LEAD:
EVALUATION OF CURRENT CALIFORNIA AIR QUALITY STANDARDS WITH RESPECT TO PROTECTION OF CHILDREN

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A. Introduction and Overview

In this section, we provide a general overview of the health effects of lead. In Section II, we discuss the evidence for the adverse outcome most relevant to current concentrations of blood lead in children, neurotoxicity. We also provide background on the Centers for Disease Control (CDC) guidelines concerning the blood level of concern for children. Since the health effects of lead are usually measured as a function of blood lead, rather than ambient lead, it is necessary to understand the relation between ambient lead and blood lead. Therefore, in Section III we review the quantitative evidence linking changes in air lead to subsequent changes in blood lead. In Section IV, we quantify the association between changes in air lead and the percent of exposed children whose blood levels would move above the CDC blood level of concern. This provides evidence about the protectiveness of the current ambient lead standard for California of 1.5 $\mu g/m^3$ averaged over one month. Section V provides a conclusion about whether significant health effects on children and infants are likely at concentrations below the current California ambient standard for lead.

The adverse health effects of lead were first described by Hippocrates in 370 BC. Pliny indicated that lead was a problem for both workers and residents of Rome during the first century AD (Kazantzis, 1989). In Britain in the 1880s, laws were enacted to control occupational exposure to lead. Over the last century, additional evidence of adverse effects from toxicological, clinical and epidemiological studies has continued to accumulate. These studies provide strong and consistent evidence for health effects related to current blood lead concentrations. A thorough review of health outcomes associated with lead exposure is provided by the U.S. Environmental Protection Agency (U.S. EPA, 1986, 1990a), the Agency for Toxic Substances and Disease Registry (ATSDR, 1990) and the National Research Council (NRC, 1993). At very high acute exposures to blood lead concentrations (> 125 micrograms per deciliter or μg /dl of blood), death can result. Brain and kidney damage have been

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reported with blood level concentrations between 80 and 100 $\mu g/dL$. Chronic exposure to lead can cause blockage of the proximal tubule in the kidney and kidney failure. Lead-induced chronic nephropathy (kidney damage) has been observed in occupationally exposed workers at blood lead levels as low as 40 $\mu g/dL$. Other renal effects, such as decreased vitamin D metabolite levels, have been observed at 30 $\mu g/dL$. The lowest blood lead level at which these effects occur has not been determined. Chronic exposure to lead in humans can also affect the blood. Anemia in adults has been reported at blood lead levels of 40 to 60 $\mu g/dL$, and in children at 30 to 40 $\mu g/dL$. In addition, increased blood pressure in adults has been reported at blood lead concentrations as low as 10 $\mu g/dL$.

Lead is also associated with several adverse reproductive and developmental outcomes. In male industrial workers, sperm abnormalities, reduced fertility, and altered testicular function have been observed at blood lead concentrations of 40-50 $\mu g/dL$ and sometimes at lower levels. Lead has also been associated with adverse effects on the fetus. Since lead in blood crosses the placenta, the fetus may be affected by maternal blood lead level elevated from current or past exposure. Several prospective studies have demonstrated an association of maternal blood lead levels of 10 to 15 $\mu g/dL$ with pre-term delivery and low birthweight (NRC, 1993). Also, studies have shown lead's effects on childhood growth. For example, using the National Health and Nutrition Examination Survey (NHANES) data, small but significant reductions in early childhood growth were observed, with no apparent threshold across a range of 5-35 $\mu g/dL$ (Schwartz et al., 1986). Lead levels of 10 $\mu g/dL$ and below have also been associated with decreased hearing acuity (Schwartz and Otto, 1987).

Levels of lead below 25 $\mu g/dL$ cause both clinical and subclinical effects on the brain and nervous system. Several long-term prospective epidemiological studies have reported an association of pre- and postnatal lead exposures with measures of intelligence, such as IQ, in

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infants and young children (U.S. EPA, 1986, 1991). These effects have been noted at blood lead levels of 10 to 20 μg/dL and lower. Since children and infants are most susceptible to the neurotoxic effects of lead, these studies are discussed in greater detail in the next section. *In vitro* and *in vivo* studies reveal changes in neurotransmission and brain mitochondrial function within minutes of exposure to submicromolar concentrations of lead. The lowest levels at which these effects occur in humans have not been determined, but these neurochemical changes could plausibly form the basis for neurodevelopmental effects observed in children. These effects have great public health significance since they are likely to occur at current ambient and blood lead concentrations.

