State of California
AIR RESOURCES BOARD

Staff Report: Initial Statement of Reasons for Proposed Rulemaking

Public Hearing to Consider the Adoption of a Regulatory Amendment Identifying Cadmium as a Toxic Air Contaminant

Agenda Item No.: 87-2-1
Scheduled for Consideration: January 22, 1987
Release Date: December 5, 1986

("ARB" or the "Board") and DHS are required to give priority to the evaluation and regulation of substances based on factors related to the risk of harm to public health, amount or potential amount of emissions, manner of usage of the substance in California, persistence in the atmosphere, and ambient concentrations in the community (Health and Safety Code § 39660). Cadmium appears on the priority list based on its evaluation using the above factors. The current priority list, approved by the Board on February 26, 1987, is appended as Attachment 1.

The Scientific Review Panel (SRP) was established to advise the Board in its evaluation of the health effects toxicity of substances (Health and Safety Code § 39670). The SRP reviewed the report on cadmium and found it to be without serious deficiency. The findings of the SRP are on pages 12 and 13 of the Initial Statement of Reasons.

A notice of public hearing on whether cadmium should be identified as a toxic air contaminant with no identified threshold was issued on December 5, 1986, and a public hearing was held on January 23, 1987 pursuant to Government Code §§ 11340 et seq. and Health and Safety Code § 39662.

The AB 1807 procedure for developing and adopting control measures to reduce toxic air contaminants is separate and distinct from the procedure for identifying substances as toxic air contaminants. After a substance is identified as a toxic air contaminant, the Executive Officer, with the participation of the air pollution control districts, will prepare a report on the need and appropriate degree of regulation for the substance (§ 39665). Sections 39665-39667 set forth the issues which the Board must consider with respect to control measures.

II. ENVIRONMENTAL AND ECONOMIC IMPACTS[1/]

The identification of cadmium as a toxic air contaminant will not result in any adverse environmental impacts. After identifying cadmium as a toxic air contaminant, the Executive Officer and the Board will evaluate the need for and appropriate degree of control measures, in accordance with the provisions of §§ 39665-39667. Potential adverse environmental impacts associated with control measures will be analyzed and addressed in the consideration of such control measures.

The identification of cadmium as a toxic air contaminant is not expected to result in any economic impacts. Economic impacts associated with any control measures which might be developed pursuant to §§ 39665-39667 will be addressed in connection with the Board's consideration of such control measures.

The Board has determined that this regulatory action creates neither costs nor savings, as defined in Government Code § 11346.5(a)(6), to any state agency or in federal funding to the state, costs or mandate to any local agency or school district whether or not reimbursable by the state pursuant to Part 7 (commencing with § 17500) of Division 4 of the Government Code, nor other nondiscretionary savings to local agencies.

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1. The staff did not consider or propose other specific regulatory alternatives to the proposed regulation because there are no alternatives to the regulation which would serve the purpose of identifying cadmium as a toxic air contaminant. In some rulemaking proceedings, Government Code § 11346.14(b) requires the consideration of a performance standard as an alternative to a proposed regulation requiring the use of specific equipment or procedures. In this case, however, the proposed regulation would not mandate the use of specific technologies or equipment or require any action by sources to reduce emissions of cadmium.

In developing the proposal, the staff considered the potential cost impact of the proposed action on private persons or businesses directly affected. The Board expects that the regulatory proposal will not cause any significant increased costs for such persons or businesses. The Board has determined that the proposed action will not have a significant adverse economic impact on small businesses.

III. STAFF RECOMMENDATION AND SUMMARY OF BOARD ACTION

Cadmium, which is known to be emitted in California, is an animal carcinogen with epidemiological evidence of carcinogenicity in humans. In its health effects evaluation, DHS concluded that in the absence of compelling evidence of a threshold, the mechanism of cadmium carcinogenesis is a nonthreshold process. The SRP found that based on available scientific information a cadmium exposure level below which carcinogenic effects are not expected to occur cannot be identified. Therefore, the ARB staff recommended that cadmium be listed as a toxic air contaminant for which there is not sufficient available scientific evidence to support the identification of an exposure level below which no significant adverse health effects are anticipated.

At a public hearing on January 23, 1987, the Board by Resolution 87-9 approved an amendment to Title 17, California Administrative Code, § 93000 which added cadmium (metallic cadmium and cadmium compounds) to the list of toxic air contaminants set forth therein. A threshold level below which no significant adverse health effects are anticipated from exposure to cadmium was not identified. At the Board hearing the Board approved the proposed amendment with modifications to the originally proposed text.

The originally proposed text listed "cadmium" as a toxic air contaminant. Airborne cadmium is generally understood to mean both airborne metallic cadmium and airborne cadmium compounds. Further, the analysis in the Staff Report to the Board applies to both metallic cadmium and cadmium compounds. Staff believes that the term cadmium refers to both forms of cadmium, but decided that the listing of cadmium in the regulation should be made explicit in order to avoid confusion as to the scope of the Board's action. The Board approved the staff's modified recommendation to include "metallic cadmium and cadmium compounds" in parenthesis after "cadmium."

In accordance with § 11346.8 of the Government Code, the Board directed the Executive Officer to adopt the approved regulatory amendments after making them available to the public for 15 days for comment regarding the changes to the regulation as originally proposed. On February 9, 1987, the 15-day availability notice, the text of the regulation as originally proposed, with the proposed change clearly indicated, and the Board's resolution were sent to all persons required by Title 1, California Administrative Code, § 44. No comments were received regarding the changes to the regulation.

IV. SUMMARY OF COMMENTS AND AGENCY RESPONSES

At the January 23, 1987 public hearing concerning the proposed regulation listing cadmium as a toxic air contaminant, oral comments were presented by a representative of the Cadmium Council, Inc. In addition, the Board received written comments regarding the proposed action from McKenna, Conner and Cuneo (on behalf of Miller Brewing) ("McKenna"), the Cadmium Council, Inc., the California Council for Environmental and Economic Balance

("CCEEB"), and the Central City South Association ("CCSA"). A summary of those comments and the agency's responses follows. Responses to comments relating to health effects have been prepared in cooperation with DHS staff.

1. Comment: The commentor supports the proposal to identify cadmium as a toxic air contaminant having no identifiable threshold. Commentor agrees with DHS' conclusion that there was a difference in health effects between ingested and inhaled cadmium. Commentor refers to the potential increase in emissions of cadmium to the atmosphere from the operation of a proposed waste-to-energy facility. (McKenna)

Agency Response: The Staff Report includes discussions of inhalation and non-inhalation health effects (pp. 19-20; pp. 167-173) and of the potential for cadmium emissions from proposed waste-to-energy facilities (pp. 55-56).

2. Comment: The commentor asserts that the Technical Support Document may understate the potential problems associated with cadmium emissions from municipal waste incinerators because no total statewide estimate of potential emissions for this source category is given and a relatively small (1,000 tons per day) incinerator was used as the basis to calculate the emission rate given in the report. The commentor urges the ARB to specifically consider municipal waste incinerators when developing control measures. (CCSA)

Agency Response: The Staff Report included cadmium emission estimates for source categories currently emitting cadmium. Because no municipal waste incinerators were operating in the state at the time the Staff Report was released, the report did not include a statewide emission estimate for this source category.

The municipal waste incinerator (North Counties) for which cadmium emissions were calculated is the largest capacity facility of this type in the state which has received a permit to operate (an emission permit). The largest capacity municipal incinerators which have been proposed are about twice the capacity of the North Counties facility; however, these larger incinerators have not received permits to operate. Because these larger facilities have not received permits, the parameters used as a basis to calculate cadmium emissions are subject to change. For this reason, we chose to provide emission estimates for the North Counties incinerator.

During the control measure phase of the AB 1807 process, the ARB will update its information on sources of cadmium emissions, including municipal waste incinerators, and will evaluate the need for control measures for each category of sources.

3. Comment: The commentor recommends that cadmium be identified as a toxic air contaminant with a risk of 0-12 cases per million persons exposed over a 70-year lifetime to an average of one nanogram of cadmium per cubic meter of air. The commentor offers the following arguments to support this recommendation.

a. "This recommendation reflects the Environmental Protection Agency's June 1985 final cadmium risk assessment conclusion that 'an empirical threshold model that is also consistent with the observed data gives a unit risk estimate of zero' (see the Council's August 20, 1986 comments)."

b. The SRP found on October 30, 1986 that "The available data are also consistent with the possibility that the risk of lung cancer from current ambient exposures to cadmium in California may be 'vanishingly small.'"

c. The commentor states, "... Health and Safety Code Sections 39660(c) and 39662(c) address a threshold of significant adverse effects. We would suggest that DHS' characterization of 'vanishingly small' risks should not be regarded as 'significant.'" The commentor further states, "The range of risk of 0-12 is 'the more health protective interpretation consistent with the data,' and fully satisfies Health and Safety Code requirements."

d. Adoption of a 0-12 range would heighten public awareness of the uncertainties regarding low-dose risk extrapolations and would give ARB and the air pollution control districts more freedom to adopt reasonable emissions control rules. (CCEEB)

Agency Response: The range of risk of 2-12 excess cancers per million persons exposed over a 70-year lifetime to an average of one nanogram of cadmium per cubic meter of air is based on the available scientific evidence and the DHS staff's and the SRP's interpretation of that evidence, all of which were summarized in the Staff Report and at the Board hearing. The following is a summary discussion of that information, corresponding to each of the commentor's points.

a. The commentor has taken the quotation from the EPA report out of context, and has intimated that the EPA's hypothetical discussion of threshold model data fit is an "EPA conclusion" which supports the commentor's recommendation of a threshold model. The CCEEB's August 20, 1986 letter (pp. 375-6 of the Staff Report) referred to by the commentor includes the statement, "In its 1985 'Updated Mutagenicity and Carcinogenicity Assessment of Cadmium,' EPA objectively presented the possibility that a threshold might exist, and indicated that under such a threshold assumption, a constant lifetime exposure to 10 micrograms per cubic meter would produce zero risk."

These assertions appear to be based on an incomplete reading of the EPA document or on a misunderstanding of its content.

In the EPA document to which the commentor referred, the EPA discussed the fit of an "empirical" threshold model to the data only to make the point that a threshold model could be consistent with the data, and that criteria other than "fit to the data" should therefore be used to choose a model. The following text is from the summary of the EPA document (from which the commentor quoted) and from the body of the EPA document (which provides a context for the earlier mention of threshold model consistency with the data).

> "Based on respiratory cancer rates from the Thun et al. (1985) study of cadmium smelter workers, and using a linear model that is consistent with the data, the upper-bound incremental cancer risk from lifetime exposure to 1 ug/m^3 of cadmium in the air is estimated to be 1.8 x 10^{-3}.
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> "The 95% confidence bound on this estimate, which takes into account only the statistical variability of the cancer rates, gives a range of 3.5 x 10^{-3} to 1.7 x 10^{-4}. However, this range does not account for possible deviations of the true (unknown) model from the linear model or of actual exposure from estimated exposure. For example, an empirical threshold model that is also consistent with the observed data give a unit risk estimate of zero. Even with the uncertainties surrounding the estimate based on human data, it is felt that this estimate is more reliable for environmentally exposed humans than the estimate based on animal data." (pp.8-9)
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> "To show how a different assumed model could influence risk estimates, the following ad-hoc 'threshold' model can be considered. This model is not based on any biological information. It simply uses the highest dose group with no observable statistically elevated risk as the threshold and assumes linearity in accumulated dose beyond that point.
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> * * * * *
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> "We note that both the 'threshold' and linear models give an adequate fit to the data. As a result, arguments other than purely statistical must be used to select the appropriate model." (p. 159)

With respect to the EPA's comment that "... arguments other than purely statistical must be used to select the appropriate model," DHS presented in the Staff Report (p. 250) the rationale it used as a basis to reject a threshold model. DHS cited the lack of evidence for the existence of a carcinogenic threshold. After evaluating the scientific evidence concerning the mechanism of cadmium carcinogenesis (as related to the existence of a carcinogenic threshold), DHS staff concluded that, because of the absence of compelling evidence of a threshold-mediated carcinogenicity mechanism, cadmium's carcinogenicity should be treated as a non-threshold phenomenon. This is a health-protective (health conservative) interpretation of the evidence. DHS made this point previously (Staff Report, pp. 488-490, 492) in response to CCEEB's similar comment (Staff Report, p. 375) on the draft staff report. The SRP's findings (Staff Report. pp. 11-13) coincide with DHS' health-protective conclusion regarding the lack of evidence to support identification of a threshold. The SRP, after review of the report, in part found that:

> "4. Based on available scientific information, a cadmium exposure level below which carcinogenic effects are not expected to occur cannot be identified."

b. The commentor referred to a statement included in the SRP's findings of October 30, 1986 which concerned the possibility of a near-zero ("vanishingly small") lower range of risk. It should be noted that these findings also included the following (SRP findings, Staff Report, P. 12).

> "Based on an interpretation of available scientific evidence by DHS, the range of lifetime excess cancer risk from exposure to one ng/m^3 of atmospheric cadmium based on the best estimate of risk and the upper 95 percent confidence limit is estimated to be 2 to 12 cases per million people

exposed; it is unlikely that the risk will exceed this range, and may be lower."

Thus, the SRP concurred with the health-protective interpretation of the scientific data presented by DHS staff in the Staff Report. The note in the SRP findings was intended to indicate that other, less health-protective interpretations of the evidence are possible.

This was explained as follows by Dr. Thomas Mack of the SRP at the Board hearing (January 23, 1987 Transcript, pp. 12-14).

> "Again, let me remind you that the goal of this particular exercise is to try and make a best estimate of what the per dose cost in human life would be for human individual cases of cancer and to make a best estimate of what the most pessimistic boundary of the reasonable range of estimates would present, and that's where the two figures, two and 12, presented themselves. In this case because we were dealing with extrapolation from humans rather than from animals, some of the problems with the usual process did not occur, but the principal problem, namely, trying to decide whether or not that line is really a straight line that goes all the way down was just as great.
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> "We recognize that we have a large room for error in these estimations. However, we have to do the best we can and the best judgment we agreed that the Health Department had provided us with.
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> "We did, however, have enough of a discussion to warrant a paragraph in the form of a note, and the note raises really one issue. The one issue is the possibility that what we refer to as dose rate might be pertinent in cadmium. I, myself, think it might well be pertinent in a number of the compounds we discussed before. The point of the note is that with cadmium it's possible that a relatively intensive exposure over the course of a shorter period of time might not be the same as that same exposure if divided out over a much longer period of time.
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> "We have no basis for saying it would or would not be, but like threshold it might be reasoned to think that our estimates are conservative and the point of the note was to say that.

"The crucial sentence in the note for your purposes, however, is the following. The available data are also consistent with the possibility that the risk of lung cancer from current ambient exposures to cadmium might be vanishingly small. That simply means that it's possible there is no risk, but our best estimate of the risk is still two per million persons exposed to one nanogram per cubic meter over their lives.

"For these reasons, we agree with the ARB staff recommendation that cadmium be listed as a toxic air contaminant. We agree that there's not sufficient available scientific evidence to support the designation of an exposure level below which carcinogenic effects would not have some probability of occurring."

c. We have responded above to the commentor's view that a risk of 0-12 is more consistent with the data than a risk of 2-12. Clearly, a range of risk of 2-12 (which predicts a greater endangerment of public health) is also more health protective than a range of risk of 0-12.

The commentor refers to two sections of the Health and Safety Code. Health and Safety Code § 39660(c) states:

"... The evaluation shall also contain an estimate of the levels of exposure which may cause or contribute to adverse health effects and, in the case where there is no threshold of significant adverse health effects, the range of risk to humans resulting from current or anticipated exposure."

Section 39662(c) states:

"If a substance is determined to be a toxic air contaminant, the regulation shall specify a threshold exposure level, if any, below which no significant adverse health effects are anticipated."

The commentor has (incorrectly) attributed use of the phrase "vanishingly small" to DHS when in fact the phrase was used in a note to the SRP's findings, wherein the SRP nevertheless found, based on DHS' interpretation of the data, that the best estimate of the range of excess cancer risk from one nanogram per cubic meter of atmospheric cadmium is 2-12 cases per million people exposed. Also, the commentor has evidently misunderstood the Health and Safety Code sections to which he referred. The commentor suggests that §§ 39660(c) and 39662(c) include a reference to "significant risk" of adverse health effects, when in fact the sections refer to "a range of risk of significant adverse health effects." Clearly, cancer is a significant adverse health effect.

d. The recommendation of a range of risk was based on DHS staff's interpretation of the available scientific evidence concerning the potential adverse health effects of cadmium and the SRP's review of and concurrence with those interpretations. The adoption of emission control rules (i.e., control measures) is not within the scope of this rulemaking action, which concerns the listing of cadmium as a toxic air contaminant only; issues related to control of cadmium emissions will be taken into account in the future development and consideration of control measures for cadmium, pursuant to Health and Safety Code §§ 39665-39667.

4. Comment: The commentor recommends that the Board delay its decision to identify cadmium as a toxic air contaminant having no identifiable threshold. This recommendation is based on the commentor's belief that it will be possible to make a more accurate judgment concerning cadmium carcinogenicity and evidence of a threshold when more definitive data on the role of cadmium exposure in producing lung cancer becomes available (within six months to a year.)

The commentor states that in a reanalysis of Globe smelter worker health data (which was the basis of the study by Thun used by DHS in their quantitative risk assessment), Dr. Steven Lamm concluded that the increase in incidence of lung cancer attributed by Thun to cadmium exposure may have been due primarily to other factors: concurrent arsenic exposure, differences in smoking histories (between the exposed workers and the control group), methodological perculiarities, or to cadmium exposures that were markedly greater for workers hired prior to 1940. Dr. Lamm is quoted by the commentor as saying, "Clearer documentation of their specific jobs and the exposure on those jobs is necessary to resolve many of the issues in this study."

The commentor submitted with his comments a draft copy of Dr. Lamm's unpublished paper titled "Analysis of Mortality Studies of Globe, Colorado Cadmium Workers," which was cited in his comments. (Cadmium Council)

Agency Response: The commentor has suggested delaying action on the identification of cadmium as a toxic air contaminant with no identifiable threshold because an alternative interpretation has been offered for the Globe smelter data. The Globe smelter study was the study used by DHS in its quantitative risk assessment. The commentor does not address other evidence discussed in the Staff Report which in the opinion of DHS staff and the SRP also establishes the carcinogenicity of cadmium. A review of this other evidence is contained in § VII.J. of the Staff Report (pp. 203-249) and includes animal exposure studies and supporting evidence from other human exposure studies (both of the Globe workers and of other exposed groups).

This review is summarized on page 229 of the Staff Report:

> "The evidence from epidemiology strongly supports the hypothesis that cadmium exposure is associated with an increased risk of respiratory cancer. Since this site has also been implicated in animal bioassays of carcinogenicity, DHS staff members concluded that there is a high probability that the observed association is not spurious and that an inference of causality is justified."

After review of the available studies of human exposure to cadmium, DHS staff concluded that the evidence is suggestive of an association between cadmium exposure and renal cancer and is inconclusive concerning an association between prostatic cancer and bladder cancer and cadmium exposure (due to limitations in the power of the studies to statistically detect an effect). (Staff Report, pp. 220-225)

Dr. Lamm's conclusion that other factors may be the primary cause of the increase in incidence in lung cancer attributed by Thun to cadmium exposure is discussed in the Staff Report: the possible effects of arsenic exposure, pp. 240-243; smoking, pp. 233-239; methodological peculiarities, pp. 229-232. DHS staff offered this summary of its consideration of these factors:

> "Finally, to summarize the DHS staff's findings with regard to the study by Thun et al.: The standardized mortality ratio (SMR) of 2.3 in those with more than two years of cadmium exposure and the dose-response relationship are unlikely to be explained by chance, by bias, or by confounding from smoking and/or arsenic exposure. The staff of DHS concludes that the excess of lung cancer deaths in the study by Thun et al. is best explained by exposure to high levels of cadmium. The DHS staff further concludes that while other confirmatory studies are desirable this study constitutes strong evidence of human carcinogenicity." (Staff Report, pp. 243-246)

The last issue raised by the commentor concerns the accuracy of cadmium exposure estimates for workers at the Globe smelter. This issue is discussed in the Staff Report (p. 265). DHS staff describes (1) adjustments made to area sample results to estimate personal exposure, and (2) adjustment of personal exposure estimates to account for respirator effectiveness. The resulting uncertainties in the exposure estimates are discussed, including the direction of possible biases. DHS staff concluded that there was no way to quantify the uncertainties. Although the accuracy of the exposure measurements has bearing on the range of risk derived by DHS, the exact level of exposure of the Globe workers was not the basis upon which DHS made its determination of causality between cadmium exposure and respiratory cancer. (See discussion of § VII.J. of the Staff Report, above.)

Dr. Thomas Mack, representing the SRP at the Board hearing, made the following comments concerning Dr. Yoder's allegation that the Thun study is inadequate evidence of cadmium's respiratory carcinogenicity (Transcript, pp. 27-29):

> "Well, I think we would like to see it [Lamm's paper] published and we'd certainly like to see a complete presentation before we'd want to incorporate it into our considerations. There do seem to be interesting questions. As you pointed out, interesting questions will come up about each of these compounds on a monthly or every six-month basis, and I think there's no way that it would be prevented that people will reevaluate the questions from time to time on each compound.
>
> "I guess I would point one thing out, and that is if we were to assume that this information was of better quality than everything we've considered and that there was no good evidence from the human studies that cadmium was a carcinogen, we would then be forced to fall back upon the animal studies. If that's the case, the extrapolation from the animal studies actually produced a higher estimate of what individual Californians would suffer from over the next years."

With respect to the commentor's specific request for a delay in the Board's decision on whether cadmium is a toxic air contaminant, there is a statutory provision for review and consideration of new information after a toxic air contaminant determination has been made. Specifically § 39662(e) of the Health and Safety Code provides that:

> "Any person may petition the state board to review a determination made pursuant to this section. The petition shall specify the additional scientific evidence regarding the health effects of a substance that was not available at the time the original determination was made and any other evidence which would justify a revised determination."

