater the likelihood that a significant result will emerge due to chance alone). The correct procedure is to apply a statistical correction to control for the error that occurs when many analyses are done at once. Several of the lead researchers have used this shotgun approach, but none have controlled for the known errors that accompany this approach. A good example is Bellinger-Needleman (1992), who interpreted a significant lead-IQ relationship at 24 months while virtually ignoring the non-significant relationship at 6 other ages. When the appropriate correction is applied to their data, the so-called significant relationship is found to be due to chance.

5. **It is inappropriate to compare the IQs of the two extreme "lead-level" groups when several additional groups are included in the study.**

Several studies of lead and IQ determined the number of IQ points that can be attributed to slight elevations of lead level by comparing the IQs of the two extreme "lead-level" groups (the ones with the highest and lowest lead levels). In these studies, the researchers simply eliminated the middle groups and focused all attention on the extremes. That approach violates the rules for interpreting the results of multiple regression analysis, a procedure that is based on the total group of children; arbitrarily eliminating subgroups of children and focusing on the extremes, once again, takes advantage of chance errors. The number of IQ points attributed to lead level, when based on the "extreme group" procedure, is bogus.

6. **The central outcome measure used in even the best studies - children's IQ - was inadequately assessed in a few studies.**

The fact that poor IQ tests were used to measure parents' IQs in numerous studies is a serious problem, but at least parental IQ is a confounding variable, not the main outcome variable. Yet, a few of the major lead-IQ studies used short forms of Wechsler's scales to measure the children's IQs, even though these short forms have an error of estimate (when predicting Full Scale IQ) that is as large or larger than the IQ effect that is attributed to slight elevations in the level of blood lead.

7. **Research suggests that differences due to lead level should be greater on Wechsler's Performance IQ than Verbal IQ, but the opposite pattern is usually found - Why?**

Based on the results of thousands of research studies, socioeconomic variables are primarily associated with Verbal IQ, and brain damage and neurological dysfunction variables are primarily associated with Performance IQ. Therefore, the logical prediction is that elevated lead level (which is presumed to affect the child's neurological integrity) would produce the largest decrements in Performance IQ. Yet, with only a few exceptions, it is Verbal IQ that has been most affected. The best explanation is that it was uncontrolled or unknown socioeconomic variables - and not lead level, per se - that produced significant IQ decrements.

8. There is no linear relationship between lead level and IQ.

There has been a tacit assumption that the relationship between blood lead and IQ is a linear one that can be extrapolated down to a minute amount of lead in the blood, and extended upward to accommodate large amounts of blood lead. Yet evaluation of the data that have been presented by lead researchers clearly does not support that assumption. When average IQs have been compared for groups that differ systematically in blood lead level, the best explanation of the relationship is as follows: If slight elevations of blood lead produce small decrements in IQ, then there is a threshold effect; no cogent arguments can be made for a linear relationship, and there is no evidence that very low levels of blood lead have any impact on IQ whatsoever.

9. Interpreting fractions of an IQ point has no meaning for public policy.

The fragmentation of an IQ point into fractions, as is commonly done in OEHHA documents, is not meaningful. The IQs have a standard error of measurement of about ± 3 points, resulting from influences such as rapport with the examiner, fatigue, boredom, luck, and mood. Also, different examiners make different administration decisions (e.g., when to query a response) and score subjective verbal items differently, leading to even greater error. The IQ tests are good, but they are not precise enough to permit an IQ point to be subdivided.

Indeed, the bands of error surrounding the IQs earned by children of all ages is even larger than the small number of points attributed by some researchers to slight elevations of blood lead levels.

10. Elevated blood lead level does not affect all children to the same extent.

When a study concludes that a certain number of IQ points are lost due to lead level, that average number is not true for every child. Some children or adults can suffer severe trauma to the head and become brain damaged, and yet suffer no loss of IQ points. The same is true for lead level and any other variable that is believed to be related to IQ. Individuals are affected to differing degrees, and some are not affected at all. IQ is so complex a concept, and is influenced so profoundly by such a plethora of variables, that it is impossible to infer for a given child whether or not a slight elevation in blood lead - or even a substantial level - had any effect at all on that child's cognitive functioning.

11. IQ is a relative concept, not an absolute concept.

Needleman has claimed that the loss of a few IQ points will create a substantial increase in the numbers of individuals with low IQs. This contention is not true, because IQ is not an absolute, determined by a specific set of skills that indicate that a person is deficient in this or that type of mental functioning. Instead, low IQs are relative concepts that define low or high functioning relative to how others of the same age perform on the same test items. The percents of children or adults who earn low IQs on tests of intelligence will remain a constant over time. Whether children are becoming smarter due to increased educational technology or less intelligent due to an impoverished environment or ingestion of lead, these changes from year to year will not change the proportion of individuals who score high or low on an IQ test. Test publishers make sure that the percentage of children earning IQs in a given range (e.g., below 70, above 120) stays the same every time an IQ test is

re-standardized. In short, there will be no increase in low-functioning individuals due to lead level or any other variable.

12. IQ is only one aspect of human intelligence.

Noted Yale researcher and theorist Robert Sternberg has provided evidence that there are three aspects of intelligence - analytic, creative, and practical. IQ tests measure only analytic skills, yet the other two aspects of intelligence are at least as important for determining success in society. Also, Sternberg has shown that the three types of intelligence correlate only modestly with each other. Therefore, losing a small amount of analytic ability does not at all imply a loss of practical or creative intelligence. The only way that an IQ loss attributed to lead level would have societal implications would be if similar losses in creative and practical abilities were shown to accompany the loss in analytic ability. And that has simply not been demonstrated.

As noted previously, Table 1 offers an overview of the 26 "best" lead-IQ tests and documents some of the various specific flaws that characterize each investigation. However, this table is intended primarily as an overview, and actually tends to minimize the problems with the studies as a whole. The table includes only a sampling of the flaws, namely the ones that I chose to elaborate on in this paper. But, as is evident throughout my extensive discussion, there are other flaws that don't even appear in the table (e.g., using a test to measure children's IQ that has 20-year-old norms), and there are other flaws that pervade every study because of limited technology or availability of pertinent data. That is to say, there are many unknown influences on IQ that we have either not yet identified or lack the technology to assess accurately. And there are other key variables that are undoubtedly crucial to a child's ingestion of lead that are virtually impossible to measure accurately and objectively - such as the specific kind of parenting and supervision the child received throughout his or her infancy and early childhood.

It is my sincere hope that you will give careful thought to the points I have summarized here and expressed in detail throughout this paper. I believe that they have sufficient scientific merit to make OEHHA rethink its response to the lead-IQ research and its applications of the results of that research. The implications of OEHHA's current conclusions are profound in view of the shaky tenets on which they are based.

TABLE I

Summary of Flaws Associated with Lead-IQ Studies

Lead-IQ Study[1]	Inferred Causality From Correlational Data[2]	Used a Global Measure of SES[3]	Parental IQ		Did Not Control for Multiple Comparisons[6]	Compared "Extreme" Lead Groups[7]	Used a Short Form to Measure Children's IQ[8]	Lead Affected Verbal IQ More Than Performance IQ[9]
			Mother: Used Poor Measure or None at All[4]	Father: Did Not Test[5]				
Baghurst, P.A., et al. 1992.				X				X
Dietrich, K.N., et al. 1993.				X		X		
Emhart, C.B., et al. 1989.			X	X				
Cooney, G., et al. 1991.			X	X				
Bellinger, D.C., et al. 1992.			X	X	X			X
Hatzakis, A., et al. 1989.	X	X		X		X		
Fulton, M., et al. 1987.	X		X	X		X		
Winneke, G., et al. 1990.	X	X	X	X			X	

Lead-IQ Study[1]	Inferred Causality From Correlational Data[2]	Used a Global Measure of SES[3]	Parental IQ		Did Not Control for Multiple Comparisons[6]	Compared "Extreme" Lead Groups[7]	Used a Short Form to Measure Children's IQ[8]	Lead Affected Verbal IQ More Than Performance IQ[9]
			Mother: Used Poor Measure or None at All[4]	Father: Did Not Test[5]				
Silva, P.A. , et al. 1988.		X	X	X			X	
Yule, W., et al. 1981.		X	X	X				
Lansdown, R., et al. 1986.		X						
Harvey, P.G., et al. 1988.				X				
Wang, T., et al. 1989.		X	X	X				
Ernhart, C.B., et al. 1985.		X	X	X				
Schroeder, S.R., et al. 1985.			X	X				
Hawk, B.A., et al. 1986.			X	X				
Winneke, G., et al. 1985.	X	X	X	X			X	

Lead-IQ Study[1]	Inferred Causality From Correlational Data[2]	Used a Global Measure of SES[3]	Parental IQ		Did Not Control for Multiple Comparisons[6]	Compared "Extreme" Lead Groups[7]	Used a Short Form to Measure Children's IQ[8]	Lead Affected Verbal IQ More Than Performance IQ[9]
			Mother: Used Poor Measure or None at All[4]	Father: Did Not Test[5]				
Ferguson, D.M., et al. 1988.	X		X	X				
Smith, M., et al. 1983.				X				
McMichael, A., et al. 1994.	X			X				
Fulton, M., et al. 1989.		X			X			
Needleman, H.L., 1979.	X		X	X		X		
Winneke, G., et al. 1983.	X	X	X	X	X			
Bergomi, M., et al. 1989.	X	X	X	X	X		X	X
Pocock, S.J., et al . 1987.				X				
Hansen, O.N., et al. 1989.	X	X	X	X	X			X

Notes - Table I

[1]Prospective studies appear first followed by cross-sectional studies. Complete citations along with the type study and additional information are presented in the appendix.

[2]The indicated studies inferred a causal relationship between blood lead level and IQ, which is an incorrect interpretation of a correlation between two variables.

[3]The indicated studies used mainly a global measure of SES (e.g., father's occupation, mother's education), instead of supplementing the global measure with a more specific assessment such as the HOME Inventory or a parent questionnaire that involved direct contact with a caretaker.

[4]The indicated studies either failed to measure maternal IQ or inadequately assessed maternal IQ with only a measure of picture vocabulary (e.g., PPVT-R, Quick Test) or with a group-administered test.

[5]The indicated studies failed to systematically assess the father's IQ.

[6]Valid scientific research requires that investigators hypothesize results, gather data, and form conclusions. Simply gathering data, subjecting it to multiple comparisons, and essentially "hunting" for results is not quality research. The indicated studies engaged in multiple comparisons, selected certain results while ignoring others, and failed to use a control for the multiple comparisons.

[7]The indicated studies compared extreme groups (high blood lead versus low blood lead), while ignoring the groups in between.

[8]The indicated studies inadequately assessed children's IQ by administering an abbreviated version of Wechsler's scales instead of the complete battery.

[9]Performance IQ is most affected by brain damage or dysfunction. Verbal IQ is most affected by SES. The expected result due to lead exposure is decreased Performance IQ in comparison with Verbal IQ. The indicated studies reported a more substantial decrease in Verbal IQ than in Performance IQ.

APPENDIX TO TABLE I

REFERENCE	TYPE STUDY[1]	Pb MEASURED IN	META ANALYSIS[2]
Baghurst PA, McMichael AJ, Wigg NR, Vimpani G, Robertson EF, Roberts RJ, et al. Life-long exposure to environmental lead and children's intelligence at age seven: the Port Pirie cohort study. *N Engl J Med* 1992; 327:1279-84.	P	Blood	P/S,S
Dietrich KN, Berger OG, Succop PA, Hammond PB, Bornschein RL. The developmental consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati lead study cohort following school entry. *Neurotoxicol Teratol* 1993; 15:37-44.	P	Blood	P/S,S
Ernhart CB, Morrow-Tlucak M, Worf AW, Super D, Drotar D. Low level lead exposure in the prenatal and early preschool periods: intelligence prior to school entry. *Neurotoxicol Teratol* 1989; 11:161-70.	P	Blood	P/S
Cooney G, Bell A, Stavron C. Low level exposures to lead and neurobehavioural development: the Sydney study at seven years. In: *Heavy metals in the environment*. Edinburgh: CEP Consultants, 1991; 16-9.	P	Blood	P/S
Bellinger DC, Stiles KM, Needleman HL. Low level lead exposures, intelligence and academic achievement: a long term follow-up study. *Pediatrics* 1992; 90:855-61.	P	Blood	P/S,S
Hatzakis A, Kokkevi A, Katsouyanni K, et al. Psychometric intelligence and attentional performance deficits in lead-exposed children. In: *Heavy metal in the environment*. New Orleans, 1987; 204-9. For more detailed information, see Hatzakis A, Kokkevi A, Maravelias C, Katsouyanni K, Salaminios F, Kalandidi A, et al. Psychometric intelligence deficits in lead-exposed children. In: Smith MA, Grant LD, Sora AI, eds. *Lead exposure and child development*. London: Kluwer, 1989; 211-23.	C	Blood	P/S,N,S