Reviewing this body of evidence, the CDC identified 10 μg/dL as a "level of concern." CDC has also recommended certain community actions dependent on the actual observed blood lead concentrations. For example, when many children in a community have blood lead levels between 10 and 14 μg/dL, community-wide childhood lead poisoning prevention activities should be initiated. All children with blood lead levels at or above 15 μg/dL should receive nutritional and educational interventions and more frequent blood lead screening. Between 15 and 19 μg/dL, environmental investigation (including a home inspection) and remediation should be undertaken if the blood lead levels persist. A child with blood lead levels between 20 and 44 μg/dL should receive environmental evaluation, remediation and a medical evaluation. Such a child may need pharmacologic treatment for lead poisoning. Above 45 μg/dL, a child would receive both medical and environmental interventions, including chelation therapy. In our analysis to determine whether effects may occur below the current ambient air standard, we will use 10 μg/dL as the level of concern for effects on intelligence, although effects may occur at lower levels. This "level of concern" is consistent with those identified by the U.S. EPA (1990a), the CDC (1991b), the National Research Council (1993) and the ATSDR (1990).

In addition to neurological effects, lead interferes with the synthesis of heme, which is essential for the functioning of cells in many organ systems, especially the brain, kidney, liver, and blood-forming tissues. Heme is a component of hemoglobin, the oxygen-carrying pigment of red blood cells. An elevated lead level can impede hemoglobin synthesis, resulting in anemia. Heme is also a constituent of cytochrome P-450 and electron transfer cytochromes. Lead can impair the function of heme-dependent liver enzymes (cytochrome P-450), which can increase vulnerability to the harmful effects of other toxic chemicals. Lead's effects on vitamin D synthesis are mediated through its effects on heme. Finally, interference with heme biosynthesis may play a role in lead's neurological effects. Decrements in an enzyme involved in heme synthesis (ALA-D) have been observed at blood lead levels as low as 10 μg/dL although the biological and medical significance of effects at this level are not well understood. Several studies using large population-based data have indicated an association of lead in blood with blood pressure in adults, particularly men, at lead levels as low as 7 μg/dL of blood (NRC, 1993).

Many of these health effects are consistent with those seen in animal and cellular studies at very low levels. Therefore, the lead levels at which these health effects are seen in humans should not be considered as threshold values, but rather as levels below which there is less certainty of the presence of adverse health effects.

Over the last 15 years, average blood lead levels have declined dramatically in both children and adults (Pirkle et al., 1994, Pirkle et al., 1998). The decline in blood lead levels is consistent with, and undoubtedly related to, continued reduction in exposure to lead from environmental sources which began in the late 1970s. From 1976 to 1990, the amount of lead used in gasoline decreased 99.8% nationally (from 205,810 tons to 520 tons). In California, dramatic decreases in average ambient air lead levels have occurred over the last two decades. The reduction and subsequent ban of lead in gasoline is most likely the greatest

contributor to the observed decline in blood lead levels during this period (Pirkle et al., 1994). The major remaining sources of environmental lead that pose a potential public health threat appear to be localized sources of lead, including but not limited to continued deterioration of lead-based painted surfaces in older buildings, and lead that has already accumulated in dust and soil, and near point sources of air emissions.

B. Neurodevelopmental Effects On Children

Lead's neurodevelopmental effects observed at low and moderate exposure levels (30 μg/dL and below) include: decreased intelligence, short-term memory loss, reading and spelling underachievement, impairment of visual motor functioning, poor perception integration, disruptive classroom behavior, and impaired reaction time (U.S. EPA, 1989d; ATSDR, 1994; Bellinger et al., 1994a; Bellinger et al., 1994b; Needleman et al., 1996).

Children are more vulnerable than adults when exposed to lead partly because they: (1) have hand-to-mouth behaviors that result in more ingestion of lead in soil and dust; (2) are more likely to exhibit pica (abnormal ingestion of non-food items); (3) absorb substantially more lead from the gut than adults, especially when they are below 2 years of age; (4) have a faster metabolic rate, resulting in a proportionately greater daily intake of lead through food; (5) have a less developed blood-brain barrier and therefore greater neurologic sensitivity (Smith, 1989); (6) have a faster resting inhalation rate; and (7) tend to breathe through their mouths when at play (less inorganic lead particulate is trapped in the nasal passages in mouth-breathers). Furthermore, children from economically disadvantaged backgrounds are especially vulnerable because they are more likely to have diets deficient in elements that suppress lead absorption, such as iron and calcium.

While teeth and bone reflect the cumulative dose of lead, blood lead levels mostly reflect recent exposures (from the past 1 to 3 months) but are also influenced by past exposures, because lead can be mobilized from bone and other storage sites. However, blood

lead levels are indicative of current soft tissue exposures. In addition, blood lead levels are reproducible (to within $\pm$ 1 μg/dL) and can be compared across studies to indicate relative levels of exposure (Smith, 1989).

Early studies of neurotoxic effects of lead were conducted by Needleman et al. (1979) using lead levels in the teeth of first and second graders. A significant association was detected between increased dentine lead level and decrements in intelligence quotient (IQ). The association was still evident when the children were tested 5 and 11 years later (Bellinger et al., 1984b; Needleman et al., 1990). Since the Needleman et al. (1979) article appeared, many studies have been published that support this finding. Most of the early studies were cross-sectional in nature, where groups with different blood lead concentrations were compared at a single point in time. Like many epidemiological studies of this type, there are concerns about exposure assessment and the ability to control for potential confounders. Despite these issues, the cross-sectional studies consistently demonstrate an association between blood lead and IQ. In an attempt to characterize the overall findings of several cross-sectional studies, Needleman and Gatsonis (1990) undertook a meta-analysis of the published IQ-blood lead studies. By pooling the results of the individual studies, the meta-analysis addressed the problem of small sample sizes with the accompanying low statistical power. The results suggested that each 1 μg/dL increase of blood lead results in a 0.24 point decrease in IQ.