ATTACHMENT 1

ATE OF CALIFORNIA

GEORGE DEUKMEJIAN, Governor

IR RESOURCES BOARD
02 Q STREET
D. BOX 2815
TO, CA 95812

March 17, 1987

Dear Sir or Madam:

This letter is to apprise you of changes in our list of substances being considered as part of our toxic air contaminant program. In January, 1986, we circulated for review and comment a draft list with three categories of substances proposed for review as possible toxic air contaminants. Based on comments received, we revised that list to clarify the meaning of the categories and to reflect the identification of several additional substances as toxic air contaminants. The Air Resources Board approved the revised list at its meeting on February 26, 1987.

The enclosed list, "Status of Toxic Air Contaminant Identification" includes three categories. Category I contains those substances which the Board has already identified as toxic air contaminants (7 substances). Category II contains those substances which the staff has nominated for review (24 substances in all). Category II-A includes substances which are already under review or have been scheduled for review (9 substances); Category II-B includes substances which are expected to be scheduled for review within three years (15 substances). It will be necessary to develop information on public exposure in California to some substances in this category (II-B) before pursuing review.

Category III contains substances which are of possible concern as toxic air contaminants, but for which evidence of adverse health effects is not yet sufficient to support review and identification (19 substances). The staff will not seek to review and identify these substances as toxic air contaminants unless and until stronger health evidence indicates that they pose a public health risk. The presence of substances in this category reflects concern about the large volumes of these compounds used in the state and the possibility of adverse health effects.

If you have questions about the list, or about the inclusion of substances in any particular category, please call Peter D. Venturini, Chief, Stationary Source Division, at (916) 445-0650.

Sincerely,

Harmon Wong-Woo
Deputy Executive Officer

cc: Kenneth Kizer, DHS
Clare Berryhill, DFA

Enclosure

State of California

AIR RESOURCES BOARD

March 1987

STATUS OF TOXIC AIR CONTAMINANT IDENTIFICATION

I. Substances identified as toxic air contaminants by the Air Resources Board, pursuant to the provisions of AB 1807.

Asbestos, Benzene, Cadmium, Chlorinated Dioxins and Dibenzofurans (15 species), Chromium(VI), Ethylene Dibromide, Ethylene Dichloride

II. Substances currently under review for identification as toxic air contaminants, scheduled for review, or nominated for review, but not yet scheduled. It will be necessary to develop information on exposure in California to some substances in this category before pursuing review.

A. (Substances already in the review process.) Carbon Tetrachloride, Chloroform, Ethylene Oxide, Inorganic Arsenic, Methylene Chloride, Nickel, Perchloroethylene, Trichloroethylene, Vinyl Chloride

B. (Substances not yet under review.) Acetaldehyde, Acrylonitrile, Beryllium, 1,3-Butadiene, Coke-oven Emissions, Dialkyl-nitrosamines, 1,4-Dioxane, Epichlorohydrin, Formaldehyde, Inorganic Lead, Mercury, N-Nitrosomorpholine, PAHs, PCBs, Radionuclides

III. Substances of possible concern for which health effects information is limited or not yet sufficient to support review which could lead to identification as toxic air contaminants. Substances in this category are produced and emitted to the air in quantities which might make them of concern at some time in the future if health effects information is strong enough to support review.

Acrolein, Allyl Chloride, Benzyl Chloride, Chlorobenzene, Chlorophenols, Chloroprene, Cresols, p-Dichlorobenzene, Hexachlorocyclopentadiene, Maleic Anhydride, Manganese, Methyl Bromide, Methyl Chloroform, Nitrobenzene, Phenol, Phosgene, Propylene Oxide, Vinylidene Chloride, Xylenes

INTRODUCTION AND RECOMMENDATION

State law defines a toxic air contaminant as an air pollutant which the Air Resources Board or the Department of Food and Agriculture finds "may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health". The staffs of the Air Resources Board and Department of Health Services have reviewed the available scientific evidence on the presence of cadmium in the atmosphere of California and its potential adverse effect on public health. Based on the conclusion of the Department of Health Services staff that cadmium meets this definition, the staff of the Air Resources Board recommends that cadmium be identified by the Board as a toxic air contaminant. The ARB staff is unable to, based on available scientific information, identify a level below which adverse health effects are not expected to occur, and therefore is unable to recommend a threshold level.

Cadmium was chosen for evaluation because: it had been identified by the International Agency for Research on Cancer (IARC) as an animal carcinogen with epidemiological evidence of carcinogenicity in humans; its presence in the atmosphere had been documented; it is emitted from many sources in the state, and may be emitted in increased amounts in the future.

SOURCES OF CADMIUM

Cadmium is emitted from both stationary and mobile sources. Stationary sources which are likely to emit cadmium include secondary smelters, cement manufacturing plants, cadmium electroplating facilities, plants burning oil or coal, and sewage sludge incinerators. Mobile sources which emit cadmium include gasoline and diesel vehicles and particles resulting from tire wear. An emissions inventory compiled by ARB staff indicates that a total of from 16 to 18 tons/year of cadmium are emitted in California; stationary sources account for eighty percent or more of cadmium emissions. Cadmium emissions from fossil fuel combustion and vehicles are projected to increase due to expected increase in fuel use.

EXPOSURE TO ATMOSPHERIC CADMIUM

General population exposure to atmospheric cadmium was estimated using data on cadmium concentrations for the first six months of 1985 in various locations in the state. We believe that these averages are reasonably representative of annual average concentrations. We estimate that 10 million people are exposed to an average cadmium concentration of 1.0 to 2.5 ng/m^3, and that one million people are exposed to an average cadmium concentration of 1.8 to 5.6 ng/m^3. Neither size distribution nor the compound forms of cadmium were determined in the ARB's measurements. Work done by others on the size distribution of atmospheric cadmium indicates that atmospheric cadmium occurs principally on the surface of respirable particles (those less than 2.5 micrometers (um) in diameter).

Exposure to atmospheric cadmium near sources is expected to be higher than general population exposure. To estimate exposure to atmospheric cadmium near sources, ARB staff used on air quality model to calculate the ambient concentration of atmospheric cadmium in the South Coast Air Basin due to emissions from three secondary copper smelters. These emissions were estimated to result in annual average exposure to atmospheric cadmium of up to 40 ng/m^3 for a population of 57,000 and 14 up to ng/m^3 for a population of 285,000.

HEALTH EFFECTS OF CADMIUM

Concentrations of cadmium measured in the atmosphere are much lower than those which are associated with chronic adverse health effects in occupational settings or which have produced acute effects in animal experiments. Because of this, and because cadmium is thought to exhibit a threshold effect for non-cancer health effects, adverse health effects other than cancer are not expected to occur due to inhalation of cadmium at current or anticipated atmospheric concentrations.

Two separate cancer risk assessments were developed, both of which assumed that cadmium carcinogenicity operates through a nonthreshold mechanism. One was based on a mortality study of workers in a cadmium production plant; for exposure to 1 ng/m^3 cadmium, a best estimate of excess lifetime cancer risk of 2 per million, and an upper 95% confidence limit (UCL) of 12 per million, were derived. The other cancer risk assessment was based on rat lung tumor incidence; risk estimates derived from these studies were higher than the human-based estimates. The DHS staff has determined that the possible roles of chance, bias and confounding factors in distorting the true dose-response relationship in the occupational study were likely to have been small. Because the human data for exposure and for response were not found to have any major deficiencies, and because a conservative linear extrapolation was used, DHS staff recommends reliance on the human-based risk assessment.

RISK DUE TO ATMOSPHERIC CADMIUM

The hazard posed by atmospheric cadmium to residents of California was estimated by applying the unit risk estimate to cadmium concentrations measured in the state. The upper-bound excess lifetime cancer risk from estimated atmospheric concentrations of cadmium in California has been estimated to be 30 per million. For people near emission sources of cadmium, the upper-bound estimated excess lifetime cancer risk from 24-hour-per-day exposure to an average of 40 ng/m^3 of cadmium is 480 per million persons exposed. These are health-conservative estimates; the actual risks may lie below these values.

Exposures to cadmium via routes other than inhalation of ambient air were not considered in the risk assessment. The major nonoccupational exposures to cadmium are through food and smoking. While the bulk of human intake is via food ingestion, this route of exposure has not been associated with an increased risk of cancer either in humans or in experimental animals.

DHS staff emphasizes that the risk estimates derived in conducting a risk assessment are not exact predictions, but rather represent best estimates based on current scientific knowledge and methods.

Based on the findings of cadmium-induced carcinogenicity and the results of the risk assessment, DHS staff finds that ambient cadmium is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

SUMMARY OF ENVIRONMENTAL IMPACTS OF THE IDENTIFICATION OF CADMIUM AS A TOXIC AIR CONTAMINANT

The identification of cadmium as a toxic air contaminant is not in itself expected to result in any environmental effects. The identification of cadmium as a toxic air contaminant by the Board may result in the Board and air pollution control districts adopting toxic control measures in accordance with the provisions of state law. Any such toxic control measures may result in reduced emissions of cadmium to the atmosphere, resulting in reduced ambient concentrations, concurrently reducing the health risk due to cadmium. Therefore, the identification of cadmium as a toxic air contaminant may ultimately result in environmental benefits. Environmental impacts identified with respect to specific control measures will be included in the consideration of such control measures pursuant to Health and Safety Code Sections 39665 and 39666.

Amend Title 17, California Administrative Code, Section 93000 to read as follows:

93000. Substances Identified As Toxic Air Contaminants. Each substance identified in this section has been determined by the state board to be a toxic air contaminant as defined in Health and Safety Code Section 39655. If the state board has found there to be a threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, that level is specified as the threshold determination. If the board has found there to be no threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, determination of "no threshold" is specified. If the board has found that there is not sufficient available scientific evidence to support the identification of a threshold exposure level, the "Threshold" column specifies "None identified."

Substance	Threshold
Benzene (C_6H_6)	None identified
Ethylene Dibromide ($BrCH_2CH_2Br$; 1,2-dibromoethane)	None identified
Ethylene Dichloride ($ClCH_2CH_2Cl$; 1,2-dichloroethane)	None identified
Hexavalent Chromium, Cr(VI)	None identified
Asbestos [asbestiform varieties of serpentine (chrysotile) riebeckite (crocidolite) cummingtonite-grunerite (amosite), tremolite, actinolite, and anthophyllite]*	None identified
Dibenzo-p-dioxins and Dibenzofurans chlorinated in the 2,3,7 and 8 positions and containing 4,5,6 or 7 chlorine atoms*	None identified
Cadmium	None identified

NOTE: Authority cited: Sections 39600, 39601 and 39662, Health and Safety Code. Reference: Sections 39650, 39660, 39661 and 39662, Health and Safety Code.

*Note: Compounds identified by an asterisk have been identified as toxic air contaminants by the Air Resources Board but not yet approved by the Office of Administrative Law.

Notice of Public Availability of Modified Text

Public Hearing to Consider the Adoption of a Regulatory Amendment Identifying Metallic Cadmium and Cadmium Compounds as Toxic Air Contaminants

Public Hearing Date: January 23, 1987
Public Availability Date: February 9, 1987

At a January 23, 1987 public hearing, the Air Resources Board ("ARB" or the "Board") considered the adoption of a proposed regulatory amendment to list cadmium as a toxic air contaminant for which there is not sufficient available scientific evidence to support the identification of a threshold exposure level. At the hearing the Board approved the proposed amendment with modifications to the originally proposed text. The modification to the originally proposed text is described below. Attached is a copy of Board Resolution 87-9 approving the proposed amendments with modifications. Attached to the resolution is the approved language, with additions to the original proposal shown by double underlining. The unchanged portion of the original proposal is shown by a single underline.

The originally proposed text listed "cadmium" as a toxic air contaminant. Airborne cadmium is generally understood to mean both airborne metallic cadmium and airborne cadmium compounds. Further, the analysis in the staff report to the Air Resources Board applies to both metallic cadmium and cadmium compounds. Staff believes that the term cadmium refers to both forms of cadmium, but decided that the listing of cadmium in the regulation should be made explicit in order to avoid any confusion as to the scope of the Board's action. The Board approved the staff's modified recommendation to include "metallic cadmium and cadmium compounds" in parentheses after "cadmium."

In accordance with Section 11346.8 of the Government Code, the Board directed the Executive Officer to adopt the approved regulatory amendments after making them available to the public for comment regarding the changes to the regulation as originally proposed for a period of at least 15 days provided that the Executive Officer shall consider written comments received and make minor modifications to the language as appropriate in response to comments, and shall present the regulations to the Board for further consideration if he determines that this is warranted in light of the written comments received. Only comments concerning the modified definition of cadmium will be considered during this comment period.

Comments must be submitted to the Board Secretary, Air Resources Board, P. O. Box 2815, Sacramento, CA 95812, no later than March 2, 1987 for consideration by the Executive Officer.

State of California
AIR RESOURCES BOARD

Resolution 87-9

January 23, 1987

Agenda Item No.: 87-2-1

WHEREAS, Sections 39600 and 39601 of the Health and Safety Code authorize the Air Resources Board (the "Board") to do such acts and to adopt such regulations as may be necessary for the proper execution of the powers and duties granted to, and imposed upon, the Board by law;

WHEREAS, Chapter 3.5 (commencing with Section 39650) of Part 2 of Division 26 of the Health and Safety Code establishes procedures for the identification of toxic air contaminants by the Board;

WHEREAS, Section 39655 of the Health and Safety Code defines a "toxic air contaminant" as an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health;

WHEREAS, Section 39662 of the Health and Safety Code directs the Board to list, by regulation, substances determined to be toxic air contaminants, and to specify for each substance listed a threshold exposure level, if any, below which no significant adverse health effects are anticipated;

WHEREAS, in California, cadmium (metallic cadmium and cadmium compounds, hereinafter "cadmium") is emitted from certain industrial processes such as secondary smelting operations, cement manufacturing, and combustion of fossil fuels, and has been measured in the atmosphere;

WHEREAS, pursuant to the request of the Board, the Department of Health Services (DHS) evaluated the health effects of cadmium in accordance with Section 39660 of the Health and Safety Code;

WHEREAS, DHS concluded in its evaluation that cadmium is an animal carcinogen with epidemiological evidence of carcinogenicity in humans; cadmium should be treated as a substance without a carcinogenic threshold; health effects other than cancer are not expected to occur at existing or expected ambient levels of cadmium; and the maximum excess lifetime cancer risk from cadmium exposure is estimated to range from 2 to 12 cases per million people exposed per nanogram per cubic meter;

WHEREAS, for the reasons set forth in its evaluation, DHS has concluded that, in the absence of strong positive evidence that cadmium acts only through mechanisms which ought to have a threshold, cadmium should be treated as acting without a threshold, and DHS has determined that there is not sufficient available scientific evidence at this time to support the identification of a cadmium exposure level below which carcinogenic effects would not have some probability of occurring;

WHEREAS, upon receipt of the DHS evaluation, staff of the Board prepared a report including and in consideration of the DHS evaluation and recommendations and in the form required by Section 39661 of the Health and Safety Code and, in accordance with the provisions of that section, made the report available to the public and submitted it for review to the Scientific Review Panel (SRP) established pursuant to Section 39670 of the Health and Safety Code;

WHEREAS, in accordance with Section 39661 of the Health and Safety Code, the SRP reviewed the staff report, including the scientific procedures and methods used to support the data in the report, the data itself, and the conclusions and assessments on which the report was based, considered the public comments received regarding the report, and on October 30, 1986, adopted for submittal to the Board findings which included the following:

"1. Cadmium is an animal carcinogen for which there is epidemiologic evidence of carcinogenicity in humans exposed in occupational settings.

"2. Cadmium is emitted into the air by a variety of sources in California, and its presence has been documented in the ambient air around the state.

The SRP notes that the sub-population of Californians who smoke tobacco or breathe second-hand tobacco smoke will be exposed to cadmium at concentrations several orders of magnitude greater than the exposure of the general population.

The SRP also wishes to emphasize that estimates of cumulative exposure to cadmium should account for cadmium levels in indoor air which, in the absence of tobacco smoke, may be lower than those in outdoor air.

"3. Adverse health effects other than cancer are not expected to occur at measured or predicted cadmium concentrations in the ambient air.

"4. Based on available scientific information, a cadmium exposure level below which carcinogenic effects are not expected to occur cannot be identified.

"5. Based on an interpretation of available scientific evidence by DHS, the range of lifetime excess cancer risk from exposure to 1 ng/m3 of atmospheric cadmium based on the best estimate of risk and the upper 95% confidence limit is estimated to be 2 to 12 cases per million people exposed; it is unlikely that the risk will exceed this range, and may be lower.

"NOTE: DHS has assumed that the carcinogenic dose response of cadmium is linear and that dose rate does not influence the magnitude of carcinogenic effects. These assumptions are justified by DHS on the basis of being health conservative. While the SRP understands the reasons for this, weighing of the available scientific evidence indicates that the upper bound of the low dose risk estimate obtained by using these assumptions is likely to be high. The available data are also consistent with the possibility that the risk of lung cancer from current ambient exposures to cadmium in Califonia may be vanishingly small."

WHEREAS, the SRP found the staff report to be without serious deficiency, and included in its findings the statement that it agreed that cadmium should be listed by the Air Resources Board as a toxic air contaminant, and that there is not sufficient available scientific evidence at this time to support the designation of an exposure level below which carcinogenic effects would not have some probablility of occurring;

WHEREAS, the California Environmental Quality Act and Board regulations require that no project having significant adverse environmental impacts be adopted as originally proposed if feasible alternatives or mitigation measures are available;

WHEREAS, a public hearing and other administrative proceedings have been held in accordance with provisions of Chapter 3.5 (commencing with Section 11340), Part 1, Division 3, Title 2 of the Government Code;

WHEREAS, in consideration of the staff report, including DHS' evaluation and recommendations, the available evidence, the findings of the SRP, and the written comments and public testimony it has received, the Board finds that:

Cadmium is an animal carcinogen with epidemiological evidence of carcinogenicity in humans;

Health effects other than cancer are not anticipated at existing ambient cadmium exposure levels;

There is not sufficient available scientific evidence to support the identification of a threshold exposure level for cadmium; and

Cadmium is an air pollutant which, because of its carcinogenicity, may cause or contribute to an increase in mortality and an increase in serious illness, and poses a hazard to human health; and

WHEREAS, the Board has determined, pursuant to the requirements of the California Environmental Quality Act and Board regulations, that this regulatory action will have no significant adverse impact on the environment.

NOW, THEREFORE BE IT RESOLVED, that the Board approves the proposed regulatory amendments to Section 93000, Title 17, California Administrative Code, as set forth in Attachment A.

BE IT FURTHER RESOLVED that the Board directs the Executive Officer to adopt the amendments, as set forth in Attachment A, after making it available to the public for a period of 15 days, provided that the Executive Officer shall consider such written comments regarding the changes in the regulations as originally proposed as may be submitted during this period, shall make such modifications as may be appropriate in light of the comments received, and shall present the regulations to the Board for further consideration if he determines that this is warranted.

I hereby certify that the above is a true and correct copy of Resolution 87-9, as adopted by the Air Resources Board.

Harold Holmes, Board Secretary

Attachment A

Amend Title 17, California Administrative Code, Section 93000 to read as follows:

93000. Substances Identified As Toxic Air Contaminants. Each substance identified in this section has been determined by the state board to be a toxic air contaminant as defined in Health and Safety Code Section 39655. If the state board has found there to be a threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, that level is specified as the threshold determination. If the board has found there to be no threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, determination of "no threshold" is specified. If the board has found that there is not sufficient available scientific evidence to support the identification of a threshold exposure level, the "Threshold" column specifies "None identified."

Substance	Threshold
Benzene (C_6H_6)	None identified
Ethylene Dibromide ($BrCH_2CH_2Br$; 1,2-dibromoethane)	None identified
Ethylene Dichloride ($ClCH_2CH_2Cl$; 1,2-dichloroethane)	None identified
Hexavalent Chromium, Cr(VI)	None identified
Asbestos [asbestiform varieties of serpentine (chrysotile) riebeckite (crocidolite) cummingtonite-grunerite (amosite), tremolite, actinolite, and anthophyllite]	None identified
Dibenzo-p-dioxins and Dibenzofurans chlorinated in the 2,3,7 and 8 positions and containing 4,5,6 or 7 chlorine atoms*	None identified
Cadmium (metallic cadmium and cadmium compounds)	None identified

NOTE: Authority cited: Sections 39600, 39601 and 39662, Health and Safety Code. Reference: Sections 39650, 39660, 39661 and 39662, Health and Safety Code.

*Note: Compounds identified by an asterisk have been identified as toxic air contaminants by the Air Resources Board but not yet approved by the Office of Administrative Law.

SCIENTIFIC REVIEW PANEL FINDINGS ON THE REPORT TO THE AIR RESOURCES BOARD ON CADMIUM

NOV 14 1986

Findings of the Scientific Review Panel on
the Report on Cadmium
as adopted at the Panel's October 30, 1986 meeting

In accordance with the provisions of Health and Safety Code Section 39661, the Scientific Review Panel (SRP) has reviewed the reports of the staffs of the ARB and DHS on the public exposure and biologic and health effects of cadmium, and the public comments on these reports. Based on this review, the SRP finds that the reports are without serious deficiency and further finds that:

1. Cadmium is an animal carcinogen for which there is epidemiologic evidence of carcinogenicity in humans exposed in occupational settings.

2. Cadmium is emitted into the air by a variety of sources in California, and its presence has been documented in the ambient air around the state.

   The SRP notes that the sub-population of Californians who smoke tobacco or breathe second-hand tobacco smoke will be exposed to cadmium at concentrations several orders of magnitude greater than the exposure of the general population.

   The SRP also wishes to emphasize that estimates of cumulative exposure to cadmium should account for cadmium levels in indoor air which, in the absence of tobacco smoke, may be lower than those in outdoor air.

3. Adverse health effects other than cancer are not expected to occur at measured or predicted cadmium concentrations in the ambient air.

4. Based on available scientific information, a cadmium exposure level below which carcinogenic effects are not expected to occur cannot be identified.

5. Based on an interpretation of available scientific evidence by DHS, the range of lifetime excess cancer risk from exposure to 1 ng/m3 of atmospheric cadmium based on the best estimate of risk and the upper 95% confidence limit is estimated to be 2 to 12 cases per million people exposed; it is unlikely that the risk will exceed this range, and may be lower.