REFERENCE	TYPE STUDY[1]	Pb MEASURED IN	META ANALYSIS[2]
Fulton M, Thomson G, Hunter R, Raab G, Laxen D, Hepburn W. Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* 1987; I:1221-6.	C	Blood	P/S,N,S
Winneke G, Brockhaus A, Ewers U, Kramer U, Neuf M. Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. *Neurotoxicol Teratol* 1990; 12:553-9.	C	Blood	P/S
Silva PA, Hughes P, Williams S, Faed JM. Blood lead, intelligence, reading attainment, and behaviour in eleven year old children in Dunedin, New Zealand. *J. Child Psychol Psychiatry* 1988; 29:43-52.	C	Blood	P/S,S
Yule W, Lansdown R, Millar IB, Urbanowicz M-A. The relationship between blood lead concentrations, intelligence and attainment in a school population: a pilot study. *Dev Med Child Neurol* 1981; 23:567-76.	C	Blood	P/S,N,S
Lansdown R, Yule W, Urbanowicz M-A, Hunter J. The relationship between blood-lead concentrations, intelligence, attainment and behaviour in a school population: the second London study. *Int Arch Occup Environ Health* 1986; 57:225-35.	C	Blood	P/S,N
Harvey PG, Hamlin MW, Kumar R, Morgan G, Spurgeon A, Delves HT. Relationships between blood lead, behaviour, psychometric and neuropsychological test perforance in young children. *British Journal of Developmental Psychology* 1988; 6:145-56.	C	Blood	P/S
Wang T, Xu S-E, Thang G-D, Want W-Y. Study of lead absorption and its effect on children's development. *Biomed Environ Sci* 1989; 2:325-30.	C	Blood	P/S
Ernhart CB, Landa B, Wolf AW. Subclinical lead level and developmental deficit: re-analyses of data. *J. Learn Disab.* 1985; 18:475-479.	C	Blood	N

REFERENCE	TYPE STUDY[1]	Pb MEASURED IN	META ANALYSIS[2]
Schroeder SR, Hawk B, Otto DA, Mushak P, Hicks RE. Separating the effects of lead and social factors on IQ. *Environ Res.* 1985; 38:144-154.	C	Blood	N
Hawk BA, Schroeder SR, Robinson G, et al. Relation of lead and social factors to IQ of low-SES children: a partial replication. *Am J Ment Def.* 1986; 91:178-183.	C	Blood	N,S
Winneke G, Beginn U, Ewert T, Havestadt C, Kraemer U, Krause C, et al. Comparing the effects of perinatal and later childhood lead exposure on neuropsychological outcome. *Environ Res* 1985; 38:155-67.	C	Blood	P/S
Ferguson DM, Ferguson JE, Horwood LJ, Kinzett NG. A longitudinal study of dentine lead levels, intelligence, school performance and behaviour. Part II. Dentine lead and cognitive ability. *J. Child Psychol Psychiatry* 1988; 29:793-809.	C	Tooth	P/S,N
Smith M, Delves T, Lansdown R, Clayton B, Graham P. The effects of lead exposure on urban children: the Institute of Child Health/Southampton Study. *Dev Med Child Neurol* 1983; 47(suppl):1-54.	C	Tooth	P/S
McMichael A, Baghurst PA, Vimpani GV, Wigg NR, Robertson EF, Tong S. Tooth lead levels and IQ in school-age children: the port pirie cohort study. *Am J Epidemiol* 1994; 140:489-99.	C	Tooth	P/S
Fulton M, Paterson L, Raab G, Thomson G, Laxen D. Blood lead, tooth lead and child development in Edinburgh. In: Vernet JP, ed. *Heavy metals in environment.* Vol 2. Edinburgh: CEP Consultants, 1989; 68-71.	C	Tooth	P/S
Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979; 300:689-95.	C	Tooth	P/S

REFERENCE	TYPE STUDY[1]	Pb MEASURED IN	META ANALYSIS[2]
Winneke G, Kramer U, Brockhaus A, Ewers U, Kujanek G, Lechner H, et al. Neuropsychological studies in children with elevated tooth-lead concentrations. II. Extended study. *Int Arch Occup Environ Health* 1983; 51:231-52.	C	Tooth	P/S,N
Bergomi M, Borella P, Fantuzzi G, Vivoli G, Sturloni N, Cavazzuti G, et al. Relationship between lead exposure indicators and neuropsychological performance in children. *Dev Med Child Neurol* 1989; 31:181-90.	C	Tooth	P/S
Pocock SJ, Ashby D, Smith M. Lead exposure and children's intellectual performance. *Int J Epidemiol*. 1987; 16:57-67.	C	Tooth	N
Hansen ON, Trillingsgaard A, Beese I, Lyngbye T, Grandjean P. A neuropsychological study of children with elevated dentine lead level. In: *International Conference on Heavy Metals in the Environment*. Edinburgh, Scotland: CEP Consultants 1987; 54-56. For more detailed information see: Hansen on, A neuropsychological study of children with elevated dentine lead level assessment of the effect of lead in different socio-economic groups. Neurotox Teratology 1989; 11:205-213.	C	Tooth	N

[1] P = Prospective; C = Cross-sectional

[2] Study included in one or more of the following meta analyses:

P/S = Pocock, Smith
Pocock SJ, Smith M, Baghurst P, Environmental lead and children's intelligence: a systematic review of epidemiological evidence. Brit Med J 1994; 309:1189-97.

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Natural Resources Defense Council

71 Stevenson Street
San Francisco, CA 94105
415 777-0220
Fax 415 495-5996

Comments on:

Proposed Identification of Inorganic Lead as a Toxic Air Contaminant

Draft SRP Version

Submitted by Gina M. Solomon, MD, MPH

Senior Staff Scientist

Public Health Program

Natural Resources Defense Council

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40 West 20th Street
New York, New York 10011
212 727-2700
Fax 212 727-1773

1350 New York Ave., N.W.
Washington, DC 20005
202 783-7800
Fax 202 783-5917

6310 San Vicente Blvd., Suite 250
Los Angeles, CA 90048
213 934-6900
Fax 213 934-1210

The Natural Resources Defense Council (NRDC) is a national environmental organization dedicated to protecting human health by reducing exposures to environmental pollutants. On behalf of our 300,000 members nationwide, and our over 55,000 members in the State of California, NRDC is submitting comments on the draft document prepared by the Air Resources Board (ARB) staff and the Office of Environmental Health Hazard Assessment (OEHHA) on the *Proposed Identification of Lead as a Toxic Air Contaminant.*

NRDC is concerned about human exposures to airborne lead because lead is a toxic metal that has well known, severe, and long-lasting health effects at environmental levels of exposure. Lead interferes with the development of the human brain and is known to result in decrements of IQ and in other neurobehavioral effects. Children, particularly those living in poverty in urban areas, are known to be at particularly high risk of lead overexposure. While much of this exposure comes from lead paint, significant exposures can also come from air sources and the deposition of lead-bearing particulate on soil and hard surfaces. A national survey revealed that an estimated 1.7 million children between the ages of one and five years have blood lead levels in excess of the current recommended guideline of 10 μg/dl. The prevalence of elevated lead levels was about two and a half times higher in nonwhite children than in white children[1]. With these numbers of children at risk, there is marked urgency in controlling all sources of lead exposure. With these concerns in mind, we are urging rapid movement to further control exposures to airborne lead in California.

As required by the California Health and Safety Code sections 39660-39662, OEHHA and the ARB staff must advise the Air Resources Board on the identification and control of toxic air contaminants. This advice must be three-fold: 1) to recommend listing of chemicals as toxic air contaminants; 2) to review the current scientific information on exposure levels and health effects; and 3) to describe a range of risk for the proposed contaminant and, if there is no threshold, to clearly present recommended guidelines for

risk management. In the *Proposed Identification of Lead as a Toxic Air Contaminant*, there are flaws in the way the scientific information on lead is presented, and the document fails to give clear guidance for risk management.

The quality of the scientific review in the report is generally excellent, and the exposure assessment and health assessment documents will be very valuable for a wide variety of organizations and individuals. We agree with the fundamental conclusions that lead should be listed as a toxic air contaminant and that there is no apparent threshold for the toxic effects of lead. However we are very concerned about four major issues: 1) Excessive delay in the recommendation to list lead as a toxic air contaminant; 2) Excessive detail and qualification in the material presented, making it difficult to understand the central points of the document; 3) the failure of the document to clearly present guidelines for risk management; 4) Overly narrow scope of adverse health effects considered along with lack of discussion of the economic impact of lead-related health-effects. These concerns are further elaborated below.

Excessive Delay in Listing Lead as a Toxic Air Contaminant

The process to list lead as a toxic air contaminant began in February of 1991 and is still not complete after almost six years. This delay is unconscionable from the point of view of science and public health. Lead is probably the most thoroughly understood environmental toxin we are exposed to today. In addition, it is known to have serious, long-term effects on human health at extremely low exposure levels. It is unfortunate that the Centers for Disease Control and Prevention (CDC), the National Academy of Sciences, and the World Health Organization have all moved more rapidly than the State of California to recognize and act on the threat of lead contamination. The fact that, despite such clear and convincing evidence, lead has not yet been added to the list of toxic air contaminants, makes it appear that something other than science is at work to delay this listing. We are particularly concerned because the current document is also a draft and therefore innumerable future iterations may be envisioned before lead is finally

(and inevitably) listed. Meanwhile the people of California continue to be exposed to airborne lead and continue to suffer from the attendant health effects.

Excessive Detail and Qualifications

The entire document has become excessively long and cumbersome. Calculations are often made using three different models when just one would amply suffice (See eg. Table 5-2, p. 5-22 in Part B: Health Assessment). The US EPA used only the aggregate model for dose-response estimation for lead[2]. This model was peer-reviewed and amply validated. The addition of two other models of dose-response in this report serves only to confuse rather than enlighten. Most of the conclusions in the report are qualified to imply lack of confidence in the conclusions. In fact, scientists understand lead better than almost any other environmental contaminant, and there is excellent scientific agreement on the type, degree, and level of health concern for lead exposure. This scientific consensus is not reflected in this document.

To improve the utility of this document to local air quality control districts and to the general public, the conclusions should be presented clearly with direct guidance for action. In addition, unnecessary qualifiers and superfluous analyses serve only to confuse and should not be included. The more unnecessarily complicated this document becomes the more lengthy the process of regulating lead will become, as a guidance document will likely be considered necessary to clarify the conclusions of this report. Such an utterly wasteful process will delay important health protective action.

Lack of a Recommended Exposure Level (REL)

We would like to remind the agency that the current ambient air quality standard for lead in the State of California is 1.5 $\mu g/m^3$. This standard was based on preventing blood lead levels in 95% of children from exceeding 30 µg/dl. This antiquated standard dates back to 1970. Since then, CDC has revised its level of concern for children down to 10 µg/dl. At an air lead level of 1.5 $\mu g/m^3$, according to your own charts and tables, over half of California children would be expected to exceed the CDC guideline from air exposure

alone (extrapolation from Table 5-2, p. 5-22, Part B: Health Assessment, although, interestingly, the tables and charts in this report do not go as high as the current air lead standard). Clearly it is not appropriate to keep the current standard in place. Yet it is OEHHA's job to recommend an appropriate health-based level of concern, or Recommended Exposure Level (REL). The lack of such a recommendation tacitly leaves the current antiquated, un-protective standard unchallenged.

A prior draft of this lead document contained an REL of 0.75 $\mu g/m^3$. The Science Review Panel (SRP) in 1994 quite appropriately criticized that level as being insufficiently health-protective because, according to your tables, between 26% and 42% of children would be expected to exceed CDC blood lead level guidelines from air exposure alone. We understood that OEHHA was, as directed by the SRP, in the process of revising their recommendations downward. Unaccountably, however, the revised REL has been omitted from the current draft. There is no justification on scientific, health, or regulatory grounds for not proposing an REL. NRDC believes that because over 10% of children in California already have blood lead levels over 10 µg/dl, all further exposures to lead should be minimized. It is clearly OEHHA's responsibility to issue a recommendation, and we will await a prompt revision of the document to this effect.

What Level of Protection is OEHHA Striving For?:

While agreeing with OEHHA's assessment that lead is a non-threshold toxin, we would like some more information about the degree of protection that OEHHA envisions. It is clearly OEHHA's job not only to recommend that lead be listed as a toxic air contaminant, but also to provide clear guidance to risk managers. In specifying a range of risk it is necessary for OEHHA to clarify whether the goal is to protect 95% of the citizens of California, 99% of the state population, or some other fraction of the general public, and of children in particular. We are simply requesting that OEHHA make it's goals clear on this issue.

Other Health Effects of Lead Were Overlooked:

The effects of lead on the neurobehavioral development of children are clearly among the most important health effects of this toxin. Unfortunately the OEHHA report focuses only on decrements in IQ. Lead is also known to affect other neurological parameters such as behavior, particularly resulting in shortened attention span[3], and increased delinquency.[4] These effects do not appear to be secondary to the effects on IQ. In light of the increasing problems of delinquency, behavioral issues, attention deficit disorder, and other similar problems among youth today, it seems strange that the data on the association of such endpoints with lead exposure should be overlooked. In addition, the economic costs of decrements in IQ and of behavioral problems are substantial. It would therefore be very useful for OEHHA to include an economic analysis of the health impacts of lead exposure on children in California.

The effect of lead on blood pressure is also an important endpoint for consideration. Unfortunately, although hypertension is known to result in a variety of adverse outcomes, only two outcomes were considered in this report: the projected increase in fatal and non-fatal myocardial infarction and all-cause mortality. In fact, hypertension is also known to predispose to cerebrovascular accident (stroke), peripheral vascular disease, cardiac failure, and hypertensive nephropathy ending in kidney failure[5]. In addition, many of these other hypertension-related endpoints result in extensive morbidity, so a focus only on mortality is inappropriate. The projected human cost in morbidity and mortality, as well as the projected economic costs of all of this lead-related disease should be clearly presented as part of the health assessment piece of this document.