Since cross-sectional studies use a single blood lead measurement as a surrogate for earlier exposures, they are more likely to suffer from exposure classification errors than prospective studies (McMichael et al., 1994). As a result, large, long-term, prospective studies were conducted in Boston, Cincinnati, and Port Pirie, Australia. In addition to minimizing recall bias, prospective studies allow investigators to measure temporal changes in outcome relative to prior levels of exposure. Because the child is followed over time, researchers can examine

the effects of lead exposure at different times as well as estimate the effects of cumulative exposure.

One of the larger cohorts studied, from Boston, Massachusetts, includes several hundred middle and upper-middle class children followed from birth to 10 years of age (Bellinger et al., 1984a, 1985, 1991, 1992; Stiles and Bellinger, 1993). These studies have consistently found an association between blood lead and IQ among different age cohorts. Among the more important findings are those of older children since their IQs may be better characterized in the standardized tests. For example, at age 10 years, the children were examined again using the Wechsler Intelligence Scale for Children-Revised (WISC-R), a measure of cognitive function, as well as the Kaufman Test of Educational Achievement (KTEA) (Bellinger et al., 1992; Stiles and Bellinger, 1993). Higher levels of blood lead at 24 months were associated with significantly lower scores on FSIQ (full scale IQ) and verbal IQ. The authors observed a decrease of almost 6 points on FSIQ and 9 points on KTEA Battery Composite score for each 10 μg/dL increase in lead level at 24 months. These estimates include adjustments for maternal age, race, marital status, number of residence changes and home environment. Visual inspection of the results and analysis of an earlier data set (Schwartz, 1993) suggest a continuous response across the entire range of blood lead levels and the lack of any threshold.

In summary, in the Boston cohort, effects on intelligence were evident from both pre and postnatal blood lead. Postnatal blood lead levels at 24 months were significantly associated with FSIQ at age 10 and to some neurological function tests requiring attention for good performance. Children from lower socioeconomic status appeared to be more sensitive to effects at lower blood lead concentrations. A more recent study found that lead impacted high school classroom behavior (Needleman et al., 1996). Therefore, evidence from these studies suggests that both prenatal and postnatal exposure may be associated with adverse

impacts on cognitive performance with effects from postnatal exposure persisting to at least 10 years of age. The effects of later postnatal exposure seem to be strongest.

Other large prospective studies of lead and neurodevelopment involve cohorts of inner-city children in Cincinnati, Ohio and children in Port Pirie, South Australia (NRC, 1993). Although there are differences in socioeconomics and demographics, experimental techniques, statistical models, and patterns of exposure among the three large cohort studies, their findings are consistent. Among the more relevant findings, changes in IQ at ages 6 to 10 are associated with blood lead measured either cumulatively over several years or in a single year. In addition, the magnitude of effect per μg/dL are similar among both the prospective and cross-sectional studies. Many of these studies report mean blood concentrations near 10 μg/dL.

Several researchers have reviewed or conducted qualitative or quantitative meta-analyses of the prospective studies relating low-level blood lead exposures to neurodevelopmental effects in young children. For example, researchers with the CDC (Thacker et al., 1992) reviewed 35 prenatal and early postnatal prospective cohort studies. They concluded that the weight of evidence suggested an adverse relationship between lead on the intelligence of children. Pocock et al. (1994) reviewed several types of studies to quantify the relationship between lead and IQ, including the WISC-R. The analysis concluded that for postnatal blood lead, both the cross-sectional and prospective studies indicate a significant inverse association between blood lead and IQ. In addition, Schwartz (1994) conducted meta-analyses of both longitudinal and cross-sectional neurodevelopmental studies. He used all studies published before 1993 that reported blood lead and measured full scale IQ. For the longitudinal cohorts, he selected those studies that measured exposure during the first 3 years of life when the neural network is most vulnerable to neurotoxicants.

Schwartz examined the IQ loss indicated by both the cross-sectional and prospective studies and concluded that the two study designs were capturing similar effects.

To provide an estimate and range of risk, the Office of Environmental Health Hazard Assessment (OEHHA) conducted a simplified meta-analysis (Hedges and Olkin, 1985) of cohort studies conducted in children older than 5 years (see Table 1). This age group was used because it is likely to provide the most accurate assessment of the impact of blood lead. Estimates of the mean effect were derived by weighting each of the regression coefficients by the inverse of its variance. This generated a mean decrease of 0.33 IQ points per μg/dL blood lead with a 95% confidence interval of 0.32 to 0.34. Thus, this central estimate suggests that a 1 μg/dL increase in postnatal blood lead is associated, on average, with a 0.33 point decrease in FSIQ. This level is close to the range of estimates derived from the earlier meta-analyses, cited above. OEHHA used this value in its identification of lead as a toxic air contaminant (OEHHA, 1997).