NOTE: DHS has assumed that the carcinogenic dose response of cadmium is linear and that dose rate does not influence the magnitude of carcinogenic effects. These assumptions are justified by DHS on the basis of being health conservative. While the SRP understands the reasons for this, weighing of the available scientific evidence indicates that the upper bound of the low dose risk estimate obtained by using these assumptions is likely to be high. The available data are also consistent with the possibility that the risk of lung cancer from current ambient exposures to cadmium in California may be vanishingly small.

For these reasons, we agree with the ARB staff recommendation to its Board that cadmium be listed by the ARB as a toxic air contaminant, and we agree there is not sufficient available scientific evidence at this time to support the designation of an exposure level below which carcinogenic effects would not have some probability of occurring.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on October 30, 1986

Dr. Emil M. Mrak, Chairman
Scientific Review Panel

State of California
AIR RESOURCES BOARD

TECHNICAL SUPPORT DOCUMENT

PUBLIC HEARING TO CONSIDER THE ADOPTION OF A REGULATORY AMENDMENT IDENTIFYING CADMIUM AS A TOXIC AIR CONTAMINANT

Agenda Item No: 87-2-1
Scheduled for Consideration: January 22, 1987
Release Date: December 5, 1986

(This report has been reviewed by the staff of the California Air Resources Board and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Air Resources Board, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.)

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OVERVIEW AND RECOMMENDATION

I. SUMMARY AND RECOMMENDATION

The staffs of the Air Resources Board and Department of Health Services collected, assessed and integrated the available scientific evidence on the presence of cadmium in the atmosphere of California and its potential adverse effect on public health. This is a summary of the information presented in the resulting report.

State law defines a toxic air contaminant as an air pollutant which the Air Resources Board or the Department of Food and Agriculture finds "may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health". Based on the Department of Health Services staff conclusion that cadmium meets this definition, the staff of the Air Resources Board recommends that cadmium be identified by the Board as a toxic air contaminant. In making this recommendation, the ARB staff is unable to, based on available scientific information, identify a level below which adverse health effects are not anticipated to occur, and is therefore unable to recommend a threshold level.

Cadmium was chosen for evaluation because: it had been identified by the International Agency for Research on Cancer (IARC) as an animal carcinogen with epidemiological evidence of carcinogenicity in humans; its presence in the atmosphere had been documented; it is emitted from many sources in the state, and may be emitted in increased amounts in the future.

Cadmium is emitted from both stationary and mobile sources. Stationary sources which are likely to emit cadmium include secondary smelters, cement manufacturing plants, cadmium electroplating facilities, plants burning oil or coal, and sewage sludge incinerators. Mobile sources which emit cadmium include gasoline and diesel vehicles and particles resulting from tire wear. An emissions inventory compiled by ARB staff indicates that a total of from 16 to 18 tons/year of cadmium are emitted in California; stationary sources account for eighty percent or more of cadmium emissions. Cadmium emissions from fossil fuel combustion and from vehicles are projected to increase due to expected increases in fuel consumption.

Available evidence suggests that cadmium from certain combustion sources undergoes atmospheric reactions which lead to increases in the water solubility of the emitted cadmium. Other reactions such as the formation of carbonate salts from cadmium oxide may also occur.

Cadmium is removed from the atmosphere through physical processes. Both wet and dry deposition have been judged to be significant. A number of deposition models have been proposed for atmospheric particles, and a wide range of cadmium deposition velocities has been measured or predicted.

General population exposure to atmospheric cadmium was estimated using data on cadmium concentrations throughout the state determined for the first six months of 1985. Review of other data, both from California and elsewhere, suggests that concentration averages calculated using data from the first six months of the year are reasonably representative of annual averages. Data from 21 sites in six air basins were used to calculate population-weighted estimates of exposure. We estimate that 10 million people are exposed to an average cadmium concentration of between 1.0 and 2.5 ng/m^3, of which one million people are exposed to an average cadmium concentration of between 1.8 and 5.6 ng/m^3.

Neither size distribution nor the compound forms of cadmium were determined in the ARB's measurements. Work done by others on the size distribution of atmospheric cadmium indicates that atmospheric cadmium occurs principally on the surface of respirable particles (those less than 2.5 um in diameter). An average mass median diameter of 0.84 um has been calculated for atmospheric cadmium from ambient air measurements including data from an urban site in California. Although the compound forms of atmospheric cadmium have not been determined, it is known that atmospheric cadmium (in California and elsewhere) is 60-80 percent water soluble. Based on the possible compounds that could be present, we conclude that most atmospheric cadmium exists as the soluble sulfate form, with the insoluble oxide and carbonate salts comprising the rest.

To estimate exposure to atmospheric cadmium near sources, ARB staff used an air quality model to calculate the ambient concentration of atmospheric cadmium due to emissions from three secondary copper smelters in the South Coast Air Basin. These emissions were estimated to result in annual average exposure to atmospheric cadmium of up to 40 ng/m^3 for a population of 57,000 and up to 14 ng/m^3 for a population of 285,000.

Concentrations of cadmium measured in the atmosphere are much lower than those which are associated with chronic adverse health effects in occupational settings or which have produced acute effects in animal experiments. Because of this, and because cadmium is thought to exhibit a threshold effect for non-cancer health effects, we do not expect adverse health effects other than cancer to occur due to inhalation of cadmium at current or anticipated atmospheric concentrations.

Two separate cancer risk assessments were developed, both of which assumed that cadmium's carcinogenicity operates through a nonthreshold mechanism. One was based on a mortality study of workers in a cadmium production plant. A direct linear model that incorporated an adjustment for the "healthy worker effect" was fitted to the exposure data and corresponding standardized mortality ratios for respiratory cancer. For exposure to 1 ng/m^3 cadmium, a best estimate of excess lifetime cancer risk of 2 per million and an upper 95% confidence limit (UCL) of 12 per million, were derived. The other cancer risk assessment was based on rat lung tumor incidence in a 31-month inhalation bioassay of soluble cadmium chloride aerosol. Application of the multistage model to these data yielded excess lifetime cancer risk estimates of 110 per million (maximum likelihood estimate) and 180 per million (upper 95% confidence limit) for exposure to 1 ng/m^3 cadmium.

Considering the degree of uncertainty associated with extrapolation of three to four orders of magnitude, the differences between the two risk assessments are relatively small. Nevertheless, the ranges of risk provided by these two sources of data do not overlap. Because the human data for exposure and for response were not found to have any major deficiencies, and because a conservative linear extrapolation was used, DHS staff has determined that reliance on the human-based risk assessment is unlikely to underestimate risk. The range of recommended risk estimates is therefore provided by the human-based risk assessment. Therefore, the excess lifetime cancer risk used in this report is 2 to 12 per million persons exposed throughout their lives to one ng/m^3 cadmium.

Exposures to cadmium via routes other than inhalation of ambient air were not considered in this risk assessment. The major nonoccupational exposure to cadmium is through food and smoking. Intake of cadmium from food and water has been estimated at 39 ug/day. While the bulk of human intake is via ingestion, this route has not been associated with an increased risk of cancer either in humans or in experimental animals. Cadmium intake from smoking 20 cigarettes per day has been estimated at 2 to 4 ug/day. Typical daily exposure to cadmium from ambient air (not in close proximity to sources) may range from less than 0.02 ug/day to 0.10 ug/day. Occupational exposure, primarily through inhalation of airborne cadmium, is the greatest source of exposure for the cadmium worker population.

DHS staff emphasizes that the risk estimates derived in conducting a risk assessment are not exact predictions, but rather represent best estimates based on current scientific knowledge and methods. Uncertainty in this risk assessment stems from: (1) limitations in the data on which the assessment was based, (2) an extrapolation from occupational exposure levels to current ambient cadmium concentrations ranging over three to four orders of magnitude, (3) generalization from the mortality experience of adult white males in Colorado to the general population in California, (4) possible differences between occupational and nonoccupational exposures in terms of particle size distribution, and (5) potential inaccuracy and variability of ambient exposure measurements.

The DHS staff has determined that the possible roles of chance, bias and confounding factors in distorting the true dose-response relationship in the occupational study were likely to have been small. The DHS staff has also concluded that inaccuracies in the evaluation of exposure and cancer mortality in that study were likely to have been small. In addition, the net direction of these potential errors was likely to result in an overestimate of cadmium's carcinogenic potency. For these reasons, the DHS staff believes that the use of these epidemiologic data in a quantitative risk assessment is appropriate.

Furthermore, the use of human data eliminates uncertainty arising from interspecies extrapolation. Since the occupational exposures were by inhalation, there is also no extrapolation between routes of exposure. Therefore the DHS staff recommends that the range of risks for ambient exposures to cadmium be based on the best estimate and upper 95% confidence limit predicted from fitting a linear model to the human data. The hazard posed by atmospheric cadmium to residents of California was estimated by applying the risk estimate to cadmium concentrations measured in the state. Noncancer health effects are not expected to occur at concentrations of cadmium measured in populated areas of the state (long-term averages ranging from 1 to 2.5 ng/m^3). The range of estimated excess lifetime cancer risks from 24-hour-per-day exposure for a lifetime to average ambient airborne concentrations, estimated to be 1 to 2.5 ng/m^3, is 2 to 30 per million persons exposed. For people near emission sources of cadmium, the range of estimated excess lifetime cancer risks from 24-hour-per-day exposure for a lifetime to an average of 40 ng/m^3 of cadmium is 80 to 480 per million persons exposed. Based on air quality modeling of three sources of cadmium emissions, the ARB staff has estimated that approximately 57,000 people may be exposed to this concentration.

Based on the finding of cadmium-induced carcinogenicity and the result of the risk assessment, DHS staff finds that ambient cadmium is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

Based on interpretation of the available scientific evidence, ARB staff concludes that cadmium meets the definition of a toxic air contaminant, and recommends that it be listed as such. In making this recommendation, the ARB staff is unable to, based on available scientific information, identify a level below which adverse health effects are not anticipated to occur, and is therefore unable to recommend a threshold level.

II. EVALUATION OF CADMIUM

Cadmium is a rare element, making up on the average between one and two parts in ten million of the earth's crust. It is found in oil and coal at higher concentrations than are normally found in the earth's crust; it is also a contaminant of zinc and copper ores, from which it is recovered commercially.

Cadmium is used in a wide range of industrial applications. Cadmium metal is used as a component of certain alloys, as a corrosion inhibiting coating, and in certain types of electrical storage batteries. Its compounds are used as pigments and stabilizers, and in semiconductor manufacturing.

This wide usage of cadmium and its compounds, its presence as a natural contaminant in fossil fuels, other metals, and industrial raw materials, along with its high volatility relative to other metals, create a high potential for release of cadmium to the atmosphere. We estimate that between 16 and 18 tons of cadmium are emitted yearly into the State's atmosphere.

Exposure

Atmospheric cadmium concentrations were measured by the ARB in urban areas of the state during 1985. High-volume (hi-vol) samplers were used to collect 24-hour samples of particulate matter of 50 micrometer and smaller diameter; atomic absorption spectrophotometry was used to determine cadmium in the acid-soluble fraction of each sample.

Data are available for the first six months of 1985 from 21 sampling sites; these data were used to estimate exposure to atmospheric cadmium in the six areas in which the samplers were located. Data on atmospheric cadmium concentrations in California collected by the U.S. EPA in 1977, and information on seasonal variation of atmospheric cadmium in England suggest that January through June averages may be representative of annual averages.

Exposures in the San Francisco Bay Area and the South Coast air basins were calculated by interpolating site values to census tract centroids, yielding population-weighted averages. Exposures in the San Joaquin Valley, San Diego, and South Central Coast air basins, and in Sacramento County, were estimated by assuming the population in each area was exposed to the arithmetic mean concentration from sampling sites in that area. Values below the limit of detection (LOD) (1.0 ng/m^3) were found in one-half of the samples. To provide a range of average concentrations, we developed two treatments for values below the LOD which are referred to below as "zero values": a minimum average estimate was calculated assuming values below the LOD equal zero; a maximum average estimate was calculated assuming values below the LOD equal the LOD. Table I presents these exposure estimates.

TABLE I

Atmospheric Cadmium Exposure Estimates
Based on Zero Value Treatments
(Jan - June 1985 data)

Air Basin/Area	Range of Average Cadmium Concentration (ng/m^3) min.	max.	Exposed Population (millions)
San Francisco Bay Area	2.3	2.5	4.34
South Coast	1.3	1.8	10.1
San Joaquin Valley	0.7	1.3	2.31
San Diego	0.8	1.0	2.13
South Central Coast	0.5	1.0	1.12
Sacramento (County)	0.3	1.0	0.89
All areas	1.3	1.8	21

The range of exposure estimates provided by different treatment of zero values does not reflect uncertainty resulting from the small number of samples collected at each site (n = 10 to 36) or from variance in measurements. To better estimate exposure, we calculated 95 percent confidence intervals for the mean concentration at each site. These confidence intervals reflect uncertainty due to sample size and the accuracy of the measurement method, in addition to the uncertainty from values below the detection limit. The estimated 95 percent confidence intervals for the average cadmium concentrations in the areas studied are given in Table II.

TABLE II

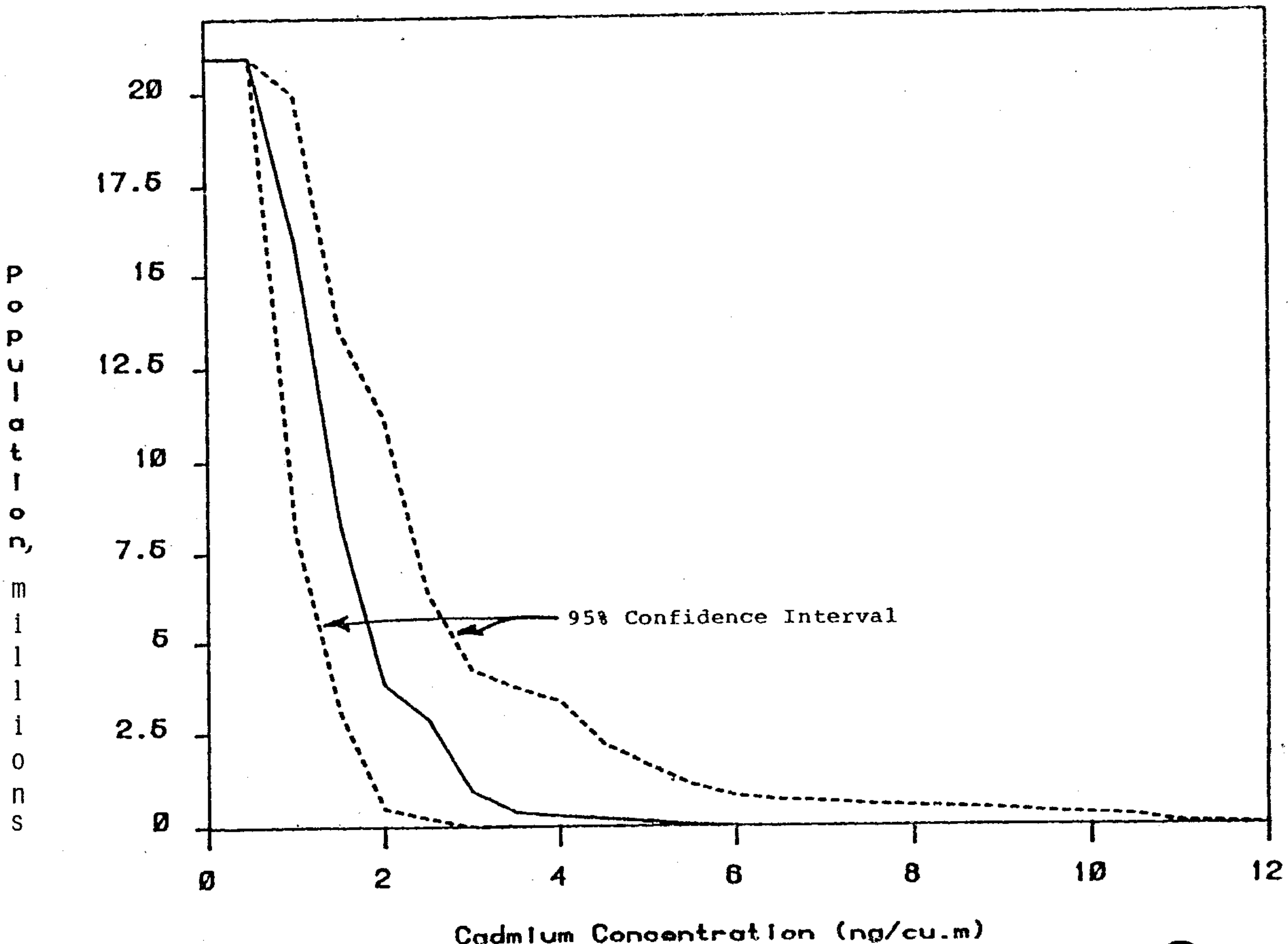
Atmospheric Cadmium Exposure Estimates
95% Confidence Intervals
(Jan - June 1985 data)

Air Basin/Area	95% Confidence Limits of Average Cadmium Concentration (ng/m^3) Lower	Upper	Population (millions)
San Francisco Bay Area	1.5	4.7	4.34
South Coast	1.0	2.3	10.1
San Joaquin Valley	0.7	1.5	2.31
San Diego	0.6	1.2	2.13
South Central Coast	0.5	1.0	1.12
Sacramento (County)	0.5	0.9	0.89
All areas	1.0	2.5	21

Comparison of the estimated ranges in average concentration shows that uncertainty from values below the detection limit is small compared to the 95% confidence intervals, except when averages are near the LOD, when both methods give comparable ranges. Figure 1 shows cadmium concentrations plotted against cumulative population for the mean and the upper and lower 95 percent confidence limits. We estimate that approximately 10 million people (50 percent of the population in the areas studied) are exposed to at least 1.5 ng/m^3 cadmium (range: 1.0 - 2.5 ng/m^3), and that approximately one million people (five percent of the population in the areas studied) are exposed to at least 3.5 ng/m^3 cadmium (range: 1.8 - 5.6 ng/m^3). The exposures discussed here are based on cadmium measured on particles 50

FIGURE 1

ESTIMATED CUMULATIVE POPULATION EXPOSURE TO CADMIUM



micrometers (um) and smaller in diameter; the fraction of cadmium on respirable particles (less than 2.5 um diameter) was not determined. The size distribution of cadmium on atmospheric particles has been found by others to be bimodal; the larger peak is seen at 0.3 - 1 um, with a smaller peak at 3 - 10 um. This tendency is observed among studies which differed in sampling location (industrial, urban, and remote/background), year (1965 - 1979), and measurement method. Milford and Davidson (1985) calculated an average particulate cadmium mass median diameter of 0.84 um from particle size distributions in 14 studies of industrial, urban, and remote areas, including an urban area in California.

Data used to assess atmospheric cadmium exposure reflect total or acid extractable cadmium. The probable compounds of cadmium occurring in atmospheric particulate matter can be inferred from data on the solubilities of atmospheric cadmium particles, the combustion chemistry of major sources, and the solubilities of cadmium salts. Analyses of emitted particulate from fossil fuel fired boilers, and from a primary copper smelter, indicate that metals are emitted principally as the sulfate, and to a lesser extent as the oxide or carbonate. This is consistent with the observed water solubilities of cadmium aerosols in California (84 percent of cadmium particulate matter collected at an urban costal location was water soluble), and elsewhere (74 percent of continental aerosol collected in rural Tennessee was water soluble).

Sources and Fate

Although cadmium occurs as a trace element of crustal materials, comparisons of the compositions of atmospheric particulate matter and crustal materials strongly suggests that atmospheric cadmium originates mainly from high temperature industrial processes. The ratio of the cadmium to aluminum concentration ratio in air to their ratio in crustal materials is defined as the enrichment factor (EF) for cadmium. EF values less than 5 are generally considered to be indicative of a crustal or soil source; higher values of EF are suggestive of sources causing enrichment in cadmium, i.e., high temperature sources (combustion or pyrometallurgical). An average EF of 1,900 for cadmium at urban, rural, and remote sites in the U.S. and elsewhere has been reported. No California-specific data are available, but the enrichment phenomenon observed elsewhere supports the supposition that atmospheric cadmium in California is emitted principally from high temperature industrial sources.

An inventory of cadmium emissions in the state indicates that most (about 90 percent) cadmium is emitted from high temperature processes. These sources have been shown to emit cadmium on particulate matter principally less than 2 um in diameter, with typical mass median diameters of 1 um. The enrichment of cadmium on smaller diameter particles has been attributed to condensation of cadmium (volatilized during combustion) on the surface of emitted particles as cooling of combustion gases occurs. Because small particles have greater surface to mass ratios than large particles, the concentration of cadmium on a mass basis is greater for small particles.

Cadmium is emitted from a number of different sources. Approximately 80% of the cadmium accounted for in a statewide emission inventory was from stationary sources with the balance emitted by motor vehicles.

Stationary sources of cadmium emissions include secondary smelters, cement manufacturing plants, cadmium electroplating facilities, sewage sludge incinerators, and industrial, commercial, and utility plants where coal or oil is burned.

Cadmium is also emitted from mobile sources. Cadmium is a component of diesel fuel and gasoline, and is emitted when these are burned. Also, cadmium is present in vehicle tires and consequently in the particles resulting from tire wear. Table III gives a summary of ARB statewide emission estimates for cadmium.

TABLE III

Statewide Cadmium Emissions

Stationary Sources	Inventory Year	Estimated Statewide Emissions (tons/yr.)
Secondary Smelters	1981	8.5
Cement Manufacturing	1984	0.02-1.1
Oil Combustion	1983	3-4
Coal Combustion	1981	0.2
Cadmium Plating	1982	0.6
Sewage Sludge Incineration	1982	0.4
Total Stationary Sources		13-15
Mobile Sources		
Motor Vehicle Fuel Combustion	1984	1.7
Vehicle Tire Wear	1984	0.9
Total Mobile Sources		2.6
Total All Sources		16 - 18

There is evidence of atmospheric reactions of cadmium emitted from coal combustion. An increase in the solubility of cadmium on coal fly ash has been attributed to reaction of emitted cadmium oxide in the plume to form cadmium sulfate, phosphate, or fluoride. In addition to this group of reactions, which would account for observed increases in the solubility of emitted cadmium, the reaction of metal oxides in fly ash with carbon dioxide to form metal carbonates has been observed. If this reaction occurs with cadmium, it would not affect the solubility of atmospheric cadmium directly, because both the oxide and carbonate salts of cadmium are insoluble.