Finally, risk managers need to know that there are a number of other important health effects of lead. Many of these other effects cannot be easily quantified but may nonetheless be important in certain situations. These effects include low birth weight, spontaneous abortions[6], male and female infertility[7], birth defects[8 9], endocrine effects[10], shortened stature[11], decreased hearing acuity[12], postural instability[13], suppression of hemoglobin biosynthesis[14], and peripheral neuropathy. While it may be premature to

perform formal risk assessments for these effects, they should certainly be mentioned in this document.

High Risk Populations Were Insufficiently Considered:

Urban children are known to be at particularly high risk from the adverse effects of lead. This is from the combined exposures to leaded paint sources, and lead in urban air. The California Department of Health Services studied children in Los Angeles, Oakland, and Sacramento in 1987-89[15]. This survey estimates that 1.2 million homes in California may have elevated levels of lead in interior paint and over 3 million homes may have leaded exterior paint[16]. When children were tested in these three cities, many were found to have elevated blood lead levels. For example, in Los Angeles almost 32% of children sampled had blood lead levels at or above the level of concern of 10μg/dl and in Oakland almost half of the children tested had blood lead levels at or above 10 μg/dl[17]. These children with already elevated blood lead levels are at exceptionally high risk of adverse neurodevelopmental effects from any further exposure to lead.

It is unclear from this report how OEHHA proposes to protect these high-risk children from further lead exposure from air sources. While average air lead levels have been decreasing in California, certain urban areas, particularly in the Los Angeles area, do have persistent air lead contamination problems. These problem areas are essentially ignored in this report. Thus the public may be left with the erroneous impression that the air lead problem has already been "solved" in California. For children in inner city areas where high air lead levels add to their already-elevated body burden, the air lead problem is far from solved.

Conclusion:

NRDC strongly encourages OEHHA to finalize the report on lead within the near future. It strains credibility that it should require nearly six years to amass the scientific data to justify classifying lead as a toxic air contaminant. We request that OEHHA provide clear guidance to risk managers by providing a benchmark level for lead, above which local

residents would be notified and emissions control strategies would be developed. We also request that OEHHA clarify what percentage of the population would be protected by such an REL. Such guidance should be a part of this document rather than in an additional guidance document, therefore confusing, unnecessary analyses and unnecessary qualification of findings should be eliminated in order to make this document more useful to local communities. Finally we urge that OEHHA consider other low-dose health effects of lead, including behavioral and attention effects as well as other end-organ effects from hypertension, and their economic costs. Although overall air lead levels have decreased greatly in California, there remain certain urban areas where children are at significant risk from their exposure to lead in the air. It is important to keep the health of these children in mind and move ahead quickly to finally, and strictly, regulate lead as a toxic air contaminant in California.

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II.

Office of Environmental Health Hazard Assessment Staff Responses to Comments

Office Of Environmental Health Hazard Assessment Staff Responses to Summarized Comments Submitted by Gradient Corporation on behalf of ARCO on October 23, 1996
12/20/96

I. Comments of ARCO submitted by Gradient Corporation

I. Comments of Gradient Corporation :

Note: Gradient corporation summarized their comments as rebuttals to OEHHA's prior responses to comments on the September 1996 document. Rather than repeating our responses, we will focus on providing clarifications where needed.

Comment 1: *The aggregate model overstates the impact of decreasing atmospheric lead concentrations on blood lead levels.* Specifically, Cal EPA should not assume that changes in atmospheric lead levels will result in changes in soil lead levels and dust lead levels as well since lead in soil and dust persists for a long period of time. The disaggregate approach isolates the impact of atmospheric lead level changes on blood lead levels and yields a slope factor that is smaller than Cal EPA's slope by a factor of approximately 2.5 (i.e. the correct value should be approximately 1.7 μg/dL per μg/m³ lead in air, rather than Cal EPA's recommended value of 4.2 μg/dL per μg/m³ lead in air. (rebuttal to comment 1). The Agency correctly notes that if (hypothetically), redistribution of lead (in soil and dust) occur very quickly, the Agency's model and equations would not overstate risks. However, Cal EPA does not provide a compelling argument as to why this hypothetical phenomenon may occur (other than hinting at mechanisms that may be relevant in an urban environment). Nor does Cal EPA provide any evidence that such quick redistributions have ever been documented anywhere. Our original comments note three sites at which US EPA has insisted on cleanup of soil long after an atmospheric lead source has been eliminated. It therefore seems that the norm is for soil lead levels to persist for extensive periods of time (or, at most, to decline relatively slowly) and hence Cal EPA's hypothetical scenario of a quick redistribution has no basis in reality . In response to the second argument (the inhalation only slope is greater than 1.7 μg/dL per μg/m³), Cal EPA does not cite the study or studies to which the Agency is referring. We therefore have no way to judge the applicability of these studies, their validity, or whether they are even representative of the literature. Moreover, Cal EPA does not even offer an alternative value for the slope factor of 1.7 μg/dL per μg/m³ lead in air that we have suggested. (Comment 1)

Response: The primary purpose of the Toxic Air Contaminant document is to evaluate the effects of increases in air lead. It is not aimed at evaluating the impacts of decreasing levels. Regardless, the document specifically states that the full impact of decreases in air lead may occur over time and not necessarily immediately. At these sites, the exposures to lead may continue to occur over a long interval due to re-entrainment. This would raise, not lower, one's concerns for air emissions of lead because of difficulty in removing lead from soil. Furthermore, it is clear that despite the usually slow turnover of lead in soil and dust, reductions in air lead can have profound effects on blood lead level. For example, based on the evidence relating to reductions of lead in gasoline, reductions in air lead have large and fairly immediate impacts on blood lead.

Still, this does not mean that increases in air lead level are only mediated by inhalation of lead. Rather, exposures are also indirectly occurring from soil and dust lead that originally came from air.

U.S. EPA agrees with this approach which partly prompted development of its multi-pathway IEUBK model. The IEUBK model, when all pathways are considered, along with the time element for accumulation of lead in the environment, generates a blood lead/air lead slope consistent with the aggregate model. Based on the evidence that lead emitted into the air is a multi-pathway problem, the effects on blood lead through soil, household dust and direct inhalation should be evaluated. Based on the empirical data, the blood lead/air lead slope of 4.2 μg/dL per μg/m³ is the best estimate for children.

Finally, the commenters requested citations of inhalation studies in which a slope greater than 1.7 μg/dL per μg/m³ was observed. U.S. EPA and OEHHA reviewed essentially the same set of studies since all chamber studies were conducted before 1985. These studies measured blood lead periodically in the same individuals exposed (usually) to very high levels of lead permitting estimates of both individual and cross-sectional slopes. Several of the individual slopes were greater than 1.7 μg/dL per μg/m³ and in all cases, the greater the air lead level, the smaller the air lead/blood lead slope. On this basis, EPA excluded those individuals exposed to extremely high levels and incorporated slopes from the only real-world non-occupational study of adults. EPA's *pooled* estimate was 1.9 μg/dL per μg/m³ indicating that several individual slopes were greater than 1.7 μg/dL. These studies are cited and critiqued in Section 4 of the document.

Comment #2: *The IEUBK model overstates blood lead levels.* There is limited documentation to support Cal EPA's hypothesis that the IEUBK model better predicts blood lead levels for typical urban populations than it does for populations in our examples (populations near mines). There has been no extensive validation of IEUBK in a typical urban setting. Moreover, even if the model were to correctly predict blood lead levels in communities where lead levels in various media have reached equilibrium, it does not follow that the model correctly predicts the impact on blood lead levels associated with decreases in atmospheric lead levels when equilibrium has not been reached. In fact the IEUBK model does overstate the impact of decreases in atmospheric lead levels on blood lead levels since the aggregate model is built into IEUBK software. Finally, the differences between observed and IEUBK-predicted blood lead levels are often too large to be explained by "measurement error" affecting empirical values. For example, in Leadville, CO, the model predicted 41% of the children would have blood lead levels exceeding 10 μg/dL; the observed fraction of children with blood lead levels exceeding this threshold was only 8.2% -- approximately 1/5 the predicted value. In Butte, MT, the predicted fraction of children with blood lead levels above 10 μg/dL (16.8%) was also greatly exceeded the observed fraction (5.1%). Thus, Cal EPA's modeling has overstated the impact of decreases in atmospheric lead levels on blood lead levels. (Comments 2 and 3).

Response: As we have stated in the document, we do not use the IEUBK model to predict blood lead concentrations. The IEUBK model is used for two other purposes. First, to evaluate the sensitivity of the aggregate model in estimating the blood lead/air lead slope. The models provide generally similar slope estimates. The second use of the IEUBK model is to evaluate the contribution to blood lead from air and other environmental pathways.

Furthermore, the primary purpose of the Toxic Air Contaminant document is to evaluate the effects of increases in air lead. It is not aimed at evaluating the impacts of decreasing levels. Also, the aggregate model is not built in to the IEUBK model. The models are entirely independent and are predicated on entirely different assumptions and empirical evidence, although Hogan (1995) showed very good prediction of the IEUBK model in terms of geometric mean and standard deviation, in three areas.

Comment #3: Cal EPA uses overly conservative values to characterize blood lead levels in California. The Agency's geometric mean estimate reflects outdated NHANES III data collected between 1988 and 1991, a period prior to the complete phase-out of leaded automobile gasoline and the complete phase-out of lead solder used in the manufacture of food cans. More recent studies (including a study of children using an HMO in California) indicate blood lead levels in California are lower than levels reported in NHANES III. By using too large a geometric mean, Cal EPA overstates the impact of lowering lead exposure on the fraction of children with blood lead levels exceeding the U.S. Center for Disease Control's) CDC's level of concern. Cal EPA also overstates the geometric standard deviation (GSD) in communities living near point sources (e.g. smelters), thereby overstating the absolute fraction of children with blood lead levels exceeding the CDC's threshold of concern. We believe that the Agency should further emphasize that since communities near point sources probably have lower GSD's (as stated in OEHHA's response) the absolute fraction exceeding 10 is smaller than that predicted by OEHHA. (This comment is subdivided below and is therefore addressed in response to comments 4 and 5.)

Comment #4: We believe that Cal EPA should add to its conclusions that it is likely that blood lead levels are lower now than suggested by the NHANES III results. By using outdated NHANES III data, OEHHA overestimates the geometric mean blood lead level. Extrapolating from a national sample to a hypothetical community living near a point source is questionable: For example, communities near point sources might not have lognormally distributed blood lead levels. To the extent that lognormality is unfounded, the risk estimates presented by Cal EPA for such communities are invalid. (Comment 4)

Response: Since generally representative data for children in California do not exist, uncertainty remains about the actual geometric mean and standard deviation of blood lead in the State or for any potentially impacted community. Therefore, to characterize the distribution of blood lead levels for California children, OEHHA used data from NHANES III. Other studies conducted among subpopulations in California have indicated both higher and lower mean blood lead levels. In Section 1, we have indicated that blood lead levels have dropped dramatically over the last decade due to the reduction in lead in gasoline. Based on our review of the available data, the data from NHANES III appears to be reasonably representative of conditions in California. Lower levels found by one study at a later date is not evidence of a continuing trend of declining lead. Also, OEHHA's sensitivity analysis indicates that the increase in children above 10 μg/dL from current (or zero) air levels is relatively robust to assumptions about the geometric mean and standard deviation. Given a large enough sample size, most evidence indicates that blood lead distributions can be well described by a lognormal distribution. Similar evidence is not availabe to substantiate other distributions.

Comment #5. A more recent study illustrates that the geometric mean (GM) and/or geometric standard deviation (GSD) estimated in NHANES III are invalid for California children. A survey conducted between 1992 and 1993 of blood lead levels in Californian children receiving Medicaid benefits reports that 2% of children had blood lead levels above 10 μg/dL. Using OEHHA's GSD of 2.1 and the reported fraction of children with blood lead levels exceeding 10 μg/dL, the GM must be no greater than 2.2 μg/dL. We do not think that any of the caveats OEHHA listed about the data explain this discrepancy. Therefore, Cal EPA must choose between our calculated geometric mean and its assumptions of lognormality and a GSD of 2.1.

When the Medicaid study was mentioned in the last round of comments, Cal EPA objected to its use for estimating distribution of blood lead levels in California children because: 1.) The study did not report a geometric mean (GM) or geometric standard deviation (GSD) (see previous comment) 2.) The population discussed in the comment were 1 to 6 year olds, a group less likely to have high blood lead levels than 1 to 2 year olds, the population that is the subject of Cal EPA's risk assessment. 3.) The editors noted several possible reasons for the low blood lead levels observed in the survey including: (a). the study was conducted in winter months when lead exposure is less severe, (b). the results may have reflected differences in study design.