In addition to the general effect magnitude, the overall population-level impact of IQ is also important to consider. Grant and Davis (1989) have demonstrated that, if one shifts down a normal distribution of IQ scores (mean=100, standard deviation=16) by 4 points, the number of children scoring 80 or below increases by 50%. The impact of such a shift applies across the entire distribution of scores, reducing the number of children who score above the norm as well as increasing the number scoring well below the norm. Thus, 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. Similarly, a shift of 3.3 points would increase the percent of children scoring 80 or below from 10.56% to 14.74%, a 39.5% increase.

The consistency of findings lends strong support to the conclusion that neurodevelopment effects are causally associated with blood lead, and that the CDC level of concern of 10 μg/dL is a reasonable action level.

C. The Contribution of Airborne Lead to Blood Lead Level

In order to evaluate the impact of change in air lead on health, it is necessary to determine the quantitative association between changes in air lead and subsequent concentrations of lead in the blood of exposed populations. This is necessary since most lead-related health effects use blood lead, rather than ambient lead, as the measure of exposure. The relationship between air lead and blood lead has been extensively studied in both field studies and experimental chamber studies. Studies in experimental chambers in which air exposures are well characterized have only been conducted using adults. Since young children have higher metabolism and inhalation rates, and since they ingest more dirt and dust (Smith, 1989; Chamberlain, 1983), one would expect air lead levels to have a greater impact on blood lead in children than in adults. Thus, when assessing risks from air lead, it is important to examine the relationship between air lead and blood lead (or the blood lead/air lead "slope") in adults and children separately. In a review of the relevant studies, OEHHA examined both the magnitude of the slope estimate and the implications for lower concentrations of air and blood lead. The review included experimental chamber studies in adults, a population-based study of adults using personal monitors, and several population-based studies of children using outdoor fixed-site monitors.

Early inhalation studies assessed the effects of inhaled lead using human subjects in experimental chambers. These chamber studies allow direct calculation of the blood lead/air lead slope through the inhalation pathway. The chamber studies are useful because exposures were well characterized. The U.S. EPA estimated that when the subjects exposed to very high air lead levels were excluded, the average blood lead to air lead slope was 1.9 μg/dL per μg/m^3 (US EPA, 1986). In their critical review, OEHHA concurred with the U.S. EPA assessment of the slope from the chamber studies, noting that the exposure levels were

relatively high in these studies and that the aggregate slope may be nonlinear, with steeper

slopes at lower air and blood lead levels (OEHHA, 1997).

For children, there are three available models to determine the contribution of air lead

concentrations to blood lead levels. These models include a disaggregate model, an

aggregate model and an uptake biokinetic model. In the disaggregate model and in the

biokinetic uptake model, the total effect of air lead is estimated by separately analyzing the

associated changes between blood lead and inhaled air lead as well as lead deposited onto

soil, dust, food and water (U.S. EPA, 1989b). Once the impact of each separate pathway on

blood lead is determined, as well as the impact of air lead on other pathways such as soil and

household dust, the total effect of a change in air lead on blood lead can be determined.

While this approach may yield more precise estimates of the relationship of air lead and blood

lead, the contribution of each pathway must be modeled and any errors or uncertainties in

these variables will reduce the precision of the estimated total exposure.

One method for developing a disaggregate model is to use a multivariate regression

approach to explain blood lead with separate explanatory variables representing soil, dust and

air lead in the model specification. Using a disaggregate model for children, U.S. EPA (1986)

estimated a slope of 2 when only accounting for the direct influence of air lead, and a slope of

5 when incorporating both direct and indirect influences of air lead (U.S. EPA, 1989b).

A second method to estimate the effects of change in air lead on changes in blood lead

is an aggregate model. This model implicitly takes into account both the direct and indirect

sources of air lead. One can derive an estimate of the slope using either of two approaches.

First, a regression model attempting to explain the variation in blood lead with air lead as an

explanatory variable, but unadjusted for soil or household dust, can be used. Ideally, such a

model would adjust for age, ethnicity and other nonenvironmental confounders. Second, a

slope can be calculated using blood lead levels and air lead levels from at least two points in

time or between two populations. For example, blood lead levels in a highly exposed and a control community can be related to corresponding ambient air lead levels. The slope (β) is calculated by comparing groups using the following formula:

$$\beta = \frac{\text{difference in blood lead between group II and group I } (\mu g/dL)}{\text{difference in air lead between group II and group I } (\mu g/m^3)}$$

The advantage of aggregate estimates is that they implicitly incorporate both direct and indirect pathways of air lead. Since we are estimating the total impact of air lead emissions on children's blood lead, aggregate slopes are most relevant in that they incorporate all air-related pathways. OEHHA staff reviewed the relevant studies for determining a slope (OEHHA, 1997). Their "best estimates" are developed from the studies using the lowest blood lead and air lead concentrations. Table 2 summarizes the best estimates and ranges from the available studies. Using 14 studies covering seven different study populations, OEHHA took the geometric mean of the study estimates to determine a combined slope estimate of 4.2 with a range of 3.3 to 5.2.