Cadmium is removed from the atmosphere through both wet and dry deposition. The rates of trace metal deposition are believed to depend on meteorology, vegetation (canopy) characterisitics, and differences in local or regional emissions.

Non-Cancer Health Effects

Cadmium has been found to induce a number of noncarcinogenic toxic effects in experimental animals and humans. Cadmium has moderate acute toxicity, producing gastrointestinal or pulmonary effects from ingestion or inhalation, respectively. Chronic and subchronic exposures to cadmium have been associated with a wide range of adverse outcomes that include cardiovascular, endocrine, hepatic, bone, hematological, immunological, respiratory, renal, reproductive, and teratogenic effects. DHS staff has concluded that renal toxicity is the most sensitive noncarcinogenic effect, in that it has been reported to occur at lower exposure levels than other effects.

The staff of the Air Resources Board has estimated that long-term atmospheric concentrations of cadmium in California are in the range of less than 1, to 6 ng/m^3. A daily retention rate of cadmium estimated to induce renal toxicity in 10 percent of the population has been estimated to be 6.6 to 24.6 ug/day over a 50-year period. Ambient air concentrations necessary to attain this range of retention rates have been estimated to be 650 to 2500 ng/m^3, assuming 50 percent pulmonary absorption. Although no threshold exposure level has been determined for renal toxicity, the staff of DHS believes that such a level does exist. The staff of DHS has concluded that the two to three orders of magnitude difference between the estimated atmospheric concentrations of cadmium and those concentrations necessary to attain a retention rate at which 10 percent of the population would develop renal toxicity is sufficiently large that atmospheric cadmium does not pose a significant hazard for renal toxicity. Since renal toxicity is the most sensitive noncarcinogenic endpoint, any other acute or chronic noncarcinogenic toxic effects from current ambient levels are not expected.

Carcinogenic Effects

Cadmium has induced cancer in experimental animals and has been associated with an increase in human cancers in epidemiological studies. Cadmium has produced injection site tumors (in rats) and remote tumors (in rats and mice) following subcutaneous or intramuscular injections, and has produced lung tumors in rats exposed to cadmium chloride aerosol. Several studies in which cadmium was given by the oral route have been negative, perhaps because of poor gastrointestinal absorption and low susceptibility of gastrointestinal epithelial tissue to carcinogenesis induced by cadmium. The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence of carcinogenicity in animals and that, for practical purposes, cadmium should be regarded as if it presents a carcinogenic risk to humans. DHS staff concurs in these conclusions.

Epidemiological evidence has suggested an association between cadmium exposure and neoplasia, including respiratory, renal, prostatic, and bladder cancers. For the latter three cancers, the evidence is suggestive or inconclusive; however, there is strong evidence of an association between cadmium exposure and an increased risk of respiratory cancer. Several occupational studies have shown some association between cadmium exposure or potential exposure and lung cancer. A recently published, well-designed study which evaluated a cohort of cadmium-exposed workers found a highly statistically significant dose-response relationship. Neither bias nor confounding appeared to be responsible for the observed excess lung cancer risk.

A variety of studies have indicated that cadmium is mutagenic and clastogenic. However, a number of similar studies have given negative results. The staff of DHS has concluded that there is only limited evidence that cadmium is mutagenic and clastogenic.

There is also evidence that cadmium can bind to DNA and cause mispairing of synthetic polynucleotides. This type of activity may cause a mutagenic or carcinogenic effect. The mechanism of action for this type of effect is believed to have no threshold associated with it. In the absence of compelling evidence of a threshold, the staff of DHS considers the mechanism of cadmium carcinogenesis to be a nonthreshold process.

Risk Due To Atmospheric Cadmium

At ambient concentrations, cadmium was estimated to present a potential carcinogenic risk to humans. This conclusion was based on two separate risk assessments, one utilizing animal data, the other utilizing human data.

In a 31-month inhalation bioassay, rats were exposed to cadmium chloride aerosol at concentrations of 0, 2.2, 4.1 and 8.3 ug/m^3 pure cadmium. The tumor incidence rates for these four dose groups were, respectively, 0%, 15%, 53% and 71%. Several models were fit to these data. The most health-conservative extrapolation was achieved by fitting the multistage model, which predicted an excess lifetime cancer risk of 110 per million persons continuously inhaling 1 ng/m^3 cadmium in ambient air throughout their lives. The upper 95% confidence limit for this risk estimate was 180 per million persons.

The human data used for a risk assessment was based on an occupational cohort study of 585 workers exposed to cadmium in a production plant. Based on cumulative exposures, the follow-up years for these workers were divided into 3 exposure categories. At median cumulative doses of 184, 796 and 2762 ug/m^3 of cadmium, standardized mortality ratios (SMRs) of 53, 152 and 280 were observed. A linear excess relative risk model with an adjustment for the healthy worker effect was fitted to these data by an iterative least squares algorithm. The model predicted an excess lifetime cancer risk of 2 per million persons inhaling 1 ng/m^3 cadmium in ambient air throughout their lives. The upper 95% confidence limit for this risk estimate was 12 per million.

The upper 95% confidence limit for lifetime cancer risk based on the rat study was about 15 times the upper 95% confidence limit of risk predicted by the epidemiological study. The maximum likelihood estimate from the animal data is about 10 times the upper 95% human-based estimate. Members of the DHS staff have concluded that the human-based quantitative risk assessment is sufficiently health conservative because: (1) it is based on a linear extrapolation, (2) potential inaccuracies in the human data regarding exposure or response are likely to be small, and (3) the net direction of these inaccuracies are likely to result in an overestimate of potency. Therefore, the DHS staff believes that the human-based risk assessment provides the most appropriate range of risks. The range of estimated excess lifetime cancer risks from 24-hour-per-day exposure for a lifetime to atmospheric concentrations of cadmium (1 to 2.5 ng/m^3) is therefore 2 to 30 per million persons exposed. In the vicinity of sources of cadmium emissions, ambient exposures may reach an annual average of 40 ng/m^3, with the estimated excess lifetime cancer risk being 80 to 480 per million persons exposed.

III. ENVIRONMENTAL IMPACTS

The identification of cadmium as a toxic air contaminant is not in itself expected to result in any environmental effects. The identification of cadmium as a toxic air contaminant by the Board may result in the Board and air pollution control districts adopting toxic control measures in accordance with the provisions of state law. Any such toxic control measures may result in reduced emissions of cadmium to the atmosphere, resulting in reduced ambient concentrations, concurrently reducing the health risk due to cadmium. Therefore, the identification of cadmium as a toxic air contaminant may ultimately result in environmental benefits. Environmental impacts identified with respect to specific control measures will be included in the consideration of such control measures pursuant to Health and Safety Code Sections 39665 and 39666.

IV. REGULATORY BACKGROUND AND PROCEDURES

Division 26, Chapter 3.5 of the Health and Safety Code** (HSC) and Food and Agriculture Section 14021 et seq. set forth the procedure for identifying and controlling toxic air contaminants in California. (These provisions were enacted in September 1983 as Assembly Bill 1807, Stats. 1983, ch. 1047.) The Department of Food and Agriculture is responsible for identifying and controlling TACs in their pesticidal uses. The ARB has authority over TACs in all other uses.

** Health and Safety Code Section 39650; all statutory references are to the Health and Safety Code, except as otherwise stated.

HSC Section 39650 sets forth the Legislature's findings about substances which may be TACs. The Legislature has declared:

> "That public health, safety, and welfare may be endangered by the emission into the ambient air of substances which are determined to be carcinogenic, teratogenic, mutagenic, or otherwise toxic or injurious to humans."

The findings also include directives on the consideration of scientific evidence and the basis for regulatory action. With respect to the control of TACs, the Legislature has declared:

> "That it is the public policy of this state that emissions of toxic air contaminants should be controlled to levels which prevent harm to the public health."

The Legislature has further declared that, "while absolute and undisputed scientific evidence may not be available to determine the exact nature and extent of risk from toxic air contaminants, it is necessary to take action to protect public health."

In the evaluation of substances, the Legislature has declared that the best available scientific evidence, gathered from both public agencies and private sources including industry, should be used. The Legislature has also determined that this information should be reviewed by a scientific review panel and by the public.

The Board's determination of whether or not a substance is a toxic air contaminant includes several steps specified by the HSC. First, we request the DHS to evaluate the health effects of a substance (Section 39660). The evaluation includes a comprehensive review of all available scientific data. Upon receipt of a report on health effects from DHS and in consideration of their recommendations, we prepare and submit a report to the Scientific Review Panel (SRP) for its review (Section 39661). The report consists of the DHS report (Part B), material prepared by the ARB staff on the use, emissions and ambient concentrations of the substance (Part A), and public comments on the draft report and responses (Part C). It serves as the basis for future regulatory action by the Board. The report is also made available to the public, which may submit comments on the report.

After receiving the SRP's written findings on the report, the Board issues a public hearing notice and a proposed regulation identifying the substance as a toxic air contaminant. If, after a public hearing and other procedures to comply with Government Code Section 11340 et seq., the Board determines that a substance is a toxic air contaminant, its findings must be set forth in a regulation (Section 39662). The HSC also sets forth procedures for developing and adopting control measures for substances identified as TACs (Sections 39665-39667).

TECHNICAL SUPPORT DOCUMENT

REPORT TO THE AIR RESOURCES BOARD ON CADMIUM

PART A - PUBLIC EXPOSURE TO, AND SOURCES OF, ATMOSPHERIC CADMIUM IN CALIFORNIA

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December 1986

Technical Support Document

Report to the Air Resources Board on Cadmium
Part A - Public Exposure to, and Sources of, Atmospheric Cadmium in California

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I. INTRODUCTION

Cadmium and its compounds are significant from both a commercial and an environmental perspective. Cadmium or its compounds is used to inhibit corrosion of other metals, to color and to stablize plastics, and to achieve a number of other unique or beneficial properties in industrial and commercial applications.

Cadmium is present at trace levels in fossil fuels and some metal ores. Because cadmium is volatile (relative to other metals), there is a high potential for its release to the atmosphere during ore smelting of some metals and during fossil fuel or waste combustion. Cadmium may also be emitted to the atmosphere during its direct industrial use. Cadmium has been measured in the atmosphere of California, both in special studies and on an ongoing basis, for more than thirty years.

This report presents statewide estimates of present and future usage and emissions of cadmium, a discussion of the available information on the nature and fate of that emitted cadmium, and an estimate of exposure to atmospheric cadmium for both the general public and for people living close to major sources of cadmium emissions. In discussion of each of these topics areas of incomplete knowledge are identified, and, where possible, inferences are drawn using available information.

II. PROPERTIES

Cadmium is a soft, silver-white metal which is found as the sulfide at trace concentrations in the earth's crust. In its elemental form, cadmium is resistant to corrosion by alkalies and salt water, and retains its metallic luster in air. The molecular weight of metallic cadmium is 112.4, and its boiling point is 767°C. The relatively high volatility of cadmium and some of its compounds compared to other metals is significant from an air pollution standpoint; cadmium vaporized during combustion or other high temperature processes, condenses on particles as the gas cools. Preferential enrichment of cadmium on fine particles (less than 2 micrometers) occurs as a result. Because some air pollution control devices have lower removal efficiencies for small particles than for large, cadmium is emitted predominantly on small particles, which are respirable.

The most common oxidation state of cadmium is +2, although there are a small number of compounds in which cadmium occurs in the +1 oxidation state (Hollander and Carapella, 1978). Commercially and environmentally significant compounds of cadmium exhibit a wide range of properties; selected properties of several compounds are given in Table II-1.

TABLE II-1

Physical Properties of Selected Cadmium Compounds

Species	Molecular Weight	Solubility[1] Water	Acid	Boiling Point (°C)
Cadmium	112.4	i	s	767
acetate	230.5	s	s	decomposes
carbonate	172.4	i	s	decomposes
chloride	183.3	s	s	960
fluoride	150.4	s	s	1758
oxide	128.4	i	s	1559
orthophosphate	527.1	i	s	---
sulfate	208.5	s	s	----
sulfide	144.5	i	s	980 (sub. in N_2)

Sources: IARC, 1976; Hollander and Carapella 1978; Weast, 1973, Germani, et al, 1981

1) i = insoluble
s = soluble

REFERENCES FOR SECTION II

Germani, Mark S., Mark Small, William H. Zoller, and Jarvis L. Moyer, 1981. Environmental Science and Technology 15: 3, 299.

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Weast, Robert C. Handbook of Chemistry and Physics, 54th ed. Cleveland: CRC Press, 1973.

III. SOURCES AND FATE OF ATMOSPHERIC CADMIUM

A. Production and Usage

Cadmium is a rare element, found mainly as a sulfide in ores of zinc, copper, and lead. Because of its rarity, because it occurs with metals of economic importance and because it can be recovered during the refining of these other metals, cadmium is not mined separately; it is always a byproduct of other mining operations. It is most commonly produced commercially as a by-product of zinc (and to a lesser extent, copper and lead) smelting and refining. During 1984, five plants in the United States produced cadmium metal from zinc ores; none of these were located in California (U.S. Department of Interior [DOI], 1985a).

Cadmium consumption in the United States during 1984 has been estimated to be 4,200 tons. Domestic production of cadmium during 1984 was 1,800 tons (48 percent of consumption), with imports and reserves making up the difference. During the period 1973 to 1984, approximately 58 percent of cadmium consumed in the United States was imported, mainly from Canada, Australia, Peru, and Mexico (U.S. DOI, 1983; U.S. DOI, 1985a,b).

Historical national production, importation, and consumption estimates are given in Figure III-1. The U.S. Department of the Interior has forecast an annual increase in cadmium consumption of approximately 1.9 percent during the period 1983-2000 (U.S. DOI, 1985a).

Figure III-2 depicts the national demand for cadmium in 1984 by major use category. The main user of cadmium (as cadmium chemicals) is the plating industry. Cadmium plating provides excellent protection for iron, steel, brass and aluminum against corrosion, especially in marine and alkaline environments. Historically, 37 percent of cadmium consumption in the United

Figure III-1

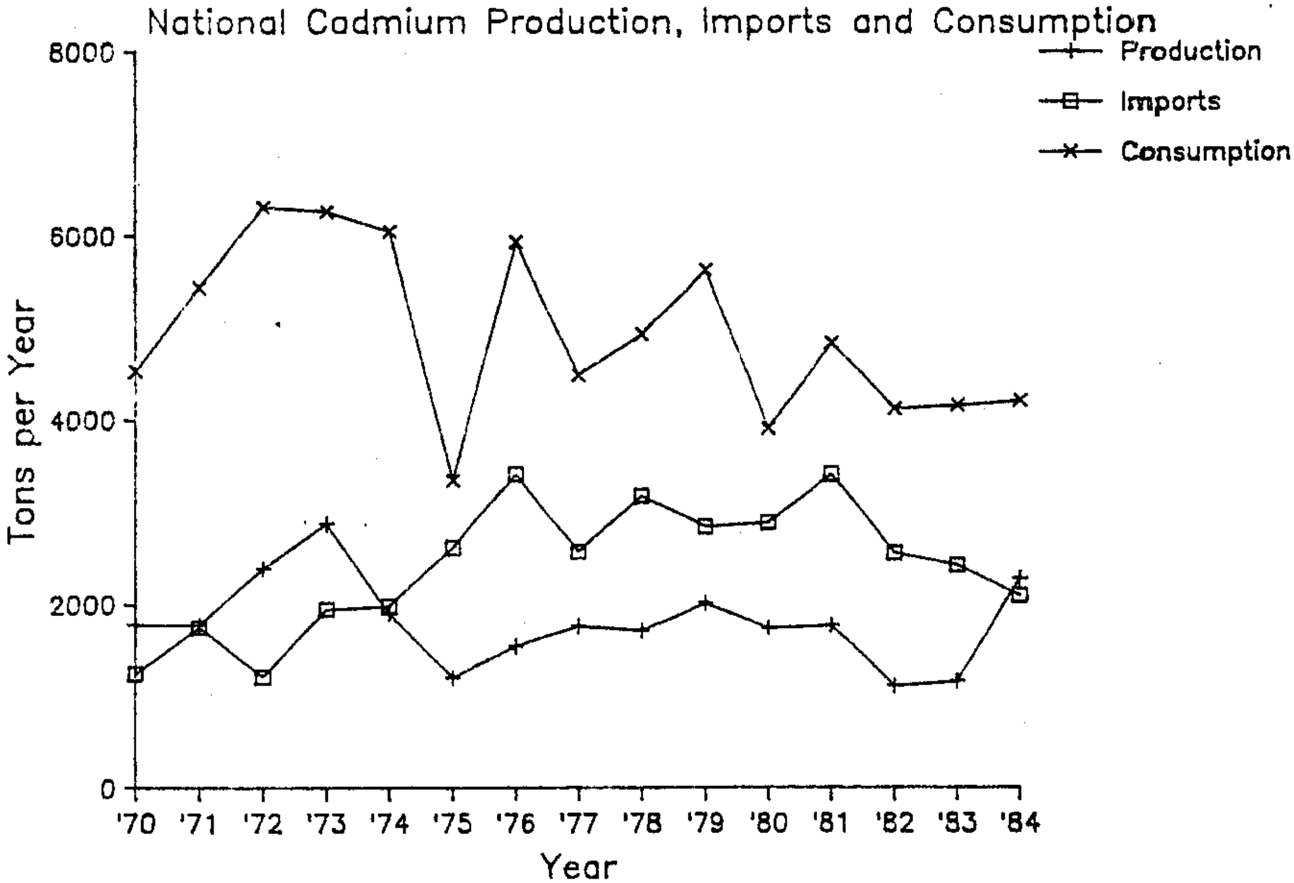
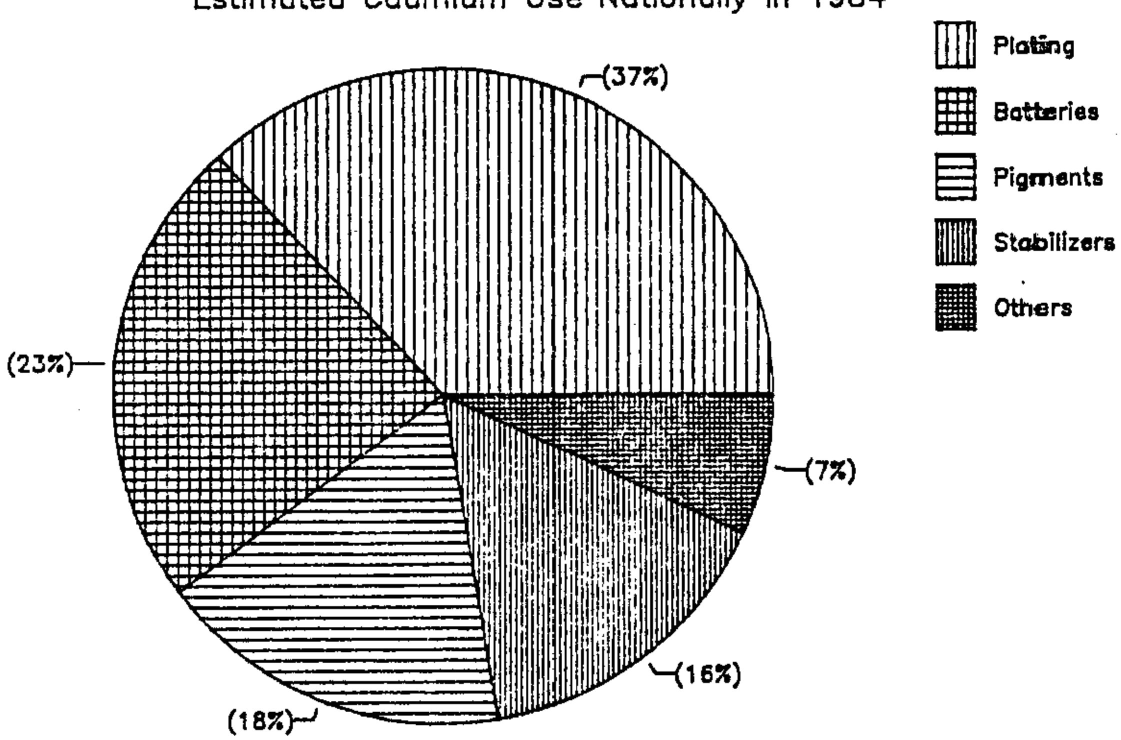


Figure III-2

Estimated Cadmium Use Nationally In 1984



States was for plating, which yields an estimate of 1,550 tons in 1984 (U.S. DOI, 1985a and 1985b; CARB, 1985a). The number of cadmium platers in California is not known; however, 31 cadmium platers have been listed in the South Coast Air Quality Management District (SCAQMD) inventory of potentially toxic chemicals (Zwiacher et al., 1983).

Cadmium is also used extensively by the battery industry to produce nickel-cadmium, silver-cadmium and mercury-cadmium batteries. Nickel-cadmium batteries are commonly used in aircraft, alarm systems, cameras, calculators, etc. (U.S. DOI, 1985a). There are no nickel-cadmium battery manufacturers in California.

Cadmium pigment and stabilizer production accounted for consumption of 1,390 tons of cadmium nationally in 1984 (U.S. DOI, 1985b and CARB, 1985a). Cadmium sulfide and sulfoselenide are the most important compounds used in pigments. High temperature stability, brilliant colors, high opacity, and resistance to chemical attack and degradation by light are characteristics of cadmium pigments. Cadmium compounds are used as stabilizing agents in many polyvinyl chloride products such as clear sheet, film and tubing, and cushioned floor covering.

Cadmium and its compounds are employed in a variety of other uses. Cadmium sulfide and cadmium telluride are used in the electronics industry to produce photocells and light emitting diodes. Cadmium metal alloyed with copper is used in the production of automobile radiators. In tires, cadmium sulfide is used as a curing agent. Cadmium salts such as cadmium sebacate, are used in fungicides, and cadmium phosphate is used in fertilizers (Anderson, 1973; Tierney et al., 1979). The cadmium content of American phosphatic fertilizers range from 3.48 to 156 ppm (Hammons et al., 1979).