We believe that the Agency's caveats are not valid. First, as stated in the last round of comments, the fraction of children above 10 μg/dL was not greater among 1 year olds (1.7%) and 2 year olds (2.2%). Second, it is unlikely that summer blood lead concentrations would increase 3-fold which would increase the annual average to 4.4 μg/dL. Prior studies (Baghurst et al., 1985; Billick et al., 1979; Marrero et al., 1983 and Johnson et al., 1996) have not identified seasonal fluctuations this great. Also, we would expect seasonal variation in California to be even less than in other parts of the country since the year-round temperate climate in California ensures that exposure to lead in soil is not interrupted in winter months. In order to discount the data from this study using the seasonal variation argument, Cal EPA must be required to demonstrate that seasonal exposure fluctuations (especially in California) are large enough to make plausible the claim that the NHANES III geometric mean of 4.1 μg/dL is still valid. Finally, Cal EPA does not explain how "differences in study design" would yield a downward bias of any magnitude, and the Agency certainly does not explain the substantial difference between the NHANES III geometric mean of 4.1 μg/dL and the geometric mean inferred from the Medicaid study data of 2.2 μg/dL. (Comments 5 and 6)

Response: Other data exist that provide both higher and lower estimates of the geometric mean of children's blood lead in California. For example, one study of children from an HMO shows only 2% of the children below age 6 have blood lead levels above 10 μg/dL. Another study using HMO data, suggests means similar to NHANES III. Finally, data from high risk census tracts in Los Angeles, Oakland and Sacramento collected by the Department of Health Services (CDHS) in 1987-1989, indicate that from 14 to 67% of the children have levels above 10 μg/dL. Clearly, one needs to be concerned about the representativeness of the data for communities as well as the state. For example, clinic-based samples might have a greater proportion of healthy children (with lower blood lead levels) than population based samples such as NHANES III or the CDHS study of "high risk" communities. This could result in a substantial downward bias in the estimated prevalence of children with blood lead concentrations at or above the CDC's level of concern. Despite the temperate climate, there is evidence of seasonality in the Los Angeles area. A recent study by Williams et al. (1996) cited in Section 5 of the document, indicates that summer and winter blood lead levels differ by a factor of about 1.5, however, an exact quantification is difficult to make from the data given. Although this is not a 3-fold difference, along with differences in study design, it could explain the observed differences between NHANES III data and that of Molina et al. Finally, the prevalence discrepancies might be explained by a continuing trend of declining lead levels, although lower levels found by one study at a later date is not evidence, in itself, of such a trend. OEHHA's sensitivity analysis indicates that the increase in children above 10 μg/dL from current (or zero) air levels is relatively robust to assumptions about the geometric mean and standard deviation.

Comment #6: The original test used by OEHHA to assess lognormality of the NHANES III blood lead distribution, nearly rejected lognormality (p=0.07) That the Agency has found a statistical test that

allegedly supports the hypothesis of lognormality does not eliminate the Agency's original finding. Choosing the second test's results and ignoring the results from the first test without explanation is arbitrary. (Comment 4)

Response: The PROC RANK test which is used in the revised draft, is more robust to outliers than the Kolmogorov-D test. The results of both tests support lognormality and reject the use of a normal distribution.

Comment #7: Using an inflated estimate of the GSD, Cal EPA incorrectly overstates the absolute fraction of individuals in the population with blood lead levels exceeding the level of concern. (Comment 8)

Response: The GSD is taken from NHANES III which is a nationally representative estimate of the distribution of blood lead. Representative estimates for California are not available.

Comment #8: We have quantified the extent to which changes in the geometric mean, along with changes to other unsupported assumptions, affect the predicted risks. Cal EPA should present its own estimates of how changes to the geometric mean affect predicted risks. Qualitative statements in the Agency's response that claim sensitivity analysis show the predicted risks are not sensitive to changes in the geometric mean are not sufficient. (Comment 6)

Response: Several sensitivity analyses are discussed in Section 5 of the document which indicate that the increase in children above 10 μg/dL from current (or zero) air levels is relatively robust to assumptions about the geometric mean and standard deviation Using NHANES III data, OEHHA compared changes in the proportion of children with blood lead levels exceeding the CDC level of concern among all children aged 1 to 2 and African-American children in the same age range. The geometric mean for the latter group was 2 μg/dL greater. The results of the analysis are depicted in Figures 5-1 and 5-2.

For the comparison of sensitive subpopulations, the GSD of the two groups hardly differed. To investigate the robustness of the findings to choice of GSD, OEHHA examined several simulations. One reported in the document compared results using the national and western NHANES III data. For the subgroup of children aged 5 and below, these data suggest a geometric mean of 2.9 and a GSD of 2.3 in the West. Data for the subgroup of 1 to 2 year olds were not provided. To determine these values for 1 and 2 year olds, for this exercise, we assumed that the ratio of the geometric mean and GSD of 1 and 2 year olds to that of those age 5 and below in the Western region was similar to the ratio reported in the national data (Brody et al., 1994). For the national data, the geometric mean and GSD are 1.139 and 1.07 higher, respectively, for 1 and 2 year olds relative to those age 5 and below. Therefore, this predicts that for 1 and 2 year olds in the Western region, the geometric mean and the GSD will be 3.30 (2.9 x 1.139) and 2.46 (2.3 x 1.07), respectively. At air lead concentrations of 0, 0.06 and 0.25 μg/m^3, in the West, the proportion of 1 and 2 year olds than are predicted to be at or above 10 μg/dL are 9.8%, 10.9%, and 14.4%, respectively. These changes in the Western data are similar to those predicted using the National data, though the GSDs are different.

Comment #9: The Western NHANES III data which estimates a geometric mean of 2.9 μg/dL, predicts only 3.5% children above 10 μg/dL. OEHHA's arguments for not using Western NHANES III data are incorrect. In the last round of comments, OEHHA stated that the Western NHANES III data is not representative of California since California is more urban than other states included in

Western NHANES III data. While it is not clear what states are included, the US Bureau of the Census includes Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Alaska and Hawaii in its Western region. This region is more urbanized (86.3% of the population) than any other region (Northeast is the next highest region with 78.9% urbanized population). Furthermore, the fact that the Western region has a lower geometric mean, calls into question the relationship of urbanization to lead level. (Comment 6)

Response: Since generally representative data for children in California do not exist, uncertainty remains about the actual geometric mean and standard deviation of blood lead in the State or for any potentially impacted community. Therefore, to characterize the distribution of blood lead levels for California children, OEHHA used data from NHANES III. Based on our review of the available data, the data from NHANES III appears to be reasonably representative of conditions in California. Even if the same regions are used for both census and NHANES III, it is not clear if the NHANES III sampling is based on preserving the level of urbanization in the entire region which is presumably driven by the coastal states, especially Washington and California. At any rate, it is clear that population lead levels are influenced by urbanization. Brody et al. reported that lead levels were greater in areas with a central city greater than 1 million versus those areas where the central city had fewer than 1 million people. This is an indication of an urban effect nationally. These findings make sense when one considers that many more sources exist in urban populations.

Comment #10: Using an inflated estimate of the GSD, Cal EPA incorrectly overstates the absolute fraction of individuals in the population with blood lead levels exceeding the threshold of concern. Cal EPA states that the baseline "severity" of lead exposure in California is uncertain with the fraction of children with lead levels above 10 μg/dL ranging from 2.5% to 10.9%, based on three models used by the Agency (see Figure 5-3 of the revised draft). Because the baseline level is uncertain, Cal EPA chooses to focus on the relative contribution of atmospheric lead to blood lead levels. We believe that Cal EPA overstates the uncertainty in the baseline "severity" of lead in exposure. Although the three models used by the Agency are uncertain, empirical data have been collected that demonstrate that the fraction of children with blood lead levels exceeding 10 μg/dL is likely to be very small. The Medicaid clinic study and the NHANES III Western Regional results indicate 2% or 3.6% of children respectively, have elevated lead levels. Federal EPA guidance indicates that when the fraction of children with blood lead levels above 10 μg/dL is so small (less than 5%), lead exposure is acceptable. Cal EPA should acknowledge this so that the reader can understand the appropriate level of priority that should be placed on this public health issue. (Comment 8)

Response: At current average ambient concentrations, the absolute number of children above 10 $\mu g/m^3$ is low relative to levels over the past two decades. However, as indicated by Tables 5-2 and 5-3, increases in air lead can have very significant impacts on the number of children above 10 μg/dL. OEHHA staff will be working with the Air Resources Board to develop risk management guidelines. The issue regarding Federal EPA guidance and acceptable levels would be more appropriate to address by risk mangers at that time.

Comment #11: In the previous round of comments, we pointed out that the three models used by Cal EPA to predict the impact of 1 $\mu g/m^3$ changes in atmospheric lead levels on the fraction of individuals with blood lead levels ≥10 μg/dL differed substantially in their predictions. Cal EPA responded that we chose a worst case scenario and that the models yield much more similar predictions at 0.06 $\mu g/m^3$.

We respond that the relative differences are at least as large. Figure 5-3 in the revised Cal EPA draft suggests the Aggregate IEUBK model (sic) predicts an increase in atmospheric lead levels increases [in] the fraction of the population with blood lead levels above 10 μg/dL by approximately 3-4%, whereas the other two models predict such a change in atmospheric lead levels would increase this fraction by 10% to 11%. The relative differences among the model predictions span a factor of as much as approximately 4. These differences can hardly be considered small when the results of the risk assessment depend in a linear manner on the magnitude of these results. That is, the predicted risks may span a factor of 4 depending on which model is selected. This level of uncertainty suggests Cal EPA's risk assessment is far from robust. (Comment 9)

Response: Some differences between the predictions (in the number of children above 10 μg/dL blood lead) from the aggregate model versus the IEUBK models are to be expected. Actually, it is notable that the results from models that are developed from such different sets of assumptions are reasonably close, particularly at the lower air lead levels. Specifically, at 0.5 μg/m3, the models predict that 21%, 27% and 30% of the children will be above 10 μg/dL. At higher ambient levels, the results are more disparate, although the aggregate model and the IEUBK (EH) model yield nearly similar predictions. In addition, the AGG model has a high slope and low intercept in the relationship of soil and dust with air. This determines the difference between those two models. The site used in the EH model has validated measurements by CDC. The AGG model represents 40 different sites with differing measurement protocols potentially lowering the quality of the data, and explaining the wide span of model predictions. In many cases in the risk assessment, differences among models may span orders of magnitude. In other cases, the magnitude of difference between model choices cannot even be quantitified. Thus, we find that given the similar predictions of the models, and the identification of areas where the models differ, that the level of uncertainty is far less than other areas of risk assessment.

Summary Comment #4. Cal EPA overstates the association between lead exposure and diastolic hypertension. (Comments 10-14).

Comment #12: Our original comment points out that at least one major study of the association between blood lead levels and blood pressure (Dolenc et al., 1993) found a negative correlation between these two quantities i.e. a protective effect association with blood lead levels). We therefore concluded that the existence of such an association is uncertain at best. Furthermore, while only one study reported a negative association, Cal EPA does not adequately highlight the fact that ten of twenty-five studies cited in the document have failed to find any association between lead and blood pressure and, especially, diastolic blood pressure. [See Table on page 11 of the comments submitted by Gradient Corporation. The table also notes that two files could not be located: Kort, 1987 and Orssaud et al., 1987]. Furthermore, as Cal EPA notes on p 3-21 of the Agency's revised report, *blood lead levels are correlated with age*, a well known predictor of blood pressure. Hence, the lack of negative associations (with one exception) may reflect improper control of the confounding effects of age.

Finally, Cal EPA reviews one literature review (Hertz-Picciotto and Croft, 1993) and two meta-analyses (Schwartz, 1995; Staessen, 1995). The Staessen (1995) failed to find a statistically significant association between blood lead levels and diastolic blood pressure, although Cal EPA contends that proper analysis of the studies reviewed by Staessen would review (sic) a statistically significant relationship. Schwartz reports a statistically significant association between blood lead

levels and diastolic blood pressure. Hertz-Picciotto and Croft come down in the middle, stating that, "the literature is strongly suggestive, but not definitive of an association."

In conclusion, Cal EPA's claim that an association between blood lead levels and blood pressure is incontrovertible is not supported by the literature, or by the reviews and meta-analyses of this literature. (Comment 10)

Response: In our review of the literature on blood lead and blood pressure in adults, we have used studies most representative of the California non-occupationally exposed population and provided quantitative measures of uncertainty around our estimates. In response to the previous set of comments, we have attempted to clearly indicate the current state of the scientific findings.

The table included with Gradient's comments was a list of every study cited in Section 3 along with findings of an association between blood lead levels and diastolic blood pressure in men. The table notes that two studies could not be located. OEHHA has confirmed that the citations used in the reference section of the document are correct. In one case, the article that commenters cite as by "Kort et al." is by "de Kort" et al. and located under "D" in the reference list. The citation for the article by Orssaud et al., similarly is correct. Five of the studies included in the table were in occupational settings. These studies were not used to provide an estimate of the magnitude of the relationship of lead on blood pressure. With removal of these eight studies, one sees that eleven of seventeen studies included in the table, were significant after adjustment. Two of the six nonsignificant studies, had small sample sizes, therefore, lack of study power may explain the nonsignificance. An even higher proportion of studies of lead and systolic blood pressure are significant. This is very persuasive evidence of a small but significant association of lead and blood pressure.