The third method for estimating blood lead levels in relationship to exposures from various media is the uptake biokinetic model such as U.S. EPA's Integrated Exposure Uptake Biokinetic (IEUBK) (U.S. EPA, 1989b). This model estimates specific intake, uptake and distribution of lead in the body, taking into account the route and rate of exposure, a child's age and baseline exposure. The processes of absorption, distribution, storage, mobilization and excretion are directly modeled. This personal computer-compatible model calculates probability distributions of blood lead levels for children of different ages based on a multipathway exposure analysis. Categories of exposure input variables include concentrations of lead in air, drinking water, soil, house dust, paint, diet and maternal blood lead (to account for newborn blood lead), with each having default or user-defined values. Exposures are converted into an uptake component based on amounts of lead absorbed from

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the lungs and gastrointestinal tract. The absorbed doses of lead are then biokinetically modeled into six different body tissues or compartments over 84 monthly time points (age 0 to 7 years) to account for age-dependent physiological parameters (e.g., increases in inhalation rates and body weights) and soil ingestion rates. Several validation efforts (Hogan et al., 1995a; U.S. EPA, 1994a; U.S. EPA, 1994b) show that the model performs well in predicting blood lead distributions. The model does not use a single blood lead/air lead slope to relate air lead concentration to blood lead concentration. Nevertheless, with some adjustment to the IEUBK model recommended by its authors (Hogan, 1995b), aggregate slopes can be determined and are presented below for comparative purposes.

Since the model assumes a nonlinear relationship between environmental and blood lead concentrations, the slope will vary with air lead concentration. Recent studies of the model carried out by the U.S. EPA using the supplemental equations based on data from either East Helena (for which a significant monitoring program was developed) or data from 40 communities, indicate aggregate slopes of 3.7 and 5.3 μg/dL per μg/m^3, respectively, for a change in air lead level from 1.5 to 2.5 μg/m^3 (Hogan, 1995b). OEHHA also looked at the slopes generated by incremental increases between 0 and 1 μg/m^3 (OEHHA, 1997). In this range of air lead concentrations, the IEUBK model predicts approximate slopes of around 6.5 μg/dL per μg/m^3. It is important to note that the slope may be a nonlinear function of the actual air lead and blood lead levels being investigated. Many researchers report a relationship that is supralinear with steeper slopes at lower air and blood lead levels (Brunekreef, 1984; Chamberlain, 1983; Hammond, 1981; O'Flaherty, 1993). If this is true, then studies conducted at higher air and blood lead concentrations might underestimate the blood lead/air lead slope associated with relatively low air concentrations.

In summary, the population-based studies provide important information not available through chamber studies; i.e., "real world" exposure scenarios encompassing a wide range of

behaviors, ages, microenvironments and climates. As expected, they indicate that "real world exposures" result in higher blood lead levels than would be directly predicted from chamber studies (slope = 2), which only measure exposure from the inhalation route. The population-based studies include deposition, accumulation and exposure from other environmental pathways as well. U.S. EPA analysis using either disaggregate (pathway specific) or aggregate models (all pathways combined) suggest a slope range of from 3 to 5 μg/dL per μg/m^3 for children (EPA, 1989a). OEHHA analysis including additional studies suggests a similar range with a best estimate of 4.2 (OEHHA, 1997). Therefore, for the subsequent analysis, we use the slope estimate of 4.2.

D. Airborne Lead and The Risks of Neurotoxicity

D.1 Background

This section examines the protectiveness of the current ambient standard for lead by providing estimates of neurodevelopment risks associated with alternative ambient air lead concentrations. Specifically, we estimate the increase in the proportion of children that will move above the CDC level of concern of 10 μg/dL as ambient lead increases from the current baseline statewide average.

To assess the impacts to children from exposure to air concentrations of inorganic lead, there are several key assumptions. These are: (1) the CDC 'level of concern' of 10 μg/dL is the appropriate blood lead concentration that should not be exceeded in order to protect the health of children; (2) 4.2 μg/dL per μg/m^3 is the most appropriate aggregate blood lead/air lead slope factor for children and is assumed to be linear over the ambient air lead concentrations of interest in California; and (3) the mean blood lead levels in one- and two-year old children in California, exposed to the baseline ambient lead average of 0.055 μg/m^3 of airborne lead, are generally comparable to one- and two-year old children in the NHANES III

survey, i.e., lognormally distributed, with a geometric mean of 3.1 µg/dL, and a geometric

standard deviation (GSD) of 2.1. Each of these issues will be discussed in turn below.