B. Sources of Current and Projected Cadmium Emissions

Approximately 90 percent of cadmium emission in California are the result of either the combustion of fuels or the smelting of metals which contain cadmium as a trace contaminant. Because of cadmium's high volatility relative to other metals, cadmium is vaporized by high-temperature processes and then condenses on the surface of particles in the gas stream. Because it is deposited uniformly on the surface of all particles, small particles with a larger surface area to volume ratio are found to contain higher concentrations of cadmium. For many types of sources significant in California, cadmium has been determined to occur on particles mostly 2 um in diameter or smaller (Milford and Davidson, 1985; Davison, et al., 1974). Available information suggests that cadmium is emitted from these source types principally as oxide and sulfate compounds, and also possibly as fluoride, chloride, and phosphate compounds.

The remaining 10 percent of cadmium emitted in the state is from either low-temperature sources such as cement manufacturing and tire wear, or from direct emission of cadmium compounds from cadmium plating operations.

Table III-1 summarizes statewide cadmium emissions.

Stationary Source Emissions

Although electroplating represents the largest use of cadmium, estimated emissions from cadmium plating in California are less than three percent of estimated total emissions. Most cadmium plating operations use cadmium-cyanide baths (Davis, 1970 and Graham, 1971), which are made up of cadmium

Table III-1

Estimated Cadmium Emissions in California

Source	Source Type	Emissions (tons/year)	Inventory Year	Reference
Secondary Smelters				
Copper	Area	8.1	1981	9,17
Steel Mills	Area	0.1	1981	9,17
Zinc	Area	0.3	1981	9,17
Fuel Combustion				
Coal	Point	0.2	1981	24,32
Distillate Oil	Area	0.6	1983	5,11,30
Residual Oil	Area	1.5-3.1	1984	1,4,5,6
Diesel	Area	0.4	1984	1,4,18
Waste Oil	Area	0.1	1983	6,10,28
Cement Manufacturing	Point	0.02-1.1	1984	5,6,7,8
Cadmium Plating	Area	0.6	1982	41
Sewage Sludge Incinerators	Area	0.4	1982	3,8,22
Motor Vehicles				
Fuel Combustion	Area	1.7	1984	5,26,36
Tire Wear	Area	0.9	1984	4,26,36

oxide or cadmium cyanide and sodium cyanide. During the plating process electric energy decomposes water in the bath, evolving hydrogen and oxygen gases; these gases carry cadmium in the plating bath to the surface of the bath and cause it to be entrained with the gases and emitted to the atmosphere. If the efficiency of the plating process is low, gassing will be high and cadmium emissions from plating process will also be high. Based on the South Coast AQMD survey (Zwiacher, et al., 1983), and assuming the distribution of cadmium platers is similar to chromium platers, it is estimated that approximately 80 cadmium platers operate in California. Cadmium emissions from this source are estimated to have been 0.6 ton in 1982 (see Appendix C for calculation).

Fuel combustion at stationary sources is responsible for approximately 3-4 tons of airborne cadmium per year. Residual oil combustion is the largest source of cadmium emissions in this category and accounts for well over half of the estimated cadmium emissions from fuel combustion.

The largest source of cadmium emissions from fuel combustion is oil and gas production activities. Utilities account for 0.7% to 17% of cadmium emissions from fuel combustion with the remainder being divided among ships, chemical manufacturers, industrial boilers, and other fuel oil users. Waste oil also contains cadmium, concentrations of which have been measured at levels as high as 110 ppm (Franklin Associates, Ltd., 1983).

During combustion, the trace levels of cadmium in distillate, residual, diesel, and waste oil are emitted into the atmosphere. There is a large variation in the cadmium content of residual oil. Southern California Edison (SCE) sampled oil in 1986 at its power plants and reported an average of 0.01 ppm cadmium (Southern California Edison, 1986). PG&E sampled oils at its power plants and reported an average cadmium concentration of 0.39 ppm (range 0.31 ppm to 0.52 ppm) (Pacific Gas and Electric Co., 1986). Several documents (Menczel, et al., 1984; U.S. EPA, 1984; John J. Yates & Associates, 1983; Krishnan and Hellwig, 1982) indicated the cadmium concentration of residual oil to be as high as 1 ppm. Lower trace metal concentrations, specifically cadmium, in residual oil burned at power plants in the South Coast are attributed to the South Coast Rule 431.2 which limits the sulfur content of any liquid fuel burned at power plants and refineries to 0.25 percent (SCAQMD, undated). This is half the 0.50 percent sulfur limit applied to liquid fuels burned in the Bay Area (BAAQMD, 1984). Staff have revised the estimated cadmium emissions from residual oil combustion based on cadmium content of fuel oil reported by SCE and PG&E (see Appendix C).

Residual fuel oil used by the California utilities has declined steadily since 1977, falling from 124 million barrels in 1977 to 4.5 million barrels in 1985 (CEC, 1986a; CEC, 1986b). In 1984, utilities used approximately 16 percent of all residual oil burned in the state (CARB, 1986a; CARB, 1986b). The California Energy Commission (CEC) forecasts a three-fold increase in residual oil use by the utility industry from 1985 to 1997 (CEC, 1986a; CEC, 1986b). By 2005, residual oil used by the utility industry would return to the 1984 level (CEC, 1986a). The use of residual oil in the industrial, commercial and transportation sectors is forecast to remain about the same through the year 2005 (CEC, 1986b). Because cadmium emissions from residual oil combustion is directly proportional to the amount used, cadmium emissions from residual oil combustion are therefore expected to increase in the next decade and then return to the 1984 level by 2005.

Cadmium emissions from coal combustion are expected to increase due to the increase in coal consumption by various industries. Five California cement plants have converted or plan to convert from a wet production process which uses natural gas as a fuel, to a more efficient dry process which often uses coal. Coal consumption from cement manufacturers will also rise due to the increase in their capacities. In 1984, 11 cement manufacturers in California produced 8.7 million tons of cement (U.S. DOI, 1985d) and consumed a total of approximately 1.6 million tons of coal. In 1985, the Department of the Interior forecast the U.S. cement production in 1990 and 2000 to be 77 million tons and 87 million tons, respectively (U.S. DOI, 1985c). Based on United States and California cement production data for 1980 through 1984, California produced an average of 11.2 percent of the cement production in the nation (U.S. DOI 1985c,d). Assuming the ratio of the California cement production to the United States is the same in the future, California cement production is forecast to be approximately 8.6 million tons in 1990 and 9.7 million tons in the year 2000.

Approximately 0.17 ton of coal is required as fuel to produce 1.0 ton of cement. Assuming the recent degree of coal use continues into the future, the California cement industry is forecast to use approximately 1.5 million tons of coal in 1990 and 1.6 million tons in 2000.

Although a number of coal gasification programs have been or are being considered by the utility industry, only one plant is currently known to gasify coal in Southern California. This plant used 108,000 tons of coal from June 1984 through January 1985 (Wolk and Holt, 1985).

The compound form of cadmium emitted from fossil fuel combustion has not been determined. The general composition of particulate matter emitted from coal and oil combustion (fly ash) has been studied, and provides some insight into the possible compound forms of cadmium. Eatough, et al., (1981) reported sulfate to be essentially the only acid-extractable anion present in oil-fired power plant fly ash smaller than 3 um (chloride was present at 0.1 mole percent of sulfate). Henry and Knapp (1980) found that an average of 65 percent of oil combustion fly ash was water soluble; exclusive of carbon, an average of 86 percent of the fly ash was water soluble. Sulfate was the only anion found above trace values in the water soluble phase; metal oxides were determined to comprise the balance of the fly ash. The range of water solubility in six samples ranged from 23.3 to 98.5 percent, and the percentage of sulfate ranged from 12 to 58. This range of values is consistent with Dietz and Weiser's (1983) conclusion that metal sulfate emissions from oil-fired power plants are related to fuel composition (sulfur and vanadium concentration), combustion parameters (excess oxygen and temperature in the combustion chamber), and air pollution control device type and performance.

Cadmium in fly ash emitted from coal combustion was determined by Hansen and Fisher (1980), and by Davidson, et al., (1974) to be concentrated at the surface of particles. Because the volatile non-metals have also been determined to occur on the surface of fly ash particles (Smith, 1981), Hansen et al., (1984) have postulated that cadmium may occur in coal fly ash as the fluoride, phosphate, or sulfate. The occurrence of these compounds on the particle surface is thought to depend on the concentration of the metal oxide at the particle surface, the concentrations of HF, SO_3, and P_4O_{10} in the flue gas, and the temperature and contact time of the particles and flue gas. Hansen and Fisher (1980) showed that 65 percent of acid-soluble cadmium on respirable fly ash from coal combustion was water soluble; this suggests that most cadmium occurs in the water-soluble sulfate, fluoride, or phosphate form, and that the balance is the acid-soluble oxide or carbonate form (Gendreau, et al., 1980).

The size distribution of cadmium on fossil-fuel combustion fly ash has been investigated by Davidson, et al., (1974), Jacko and Neuendorf (1977), Toca, et al., (1973), Hansen and Fisher (1980), and others. The common conclusion is that cadmium shows a pronounced concentration trend with particle size, occurring at increasing concentrations on smaller particles.

Emissions from secondary smelters result from cadmium present in scrap metal or feedstock. Cadmium is not recovered from these smelters, cadmium present in the feed materials which is not collected by air pollution control devices is released to the environment. Table III-1 lists cadmium emissions from secondary copper, steel and zinc smelters. Together, secondary smelters are estimated to have emitted 8.5 tons of cadmium in 1981.

The compound forms of cadmium emitted from primary smelters have been determined to be the sulfate and oxide forms (Eatough, et al., 1981; Radian, 1985). The extent to which cadmium would be emitted from secondary smelters as the sulfate would depend on the amount of sulfur present during smelting. We expect this to be much less in secondary than in primary smelting; therefore, we hypothesize that cadmium will be emitted from secondary smelting primarily as cadmium oxide, and to a lesser extent as cadmium sulfate.

Cadmium emitted from secondary smelters is expected to exhibit tendencies of surface enrichment, and therefore, a trend of increasing concentration on small particles. Jacko and Neuendorf (1977) showed that particles emitted from pyrometallurgical processes have mass median diameters of less than 1.0 um, and that a large percentage (30 to 50) of cadmium emitted from such processes is found on particles in the respirable range (less than 2.5 um diameter). This observation has been corroborated by Van Graen, et al., (1983), who concluded that surface enrichment of trace elements in particles from high temperature processes is universal, based on analysis of dust from an electric steel making furnace.

Of the secondary smelters, copper smelters are the largest source of cadmium emissions. Processes in secondary copper include: a) sweating scrap to remove low melting point metals or burning to remove insulation from copper wire; b) smelting and refining to obtain a certain type of copper; and c) alloying to modify the final product. No control devices are employed in wire burning; however, smelters and furnaces are usually equipped with hoods and baghouses (Coleman, 1970) to reduce direct cadmium emissions.

The ARB's Emissions Data System (EDS) includes 71 secondary copper smelters in California for inventory year 1981. Using an emission factor of 3 lb. Cd per ton of scrap and assuming 90 percent control, cadmium emissions from copper smelters were estimated to be 8.1 tons in 1981 (Coleman, et al., 1979 and CARB, 1985d).

The production of secondary lead consists mainly of melting down lead batteries, lead oxide drosses, recycled dust and metal scrap in reverberatory or blast furnaces at 930 degrees C. Cadmium is released in this process (Anderson, 1973). Based on available data, the amount of cadmium emitted from secondary lead smelters in California is expected to be small.

Cadmium is present as an impurity in the material used to produce cement and is emitted during cement production. Currently, cement is produced by either a dry or a wet process. Particulate emissions, including cadmium, from these two processes differ primarily due to the nature of the processes involved. Cadmium emissions from cement production for both the dry and the wet process were estimated to be between 0.02 and 1.1 ton during 1981 (see Appendix C).

As the result of conversion from wet to dry processes in the cement industry and expected increases in the plants' capacities (Sierra Energy & Risk Assessment, Inc., 1982), cadmium emissions from cement manufacturing are expected to increase.

The estimate of cadmium emitted from sewage sludge incineration was based on the fraction of cadmium in the particulate matter and particulate matter emissions from municipal sewage sludge incinerators (Bennett, et al., 1982, Jacko et al., 1977 and CARB, 1985b). Cadmium emitted from sewage sludge incineration has been shown to occur primarily on particles less than 2 um in diameter (Bennett, et al., 1984; Radian, 1985); this is consistent with the distribution observed in other combustion emissions. The compound form emitted may be similar to those from municipal waste incineration, which is believed to be the oxide.

Resource recovery facilities are potential sources of cadmium. In municipal solid waste (MSW)-to-energy plants, cadmium present in batteries, in plastics (as stabilizers or dyes), or in other forms, will volatilize during the combustion process. The amount of cadmium emitted depends on the amount of cadmium present in the waste burned and on the efficiency of emission control equipment used to remove particulate matter from the exhaust gas. For example, the potential emission of cadmium from a planned MSW-to-energy facility (based on certain assumptions about the concentration of cadmium in the feed and the efficiency of removal), is about 5.9 kilograms (Kg) (13 pounds) per year (see Appendix C).

At this time, one MSW-to-energy facility is operating in California. Four more have received the approval of regulatory agencies. More than thirty resource recovery facilities are proposed for construction in California.

Cadmium emitted from municipal incinerators has been determined to be in the oxide form, and in the respirable size range (Radian, 1985). Measurements by Greenberg et al., (1978a, b), of cadmium on particles from municipal incinerators showed that 80 to 95 percent of cadmium is found on particles of 2 um diameter or smaller.

Cadmium emissions from fertilizer and pesticide applications are estimated to be less than 4.6 Kg (10 pounds) per year statewide.

Mobile Source Emissions

Cadmium occurs as a trace component in diesel fuel, gasoline and lubricating oil. When these fuels are burned, cadmium is emitted. Combustion of gasoline, diesel, and lubricating oil from motor vehicles is a source of 1.7 tons of cadmium emissions per year. Cadmium emitted on exhaust particulate exhibits surface enrichment (Keyser, et al., 1978).

Because cadmium sulfide is used as a curing agent in tires, particulate matter resulting from tire wear contains cadmium. Based on the number of vehicle miles traveled in California and estimates of cadmium emitted in tire particulate, tire wear is a source of 0.9 ton/year of cadmium emissions (see Appendix C for details).

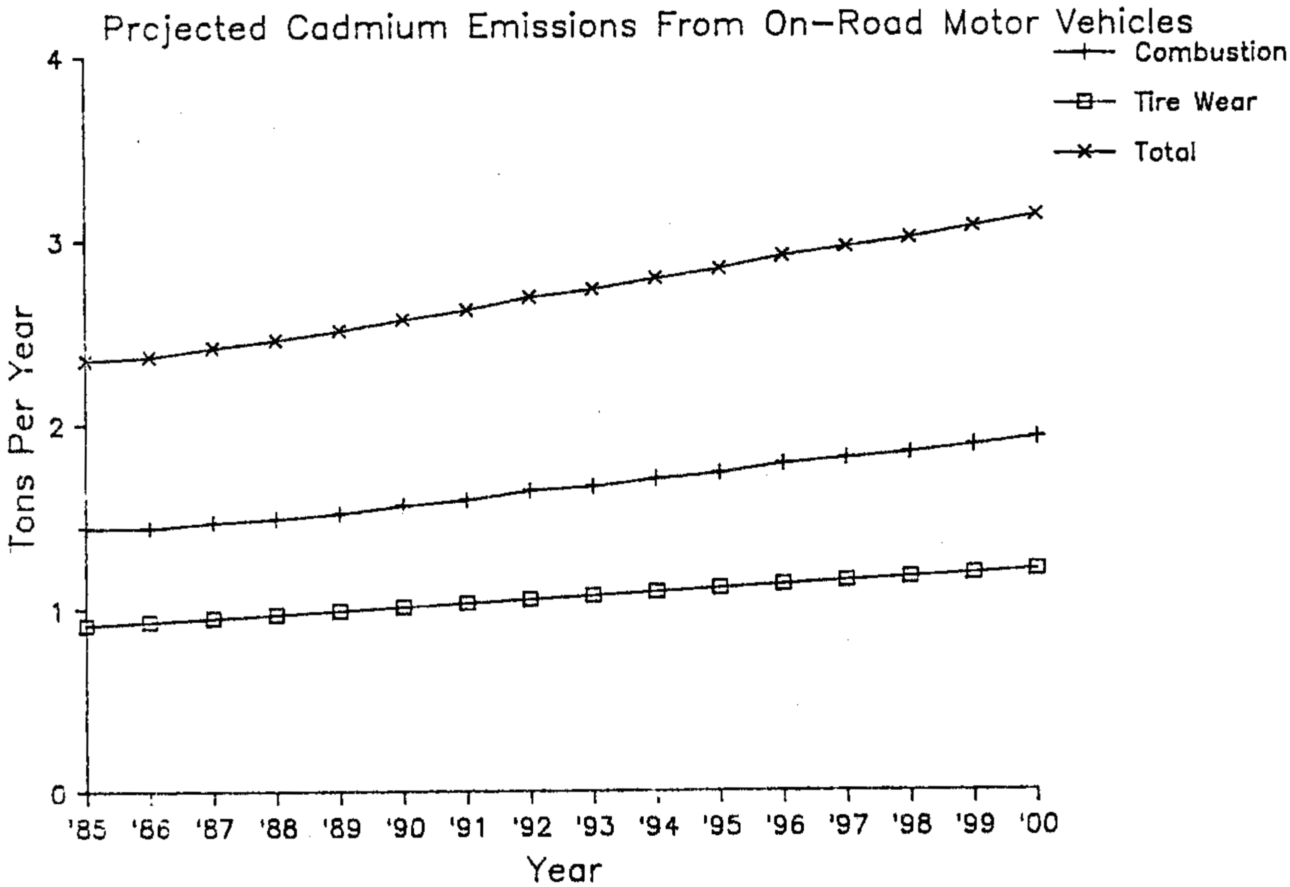
Cadmium emitted from attrition of tire rubber is thought to occur as large particles which are rapidly deposited in the immediate vicinity of the roadway. This is consistent with a study by Johnston and Harrison (1984), who measured cadmium deposition rates near a major English motorway; they found the highest concentration of particulate-associated cadmium occurred 3.8 meters from the road. Deposition rates declined to background levels at a distance of 25 meters.

Cadmium emissions from on-road motor vehicles are expected to increase due to an increase in vehicle population, vehicle miles traveled, and changes in fuel consumption. Projected cadmium emissions from on-road motor vehicles from 1985 to 2000 are depicted in Figure III-3.

C. FATE OF ATMOSPHERIC CADMIUM

Consideration of the fate of atmospheric cadmium includes both atmospheric reactions of cadmium compounds emitted from sources, and mechanisms of removal of atmospheric cadmium. There has been no characterization of atmospheric reactions of cadmium; however, there is evidence of atmospheric reactions of metal oxides. It is possible that such reactions occur with cadmium oxides emitted from combustion sources. The mechanisms of removal of cadmium from the atmosphere have been studied in many areas, including an urban area in California.

Figure III-3



Prcjected Cadmium Emissions From On-Road Motor Vehicles
Combustion
Tire Wear
Total
Tons Per Year
4
3
2
1
0
'85
'86
'87
'88
'89
'90
'91
'92
'93
'94
'95
'96
'97
'98
'99
'00
Year

Atmospheric Reactions

Study of the plume constituents of a large coal-burning power plant by Lindberg and Harriss (1980) indicated that as the distance from the stack increased, the water solubility of cadmium increased, and that cadmium on the smallest particles (less than 0.14 um) exhibited the greatest relative increase in concentration. It was hypothesized that this increase in solubility was due mostly to vapor condensation on the fine aerosols, causing the formation of thin, highly soluble coatings. Hansen, et al., (1984) have subsequently identified possible specific reactions of metal oxides (on coal fly ash) which explain the increase in cadmium solubility with plume "aging". These reactions include the formation of phosphates, fluorides, and sulfates:

$$6\ MO_{(s)} + P_4O_{10(g)} \rightarrow 2M_3\ (PO_4)_{2(s)} \qquad (1)$$

$$MO_{(s)} + SO_{3(g)} \rightarrow MSO_{4(s)} \qquad (2)$$

$$MO_{(s)} + 2HF_{(g)} \rightarrow MF_{2(s)} + H_2O_{(g)} \qquad (3)$$

Another reaction of metal oxides on coal fly ash has been identified (Bauer and Natusch, 1981). It was observed that metal oxides in coal fly ash could react with CO_2 to form metal carbonates. The rapid chemisorbtion of CO_2 was judged favorable for some metal oxides. If such a reaction occurs for emitted cadmium oxide, the solubility of the cadmium aerosol will remain unchanged, since both the oxide and carbonate salts are insoluble in water.

Removal of Cadmium

Cadmium is removed from the atmosphere through physical processes; both wet and dry deposition have been judged to be significant. Lindberg, et al., (1982) identified a wide range of trace metal deposition mechanisms and rates, depending on the meteorology, canopy characteristics, or differences in local or regional emissions.

Davidson (1980) found that the rough surface dry deposition velocities of cadmium varied over a 20-fold range. For a site in Pasadena, estimates of the deposition flux of cadmium ranged from 0.30 to 0.67 ng/cm^2 day, depending on the deposition model used. Struempler (1975) measured the deposition of cadmium in precipitation to be 0.033 $ng/cm^2/day$ at a rural site in Nebraska. Based on the residence time of aerosols, cadmium is expected to have a residence time of seven days in the atmosphere (U.S. EPA, 1980).

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IV. PUBLIC EXPOSURE

A. Atmospheric Concentrations

During 1985, ARB staff collected total suspended particulate matter (TSP) at 21 air monitoring stations in California to be analyzed for trace metals. High-volume samplers were used to collect 24-hour samples of atmospheric particulate matter of 50 um and smaller diameter (50 percent size cutoff). Monitoring sites met criteria for population oriented exposure for the criteria pollutants; all sites are in populated urban areas. Samples were collected at most sites every eight days.

Analysis was performed for each filter individually; flame atomic absorption spectrophotometry was used to determine acid-extractable cadmium. The method is described in Appendix D. A detection limit of cadmium in air of 1.0 ng/m^3, with a precision and accuracy of $\pm$ 15 percent, has been determined for the method.

Data are available for the first six months of 1985. The range of samples at each site above the detection limit ranged from 13 to 100 percent, with an average of 50 percent above the detection limit for all data. To estimate the possible range of the average concentration at each site, two methods were used. The first, termed the zero treatment method, estimates the range by assigning maximum and minimum values to all data below the detection limit. The second method, a statistical treatment, assumes the data is lognormally distributed and uses standard statistical techniques to estimate the uncertainty of the average concentration. Although the second method is believed to give the best estimate for the average, the first method is included to show the uncertainty that arises from having data below the detection limit. In most cases the possible range of average concentration is small.