All of the studies adjusted for BMI, age, and sex. The studies adjusted for age, either by using age as a covariate in the model, or stratifying by age group. One of the reasons OEHHA focuses its review on men aged 40-59, is to reduce the confounding and potentially modifying effects of age and sex. This is also why we think the magnitude of the effects indicated by the Staessen meta-analysis needed to be adjusted. Given the number of confounders some studies have included for adjustment, it is highly unlikely that the results are consistently explained by residual confounding. In their literature review, Hertz-Picciotto and Croft (1993) state that "while not ruled out, the probability that confounding explains most of the findings appears to be low". The authors also indicate that several covariates included in the regression analyses that relates blood pressure to blood lead may have reduced the apparent lead effect.

In response to interpretation of the meta-analyses and literature reviews, although Hertz-Picciotto et al. do not conclude that there is a definitive association between blood lead and blood pressure, they also state that no other explanation for the association, other than the causal one, was felt to exist. Finally, they concluded that, based on the available evidence, "public health measures to reduce exposures may be deemed justified." There are several decision points when one is doing a meta-analysis. Consequently, two meta-analyses using the same set of studies might come up with quite different conclusions. Staessen's meta-analysis included populations not relevant to OEHHA's risk assessment. When these populations were removed, the results were not contradictory to OEHHA's.

Comment #13: Cal EPA's conclusion as to the impact of blood lead increases on blood pressure (1.9 mm Hg per doubling of blood lead level) is based on a very selective review of the available data.

[NOTE: OEHHA summarized the points made by Gradient into a table].
Alternative findings to high-end estimate reported by OEHHA using studies from the report.

Study	Section/Page	mm Hg increase	Lead Increase
NHANES II	3-20	1	doubling
Pocock et al. (1988)	3-20,21	1.2-1.4	doubling
Sharp et al. (1988)	3-22	1.26-1.69	doubling*
Staessen et al. (1995) as modified by Cal EPA	3-24,25	1.2	doubling

We take issue with Cal EPA's conclusion that there is reasonable agreement about the size of the effect. Moreover, we do not understand how the Agency's estimate ... can be considered a central estimate. In the very same paragraph in which Cal EPA concludes there is reasonable agreement between studies on this issue, the Agency states that " the U.S. EPA's external Science Advisory Board, and the National Research Council (NRC 1993) have concluded that a doubling of blood lead is associated with a 1 to 2 mm Hg increase in *systolic* blood pressure [emphasis added by the commenters]." Since the reported impact of lead on diastolic blood pressure is often smaller than the impact of lead on systolic blood pressure, these statements cast a great deal of doubt on the Agency's use of 1.9 mm Hg as the central estimate for the magnitude of this effect.

We conclude that Cal EPA should use a range of values to represent the central estimate of the association between blood lead levels and diastolic blood pressure. The Agency should then consider stochastic uncertainty (see p. 6-3 of Cal EPA's revised report) in addition to the uncertainty in the central estimate introduced by the range of results reported in the literature. The set of plausible central estimates for this relationship should probably range from approximately 0.6 mm Hg per doubling of blood lead levels (the magnitude reported by Staessen, 1995) to approximately 1.9 mm Hg (the result now used by Cal EPA). Use of this range will place more weight on relatively small predictions of risk for CHD and CHD mortality in the population than Cal EPA currently reports. (Comment 11)

Response: In the document, we have clearly stated the conditions from which we made our estimate of the effect of a doubling of blood lead on blood pressure. First, we have reviewed all available studies and explicitly responded to the request in the last round of comments to provide an objective review. Second, we focus on ages 40 to 59 years. Third, we have included all studies in that range and cite meta-analyses of Schwartz and Staessen which are generally consistent when this subpopulation is considered. Fourth, we then rely on NHANES II results, since these data are most closely related to our population (Americans, non-occupationally exposed). In this population, OEHHA's estimate of the magnitude of the association is a reasonable central estimate. Higher estimates, cited in the document, have been reported. The estimated effect per unit would be lower if all age groups were considered in our cardiovascular risk assessment. However, the calculations were calculated for only for the 40 to 59 age group so the related risk estimates were used.

Comment #14: Cal EPA states that it uses a logistic relationship to model the probability of hypertension (diastolic blood pressure exceeding 90 mm Hg) as a function of blood lead levels, and a semi-log function to model diastolic blood pressure as a function of blood lead levels.

The Agency also argues that a supralinear relationship is not unreasonable, and has been observed for other lead-related biological phenomenon, including the relationship between lead intake (e.g., from food, water, or air) and blood lead levels.

Finally, Cal EPA states that researchers have found that a semi-log model provides the best fit to the data. For example, Cal EPA quotes Schwartz (1988) as stating that "the natural log of blood

lead was more normally distributed, more significant, and gave a higher R^2 than untransformed blood lead....All of the results reported here are for the natural log of blood lead, our regressions for untransformed lead gave very similar results" (p 370 of the Response to Comments).

We respond to each of these arguments in turn. First, use of the logistic function to model the probability of hypertension as a function of blood lead levels is irrelevant since the risk assessment calculates the risk of probability of CHD as a function of diastolic blood pressure, not as a function of whether an individual is hypertensive.

Second, each of the examples of supralinear relationships provided by OEHHA are not dose-responses but instead exposure-dose relationships. In the case of an exposure-dose relationship, a supralinear function would be expected since at higher exposure levels, active transport mechanisms become saturated....[T]his saturation principal supports the hypothesis that dose-response relationships tend to be sub-linear. At low dose levels (i.e. low blood lead levels) mechanisms to protect the body's organs from toxicity can safely handle the challenge.... Hence, the incremental toxicity (response) associated with each incremental increase in blood lead levels (dose) will be smaller at low blood lead levels than at high blood lead levels. This sub-linear dose response is at odds with the semi-log relationship advanced by Cal EPA since the semi-log [is supralinear].

Third, the rationale that Cal EPA quotes from Schwartz (1988) is not compelling. The fact that the log-transformed blood lead data are more normally distributed than the untransformed data means that ordinary least squares techniques are not valid if the data are left untransformed. However, this fact does not mean that the shape of the dose-response relationship should be dictated regressing blood pressure against the log of the blood lead levels (i.e., by the semi-log regression). Likewise, the fact that the semi-log regression yielded a higher R2 and was more significant than the untransformed regression does not mean that it better describes the shape of the relationship. With skewed data (in this case, the blood lead data), one would expect failure to transform the data (e.g., by taking the logs) would yield a model that does not fit as well because transformation of the data eliminates "outliers" that degrade the model fit. Finally, it is not clear what Schwartz meant when he stated that "regressions for untransformed lead gave very similar results" as the transformed regressions. We are interested in the strength of the association between blood lead levels and blood pressure at the particularly low blood lead levels that are relevant to contemporary populations. The extracted quote from Schwartz does not show that a linear (i.e., untransformed) dose-response relationship yields the same predictions in this low range of blood lead levels as the semi-log relationship used by Cal EPA. (Comment 12)

Response Based on the original article (Schwartz, 1988), the probability of hypertension, as indicated in Section 5, is a logistic function of blood lead. We have modeled this outcome using the same functional form. Likewise, in examining the association between blood pressure and more serious outcomes, we have directly applied the results of several long-term cohort studies. The concentrations of blood lead that we are currently considering was within the range of the original studies conducted on this issue. Therefore, use of these studies and the reported functional forms, is appropriate. A similar conclusion was reached by U.S. EPA's Science Advisory Board which reviewed the use of these models as part of the Regulatory Impact Analysis related to the reduction of lead in gasoline. Regarding the quote from Schwartz (1988), we assume that "very similar results" means that the strength of the association and the magnitude of the association were similar. Thus, although a non-linear model was determined to provide the best fit of the data, this means that the non-linearities were not that great. Regardless, we have used the most appropriate results from the published literature and are not able to determine other fits of the data.

Comment 15: While it is true that no threshold has been identified for the relationship between blood lead levels and blood pressure, it is also true that the vast majority of studies have focused on populations with substantially higher blood lead levels than the levels typical of contemporary Californian adults. It is also difficult to see how Harlan demonstrated effects down to 4 μg/dL given that he was using the same data set (NHANES II) for which Pirkle observed effects down to 7 μg/dL. Even so, since the geometric mean of PbB in California adults is 3 μg/dL, then about half of the blood levels are below this level. The lack of evidence on the shape or existence of the dose-response relationship below the geometric mean makes the risk assessment results presented by Cal EPA even more uncertain. (Comment 13)

Response: The studies of Harlan et al. and Pirkle et al, while both using data from NHANES II, studied different age groups. In the case of Harlan et al., the relationship of lead and blood pressure in males aged 20 to 74 years was studied. In this subgroup, blood lead levels were as low as 4 μg/dL. This was inadvertently omitted from the document but was mentioned in response to the last round of comments. Therefore, the findings could be extended down to the lowest blood concentrations for that group. In the study of men aged 40-59 by Pirkle et al., the lowest blood concentrations were 7 μg/dL. Although blood lead levels have dropped dramatically since NHANES II was conducted, the level of extrapolation is small relative to other risk assessments of toxic air contaminants and reasonable, given the absence of evidence of a threshold for this effect. Specifically, the NHANESIII data indicate that the current means for adults in this age range are between 3 and 4 μg/dL. Therefore, they are within the range where effects from blood lead have been found. Since the original authors examined several alternative functional forms, we can only assume that the "best" model was closer based on the best fit of the data. There is no reason, at this point, to assume another shape of the dose-response function.

Comment #16: Cal EPA has not identified any data directly documenting an association between blood lead levels and adverse cardiac events. According to OEHHA, the reason why no data have directly linked elevated blood lead levels to adverse cardiac events is that the relative risk of an adverse event is small and to date, no study has had sufficient power to detect such an association. Even so, we believe that the lack of such evidence leaves the existence and magnitude of the alleged association highly uncertain. Moreover, we believe that Cal EPA has not fully characterized this uncertainty in the Agency's revised report. The only uncertainty reflected in the Agency's risk assessment is the stochastic uncertainty in the one study used by Cal EPA to quantify the strength of the association between elevated blood lead levels and blood pressure, and the stochastic uncertainty in the association between elevated blood pressure and adverse cardiac events. We believe in particular that Cal EPA has neglected many sources of uncertainty underlying our understanding of the first of these two causal links. A full characterization of uncertainty should address the following additional issues:

1. Whether there exists a relationship between blood lead levels and diastolic blood pressure (see Comment 10).
2. The magnitude of the central estimate of the relationship between an incremental increase in blood lead levels and the associated change in diastolic blood pressure (see Comment 11)
3. The shape of the dose-response relationship (see Comment 12) and whether there may be a threshold at levels relevant to the current population of adults living in California (see Comment 13).

It is our opinion that taking all these factors into account would lower the bottom end of the range of plausible adverse cardiac mortalities to per year to zero, and would place much less probabilistic weight at the current upper end of this range. Without addressing these issues, Cal EPA has failed to reflect the current state of knowledge in its risk assessment and has therefore not produced a valid characterization of the range of possible risks associated with slightly elevated atmospheric lead levels. (Comment 14)

Response: OEHHA has fully characterized the uncertainty in the models by applying the @risk software program. By doing so, we have characterized the central estimate, the confidence intervals, and the joint uncertainties in moving from changes in blood lead through changes in blood pressure, to changes in more serious cardiovascular health outcomes. Based on this analysis, there is no evidence that zero is included in the confidence interval. No judgments are made in the document as to whether the risks are substantial or not. The report objectively determines the health implications of the available scientific evidence.

Office Of Environmental Health Hazard Assessment Staff Responses to Summarized October 22 1996 Comments Submitted by Natural Resources Defense Council

II. Comments of NRDC

Comment #1: There has been excessive delay in listing lead as a Toxic Air Contaminant. This delay is unconscionable from the point of view of science and public health. Lead is probably the most thoroughly understood environmental toxin we are exposed to today. It is unfortunate that the Centers for Disease Control and Prevention (CDC), the National Academy of Sciences, and the World Health Organization have all moved more rapidly than the State of California to recognize and act on the threat of lead contamination. The fact that, despite such clear and convincing evidence, lead has not yet been added to the list of toxic air contaminants, makes it appear that something other than science is at work to delay this listing.

Response: OEHHA acknowledges that the process involved in listing lead as a Toxic Air Contaminant has been a lengthy one. As a result of multiple revisions, the document now reflects state of the art knowledge on specific aspects of lead toxicity and incorporates many of the concerns expressed in public comments.

Comment #2: There are excessive details and qualifications in the report. For example, OEHHA used three different models when one would amply suffice. The US EPA used only the aggregate model for dose-response estimation for lead. This model was peer reviewed and amply validated. The addition of two other models of dose-response in this report serves only to confuse rather than enlighten. Most of the conclusions in the report are qualified to imply lack of confidence in the conclusions. In fact, scientists understand lead better than almost any other environmental contaminant, and there is excellent scientific agreement on the type, degree, and level of health concern for lead exposure. This scientific consensus is not reflected in this document. To improve the utility of this document to local air quality control districts and to the general public, the conclusions should be presented clearly with direct guidance for action.

Response: The SRP Draft Version of the lead document reflects the complexity of the issues surrounding lead-induced health effects. As a result of multiple workshops and many comments received, OEHHA has included sensitivity analyses in the lead document. This has contributed to the level of detail in this version of the report. The Executive Summary provides a simpler version of the longer document and is easier to read. Specific guidance to the district and to the general public have been recommended to be developed by the Air Resources Board, with assistance from OEHHA staff.