D.2 Identification of the Blood Lead Level of Concern

Based on current information it is not possible to identify a clear threshold blood lead

level associated with adverse health effects in humans. As discussed in Section III, a level of

concern where human neurodevelopmental effects are seen in children exposed either

prenatally or postnatally has been identified at 10 µg/dL. The CDC has concluded that blood

lead concentrations at or near 10 µg/dL present a public-health risk to infants, children and

pregnant women (CDC, 1991). This blood lead level is the CDC level of concern for

communities as a whole, as well as for individuals (CDC, 1991). The level of 10 µg/dL has

been designated by the U.S. Public Health Service as the maximum permissible concentration

from the standpoint of protecting the health of children and other sensitive populations (NRC,

1993). In 1990, the Science Advisory Board of the U.S. EPA identified a blood lead

concentration of 10 µg/dL as the maximum to be considered safe for individual young children

(NRC, 1993). The National Research Council and U.S. EPA concur that neurodevelopmental

effects in children are likely to occur at 10 µg/dL and possibly lower (NRC, 1993; U.S. EPA,

1990a). However, as the evidence continues to grow, it is possible that future levels of

concern may drop below 10 µg/dL (NRC, 1993).

D.3   Blood Lead/Air Lead Slope

Based on the studies described in Section II, we estimate that increases in airborne lead concentrations can result in an increase in blood lead levels in children at an estimated rate of 4.2 µg/dL per µg/m^3, after all air-related exposure pathways are included and a steady state has been reached. Reasonable lower and upper bounds for the slope are 3.3 and 5.2, based on the range of geometric means. Although the studies reviewed in Section III include many different age groups, applying the results to the younger children appears reasonable.   We have assumed that the slope is linear near current ambient air lead concentrations and blood lead levels so that calculations for varied exposures near these levels can be made using the aggregate slope factor.

D.4.   Geometric Mean Blood Lead Level for California

There currently are no population-based blood lead data that are both specific to and representative of California as a whole. For this reason, estimates of the geometric mean blood lead level for California children are derived from the third National Health and Nutritional Examination Survey, Phase 2 (NHANES III), conducted by the National Center for Health Statistics/Centers for Disease Control and Prevention.   This survey provides nationally representative estimates of blood lead levels for several population subgroups, by age, sex and race/ethnicity (Pirkle et al., 1998). Specifically, data for the years 1991 through 1994 were provided for one- and two-year old children and disaggregated for non-Hispanic whites, African-Americans (defined as "non-Hispanic blacks" in NHANES III), and Mexican-Americans. Survey sample weights were applied to the data in order to generate estimates that were representative of the U.S. population. Blood lead concentrations were measured by graphite furnace atomic absorption spectrophotometry, with a detection limit of 1 µg/dL. Analysis of each specimen was performed in duplicate, and the mean of the duplicate measurements was

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reported.   "Blind" sample analyses and laboratory analytical quality control and quality assurance in the blood lead data analysis.

This data set of 13,642 people provides the best blood lead concentration information available for the United States. It contains standardized estimates with a high degree of both analytical standardization and laboratory quality control and quality assurance. For these reasons, it is reasonable to extrapolate the blood lead distribution characterized by NHANES III to California. However, we are uncertain about how the levels in California compare to the national averages, since the sampling design used in the NHANES III survey does not provide representative state-level data. Consequently, we conducted sensitivity analyses to determine the impact of assuming a lower mean for California than the national average. Our sensitivity analysis indicates that the general results of our analysis are relatively insensitive to the choice of the geometric mean or geometric standard deviation (GSD).

Although several recent studies have investigated the distribution of blood lead in certain cohorts of California children, they are not necessarily representative of the state's population. For example, Haan et al. (1996) examined blood lead levels in 305 healthy children recruited from HMOs over a 5-month period in 1991-1992. In this study, the mean blood lead levels was 4.65 µg/dL, for children ages 1 through 5. However, this sample is not representative of the state as a whole. Although this clinic-based sample was representative of the HMO studied, its characteristics differ from the overall state population. Specifically, the population consists of employed families, most with two parents, with pre-paid health insurance. In addition, the sample used in this study consists of regular visitors to the well baby clinic.

As part of a cost analysis of the lead-testing program in Orange County, California, Gellert et al. (1993) analyzed a nonrandom sample of venous blood lead tests in 5115 children aged 1-5 years in the Child Health and Disability Prevention Program. The ethnicity of the

sample was not representative of the state of California: 73.5% Hispanic; 12.2% Asian; 9.8% white; 1.2% African-American; 3.3% other. Blood lead levels were found to be greater than 10 μg/dL in 7.25% of children. The geometric mean and standard deviation were not provided. The major sources of blood lead levels ≥ 20 μg/dL in children in this study were shown to be related to pica, folk remedies, use of unglazed earthenware or peeling paint. While only 7.54% of houses in the county were constructed prior to 1950, no information on the location of residence were provided for the study population. The results of this study should not be generalized to the California population because the sample was not randomly chosen, the ethnic makeup of this community differed from the state and because the age distribution of housing may not be representative of those in other urban areas.