The zero treatment method assigns two different values to the data below the LOD in order to calculate the range arithmetic averages: in the first, or minimum average estimate, zero was substituted for values reported below the detection limit. For the second, or maximum average estimate, the detection limit value (1.0 ng/m^3) was substituted for values reported below the detection limit. These "min" and "max" values therefore represent the upper and lower estimates of mean concentrations possible at each site. The average concentrations derived using this zero treatment method are shown in Table IV-1.

Data from the ARB's monitoring stations were used to estimate residential population exposure to ambient cadmium in the South Coast Air Basin, the San Francisco Bay Area Air Basin, the San Joaquin Valley Air Basin, San Diego Air Basin, South Central Coast Air Basin, and Sacramento County. Exposures over the San Francisco Bay Area and the South Coast air basins were estimated by interpolating station values to census tract centroids. Exposures for the remaining air basins were estimated by assuming the entire population in each area is exposed to the arithmetic mean concentration from all sampling sites in the air basin. Table IV-2 shows the average cadmium concentrations for each air basin. For the South Coast and the San Francisco Bay Area air basins, these are population weighted averages based on the concentrations interpolated to each census tract. Average cadmium concentrations range from less than 1 ng/m^3 for Sacramento County to 2.5 ng/m^3 for the San Francisco Bay Area air basin.

The 1985 residential population in California was about 26.4 million people. The population represented in the six areas covered in our exposure

TABLE IV-1

1985 AVERAGE CADMIUM CONCENTRATIONS
ARB CADMIUM SAMPLING NETWORK
BASED ON DIFFERENT ZERO VALUE TREATMENTS*
(Six Months Data)

RANK	SITE ID	LOCATION	AVERAGES (ng/m^3) MIN	MAX	# SAMPLES ABOVE LOD	TOTAL # SAMPLES
1	0700440	Concord	5.5	5.9	22	36
2	7000579	El Monte	4.1	4.1	15	15
3	0700433	Richmond	2.9	3.0	30	32
4	9000304	San Francisco	2.5	2.6	26	31
5	2400521	Merced	1.9	2.0	13	15
6	7000087	Los Angeles	1.1	1.7	13	29
7	4300382	San Jose	1.1	1.5	18	30
8	6000336	Fremont	0.8	1.3	16	31
9	1000234	Fresno	0.7	1.1	12	22
10	8000131	El Cajon	0.8	1.0	15	21
11	4200378	Santa Barbara	0.7	1.1	11	18
12	0700430	Pittsburg	0.7	1.0	7	10
13	7000085	Pico Rivera	0.5	1.2	11	31
14	3600175	Upland	0.5	1.2	9	30
15	3900252	Stockton	0.4	1.1	8	24
16	5000558	Modesto	0.4	1.0	12	29
17	5600413	Simi Valley	0.3	1.0	6	19
18	7000072	No. Long Beach	0.3	1.0	8	31
19	1500203	Bakersfield	0.3	1.0	6	27
20	3400293	Citrus Heights	0.3	1.0	7	24
21	3300144	Rubidoux	0.2	1.0	4	31
		STATEWIDE	1.2	1.7	269	536

*Zero value treatment assigns all observations which are below the LOD a value of 0 ng/m^3 in calculating the "MIN" average and 1 ng/m^3 in calculating the "MAX" average.

TABLE IV-2

1985 AVERAGE AMBIENT CADMIUM EXPOSURE ESTIMATES
BASED ON ZERO VALUE TREATMENTS
(Six Months Data)

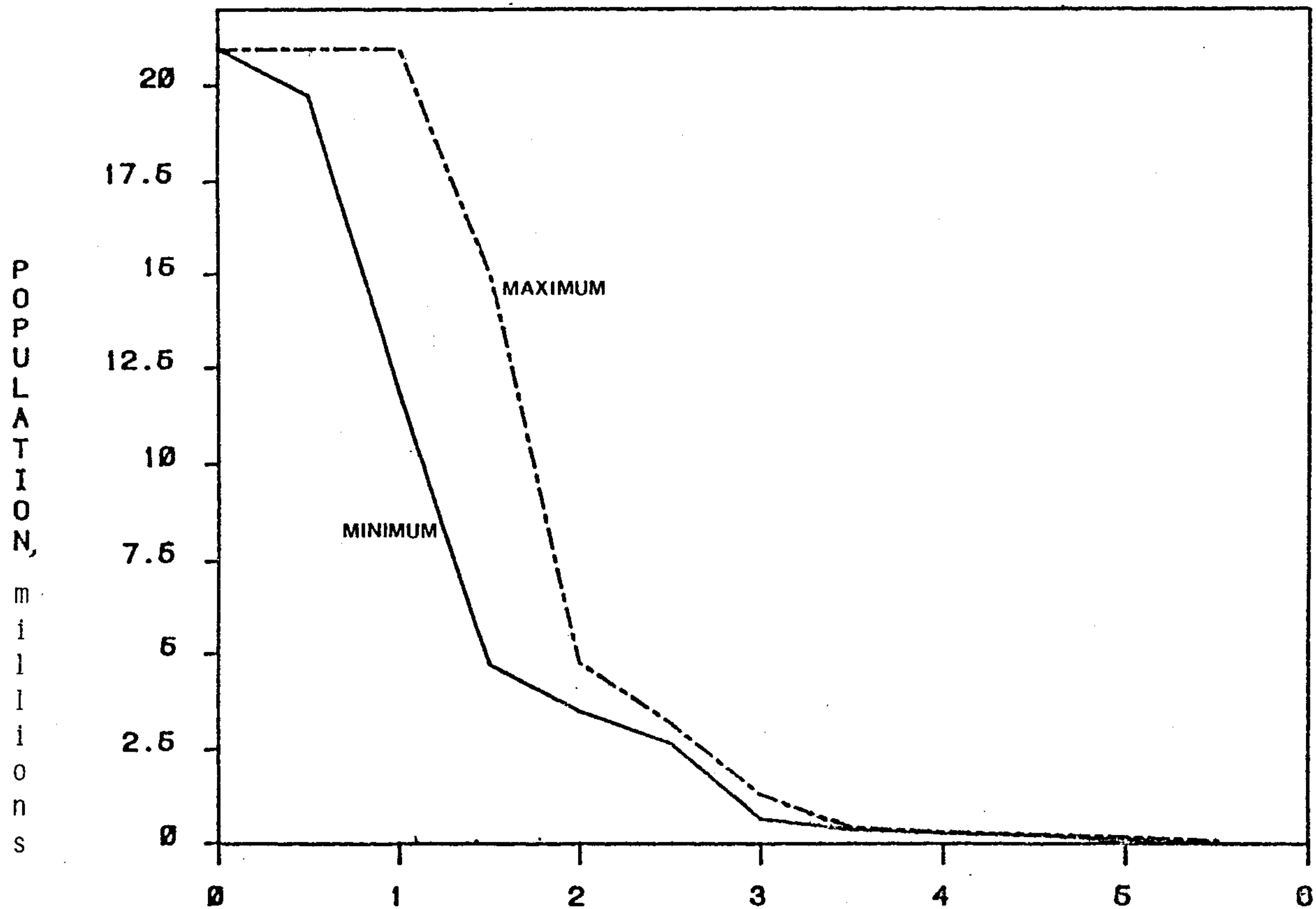
AIR BASIN/COUNTY	1985 AVERAGE CADMIUM EXPOSURE (ng/m^3) MINIMUM	MAXIMUM	POPULATION (millions)
San Francisco Bay Area	2.3	2.5	4.4
South Coast	1.3	1.8	10.1
San Joaquin Valley	0.7	1.3	2.3
San Diego	0.8	1.0	2.1
South Central Coast	0.5	1.0	1.1
Sacramento County	0.3	1.0	0.9
TOTALS	1.3	1.8	20.9

analysis is about 21.0 million people. No cadmium exposure estimates have been made for the remaining 5.4 million people. The following discussion of population exposure is based on an exposed population of 21 million people. Figure IV-1 shows estimated cumulative population exposure for this population using both zero value treatments. The results of this method indicate that approxmately 10 million people are exposed to annual average concentrations of cadmium of at least 1.3 ng/m^3 for the minimum average estimate and 1.8 ng/m^3 for the maximum average estimate. Approximately one million people are exposed to annual average concentrations of cadmium of at least 3.4 ng/m^3 for the minimum average estimate and 3.6 ng/m^3 for the maximum average estimate.

The range of cadmium exposure by using the two zero treatments does not include uncertainty arising from the small sample size or from large variance in measurements. To better estimate the probable range of the average atmospheric cadmium concentration, we have developed a statistical treatment for calculating 95% confidence intervals for the mean concentration at each

Figure IV-1

ESTIMATED CUMULATIVE POPULATION EXPOSURE TO CADMIUM



station. This treatment is believed to provide a better estimate of the true uncertainty associated with the calculated mean because it includes factors such as sample size, standard deviations of the data, and an estimate of the uncertainty of the sample collection and analysis procedures. A description of the statistcal treatment follows. A scatter plot of the cadmium data indicated that the data is probably distributed lognormally. Since this is commonly the case for atmospheric pollutants, it was assumed that the real distribution of ambient cadmium concentrations is lognormal. Because available software only analyzes data that is normally distributed, the cadmium data was first converted from a lognormal distribution to a normal distribution. This was done by using the logarithm of the data for the analysis. The logarithms of data that is lognormally distributed are themselves normally distributed. The statistical analysis system (SAS, 1982) software package was used for the analysis. Additionally, to complete the analysis, it was assumed that the uncertainty for sampling and analysis was 15% and that data below the LOD was equal to 1/2 of the LOD value. Setting all values below LOD to 1/2 the LOD value is expected to bias the confidence intervals to the high side of the mean.

The resulting 95% confidence interval for each station is shown in Figure IV-2. The calculated range of exposure is different from that based on the zero value treatment. For example, the 95% confidence interval calculated using the statistical treatment for the Concord site is 2.96 to 10.81 ng/m^3. This compares to a range of 5.5 to 5.9 ng/m^3 calculated using the previously discussed zero value treatment. We believe the confidence intervals from the second method provide a more realistic estimate of the uncertainty in the actual average. The estimated 95% confidence intervals for each station are shown in Table IV-3. Comparisons of the estimated

Figure IV-2

Cadmium Averages at Stations (ng/m3)
With 95% Confidence Intervals

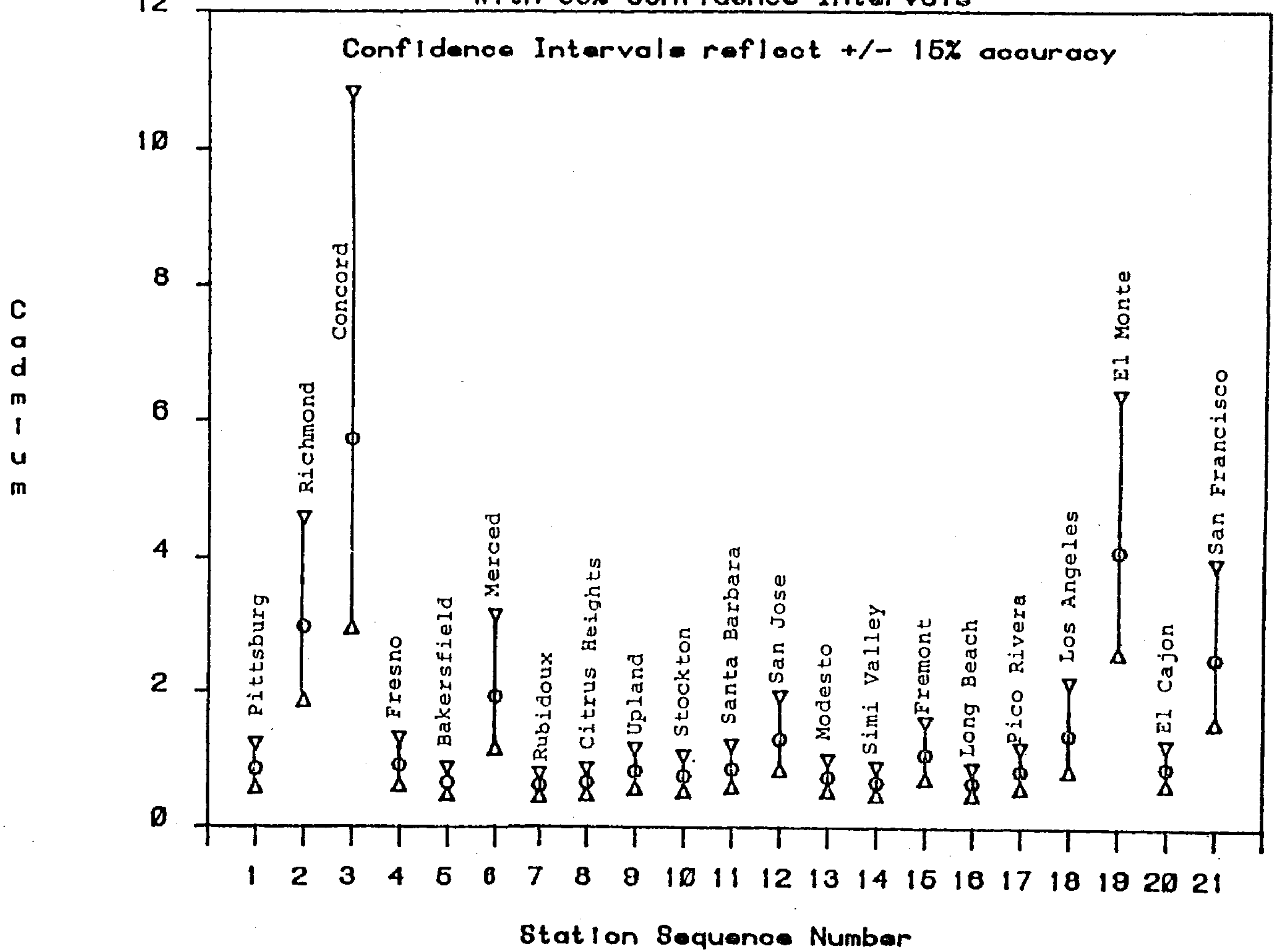


TABLE IV-3

1985 AVERAGE CADMIUM CONCENTRATIONS

ARB CADMIUM SAMPLING NETWORK

WITH 95% CONFIDENCE INTERVALS

(Six Months Data)

RANK	SITE ID	LOCATION	AVERAGES (ng/m^3) MIN	MID	MAX	# SAMPLES ABOVE LOD	TOTAL # SAMPLES
1	0700440	Concord	3.0	5.7	10.8	22	36
2	7000579	El Monte	2.6	4.1	6.4	15	15
3	0700433	Richmond	1.9	3.0	4.6	30	32
4	9000304	San Francisco	1.6	2.5	4.0	26	31
5	2400521	Merced	1.2	1.9	3.1	13	15
6	7000087	Los Angeles	0.9	1.4	2.2	13	29
7	4300382	San Jose	0.9	1.3	1.9	18	30
8	6000336	Fremont	0.7	1.1	1.6	16	31
9	1000234	Fresno	0.6	0.9	1.3	12	22
10	8000131	El Cajon	0.6	0.9	1.2	15	21
11	4200378	Santa Barbara	0.6	0.9	1.2	11	18
12	0700430	Pittsburg	0.6	0.8	1.2	7	10
13	7000085	Pico Rivera	0.6	0.8	1.2	11	31
14	3600175	Upland	0.6	0.8	1.1	9	30
15	3900252	Stockton	0.5	0.8	1.0	8	24
16	5000558	Modesto	0.5	0.7	1.0	12	29
17	5600413	Simi Valley	0.5	0.7	0.9	6	19
18	7000072	No. Long Beach	0.5	0.7	0.9	8	31
19	1500203	Bakersfield	0.5	0.6	0.9	6	27
20	3400293	Citrus Heights	0.5	0.6	0.9	7	24
21	3300144	Rubidoux	0.4	0.6	0.8	4	31
		STATEWIDE	0.9	1.5	2.3	269	536

ranges in annual average concentrations on a site by site basis (Tables IV-1 and IV-3) show that the zero value treatments become unimportant when confidence intervals are used to estimate uncertainty in annual averages. The use of the zero value treatment usually results in a smaller range of uncertainty when compared to the confidence interval method except when averages are near the LOD. When averages are near the LOD both methods give comparable ranges.

Population exposures were also interpolated for the upper and lower confidence intervals for the South Coast and San Francisco air basins. The resulting average exposures for each air basin are shown in Table IV-4. The range in exposure is greater than shown in Table IV-2, while the mean is the same. The annual average cadmium concentration (weighted by population) for the San Francisco Bay Area Air Basin is now estimated to range from 1.5 to 4.7 ng/m^3 while Table IV-2 (reflecting differences due to zero value treatments) shows a range of 2.3 to 2.5 ng/m^3. On a statewide basis, we now estimate that 50% of the population is exposed to at least 1.5 ng/m^3 cadmium, as before, but the range increases from 1.3 - 1.8 ng/m^3 to 1.0 - 2.5 ng/m^3. Five percent of the population is exposed to at least 3.5 ng/m^3 with the range being expanded from 3.4 - 3.6 ng/m^3 to 1.8 - 5.6 ng/m^3. Figure IV-3 shows cadmium concentrations plotted against cumulative population for the mean and the lower and upper 95% confidence limits.

Data are available for only six months (Jan - June) of 1985. To investigate whether concentration averages calculated from this data are representative of annual average concentrations averages, data from other sources were used to compare seasonal averages with annual averages.

FIGURE IV-3

ESTIMATED CUMULATIVE POPULATION EXPOSURE TO CADMIUM

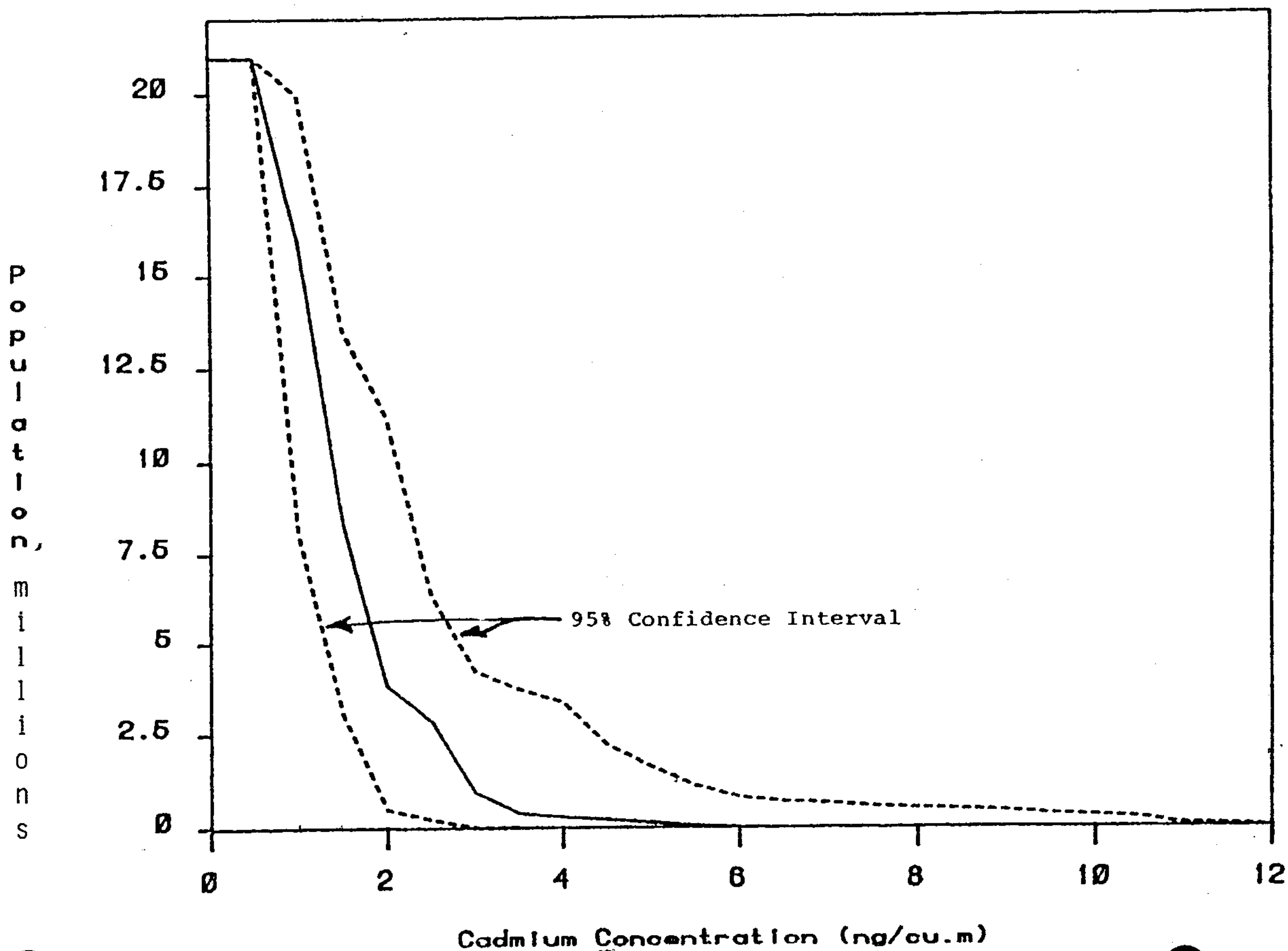


TABLE IV-4

1985 ANNUAL AMBIENT CADMIUM EXPOSURE ESTIMATES
WITH 95% CONFIDENCE INTERVALS
(Six Months Data)

AIR BASIN/COUNTY	1985 AVERAGE CADMIUM EXPOSURE (ng/m^3) LOW	MEAN	HIGH	POPULATION (millions)
San Francisco Bay Area	1.5	2.4	4.7	4.4
South Coast	1.0	1.5	2.3	10.
San Joaquin Valley	0.7	1.0	1.5	2.3
San Diego	0.6	0.9	1.2	2.1
South Central Coast	0.5	0.8	1.0	1.1
Sacramento County	0.5	0.6	0.9	0.9
TOTALS	1.0	1.5	2.5	20.9

Data collected by EPA in 1977 in California show that the ratio of the first half (Jan-June) to second half (July-Dec) of the year for cadmium concentration averages varied from site to site (Table IV-5). The ratios ranged from 0.4 to 2.4 with the overall ratio being 1 for all sites. We are uncertain whether the differences in ratios between sites represent real differences in the ambient concentration or a consequence of the small sample size. When the data is examined for regional trends, the ratios for the San Francisco Bay Area range from 0.6 to 2.4 and there is a range of 0.4 to 1.9 for the Los Angeles Area. The seasonal variation of atmospheric cadmium at an urban and at a rural site in England showed that winter (Oct-March) means were higher than summer (April-Sept) means. (Harrison and Williams, 1982). This may be due to increased emissions of cadmium from increased fossil fuel

TABLE IV-5

AVERAGE CADMIUM CONCENTRATIONS DURING 1977 - FROM EPA DATABASE

LOCATION	FIRST HALF CADMIUM (ng/m^3)	FIRST HALF TOTAL # SAMPLES	SECOND HALF CADMIUM (ng/m^3)	SECOND HALF TOTAL # SAMPLES	RATIO
Anaheim	0.7	15	1.3	15	1.9
Berkeley	1.0	15	0.8	15	0.8
Burbank	2.7	15	2.5	15	0.9
Fresno	1.2	15	1.0	15	0.8
Los Angeles	2.6	15	2.6	15	1.0
Long Beach	1.0	13	1.2	14	1.2
Oakland	1.8	15	1.1	14	0.6
Ontario	2.4	15	2.2	15	0.9
Pasadena	1.3	14	1.0	15	0.8
Sacramento	0.6	15	0.7	15	1.2
San Bernadino	1.7	14	1.8	15	1.1
San Diego	1.1	15	0.9	15	0.8
San Francisco	0.7	15	1.7	15	1.0
San Jose	0.7	15	1.7	15	2.4
Santa Ana	0.7	15	0.8	14	1.1
Torrance	3.8	14	1.7	15	0.4
				OVERALL AVERAGE	1.0

combustion during the colder winter months. The January through June period for which we have 1985 data includes both 'winter' months and 'summer' months. Because the overall ratio for the first-to-second half mean concentrations was 1.0, the sampling period includes both summer and winter months, and ARB sites cover the same areas as the EPA sites, we believe that the average concentrations calculated from the available data provide a reasonable estimate of annual average concentrations for all of the California sites. However, the annual average concentration at individual sites could be different from the ARB average by a factor of 2; site-specific ratios are between one-half and two.