Comment #3: The document does not include a Recommended Exposure Level (REL). The lack of such a recommendation tacitly leaves the current antiquated, un-protective (sic) standard unchallenged. A prior draft of this lead document, which contained an REL of 0.75 μg/m3 was appropriately criticized as being insufficiently health protective. We understood that OEHHA was, as directed by the SRP, in the process of revising their recommendations downward. It is clearly OEHHA's responsibility to issue a recommendation and we will await a prompt revision of the document to this effect. Also, in specifying a range of risk it is necessary for OEHHA to clarify whether the goal is to

protect 95% of the citizens of California, 99% of the state, or some other fraction of the general public, and of children in particular.

Response: The presence of a Reference Exposure Level calculation in the previous report assumed the presence of a threshold for lead neurotoxicity. Under such an assumption, standard methodologies were used to derive the point estimate. The current document incorporates the distribution of blood lead levels in the population and reflects an estimation of neurodevelopmental risks to children without a threshold assumption. We provide alternative methodologies for assessing the impacts of air lead on blood lead concerning the 10 μg/dL level of concern. Standard methodology for developing a single point estimate from the current analysis is not available. Level of protection is an issue that can be discussed in the risk management guidance to be developed by the Air Resources Board.

Comment #4: Other health effects of lead were overlooked. For example, lead is known to affect other neurological parameters such as shortened attention span (Tuthill, 1996) *a hair lead study,* and increased delinquency (Needleman et al., 1996). In addition, the economic costs of decrements in IQ and of behavioral problems are substantial. It would therefore be very useful for OEHHA to include an economic analysis of the health impacts of lead exposure on children in California. Because increased lead is associated with increases in blood pressure, in addition to the cardiovascular outcomes mentioned in the document, it could also predispose individuals to stroke, peripheral vascular disease, cardiac failure and hypertensive nephropathy ending in kidney failure (Kannel, 1996). The projected human cost in morbidity and mortality, as well as the projected economic costs of all of this lead-related disease would be clearly presented as part of the health assessment piece of the document. Finally, risk managers need to know that there are a number of other important health effects of lead. These include spontaneous abortions (Lindholm et al., 1991), birth defects (Thomas et al., 1993; Needleman et al., 1984), endocrine effects (Uzych, 1985), shortened stature (Kim et al., 1984), decreased hearing acuity (Schwartz and Otto, 1987), postural growth (Bhattacharya et al., 1988), suppression of hemoglobin biosynthesis (Hu et al., 1994), and peripheral neuropathy. While it may be premature to present formal risk assessments for these effects, they should certainly be mentioned in the document.

Response: OEHHA acknowledges that lead exerts multiple health effects, including those mentioned in the NRDC comment. Some of these health effects are briefly mentioned in Section 2 of the document. The effects on postural sway and decreased hearing acuity have been added to Section 2. This section is intended to be an overview of potential health effects of lead not investigated in detail in the document. The overall intent of Part B of the lead TAC document was to review the major findings from well-designed studies that address the health effects of lead that are of greatest known public health significance. These effects are: neurodevelopmental effects in children, effects on blood pressure and related cardiovascular events in adults and cancer. Because the health effects mentioned in the comment are not considered in the quantitative analyses, the true public health impacts of lead may be underestimated.

Comment #5: High risk populations were insufficiently considered. In a study conducted by the California Department of Health Services from 1987 to 1989 (Sutton et al., 1995), many children in Los Angeles, Oakland and Sacramento were found to have elevated blood lead levels. For example, almost 32% of children sampled in Los Angeles and almost half of the children tested in Oakland had blood lead levels at or above the 10 μg/dL level of concern. It is unclear from this report how OEHHA proposes to protect these high risk children from further lead exposure from air sources. While

average air lead levels have been decreasing in California, certain urban areas, particularly in the Los Angeles area, do have persistent air lead contamination problems. These problems are essentially ignored in this report. Thus the public may be left with the erroneous impression that the air lead problem has already been "solved" in California.

Response: The CDHS study mentioned in the comment is discussed in the document in Section 5. While an extraordinary number of children had elevated lead levels in this study, there are two caveats. First, as mentioned in the comment, the communities studied in each of these areas were defined as high risk because they contained a high proportion of housing constructed before 1950, children below the age of 6 and families beneath the poverty line. Second, the study was conducted from 1987 to 1989. Blood lead levels have dropped since that time due to the ban of leaded fuel. Following the identification phase, OEHHA has recommended that the Air Resources Board develop further guidance to manage the risks from environmental lead exposure.

Office Of Environmental Health Hazard Assessment Staff Responses to Summarized Comments Submitted by Lead Industry Associates, Battery Council International and GNB Technologies

III. Comments of Lead Industry Associates, Battery Council International and GNB Technologies

Comment #1: The report fails to carry forward into the Executive Summary key acknowledgments that appear in other sections. There are four areas where these problems remain: (1) the minor role that air lead plays; (2) the question of persistence of effects of lead exposure on pre-natal neurodevelopmental; (3) the insignificance of lead's impact on cardiovascular disease; and (4) the overestimation resulting from the use of an aggregate slope. (pages 1, 2-5)

Response: OEHHA has attempted to ensure that the Executive Summary serves as an accurate reflection of the conclusions and uncertainties expressed in Part B of the Identification of Inorganic Lead as a Toxic Air Contaminant. However, in response to these comments, we have produced several amendments in the Executive Summary. Briefly, our specific responses to the four questions are listed in order.

1. We will amend the executive summary on page 3 to read (the underlined section represents the addition), "However, new information has been reported on the health effects of lead since the adoption of the ambient air quality standards approximately 20 years ago. Therefore, although at current average ambient levels, air lead is a minor contributor to children's exposures, ambient and near-source exposures may still present a public health concern.

2. Section 3 includes a discussion of persistence of effect which concludes that "studies of effects at later ages appear stronger and more consistent than effects from pre-natal exposures". This sentence could be added to the executive summary. However, it is not correct to say that pre-natal exposures are not persistent. Rather, diminishing of a reported effect may be due to loss of study power, changing intelligence tests, lack of reliability of IQ tests at younger ages, declining study power and imperfect measures of lead exposure.

The executive summary now reads (the underlined section represents the addition), "At low blood lead concentrations, several carefully conducted prospective human epidemiological studies have shown an association between general measures of intelligence and both pre- or post-natal blood lead concentrations. Studies of effects at later ages appear stronger and more consistent than effects from pre-natal exposures. Based on these studies, a blood lead level of 10 micrograms per deciliter has been identified as the level of concern for children.

3. The commenters point out that although we state that the effects of lead on cardiovascular disease are small in the body of the report, in the executive summary, we refer to effects that do not sound small at all. What the document actually states is that the association between blood lead and blood pressure is small. But as stated by Hertz-Picciotto and Croft, among others in their 1993 review of blood lead/blood pressure studies, "a small influence of blood lead on blood pressure might have rather large consequences for debilitating and fatal cardiovascular disease" This is reflected in the estimates of impact provided both in the executive summary and in Section 6 of the document. We have

responded to the comment about uncertainty in the estimates by adding the following to the executive summary (page 10): These effects are based on epidemiologic studies from which it is generally difficult to prove causality. Therefore, controversy remains about the precise magnitude of the effect of blood lead on cardiovascular disease.

4. The scenario that the commenters refer to as overestimates from use of the aggregate slope model reflects the impact of short-term decreases in air lead concentrations. The primary focus of this document, however, is to evaluate the effect of increases in air lead. It is not aimed at evaluating the impacts of decreasing levels or of remediation, per se so this point was not included in the Summary.

Comment #2: By including health effects at current ambient air lead concentrations, the report implies that ambient air lead levels OEHHA present a public health concern. Elsewhere, the report makes clear that ambient exposures are not a public health threat, and accordingly, they should not be used in risk assessment calculations. Furthermore, far more certainty is implied by these calculations than is scientifically justified. For example, in determining the health impact of ambient air lead concentrations on IQ, contributions to uncertainty include uncertainty in the blood lead to air lead relationship, the relationship of blood lead to IQ and even use of the NHANES III distribution to estimate blood lead distribution in California's children. In the face of such uncertainty, it is inconceivable that a 0.1% shift in the percentage of children with IQs below 80 could be attributed with any confidence to a 0.06 $\mu g/m^3$ differential in air lead levels. The same concerns apply to OEHHA's projections of increased cases of hypertension and related cardiovascular effects purportedly arising from exposure to background air lead levels in California. Further, OEHHA should acknowledge clearly and explicitly in the Executive Summary and elsewhere in the report that at worst, air lead may pose health risks only in extreme near source situations. (pages 1-2, 5-6)

Response: Most, if not all of the model parameter values are within the range of the values used in the original health effects studies. At a minimum, the level and kind of extrapolation is far less than that usually employed in toxic risk assessments. Therefore, given the lack of evidence for a threshold for either the neurodevelopmental or cardiovascular effects, OEHHA has calculated the health effects that may be expected at current air lead and blood lead levels. The evidence indicates that although at current ambient air lead concentrations lead is a relatively minor contributor to blood lead levels, health effects due to air exposure are still likely, particularly if air lead concentrations increase.

Comment #3: The document has failed to indicate the disagreement in the scientific community regarding the impact, if any, of lead on these health endpoints. At most, OEHHA deals with these concerns by sprinkling statements in the Executive Summary with a host of qualifiers, using words such as "may", "might," "could," and "potential" rather than recognizing that fundamental questions remain about the validity of these conclusions. Indeed, we have attached as Attachment 1, a major new review of the neurodevelopmental studies by one of the worlds' foremost experts on IQ test design and interpretation, Dr. Alan Kaufman, who concludes that OEHHA's views are not supported by the best available scientific evidence. Even if OEHHA is determined to reject all of the evidence contrary to its position on neurodevelopmental and blood pressure-related health effects, at a minimum the Executive Summary should acknowledge that there is continuing disagreement in the scientific community regarding the impact, if any, of lead on these health endpoints. (page 2)

Response: The Executive Summary is intended to present the conclusions of the Parts A and B. In Section 3, alternative views of the relationship of low level lead exposure to neurodevelopment are discussed and critiqued. The wording in the executive summary in reference to the effects of low level lead exposures on neurodevelopment has been changed to reflect the greater evidence for postnatal effects of lead. It now reads: "At low blood lead concentrations, several carefully conducted prospective human epidemiological studies have shown an association between general measures of intelligence and both pre- or post-natal blood lead concentrations. Studies of effects at later ages appear stronger and more consistent than effects from pre-natal exposures. Based on these studies, a blood lead level of 10 micrograms per deciliter has been identified as the level of concern for children." The criticisms submitted by Dr. Kaufman include a number of studies not considered in OEHHA's risk assessment. In addition, some of his criticisms would suggest it would be harder to find an association between blood lead and intelligence but do not serve to explain away an association that has already been detected. OEHHA has responded to his specific concerns to studies discussed in the health assessment in its responses below. While some scientists have disputed the relationship of low levels of lead and IQ loss, OEHHA's conclusions are supported by the vast majority of researchers, including those represented by U.S. EPA, NRC, ATSDR and CDC.

Similarly, the association of lead and elevated blood pressure has been demonstrated in several high quality studies. OEHHA critiques both significant and nonsignificant studies in Sections 3 and 6 of the TAC. The overall conclusions are presented in the Executive Summary. We have responded to the comment about uncertainty in the estimates of lead's impact on cardiovascular disease (via its effects on blood pressure) by adding the following to the executive summary (page 10): These effects are based on epidemiologic studies from which it is generally difficult to prove causality. Therefore, controversy remains about the precise magnitude of the effect of blood lead on cardiovascular disease.

Neurodevelopment

Comment #4: Controversy continues to surround the entire question of linkages between low blood lead levels and neurodevelopmental effects. As noted above, there seems to be emerging consensus that prenatal health effects dissipate after age two in the absence of independent exposures to the child. The issues of postnatal effects is more complicated, but more and more scientists in the field are questioning the strength of the evidence to support conclusions like those OEHHA seeks to draw. As LIA pointed out in its March 1996 comments, Dr. Sergio Piomelli, one of the original pioneers in the effort to prevent lead poisoning in children, wrote as a member of the CDC Committee on Environmental Health Hazard and Health Effects in September 1995 (see Attachment 1) that the evidence on IQ losses claimed to be associated with low level lead exposure reflects effects that are "trivial and unmeasurable in the individual children" even at levels of 15 to 20 μg/dl. As to the claim that such negligible impact "across a population may have important effects," (Part C, p. 375),Dr. Piomelli takes a view completely contrary to OEHHA's, concluding that "the effect that an average loss of [as much as] 2.6 IQ points may have on the frequencies of children with an IQ ≤ 80 points or ≥125 points, appears insignificant, when calculated statistically." Att. 1, p. 2. (page 6)

Response: OEHHA has clearly stated that the evidence for effects of postnatal lead exposure on intelligence is stronger than that for prenatal effects. The Executive Summary has been revised to reflect this. It now reads: "At low blood lead concentrations, several carefully conducted prospective human epidemiological studies have shown an association between general measures of intelligence and both pre- or post-natal blood lead concentrations. Studies of effects at later ages appear stronger and more consistent than effects from pre-natal exposures." OEHHA has conducted an independent review of the literature (see Section 3) and concurs with U.S. EPA, CDC and the NRC that the IQ

declines observed at lead concentrations of 10 μg/dL and above, have public health significance at the population level. As stated in the document, the loss of a fraction of an IQ point is meaningless at the individual level, but not at the population level when the community burden of lowered IQ is being assessed. Finally, whether or not the predicted change in IQ are important from a public health perspective is a risk management issue. OEHHA has attempted to provide best estimates of the quantitative effects based on the available evidence.