Since it is difficult to generalize these available data to the entire state, we use the data from NHANES III, Phase 2. The NHANES III results are tabulated by age, so that data can be disaggregated for one- and two-year olds, the age group at greatest risk for lead poisoning. For this subgroup, the geometric mean blood lead was 3.1 μg/dL with 5.9% above 10 μg/dL. The NHANES III data also reveal that the geometric mean blood lead values vary by race/ethnicity within this and all other age groups. Blood lead levels for non-Hispanic white male children are the lowest (GM = 2.7 μg/dL) relative to that of Non-Hispanic blacks and Mexican-Americans (GM= 4.8 and 3.2, respectively). Regression analysis for all age groups together indicated that higher blood lead levels were found among males, those residing in large urban areas, blacks, those from families with lower education and lower income (Brody et al., 1994). Also, the data demonstrated that children between ages one and two tended to have higher blood lead levels.

Since the distribution of blood lead is log normal, the GSD provides the best summary measure of dispersion. The GSD can be used to calculate the percent of a cohort that exceeds 10 μg/dL, and *vice versa*. For the one- and two-year old age group, we use the GSD

of 2.1 based upon the NHANES III data, Phase 2. We assume that this distribution is associated with average national lead exposure occurring from 1991 – 1994. During this period, the ambient average for lead was 0.055 $\mu g/m^3$. Children exposed primarily to a local point source may be more homogeneous and have more similar exposures patterns relative to the California population as a whole. As a result, this group may be expected to have a higher mean blood lead and a lower GSD. For example, White et al. (1998) suggest a GSD values for a single city of around 1.8.

D.5. The Range of Neurodevelopmental Risks for Children Using the Aggregate Model Approach

When the U.S. EPA developed its lead standard in 1978, the aim was to protect those children most exposed to lead. U.S. EPA considered the range of blood lead concentrations, the geometric mean and the GSD reported at that time. Then they identified 30 $\mu g/dL$, the level of concern at the time, as a level not to be exceeded by 99.5% of children (U.S. EPA, 1978). From these assumptions the U.S. EPA developed the current ambient air quality standard of 1.5 $\mu g/m^3$ (arithmetic average). In a more recent analysis, U.S. EPA chose a ninety-five percent level of protection for site-specific preliminary soil remediation goals for lead at Superfund sites and soil cleanup standards for lead at RCRA (The Resource Conservation and Recovery Act) sites (Laws, 1994). The U.S. EPA calculation is designed to limit exposure such that children would have an estimated risk of no more than 5% of exceeding the 10 $\mu g/dL$ level.

We have adopted a similar approach to that used in the U.S. EPA (1978) methodology, while incorporating more current information. The current level of concern is 10 $\mu g/dL$, not 30 $\mu g/dL$ used by U.S. EPA in 1978. Second, at the current time approximately 5.9 percent of all one- and two-year old children already have blood lead levels exceeding 10 $\mu g/dL$ blood lead (Pirkle et al., 1998) due to exposure to lead from various media, such as air, water, food,

consumer products, soil, and paint. Also, decreasing the current statewide average air lead concentration of 0.055 μg/m^3, even to zero air lead, would still not protect 99.5%, or even 95% of children from exceeding 10 μg/dL, based on NHANES III data. For these reasons we focus on quantifying the incremental change in the proportion of children with blood lead levels exceeding 10 μg/dL that would result from exposures to various concentrations of air lead. This will help determine whether the current standard is protective.

A first step in this analysis requires calculating the geometric mean blood lead levels associated with alternative air lead concentrations using a blood lead/air lead slope of 4.2. Next, assuming a constant GSD of 2.1, we calculate the proportion of the cohort expected to have a blood lead level of 10 μg/dL or greater. We assume all other sources are constant, except those that are impacted directly by increases in air emissions (i.e., soil and dust concentrations). The results are summarized in Table 3. The analysis indicates that an air lead concentration of 0.10 μg/m^3, an additional one percent of the population of one and two year old children in California would be predicted to exceed 10 μg/dL. At an air lead concentration of 0.25 μg/m^3, an additional 4.5 percent of the exposed population of one- and two-year old children would be predicted to have blood level concentrations exceeding the CDC guideline of 10 μg/dL. At an air lead concentration equivalent to the current ambient standard of 1.5 μg/m^3, more than 45% of children aged one and two would have blood lead levels above the CDC guideline of 10 μg/dL according to the aggregate model. We next examined the implication of lowering the GSD from 2.1 to 1.8. Moving from the baseline ambient concentration of 0.055 to 0.10 adds 0.6% to the number of children expected to exceed 10 μg/dL and adds 3% if the ambient concentration moves to 0.25 μg/m^3. Even with this lower GSD, 44% of the children would be expected to have blood lead levels above 10 μg/dL at an ambient concentration of 1.5 μg/m^3. Finally, if we lower the assumed GSD to 1.8

and lower the GM to 2.5, we still estimate that at the current ambient standard, almost 40% of the children would exceed the CDC level of concern. If we considered the effects on African-American children, who have a much higher baseline GM, the percent moving above 10 μg/dL in any of these scenarios would be much greater.