Further evidence to support this conclusion is that the estimates of average cadmium concentration presented in this report are in the range of concentrations measured by others in California. Saltzman, et. al., (1985) calculated a annual geometric mean cadmium concentration for the Los Angeles area of 2 ng/m^3. Sampling was carried out at eight sites during 1968-1969; 1,841 samples were collected. The authors reported that only a small fraction of samples were below the detection limit, in which case a value of one-half the detection limit was substituted. Also, data on atmospheric cadmium in California were gathered by the U.S. EPA during 1977. The U.S. EPA data contained a greater percentage than current ARB data of values below the detection limit, which limited its usefulness in estimating population exposure.

The size distribution of cadmium and its compounds were not determined in the ARB's measurements. Work by others on the size distribution of cadmium have shown that cadmium exhibits a tendency to be present at higher concentrations on small particles and to be distributed bi-modally, with a concentration maximum in the 1 um range, and a smaller mode in the 3-10 um range. Harrison, et. al. (1971) showed this for urban aerosols in Michigan. Lee, et. al., (1968) estimated the mass median diameter of cadmium at an urban and a rural site to be 3.1 um and 10 um respectively. However, the sampling procedure employed by Lee, et. al., has been shown to bias the data to larger particle sizes, due to large particle bounce off in the first impactor stages (Dzubay, et. al., 1976; Lawson, 1980). Work on cadmium size distribution in remote areas (Davidson, et. al., 1985) yielded estimates of mass median diameters (MMD) of 0.28 and 0.56 um. Measurements in Europe of cadmium particle size (away from known cadmium sources) gave an estimate of 0.4 um MMD, and a fraction of 64 percent below 1.1 um (Duc and Favez, 1981). The MMD of atmospheric cadmium in Glasgow was determined to be 0.6 um (McDonald and Duncan, 1979). Davidson, et. al., (1981) reported a cadmium MMD of 1.5 um in an industrial section of Pittsburg, with a bimodal distribution observed. Milford and Davidson (1985) have surveyed work done on atmospheric cadmium particle size and calculated the MMD for atmospheric cadmium using data from 14 studies of remote, urban, and industrial sites. This average MMD was 0.84 um. Data from an urban California site was included in that survey; the California data (Davidson, 1977) were consistent with data from other locations. Because of the nature of cadmium sources in the State (principally high-temperature sources emitting cadmium on particles in the micron to submicron range), and the surprisingly consistent size distribution of cadmium in different studies, we believe that atmospheric cadmium in California occurs largely on particles in the respirable size range (less than 2.5 um diameter).

The compound forms of cadmium present in the atmosphere have not been determined. Study of the solubility of cadmium on particulate matter both in California (Hodge, et. al., 1978), and elsewhere (Lindberg and Harriss, 1983), indicate that 70 - 84 percent of atmospheric cadmium that is acid soluble is also water soluble. Based on the expected forms of metals emitted from combustion processes and other high-temperature processes (see Section III B, C), we conclude that atmospheric cadmium occurs primarily as the soluble sulfate, phosphate, and fluoride, with the balance occuring as the insoluble oxide or carbonate.

Because cadmium is an element found in most crustal materials at concentrations typically between 0.1-0.2 parts per million, we evaluated the contribution of crustal materials to concentrations of atmospheric cadmium. Comparison of the composition of atmospheric particulate matter to that of crustal materials has been made to assess the contribution of crustal materials to the atmospheric burden of trace metals, including cadmium. The degree of enrichment of an element in atmospheric particulate matter had been calculated as the enrichment factor, EF:

$$\frac{C_{Cd}/C_{Al}}{C_{Cd,crust}/C_{Al,crust}}$$

where C_{Cd} and C_{Al} are the atmospheric concentrations of these metals, and $C_{Cd,\ crust}$ and $C_{Al,\ crust}$ are the concentrations in the earth's crust. In most cases, average values of crustal abundance are used; in some cases, values specific to the region under study are employed. Values of EF less than 5 are considered indicative of a crustal or soil source of the element;

higher values suggest non-soil sources, including high temperature industrial processes and fuel combustion. Certain natural processes may also lead to atmospheric enrichment of certain elements: volcanism, direct sublimation from crustal materials, emissions from vegetation, and sea spray enrichment (Duce, et. al., 1975). In most urban areas, however, large EF factors are considered to be indicative of anthropogenic sources (Heindryckx, 1976). Large cadmium enrichment factors have been determined in remote areas (EF 2500) with increasing value of EF with decreasing particle size (Davidson, et. al., 1985). Lindberg and Harriss reported lower EFs for cadmium (7-23) in continental aerosols; the tendency to increasing EF with decreasing particle size was observed. Davidson et. al., (1981) found an average cadmium EF in Pittsburg of 630, and observed the same trend in increasing EF, with decreasing particle size. McDonald and Duncan (1979) reported EFs for cadmium in Glasgow ranging between 750 and 8,400, with particles in the range of 0.43 - 0.7 um exhibiting the highest EF.

Milford and Davidson (1985) provided summary statistics including cadmium EF from 14 studies in urban, rural, and industrial sites; an average EF of 1,900 was reported. This average included results of one study done in California urban area (Davidson, 1977). Based on this information, we believe it is reasonable to assume that atmospheric cadmium in California results predominantly from non-crustal sources.

Because a large amount of data on total suspended particulate matter (TSP), collected by both ARB and EPA, is available, an effort was made to determine a relationship between TSP and cadmium measurements. First, EPA cadmium data from samples collected throughout California during 1977 through 1983 were analyzed to determine if cadmium concentrations were correlated to simultaneous occurences of total suspended particulate matter (TSP). If the two were highly correlated, cadmium concentrations could be estimated using the much larger TSP database. No significant correlations were found using the EPA data. Correlation coefficients (r) were below 0.53 at nineteen of twenty sampling sites having both cadmium and TSP data; an r value of 0.69 was calculated for cadmium-TSP data collected at Long Beach. The overall correlation coefficient was less than 0.01 for 1409 paired observations.

The same statistical calculations were made using the 1985 ARB cadmium data. To do this, we extracted twenty-four hour average TSP concentrations from the ARB air quality database for the same time periods and stations as the cadmium data contained in the ARB toxics air quality database. The cadmium-TSP correlation results for each station are given in Table IV-6. As with the EPA data, the ARB data showed little correlation between ambient cadmium and TSP concentrations, except for a few stations. Fresno data showed a very high correlation coefficient, 0.94, for a sample size of 22. The next highest correlation coefficient was 0.68 at the Upland station for 30 samples. The r value calculated for the North Long Beach site, 0.67, was quite similar to the r value calculated for the EPA Long Beach data (0.69). Thirteen of the seventeen ARB stations having comparable cadmium and TSP data had correlation coefficients below 0.54; scatter plots for the remaining three stations showed almost no variation in cadmium concentrations. As a result, we again find no useful correlations between cadmium and TSP.

All of the above discussion concerns outdoor concentrations of cadmium. ARB has made no measurement of indoor concentrations. However, Seifert, et. al., (1984) studied indoor heavy metal exposure near a secondary lead smelter,

TABLE IV-6

1985 CADMIUM AND TSP CORRELATIONS
ARB CADMIUM SAMPLING NETWORK

STATION ID	LOCATION	CORRELATION COEFFICIENT	# SAMPLES ABOVE LOD	TOTAL # SAMPLES
0700430	Pittsburg	-	7	10
0700433	Richmond	0.07	30	32
07004400	Concord	0.22	22	36
1000234	Fresno	0.94	12	22
1500203	Bakersfield	- 0.16	6	27
2400521	Merced	- 0.11	13	15
3300144	Rubidoux	- 0.06	4	31
3400293	Citrus Heights	-	7	24
3600175	Upland	0.68	9	30
3900252	Stockton	- 0.57	8	24
4200378	Santa Barbara	- 0.02	11	18
4300382	San Jose	0.53	18	30
5000558	Modesto	0.16	12	29
5600413	Simi Valley	-	6	19
6000336	Fremont	0.23	16	31
7000072	N. Long Beach	0.67	8	31
7000085	Pico Rivera	- 0.26	11	31
7000087	Los Angeles	0.47	13	29
7000579	El Monte	-	15	15
8000131	El Cajon	0.19	15	21
9000304	San Francisco	- 0.41	26	31
	STATEWIDE	- 0.09	269	536

and found that the indoor metal burden could be very different in adjacent houses. The "maintenance conditions" (window tightness) of the building (i.e., air exchange rate), and the nature of the building's immediate surroundings (vegetation), which would effect deposition and reentrainment, were believed to be significant factors in influencing indoor metal concentrations. Because of the lack of available data relating indoor to outdoor cadmium concentrations, no attempt was made to estimate indoor cadmium exposure.

C. Exposure Close to Sources

To evaluate increased exposure to atmospheric cadmium for people living close to sources, we calculated the cumulative air quality impact of three secondary copper smelters in the South Coast Air Basin. Emissions from the three facilities were estimated by making certain assumptions using information on the process rate (tons of scrap processed per year), facility operations, and an emission factor of 3.0 lb cadmium/ton of scrap. This emission factor was derived from information on the cadmium content of various types of copper scrap. Cadmium emissions from three smelters were estimated to range from 1.7 to 3.4 tons/year. The upper value represents maximum worst case emissions, and the lower value represents a worst-plausible case.

The range of emissions rate estimates for each of the three smelters was used as input to the Gaussian air quality model Industrial Source Complex - short term (ISCST). Historical meteorological data from a station close to the sources for three years (1976, 1977, and 1978) were used to run the model. The difference in meterological conditions among the three years yield variations in results which were small. Results produced using 1977 met data (the highest results) are discussed here. Where stack parameters for the sources were available, they were used; in some cases, the data were unavailable and the stack parameters which would yield worst case ambient concentrations were used. Because of the assumptions made, we do not expect cadmium exposure due to emissions from these sources to be higher than our estimates. To estimate exposure, the modeled ambient concentrations were superimposed on the population data for the area based on census tract centroids. The area contained a residential population of 5.7 million.

Figure IV-4 shows the cumulative exposure attributable to emissions from these three smelters.

The modeled exposure levels were: 20 ng/m^3 for 57,000 people; 5-7 ng/m^3 for 290,000 people; and 0.5 - 0.6 ng/m^3 for 2.8 million people. The exposure estimates, based on maximum possible emission rates, are double these values. The general exposure in the South Coast Air Basin (based on direct measurement of atmospheric cadmium) has been estimated to be 1-2.3 ng/m^3.

In interpreting data on exposure close to sources of cadmium, it should be realized that sources of cadmium emissions usually are also emitters of other metals and compounds which may have potential adverse health effects. It is beyond the scope of this report to quantitatively address this issue; Table IV-7 lists non-criteria pollutants which may have chronic health effects which are known or are likely to be emitted from cadmium emission sources.

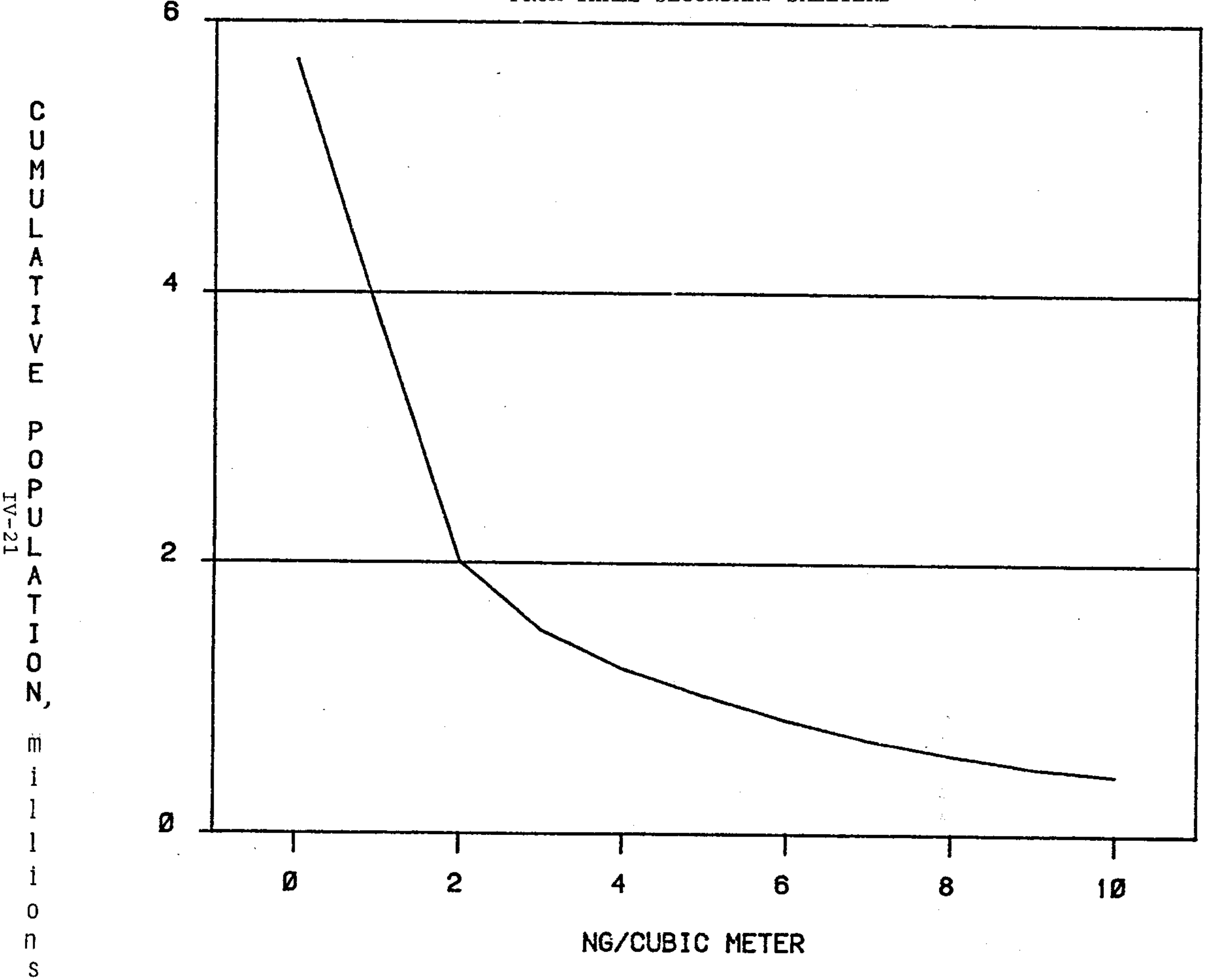
Table IV-7

Selected Non-Criteria Pollutants Which May be Emitted from Sources of Cadmium

Source Type	Pollutants
Combustion processes	Metals (arsenic, mercury, nickel); polycyclic aromatic hydrocarbons (PAH); chlorinated dioxins
Smelters	Lead; arsenic; chromium; chlorinated dioxins
Gasoline-powered vehicles	Benzene, ethylene dibromide; ethylene dichloride; PAH

Figure IV-4

ESTIMATED CADMIUM EXPOSURE DUE TO EMISSIONS
FROM THREE SECONDARY SMELTERS



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APPENDIX A

INFORMATION REQUEST LETTER WITH
ATTACHMENTS AND RESPONSES

AIR RESOURCES BOARD
1102 Q STREET
P.O. BOX 2815
SACRAMENTO, CA 95812

February 4, 1985

Dear Sir or Madam:

Subject: Request for Information Regarding Cadmium

I am writing to request information on the health effects of cadmium as part of our toxic air contaminant program. This program is based on Health and Safety Code Sections 39650, et seq. which require the ARB to identify compounds as toxic air contaminants and once identified to develop and adopt control measures for such compounds. After consultation with the staff of the Department of Health Services (DHS), we have selected cadmium as a candidate toxic air contaminant to be evaluated in accordance with the provisions of Health and Safety Code Sections 39650, et seq. During our evaluation of cadmium, we will consider available health information on all forms and compounds of cadmium. Additionally, we are soliciting information regarding environmental and biological transformations of cadmium and its compounds.

Before the ARB can formally identify a compound as a toxic air contaminant, several steps must be taken. First, the ARB must request the Department of Health Services to evaluate the health effects of candidate compounds. Second, the ARB staff must prepare a report which includes the health effects evaluation and then submit the report to a Scientific Review Panel for its review. The report submitted to the Panel will be made available to the public. Information submitted in response to this request will be considered in the ARB report to the Panel. Although any person may also submit information directly to the Panel for its consideration, I urge you to submit all information at this time for our consideration in the development of the report for the Panel. The Panel reviews the sufficiency of the information, methods, and data used by the DHS in its evaluation. Lastly, after review by the Scientific Review Panel, the report with the written findings of the Panel will be considered by the Air Resources Board and will be the basis for any regulatory action by the Board to officially identify a compound as a toxic air contaminant.

Prior to formally requesting the DHS to prepare a health effects evaluation of cadmium, we are providing, pursuant to the provisions of Section 39660(e) of the Health and Safety Code, an opportunity to interested parties to submit information on the health effects of cadmium which he or she believes would be important in DHS's evaluation of cadmium as a candidate toxic air contaminant.

In January 1985, ARB staff received a reference search on cadmium health effects using the MEDLINE and TOXLINE Information Services. These information services include material available to the public on or before September 1984. The attached bibliography lists the references from this information search. We are requesting pertinent information on cadmium health effects, including any material that may not be available to the public, that is not included in the attached bibliography.

Pursuant to the provisions of the Public Records Act (Government Code Sections 6280 et seq.), the information you provide will be a public record and subject to public disclosure, except for trade secrets which are not emission data or other information which is exempt from disclosure or the disclosure of which is prohibited by law. The information may also be released to the Environmental Protection Agency, which protects trade secrets and confidential information in accordance with federal law, and to other public agencies, which are also required to protect such information.

To expedite the review process, we ask that any information which you believe should be regarded as "trade secret" be clearly marked and separated from other information. You may identify portions of the information you submit as "trade secret" in accordance with Health and Safety Code Section 39660(e). The claim of trade secrecy must be supported upon the request of the Air Resources Board. Other information claimed to be trade secret and information otherwise claimed to be exempt from disclosure may be identified as confidential in accordance with Section 91011, Title 17, California Administrative Code. Section 91011 requires that the claim of confidentiality be accompanied by specified supporting information.

I would appreciate receiving any relevant information you wish to submit by March 22, 1985. Your help in expediting our review will be greatly appreciated. Please send the information to the attention of:

William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Cadmium
California Air Resources Board
P. O. Box 2815
Sacramento, CA 95812

If you have any further questions regarding health effects information, please contact Mr. John Batchelder at (916) 323-1505. For any other questions, please contact Mr. Robert Barham at (916) 322-7072.

If you are not the person to whom this request should be addressed, please forward it to the appropriate person in your organization. Also, please let us know whether you would like to continue to receive information inquiries for other candidate compounds, and if not, if there is anyone in your organization to whom such requests should be sent.

Sincerely,

Peter D. Venturini, Chief
Stationary Source Division

cc: Alex Kelter, DHS
Lori Johnston, DFA
Wayne Morgan, President, CAPCOA
Jan Bush, Executive Secretary, CAPCOA
David Howekamp, EPA Region IX
Assemblywoman Sally Tanner, Chairwoman, Committee on Toxic Materials
Senator Ralph Dills, Chairman, Committee on Governmental Organization
Senator Art Torres, Chairman, Committee on Toxics and Public Safety Management
Emil Mrak, Chairman and Scientific Review Panel Members
APCOs

Attachment

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Oil, Chemical and Atomic Workers International Union

J. E. (JACK) FOLEY
DIRECTOR, DISTRICT NO. 1

304 FREEWAY CENTER BUILDING
3605 LONG BEACH BOULEVARD
LONG BEACH, CALIFORNIA 90807
PHONE: (213) 426-6961

February 8, 1985

Mr. Peter D. Venturini, Chief
Stationary Source Division
AIR RESOURCES BOARD
1102 "Q" Street
Sacramento, California 95812

Dear Mr. Venturini:

In response to your communication regarding Cadmium that matter has been referred to our headquarters in Denver, Colorado Health & Safety Dept.