Comment #5: For the studies of lead's effects on intelligence, OEHHA should [also] respond to the major concerns mentioned by Dr. Kaufman in his report (Attachment 2). (pages 7-8)

1. Many of the studies [cited in Dr. Kaufman's report] infer causality from correlational data.

Response: No one study can be used to infer causality. For this reason, OEHHA has reviewed all of the prospective studies of lead and neurodevelopment, critiqued each one and drew conclusions from the weight of evidence. The replication of independent epidemiologic studies resulting in statistically significant and consistent findings is a powerful finding. Any given study may have problems or uncertainties due to questions about measurement, design, and analysis. However, it is extremely unlikely that the three long-term prospective cohort studies that have examined older children will have similar confounders and measurement errors impacting the diverse samples in a similar way. Therefore, the consistency of these findings on blood lead and IQ is compelling and cannot be dismissed. In this regard, OEHHA concurs with NRC, CDC, U.S. EPA and WHO, that there is a likely association between blood lead and measures of intelligence.

Comment #5 continued:

2. Effects could be due to residual confounding.

Response: In order for residual confounding to explain the observed effects, each of the long-term prospective cohort studies would have to have similar distributions of the unadjusted confounders. This is extremely unlikely. In fact, some of the prospective studies potentially overadjusted for confounding by including several interrelated covariates. In this situation, the lead coefficient could be underestimated.

Comment #5 continued:

3. Many researchers conducted multiple comparisons but failed to correct for this. An example is the frequently cited Bellinger Needleman study (sic), upon which OEHHA principally relies.

Response: Because so many comparisons were used within each study, it is important to examine the consistency of findings across studies. The replication of independent epidemiologic studies resulting in statistically significant and consistent findings is a powerful finding. In this regard, OEHHA concurs with NRC, CDC, U.S. EPA and ATSDR, that there is a likely association between blood lead and measures of intelligence. Also, OEHHA does not rely principally on any one study. OEHHA has continued to incorporate results from all of the ongoing prospective studies. These include studies of cohorts in Cincinnati, Port Pirie, Australia and Cleveland as well as Boston. The prospective studies are discussed extensively in Section 3 of the document.

Comment #5 continued:

4. The extent to which verbal IQ rather than performance IQ was affected, suggesting confounding by socio-economic status rather than a true relationship with lead exposure.

Response: Dr. Kaufman indicates that if lead is associated with neurological dysfunction, this would be exhibited through effects on Performance IQ (PIQ), rather than Verbal IQ (VIQ). Since VIQ is associated with socioeconomic factors, Dr. Kaufman believes that its association with lead might be spurious due to poor adjustment for confounding by sociodemographic characteristics. He mentions two reports from the prospective studies in which there were findings for VIQ but not PIQ (Baghurst et al., 1992 ; Bellinger et al., 1992). Dr. Kaufman considers PIQ to be the most likely target of lead's effects because of research showing that excessive alcohol intake and brain trauma are correlated with decrements in PIQ but not VIQ. While the literature may support a relationship of alcoholism and trauma with PIQ and while socioeconomic factors such as income and parental education level correlate with verbal IQ, it is possible that lead effects both performance and verbal development. Indeed, the most consistent findings have been for lead and full scale IQ a scale which is comprised of both VIQ and PIQ.

Comment #5 continued:

5. Incorrect measures of IQ were employed

Response: In his report, Dr. Kaufman mentions several instances in which IQ tests were improperly used. Since many of the studies critiqued by Dr. Kaufman were not used in OEHHA's quantitative risk assessment, OEHHA confines its responses to the prospective studies mentioned by Dr. Kaufman. For example, the study by Bellinger et al. (1992) used the out-of-date WISC-R (scores had been normalized 18 years earlier). These investigators also measured adult intelligence with the Peabody Picture Vocabulary Test (PPVT) which is not a true intelligence test. Furthermore, the test was standardized 30 years earlier on an all-white sample from Tennessee.

Dr. Kaufman acknowledges that WISC-III, the update of WISC-R was not available when the study by Bellinger et al. was conducted. Furthermore, by using the WISC-R, results could be compared both to their earlier studies, and other prospective studies that were ongoing such as the cohorts in Port Pirie and Cincinnati. Furthermore, given the historic increase in IQ, if the increase were uniform across exposure groups as would be expected, it wouldn't have effected the results. Finally, with regards to use of the PPVT, blood lead levels at age 24 was relatively unaffected by adjustment for intellectual climate within a family. In the Port Pirie cohort, the Wechsler Adult Intelligence Scale-Revised (WAIS-R), an "accepted criterion of adult intelligence", was used. With better adjustment of maternal IQ, the relationship of blood lead and FSIQ persisted.

Comment #5 continued:

6. In some cases "extreme" lead groups were compared.

Response: Most of the papers Dr. Kaufman mentioned as having used extreme lead groups, were not included in OEHHA's risk assessment. Therefore, while this is a valid criticism, it doesn't seem to have effected OEHHA's findings. Our risk assessment used studies that considered the enitre data set.

Comment #5 continued:

7. The relationship of lead and IQ was incorrectly assumed by OEHHA to be linear.

Response: In his report, Dr. Kaufman also says that for the 1992 study of Bellinger et al., a threshold effect at about 10-15 μg/dL is a more realistic explanation than is a linear one. Using the data of Bellinger et al. (1991), Schwartz (1993) used nonparametric smoothing (LOWESS) to examine the shape of the relationship between 24 month blood lead concentrations and McCarthy General Cognitive Index scores after adjustment for 13 covariates. The results of this analysis indicated that although slopes appeared to get steeper at concentrations greater than 6 μg/dL, there was no evidence of a threshold down to 1 μg/dL. A hockey stick regression of the same data yielded an estimated threshold of 0.000001 μg/dL. The 1992 publication of Bellinger et al. had very low power to detect significant differences in IQ by blood lead quartile, with the greatest power in the lowest lead group. There were only 18 and 10 children respectively in the two highest categories (10-14.9 and ≥ 15 μg/dL). One of the three groups which continued to investigate lead's effects at later ages used semi-log models, two used a linear relationship. Keeping things in semi-log terms (versus OEHHA's linearization) would increase the effects of a given change.

Comment #5 continued:

8. Even if OEHHA's risk assessment was accurate, the societal impact of IQ loss would be trivial. Att. 2, pp. 32-38,39-40). Furthermore, fractionalizing an IQ point is irresponsible and meaningless.

Response: Dr. Kaufman states that since IQ is a complex measurement, measurement error is a result. In fact, inexperienced examiners can have a greater impact on IQ than the lead level in the child's blood. Care must be exercised in distinguishing between error in measurement of IQ, which may be large, versus predicted changes in IQ. Measuring IQ with error in the original studies will not bias the concentration-response relationship if the error is random (i.e., not related to actual blood lead), but will increase the error, that is, the power to detect an effect. Therefore, it is not inconsistent or inappropriate to have relatively small average changes in IQ. As stated in the document, the loss of a fraction of an IQ point is meaningless at the individual level, but not at the population level when the community burden of lowered IQ is being assessed. Whether or not the predicted changes in IQ are important from a public health perspective is a risk management issue. OEHHA has attempted to provide best estimates of the quantitative effects based on the available evidence.

Comment #6: In 1991, EPA acknowledged that "[t]here remains uncertainty in the global scientific community about the causal relationship between low level lead exposure and certain health effects in children", and that the issue as to neurological effects is "controversial." (Report to the Organization for Economic Cooperation and Development, "Cooperation on Existing Chemicals: Risk Reduction Lead Country Report" (May 1991) p.54). That controversy is, if anything, intensifying, as both Dr. Piomelli's and Dr. Kaufman's statements make abundantly clear. If OEHHA is unwilling to withdraw or temper its conclusions despite the mounting questions on this issue, it should nonetheless at a minimum amend the Executive Summary discussion to acknowledge the existence of responsible contrary scientific opinion that differs with OEHHA's analysis and conclusions. (page 8)

Response: OEHHA has updated its literature review of studies of the association of lead and neurodevelopment in children. That review is contained in Section 3 of the document and is an

objective reflection of the level of controversy on this issue. OEHHA's conclusions are in harmony with several organizations including U.S. EPA, CDC, NRC and ATSDR.

Blood Pressure

Comment #7: OEHHA's qualifications in its Executive Summary are a reflection of the tenousness of the relationship between lead and blood pressure. OEHHA's September 1996 response to this comment was that each of these qualifiers indicated a different element of uncertainty... This illustrates why there is unabated controversy in the scientific literature on whether or not a causal relationship exits at all. The questionable scientific validity of OEHHA's interpretations is further complicated by applications of [conclusions] to low blood lead levels. Rather than acknowledging the combined effect of these qualifiers, OEHHA simply revised its Executive Summary language, presumably to make the wording appear less tentative, and for good measure, extended the range of effects even lower, to 4 μg/dl. (page 8)

Response: Each one of the qualifiers deals with different aspects of uncertainty in the blood lead - blood pressure association including the identification of a threshold and the evidence for other groups besides adult males. A relationship between systolic and diastolic blood pressure and blood lead has been demonstrated in several, but not all studies. The exact threshold for the effect is not known, but effects have been observed in one study down to 7 μg/dL (Pirkle, 1985) with effects at lower levels of 4 μg/dL observed in other groups. For example, the mean blood lead in males reported by Morris et al. (1990) was 8.0 μg/dL, while in the analysis of males age 20 to 74 in the NHANES II study (Harlan et al., 1985), the minimum level was around 4.0 μg/dL . We have attempted to responsibly depict the uncertainty in the best scientific manner and have now explicitly modeled the cascading confidence intervals.

Second, although lead levels have dropped substantially since NHANES II was conducted (the mean blood lead concentration for men aged 40-59 in NHANES III is 4 μg/dL), the amount of extrapolation used by OEHHA is very small relative to other toxic air contaminant risk assessments. OEHHA considers its extrapolation down to a geometric mean blood lead level of 3 μg/dL to be quite reasonable.

Comment #8: While it may technically be true that "a relationship between systolic and diastolic blood pressure and blood lead has been reported over a wide range of blood lead levels, as low as 4 micrograms per deciliter for middle-aged Caucasian men, OEHHA's discussion makes it clear that these statements strain at the outer edges of reported findings – the support for them consists of "one study" that found an effect at 7 μg/dl (Part C, p. 379), and a vague reference in the Part C document to the fact that "in the analysis of males age 20 to 74 in the NHANES II study, the minimum level was around 4.0 μg/dl," *id.* The analysis in part B does not appear to refer to any study showing effects at 4.0 μg/dl, and the lowest effect level asserted occurs in a sentence referring to a study showing a relationship between diastolic blood pressure and "blood lead values extending possibly down to 5 μg/dl for middle-aged men." Part B, p3-21. (page 8)

Response: OEHHA's statement is based on a comprehensive and objective review of the literature. Our conclusions are also in agreement with those of the U.S EPA, the NRC and several meta-analyses. OEHHA discussed the study by Harlan et al. (1985) in Section 3 of the document but inadvertently

omitted the statement that the investigators found effects in their study down to 4.0 μg/dL. Because blood lead levels have fallen substantially since the NHANES II data were collected, many adults have blood lead levels below the range observed in these analyses. However, the level of extrapolation is far less than that of other risk assessments, and is credible.

Comment #9: The questionable scientific validity of making health interpretations upon such a tenuous health endpoint is further complicated by application of this speculative effect to low blood lead levels. Extrapolation to exposure levels lower than those which were studied can produce significant inflation of estimates of health impact. Moreover, estimates of blood lead's impact upon blood pressure are relative, as opposed to absolute, lead measures. Thus, an 0.06 μg/m^3 air lead yields a presumed 0.108 μg/dl increase in blood lead. Use of a baseline of 3 μg/dl yields hypertension impacts 3.3-fold greater than that which would be predicted to occur at a baseline of 10 μg/dl, and five-fold greater than would be calculated at an average of 15 μg/dl. The present OEHHA analysis suggests that estimates of lead's impact are relatively independent of baseline blood lead values. This statement is difficult to reconcile with the preceding analysis, but one is not able to evaluate the range of baseline values considered in this portion of OEHHA's analysis. It is important to recognize that the calculations made by Schwartz represent an extrapolation of existing studies to blood lead levels lower than those actually studied. The presence of an effect in the blood lead range of 5-10 μg/dl largely rests upon the results of a single study (Hense, 1993). Even in that study, the suggestion of an effect is largely contingent upon data that are not corrected for confounders most investigators feel should be included in an analysis. Confounder correction largely eliminates the suggestion of an effect at low blood lead levels. (page 9)

Response: Three studies were cited in which the range of effects were observed down to the lowest blood lead levels of 4, 5 and 7 μg/dL(Morris et al., 1990; Harlan et al., 1985; Pirkle et al., 1985),. Two of those studies analyzed data from NHANES II which adjusted for workplace exposures to other toxic agents, selection bias, age, BMI, cigarette and ethanol consumption as well as nutritional variables. The geometric mean lead level is now about 4 μg/dL in adults older than fifty. Thus, while lead levels have dropped substantially, the level of extrapolation used in OEHHA's risk assessment of lead and hypertension, is far less than that of other risk assessments, and is credible.