With an estimated total California population of 33.9 million, the estimated number of one- and two-year old children is 1.2 million (California Department of Finance 1996 projections). With a GM = 3.1 and GSD = 2.1, at the current state standard an additional 476,000 children would move above the CDC level of concern.

E. Summary

In summary, the estimates indicate that exposure to an airborne lead concentration up to the current state standard of 1.5 μg/m^3 is associated with an increase of approximately 40% of the cohort of one- and two-year old children to levels exceeding the CDC level of concern. Even an increase to only 0.50 μg/m^3, would theoretically result in an additional 10% of children having blood lead concentrations above the CDC level of concern of 10 μg/dL.

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Table 1. Regression Coefficients Indicating Change in IQ per 1.0 µg/dL Increase in Blood Lead for Crude and Adjusted Models in Prospective Studies at Later Ages

Crude Model:

Study	Intelligence Measure	Coefficient (s.e.)
Boston[a]	WISC-R (FSIQ)	-0.71 (0.25)
Cincinnati[b]	WISC-R (FSIQ)	-0.58 (0.13)

Adjusted Model:

Study	Intelligence Measure	Coefficient (s.e.)
Boston[c]	WISC-R (FSIQ)	-0.58 (0.21)
Cincinnati[d]	WISC-R (FSIQ)	-0.33 (0.14)
Port Pirie[e, f]	WISC-R (FSIQ)	-0.24 (0.12)

Meta-Analyses:

Study	Intelligence Measure	Coefficient (s.e.)
Needleman and Gatsonis[g]	Varied	-0.25 (0.04)
Schwartz[h]	Varied	-0.24 (0.04)
OEHHA[I]	WISC-R (FSIQ)	-0.33

a. Blood lead at age 2, WISC-R at age 10, unadjusted analysis.
b. Mean blood lead at age 6, WISC-R at age 6.5
c Adjusted for HOME score at 10 years, maternal age, race, marital status, and number of residence changes prior to 57 months.
d. Adjusted for HOME score, maternal IQ, birth weight, birth length, child sex, and cigarette consumption during pregnancy.
e. Averaged blood lead at ages 0-4, linearized using PbB mean of 19.59, WISC-R at age 7.
f.. Adjusted for sex, parent's level of education, maternal age at delivery, parental smoking status, SES, HOME score, birth weight, birth order, feeding method, duration of breast feeding and whether or not child's parents were still living together.
g. Meta-analysis of six cross-sectional studies of blood lead and intelligence.
h. Meta-analysis using same six cross-sectional studies and one additional prospective study by Bellinger et al. (1991).
i. Meta-analysis using the three above "Adjusted Models."

Sources: Stiles and Bellinger (1993); Bellinger et al. (1992), Dietrich et al. (1993), Baghurst et al. (1992), Needleman and Gatsonis (1990), Schwartz (1993), OEHHA (1997).

Table 2. Best Estimates and Range of Slopes from Population-Based Studies in Children using Aggregate, Disaggregate and IEUBK Models..

Aggregate Study	Location	Ages (yrs)	Best Estimate[a]	Range[a]
Brunekreef et al., 1983	4 Dutch cities	4 to 6	8.50	NR
Angle et al., 1984	Omaha	1-18	1.92	NR
Roels et al., 1976; 1978; 1980 Brunekreef, 1984	Antwerp, Belgium	10-15	5.30	4.6 to 13.7
Cavalleri et al., 1981	NR	3-6, 6-11	3.65	3.3 to 4.0
Yankel et al., 1977; Walter et al., 1980; Snee, 1982.	Silver Valley, Idaho	1-10	1.70	1.0 to 2.4
Billick et al., 1979, 1980; Billick, 1983	New York	NR	2.90	NR
Hayes et al., 1994	Chicago	6 mo - 5	16.2	5.6 to 16.2
Combined estimate from Aggregate Models			4.2	3.3 to 5.2
Estimate from Disaggregate Model			5	NR
Estimate from IEUBK Models			4.5	3.7 – 5.3

a μg/dL per μg/m^3

NR= Not reported

Table 3. Association Between Ambient Air Lead and the Expected Percent of One and Two Year Old Children Equal to or Above 10 µg/dL Blood Lead.

	GM = 3.14 GSD = 2.1	**GM = 3.14** GSD = 1.8	GM = 2.5 GSD = 1.8
Average Air Lead Concentration ($\mu g/m^3$)	Percent ≥ 10 µg/dL	Percent ≥ 10 µg/dL	**Percent ≥ 10 µg/dL**
0.055*	5.9	2.4	1.0
0.10	6.9	3.0	1.3
0.25	10.6	5.7	3.0
0.50	17.6	12.0	8.0
1.00	32.2	28.1	22.9
1.50	45.6	44.4	39.6

* National average air lead concentration during the period of data collection of NHANES III, Phase 2. Calculation assumes that baseline non-air sources of lead exposure including paint, household dust, soil, pottery, and tap water are constant. GM = 3.14 and GSD = 2.1 are taken from NHANES III, Phase2, and represent the blood lead distribution for children ages one and two (Pirkle et al., 1998).