The commenters dispute OEHHA's statement that changes in estimates of lead's impact are relatively independent of baseline blood lead values. OEHHA's statement applied to relatively small changes around the mean values since only small changes are expected. The scenario used in the comment compares much larger changes. Also, there is no reason to think the dose-response changes drastically at 3 or 4 μg/dL.

Comment #10: The Part C response rejects all contrary evidence, even going so far as to extract from Staessen's meta-analysis one data subset (40 to 49 year old males) and use it to support a conclusion opposite to that reached by the authors (Part C, p. 385). [In footnote], The report incorrectly states that the International Conference on Lead and Blood Pressure (Victery, 1988) supports a finding that there is "overwhelming evidence for the causality of the association,"(Part C, p. 380). In fact, the opposite is true. No collective conclusions were produced at this conference; instead each panelist summarized his or her own view of the evidence. Of the seven, only one took the position that the evidence on causality was sufficient and even that panelist, Dr. Joel Schwartz, found it only "probably although not definitively established" (Discussion Report, p. 155). (page 10)

Response: In its evaluation of the relationship of blood lead to blood pressure, OEHHA conducted a thorough and objective literature review. Risk assessments were applied to the subset of men aged 40 to 59 who were non-occupationally exposed, because the strongest evidence existed for this subgroup, although effects were found in NHANES II for the broader age group of men aged 20 to 74. In addition, the 8 or 9 prospective cohort studies linking blood pressure to cardiovascular disease focused on this age group. Other studies found effects in women as well. Given that this subset of men were being examined, it was entirely appropriate to extract the data subset of 40 to 59 (not 40 to 49) year old males from the meta-analysis conducted by Staessen. Furthermore, because age seems to have a modifying effect on blood pressure, it is important to examine the lead-blood pressure relationship in narrower age groups than used by Staessen in his meta-analysis.

Comment #11: It is difficult to understand why the report is so determined to stake out the most extreme ground on this issue and to do so only in the direction of finding an effect. If the report's apparent one-study prerequisite were applied fairly, for example, the Executive Summary ought to note the findings by Staessen et al. (1996) of an inverse (protective) effect from lead exposure in males (page 9).

Response: Though Staeesen's own empirical work failed to detect an association between blood lead and blood pressure, the meta-analysis by Staessen et al. (1995) indicates that an association does exist for both males and females. Furthermore, when studies involving occupationally exposed individuals and those outside the age range of 40 to 59 are removed, the magnitude and precision of the effect estimate improves.

Comment #12: We submit that several highly credible studies including more recent ones, find zero within the range of effects and this possible outcome should be included in the "body count" projections shown in the Executive Summary.

At a minimum, the Executive Summary should be amended to acknowledge that the question of causality remains an issue, as the amended language suggested above for page 8 of the Executive Summary would make clear. In addition, the risk assessment on page 10 of the Executive Summary should be modified to include zero in the range of possible effects. (page 9-10)

Response: . We have responded to the comment about uncertainty in the estimates by adding the following to the Executive Summary (page 10): These effects are based on epidemiologic studies from which it is generally difficult to prove causality. Therefore, controversy remains about the precise magnitude of the effect of blood lead on cardiovascular disease. OEHHA conducted a thorough and objective evaluation of all recent published studies of blood lead and blood pressure. Quantitative risk estimates were based on non-occupational exposures among men aged 40-59. In Section 3, OEHHA discusses potential confounders of the association and both strengths and limitations of the studies reviewed. Finally, uncertainty was propagated throughout the risk calculations. The final range of effects did not include zero.

Air lead-blood lead slope issues.

Comment #13: OEHHA has greatly overestimated the direct incremental benefits or risks that might arise from changes in air lead levels. OEHHA should state explicitly how long one would have to wait for its calculations to be appropriate. As we have pointed out before, OEHHA should include calculations for shorter term exposures for which a direct inhalation slope factor on the order of 1 μg/dl

per μg/m³ would be far more appropriate as is borne out in some of OEHHA's worked examples of IEUBK-projected effects on blood lead levels of changes in air lead.

Table IV-8 of Part A shows that the average indoor concentration of airborne lead in California is about 0.03 μg/m³, about half the ambient average level of 0.06 μg/m³ used by OEHHA in its calculations. This table further show that both children and adults spend about 86 percent of their time indoors. They are thus not exposed to a background level of 0.06 μg/m³ air lead on a continuous basis. OEHHA's projections of health effects attributable to ambient levels of 0.06 μg/m³ accordingly appear to be greatly exaggerated.

OEHHA has also failed to make adjustments for the differential proximity of individuals to sources and the intermittency of their exposures, both of which would serve to reduce the effective blood lead-air lead slope factor from its controlled laboratory study value. No one is exposed continuously to maximum receptor site concentrations all day, every day, yet OEHHA's calculations have assumed exactly that. (pages 10-11)

Response: The air lead/blood lead slope of 1.8 μg/dL per μg/m^3 in adults refers to immediate effects of exposure. The slope of 4.1 μg/dL per μg/m^3 in children includes indirect effects and is therefore over a longer period of time. However, air lead changes in the last decade have resulted in a substantial decline of blood lead concentrations due to the removal of lead from gasoline demonstrating that blood lead concentrations can drop rather quickly.

A. Neurodevelopmental Effects

Comment #14: Both the mean and the GSD [which OEHHA estimated from NHANES III data] are assumed to be known without error. However, the original publications of NHANES III findings reported that the 4.1 μg/dl geometric mean has a 95 percent confidence interval of 3.7 to 4.5 μg/dl. This represents a range of uncertainty of nearly 20 percent, a range far greater than any effect that estimated air lead levels would have on blood lead. A similar and perhaps even greater degree of uncertainty applies to OEHHA's estimated GSD of 2.1. OEHHA needs to take into explicit account the substantial uncertainties in these estimates. How is it possible that OEHHA can confidently project a 0.6 percent change in the proportion of children with blood leads above 10 μg/dl (10.9 to 11.5 percent) from an air lead increment of 0.06 μg/m³ when the distributional parameters that go into its calculations are so much more uncertain? Similar concerns apply to OEHHA's calculations of blood lead levels and changes arising from various air lead differentials for sensitive subpopulations.

Response: We have characterized the uncertainty in the risk estimates by using 95% confidence intervals and, when necessary, by propagation of uncertainty using available software packages. This technique should give the reader a reasonable idea of the uncertainty in the estimates.

Comment #15: OEHHA has assumed that the distribution of blood lead levels is lognormal in form. This theoretical curve is not necessarily valid for particular narrow regions of that distribution, such as for blood lead levels near 10 μg/dl, or for still higher levels. For example, in the NHANES III data, there is a readily apparent cluster of children with blood lead levels in the 20 to 26 μg/dl range that constitutes a far greater percentage of the total than would be predicated (sic) by any log-normal curve fitted to the overall blood lead distribution (cf., Figure 2 in Pirkle et al., 1994). This is simply an

indication that the general population distribution is in reality a mixture of distinct distributions that apply to individual subpopulations of children. The resulting distribution for the overall mixture could not possibly be log-normal, even if each of the individual subpopulations were log-normal. (pages 11-12)

Response:. Given a large enough sample size, most evidence indicates that blood lead distributions can be well described by a lognormal distribution. Although outliers are likely to exist in many distributions, the weight of evidence supports the general log normal distribution.

B. Blood Pressure and Cardiovascular Disease

Comment #16: In developing its estimate of increased probability of hypertension, OEHHA used blood pressure data from various age groups and data sets. For example, OEHHA used NHANES II data for adult males aged 20 to 74, as well as NHANES III data, for adult males aged 20 to 69 in combination with other NHANES II data for men aged 40 to 59. OEHHA also employed blood pressure data for 1979 California white males (age category unspecified) in combination with relationships derived from analyses of Pooling Project data. OEHHA should go further to characterize uncertainty inherent in its combinations of data and relationships from the various studies. The only factors that were considered explicitly as uncertain by OEHHA were the regression coefficients, either for the logarithm of blood lead or for diastolic blood pressure, in each of the empirical relationships it employed. The other important factors in the relationships, namely, the means of the many covariates that are in fact far more significant explanatory variables than is blood lead, and the mean blood lead or blood pressure levels, were apparently presumed to be known without error. OEHHA must adequately characterize these additional sources of uncertainty. (page 12)

Response: The uncertainty in the association of blood lead with blood pressure should not be unduly influenced by other important factors if they are controlled for in the studies. To our knowledge, most important covariates were included in the original regression analysis. Random error in measures of lead should not bias the estimated effect magnitude buy may have added more uncertainty (already included in the standard error of the coefficient).

Comment #17: OEHHA needs also to address explicitly the critical questions of model uncertainty, since it is entirely inappropriate to simply assume the (1)the mathematical form of the true dose-response relationship (if there is one) is exactly and correctly specified, and (2) the relationship extends smoothly all the way down to zero air lead levels. Satisfactory consideration of these additional sources of uncertainty should widen substantially the confidence intervals that surround OEHHA's estimates of potential adverse health effects among adults, and would likely lead to the conclusion that no such effects are confidently attributable to levels as small as or smaller than the near source or statewide ambient air lead levels of interest to OEHHA. (page 12)

Response: We have characterized the uncertainty in the risk estimates by using 95% confidence intervals and, when necessary, by propagation of uncertainty using available software packages. This technique should give the reader a reasonable idea of the uncertainty in the estimates. We have not suggested that the results continue smoothly down to zero air lead levels. The model already incorporates uncertainty due to lack of knowledge about the true dose-response function. Schwartz (1988) showed that the probability of hypertension, as indicated in Section 5, is a logistic function of blood lead. We have modeled this outcome using the same functional form. Likewise, in examining the association between blood pressure and more serious outcomes, we have directly applied the

results of several long-term cohort studies. Therefore, use of the reported functional forms, is appropriate. A similar conclusion was reached by U.S. EPA's Science Advisory Board which reviewed the use of these models as part of the Regulatory Impact Analysis related to the reduction of lead in gasoline. Also, although blood lead levels have dropped dramatically since NHANES II was conducted, the level of extrapolation used in OEHHA's risk assessment is small relative to other risk assessments of toxic air contaminants and reasonable, given the absence of evidence of a threshold for this effect.

III.

Air Resources Board Staff Responses to Comments

Air Resources Board Staff Responses to Summarized Comments on the Draft September 1996 Executive Summary and Part A Exposure Assessment

National Resources Defense Council. Submitted by Gina M. Solomon, MD, MPH. October 22, 1996

Comment: The NRDC would like more information about the degree of protection that the Office of Environmental Health Hazard Assessment (OEHHA) envisions, and that it be included in this document.

Response: The OEHHA provided a scientific assessment based on the existing information. The degree of protection for these children will be a risk management issue which will be discussed in the development of guidelines for risk managers during the risk management phase of the toxic air contaminant program.

Lead Industries Association, Inc., Battery Council International, and GNB Technologies, Inc. October 23, 1996

Comment 1: The estimate of 2500 individuals exposed to a near source concentration of 0.20 micrograms per cubic meter has no basis.

Response: The analysis the Lead Industries Association (LIA) is referring to comes from the near source analysis in Part A, Table IV-4, page 60. This analysis shows that 2,000 individuals in the near source estimate were used instead of 2,500. The approximately 2,000 individuals could potentially be exposed to a minimum of 0.03 micrograms per cubic meter and a maximum of 0.2 micrograms per cubic meter. For the purposes of the near source analysis, the ARB staff used the upper end of the range. It is also noted that Tables IV-5 and IV-6 show that these concentrations are not unreasonable.

Comment 2: The LIA objects to the characterization in Part A that near source monitoring results conducted near the two secondary lead recycling facilities in Southern California showed significant concentrations near the facilities without qualification as to the meaning of the term "significant," and they want the 52-fold value removed from the document.

Response: The monitoring results are viewed as significant because they were 52 times higher than the surrounding ambient concentrations, and were over our ambient air quality standard. The ARB maintains the position that this example is informative and could occur, but have placed this example in context as far as its occurrence and possible causes (page 58-59).

Comment 3: The LIA believes that relying on emissions data from 6 to 7 years before the implementation of the South Coast Air Quality Management District Rule 1420 is inappropriate.

Response: The ARB staff have updated the emissions inventory to include 1996 Rule 1420 data.

During the risk management phase, a more thorough evaluation of source contributions to the ambient air will be conducted.

Comment 4: The LIA objects to the characterization that stationary area source release small quantities of pollutants from many closely located sites over a relatives large geographic area, and finds this to be confusing and unfounded.

Response: The ARB have added clarification on what stationary area sources are in general (page 22).

Comment 5: The LIA states that the report fails to reconcile the revised emission estimates with the prioritization criteria found in the California Health and Safety Code. If reconciled, lead would rank as a low priority for evaluation.

Response: The prioritization occurs before substances are entered into the identification process. However, even though the emission estimate has been revised and is lower, it does not result in the ranking of inorganic lead as a low priority because emissions are only one of five factors considered in prioritization. The others are risk of harm to public health, usage in California, persistence in the atmosphere, and ambient concentrations in the community (Health and Safety Code, section 39660 (f)).