I would appreciate any request of this nature, in the future, be sent our Denver office, addressed as follows: Oil, Chemical and Atomic Workers International Union, Health & Safety Department, P.O. Box 2812, Denver, CO. 80201 to the attention of Director Dan Edwards with a copy to this office, as addressed on this letterhead.

Thank you for keeping us informed.

Very truly yours,

J.E. Foley
Director, District #1

JEF:rmm
cc: D. Edwards
File

Memorandum

To : Peter D. Venturini, Chief
Stationary Source Division
Air Resources Board
1102 Q Street
Sacramento, CA 95814

Date : February 13, 1985

Place : Sacramento

From : **Department of Food and Agriculture**

Subject: Request for Information Regarding Cadmium

Thank you for your letter regarding your information search on cadmium. Currently, only one pesticide containing cadmium is registered for agricultural use in California. Cleary's Granular Turf Fungicide which contains 0.75% cadmium (as cadmium chloride) is not widely used in California.

The Department has no additional health effects data on cadmium to contribute.

R. V. Petersen for

Lori Johnston, Assistant Director
Pest Management, Environmental
Protection and Worker Safety
(916) 322-6315

cc Assemblywoman Sally Tanner
William V. Loscutoff, Chief, ARB
Alex Kelter, DHS

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February 13, 1985

Peter D. Venturini, Chief
Stationary Source Division
State of California
Air Resources Board
1102 Q Street
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Venturini:

Your request for information on cadmium was forwarded to me. I must report that we have no health effects information on cadmium. But, please add me to your mailing list to receive your information inquiries in the future.

Sincerely yours,

Eugene G. Wood

Eugene G. Wood
Manager,
Industrial Hygiene

EGW:mse

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FEB 19 1985

Stationary Source
Division
Air Resources Board

JAN 1 9 1985

UNIVERSITY OF WASHINGTON
SEATTLE, WASHINGTON 98195

School of Public Health and Community Medicine
Department of Environmental Health, SC-34

February 12, 1985

William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Cadmium
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Dr. Loscutoff:

Please find listed below several references on the developmental toxicity of Cadmium:

Barr, M: The teratogenicity of cadmium chloride in two stocks of Wistar rats. Teratology 7:237-242, 1973.

Chernoff, N.: Teratogenic effects of cadmium in rats. Teratology 8:29-32, 1973.

Cvetkova, R.P.: Materials on the study of the influence of cadmium compounds on the generative function. Gig. Tr. Prof. Zabol. 14:31, 1970.

Dencker, L.: Possible mechanisms of cadmium fetotoxicity in golden hamsters and mice: uptake by the embryo, placenta and ovary. J. Reprod. Fert. 44:461-471, 1975.

Levin, A.A. and Miller, R.K.: Fetal toxicity of cadmium in rat: Decreased utero-placental blood flow. Toxicol. Appl. Pharm. 58: 297-306, 1981.

Mulvihill, J.E.; Gamm, S.H. and Ferm, V.H.: Facial formation in normal and cadmium-treated golden hamsters. J. Embryol. Exp. Morphol. 24:393-403, 1970.

Thueraut, J.; Schaller, K.H.; Engelhardt, E. and Gossler K.: The cadmium content of the human placenta. Int. Arch. Occup. Environ. Health 36:19-27, 1975.

An excellent reference source for further information on the developmental toxicity of compounds is:

T.H. Shepard
Catalog of Teratogenic Agents
Johns Hopkins Press, Baltimore, 1984.

Several general references that you may want to include in your references are:

Hutton, M.: Sources of Cadmium in the Environment. Ecotoxic. and Environ. Safety. 7:9-24, 1983.

Itokawa, Y., Abe. T., Tabei, R., and Tanaka, S.: Renal and Skeletal Lesions in Experimental Cadmium Poisoning. Arch. Environ. Health. 28:149-154, 1974.

Korte, F. Ecotoxicology of Cadmium: General Overview. Ecotoxic. and Environ. Safety. 7:3-8, 1983.

Lauwerys, R.R., Buchet, J.P., Roels, H.A., Brouwers, J., and Stanescu, D.: Epidemiological Survey of Workers Exposed to Cadmium. Arch. Environ. Health. 28:145-?, 1974.

Lemen, R.A., Lee, J.S., Wagoner, J.K., and Blejer, H.: Cancer Mortality Among Cadmium Production Workers. Ann. N.Y. Acad. Sci. 271:273-?, 1976.

Rieth, F.H., Stocker, W.G., and Thiess, A.M.: Chromosome Investigations of Workers Exposed to Cadmium in the Manufacturing of Cadmium Stabilizers and Pigments. Ecotoxic. Environ. Safety. 7:106-?, 1983.

Quaife, C., Durnam, D., Mottet, N.K.: Cadmium hypersusceptability in C34 mouse liver: cell specificity and possible role of metallothionein. Tox. Applied Pharm. 76:9-17, 1984.

I hope this information is useful.

Sincerely,

Elaine Faustman-Watts

Elaine Faustman-Watts
Assistant Professor

EFW:jt

Ford Motor Company

The American Road
Dearborn, Michigan 48121

February 18, 1985

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Cadmium and Asbestos
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Subject: Response to Mr. P. D. Venturini's Requests for Information Regarding Cadmium and Asbestos

Dear Mr. Loscutoff:

The Ford Motor Company has not undertaken independent scientific studies to evaluate the health effects relating to either cadmium or asbestos. Rather, the Company quantitatively measures the ambient concentrations within the plant environments of regulated and-suspected toxic air contaminants. These concentrations are evaluated with respect to the current Occupational Safety and Health Administration permissible exposure limits, National Institute of Occupational Safety and Health recommended standards, and American Conference of Governmental Industrial Hygienists Threshold Limit Values.

We regret that we are unable to submit information pursuant to your inquiries concerning health effects but we would like to continue to receive information on your progress in regulating toxic air contaminants.

Yours truly,

Frank P. Partee

F. P. Partee
Principal Staff Engineer
Air/Noise Compliance
Stationary Source Environmental
Control Office

10:DKJ7/L

UNIVERSITY OF CALIFORNIA, RIVERSIDE

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COLLEGE OF NATURAL AND AGRICULTURAL SCIENCES
CITRUS RESEARCH CENTER AND
AGRICULTURAL EXPERIMENT STATION
DEPARTMENT OF SOIL AND ENVIRONMENTAL SCIENCES

RIVERSIDE, CALIFORNIA 92521

February 27, 1985

RECEIVED
MAR 4 1985
Stationary Source
Division
Air Resources Board

Mr. Peter D. Venturini, Chief
Stationary Source Division
Air Resources Board
1102 Q. Street
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Venturini:

This is a response to your request for information regarding cadmium. The following two articles published by the Department of Soil and Environmental Sciences, University of California, Riverside, CA, are submitted, herewith, for your consideration. Both papers dealt with environmental and biological transformations of cadmium:

1. Page, A. L. and A. C. Chang. 1979. Contamination of soil and vegetation by atmospheric deposition of trace metals. Phytopathology 69(9):1007-1011.

2. Page, A. L., M. M. El-Amamy and A. C. Chang. 1984. Cadmium in the environment and its entry into terrestrial food chain crops (to be published as Chap. 2 in a cadmium book; publisher Springer-Verlag, New York, N.Y.).

Sincerely,

A. C. Chang

ACC:jt
Enclosures

cc: M. M. El-Amamy
A. L. Page

Cadmium Council, Inc.
292 Madison Avenue
New York, N.Y. 10017

212 578-4750

February 28, 1985

Lynn Terry
Stationary Sources Division
Air Resources Board
1102 Q Street
P.O. Box 2815
Sacramento, CA 95812

Dear Ms. Terry,

I am sorry for the delay in responding to your request but the Yost and Greenkorn study was being printed. I have also included the proceedings from the Second, Third, and Fourth International Cadmium Conferences, a book entitled Cadmium Chemicals, and an article, "The Effect of Cadmium on the Environment" by J. F. Cole and R. A. Volpe.

I will gladly carry out a data search for you if you need further information. I will be calling you shortly to see ~~if~~ I may be of further assistance.

Sincerely,

Giovina L. Leone
Assistant Director
Environmental Regulations

mfcl

enclosures

DEPARTMENT OF THE ARMY
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY
ABERDEEN PROVING GROUND, MARYLAND 21010-5422

MAR 12 1985

REPLY TO
ATTENTION OF
Occupational and Environmental
Medicine Division

William V. Loscutoff
Chief, Toxic Pollutants Branch
California Air Resources Branch
P.O. Box 2815
Sacramento, California 95812

Dear Mr. Loscutoff:

While the US Army Environmental Hygiene Agency does not conduct research into the toxicology of cadmium, we are aware of an EPA draft document, not included in your bibliography, which you may wish to review. The title is: Updated Mutagenicity and Carcinogenicity Assessment of Cadmium, Addendum to the Health Assessment Document for Cadmium (May 1981, EPA-600/8-81-023), EPA-600/8-83-025B, April 1984, External Review Draft. This document is available from the National Technical Information Service.

Any comments should be directed to Major Robert W. Petzold, M.D. at this Agency.

Sincerely,

Joel C. Gaydos
Joel C. Gaydos
Colonel, Medical Corps
Director, Occupational and
Environmental Health

MANUFACTURERS'
ASSOCIATION

Representing the Color Pigments Industry

SUITE 202, 206 NORTH WASHINGTON STREET
ALEXANDRIA, VA 22314 (703) 684-4044

Mailing Address:
P.O. BOX 931, ALEXANDRIA, VA. 22313

Item 1r

March 29, 1985

William V. Loscutoff, Chief
Toxic Pollutants Branch
Re: Cadmium
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Sir:

Subject: Request for Information Regarding Cadmium

The Dry Color Manufacturers' Association (DCMA) appreciates receiving from the California Air Resources Board their request to furnish information on the health effects of cadmium.

The DCMA is an industry trade association representing small, medium and large pigment color manufacturers throughout the United States and Canada, accounting for approximately 95% of the production of color pigments in these countries. Included within the DCMA structure is the Cadmium Pigments Committee (the "Committee") which is composed of major manufacturers of cadmium pigments.

The Committee has been active for many years in addressing safety and health concerns with respect to cadmium pigments. It has provided information to interested government agencies and to the public with respect to these concerns. In addition, the Committee has sponsored various testing activities and studies conducted by consulting professors, laboratories and institutions. Consequently, the Committee is generally recognized as the primary source of expert industry information on cadmium pigments, including safety and health concerns. The Committee is vitally interested in any development that might affect the use of these important color pigments.

While the Committee has undertaken animal studies to demonstrate that cadmium pigments, owing to their insolubility, are less bio-available than more soluble cadmium compounds both by ingestion and inhalation, no studies have been undertaken by the Committee that relate to levels of cadmium in the atmosphere over urban and rural areas.

It is noted that the list of references on cadmium enclosed with your letter of February 4, 1985 does not include a comprehensive review of environmental levels of cadmium prepared by Dr. Kenneth J. Yost of Purdue University, West Lafayette, Indiana entitled "Cadmium, the Environment and Human Health: An Overview" (Experientia 40 (1984) Birkhauser Verlag CH-4010 Basel/Switzerland). A copy is enclosed. Taking into account the production of cadmium, its major uses and disposal into the environment, this report concludes that "respiratory (inhalation) intake of cadmium by urban populations is negligible".

You are no doubt aware of the data available on ambient air levels of cadmium in Section 4.2.1 of the U.S. Environmental Protection Agency "Health Assessment Document for Cadmium" prepared by L.D. Grant, et al. and dated October 1981 (EPA 600/8-81-023). With few exceptions the average annual cadmium levels for U.S. cities studied fall within a range of 1 to 20 nanograms/cubic meter of air. From these values it has been estimated that cadmium in the ambient air contributes less than 2 micrograms per day of an adult's uptake of cadmium as compared to from 20 to 50 micrograms per day by way of the diet.

You may find of interest the enclosed booklet published by the Committee several years ago entitled, "Cadmium Pigments - An Encouraging Outlook".

It is understood that the health evaluation report to be prepared by the ARB staff for submission to a Scientific Review Panel will be made available to the public. Your sending us a copy of this report when available or advising us how to obtain a copy will be very much appreciated.

Sincerely,

J. Lawrence Robinson
Executive Vice President

Enclosures (Yost Paper and "Cadmium Pigments - An Encouraging Outlook")

cc: Peter D. Venturini, Chief
Stationary Source Division
State of California
Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

PACIFIC GAS AND ELECTRIC COMPANY

PG&E 77 BEALE STREET • SAN FRANCISCO, CALIFORNIA 94106 • (415) 781-4211 • TWX 910-372-6587

April 16, 1985

Mr. William V. Loscutoff, Chief
Toxic Pollutants Branch
RE: Cadmium
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Loscutoff:

Request for Public Health
Information Regarding Cadmium

Pacific Gas and Electric Company received your February 4, 1985 request for additional public health information regarding Cadmium. We reviewed the bibliography and concluded that we are unaware of any additional information which would be of use to you.

PGandE would like to remain on the Cadmium mailing list, and would appreciate receiving copies of future announcements on reports. Please send future communications to me at:

Pacific Gas and Electric Company
77 Beale Street - Room 1357
P.O. Box 7640
San Francisco, CA 94124

Thank you.

Sincerely,

J. F. McKENZIE

DEPARTMENT OF FOOD AND AGRICULTURE

1220 N Street
Sacramento
95814

April 22, 1985

William V. Loscutoff, Chief
Toxic Pollutants Branch
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Dear Mr. Loscutoff

Subject: Public Records Request
Our File PR-85-05

Enclosed please find the data on cadmium requested in your letter of February 4. Mr. Bill Erdman of Mallinckrodt Inc. has indicated his company will not respond to our letter of February 27. Accordingly, the two studies are being released.

Sincerely

Lisa Brown

Lisa Brown, Staff Counsel
Pesticide Enforcement Unit
(916) 445-5895

cc Bill Erdman
Alex Kelter, DHS
Lori Johnston, CDFA
Wayne Morgan, President CAPCOA
Jan Bush, Executive Secretary, CAPCOA
David Howekamp, EPA Region IX
Assemblywoman Salley Tanner
Senator Ralph Dills
Senator Art Torres
Emil Mrak, Chair, SAP

APPENDIX B

HEALTH EFFECTS REQUEST TO DHS AND LETTER OF RESPONSE

Memorandum

To : Kenneth Kizer, M.D., Director
Department of Health Services
714 P Street
Sacramento, CA 95814

Date : April 12, 1985

Subject: Evaluation of Cadmium

James D. Boyd
Executive Officer
From : Air Resources Board

I am writing to formally request that the Department evaluate the health effects of cadmium as a candidate toxic air contaminant in accordance with Assembly Bill 1807 (Tanner). According to Health and Safety Code Sections 39660-62, your Department has ninety days to submit a written evaluation and recommendations on the health effects of cadmium to the Air Resources Board and may request a thirty day extension.

Attached for your staff's consideration in evaluating cadmium are: Attachment I - a suggested list of topics that we believe should be included in your cadmium evaluation and recommendations; Attachment II - a list of references on cadmium health effects which were identified in an ARB letter of public inquiry or received in response to the inquiry letter; and Attachment III - ambient cadmium concentration data which should be used to estimate the range of risk to California residents as required in Health and Safety Code Section 39660(c).

My staff is available for consultation in conducting this health effects evaluation. We look forward to continuing to work closely with you and your staff in carrying out this legislative mandate. If you have any further questions regarding this matter, please contact me at 445-4383 or have your staff contact Peter D. Venturini, Chief of the Stationary Source Division, at 445-0650.

Attachments

cc: Gordon Duffy
Alex Kelter, DHS, w/attachments
Raymond Neutra, DHS, w/attachments
Peter D. Venturini, ARB
Assemblywoman Sally Tanner
Claire Berryhill, DFA
Emil Mrak, Chairman and Members of the Scientific Review Panel
Senator Ralph Dills
Senator Art Torres
John Holmes, ARB

ATTACHMENT I
Suggested List of Topics

CADMIUM

I. Chemical and Physical Properties

A. Cadmium compounds

B. Production and use

C. Occurrence

1. Natural

2. Airborne, water, soil

3. Cigarette Smoke

4. Occupational

5. Plant Uptake

6. Food

II. Acute Health Effects

A. Acute pulmonary toxicity

B. Chronic renal damage (see Section V)

III. Carcinogenicity

A. Epidemiologic evidence

1. Lemen study (1976). Cohort of 292 workers in cadmium production facility. An excess of malignant respiratory disease found as was an excess of cancer of the prostate.

2. Varner study (1983). An enlarged version of the Lemen study that demonstrated dose-response relationships for both lung cancer and total neoplasms.

3. Thun study (1984). Cohort of Lemen expanded to 602 white males with at least 6 months work in cadmium production between 1940 - 1969. This cohort was followed through 1978. Mortality from cancer of the respiratory tract (but not the prostate) was significantly greater in the cohort than the general population (2:1). Cancer mortality increased with an increase in exposure. Thun considered both possible confounders of arsenic and smoking and dismissed both.

4. Other epidemiologic studies: Sorahan et al., 1983; Armstrong et al., 1983, 1982; Holden H., 1980; providing limited additional epidemiologic evidence for excess lung cancer mortality.

5. C. A. G.: Further evidence provided by CAG under the assumption that arsenic is additive to the risk of lung cancer from cadmium while smoking is multiplicative indicates that the upper bound for the expected number of lung cancer cases is still significantly below that of the observed number of cases at the $P < 0.05$ level in the Thun study.

6. NIOSH: Cadmium is a potential occupational carcinogen.

7. IARC: The human epidemiologic evidence appears to provide limited evidence of lung cancer risk from exposure to cadmium. (conclusion prior to Thun study).

B. Animal evidence

1. Takenaka et al. study, 1983. Exposure of rats to cadmium chloride aerosol by inhalation was done over the lifetime of the animals. A positive dose-response relationship for the development of primary lung carcinoma was shown. 25 out of 35 rats (71.4%) exposed to 50 ug/m^3 cadmium chloride aerosol developed lung cancer compared to O out of 38 unexposed controls. 20 out of 38 (52.6%) exposed to 25 ug/m^3 and 6 out of 39 (15.4%) exposed to 12.5 ug/m^3 developed lung cancer.
2. Additional animal studies contributing information to the carcinogenic potential of cadmium.

    a. Lung cancer in rats by single inhalation exposure (Hadley et al., 1979).

b. Mammary tumors in males by multiple tracheal instillation (Sanders et al., 1984).

c. Pancreatic Islet cell tumors following parenteral administration of cadmium chloride (Poirier et al., 1983).

d. Prostate tumors by injections of cadmium chloride into prostate (Scott and Aughey 1979)

e. Interstitial-cell tumors of the testis observed following testicular atrophy in rats and mice given s.c. injections of soluble cadmium salts, cadmium sulphate and cadmium chloride (Gunn 1964 and 1963).

f. Local spindle-cell sarcomas in Wistar rats by single s.c. injections of cadmium chloride, sulphate, sulfide, and oxide (Knorre, 1970).

C. Short-term tests for genotoxicity

1. Gene mutation on DNA damage in bacteria or fungi

a. Data is conflicting. Negative results reported by Heddle and Bruce 1977, Milvy and Kay 1978, Polukhina et al. 1977, Putrament et al. 1977.

b. Positive results reported by Hedenstedt et al. 1979, Nishioka 1975, and Kanematsu 1980, Takahashi 1972.

2. Gene mutations in mammalian cell cultures

a. Mouse lymphoma assay - mutagenic by Amacher and Paillet, 1980. Also positive results by Oberly et al. 1982.

b. Chinese hamster cell assay

Mutagenic results reported by Casto (1976). Ochi and Ohsawa (1983) demonstrated single strand DNA scission by cadmium indicating formation of DNA-protein cross-linking by cadmium.

3. Studies of cadmium mutagenesis via sex-linked recessive lethal test in Drosophila Melanogester

   a. Majority of studies show no mutagenic response.
   b. Dominant lethal test in Drosophila resulted in a positive response with a dose-response relationship.

4. Chromosomal aberrations

   a. In human lymphocytes and human cell lines results have been conflicting and contradictory.

   b. Chinese hamster cells - Chromosomal aberrations followed treatment with cadmium.

   c. Mouse carcinoma cells - no aberrations noted following cadmium treatment.

   d. No observed chromosomal aberrations in bone marrow cells of rodents treated with cadmium.

   e. Plants exposed to cadmium demonstrated chromosomal aberrations and gene mutations.

   f. Cadmium is a mutagen that interfers with spindle formation *in vitro* and *in vivo* studies in mammals.

   g. Cadmium induces aneuploidy in CHO cells and in whole mammals and germ cells of female mice and Syrian hamsters.

h. Numerical aberrations induced by cadmium chloride in female germ cells of mice are inherited in embryos.

IV. Reproductive Effects

A. Animal studies

1. Testicular damage by cadmium in mice (Gunn et al. 1965, Parizek 1960)

a. Pretreatment of mice with zinc reduces incorporation of cadmium into spermatids.

b. Cadmium inhibits incorporation of Thymidine into spermatogonia-(significant reduction of litter size by injection of 1 mg/kg b.w. cadmium chloride into male mice 15 to 48 days before mating with untreated female mice)

c. Destruction of testicular cells (spermatogenic and interstitial) of rabbits given 1 to 8 s.c. injections of 9-18 mg/kg b.w. cadmium chloride.

d. One s.c. injection of 0.05 mmol/kg b.w. (9.2 mg/kg b.w.) cadmium chloride into rabbits produced aspermia within 4 weeks with no recovery. (Paufler and Foote, 1969).

e. Schroeder and Mitchner (1971) exposed mice for 3 generations to cadmium in drinking-water (10 ug/ml) which produced a reduction in litter size in breeding mice with loss of strain characteristics after two generations and congenital anomalies.

f. Teratogenesis in rats by daily oral administration of 20-80 mg/kg b.w. cadmium chloride days 6 to 19 gestation. (Scharf et al 1972).

g. Teratogenesis in golden hamsters by i.v. injection of 2-4 mg/kg b.w. cadmium sulphate on day 8 of gestation produced cleft lips and palates and other facial defects (Ferm and Carpenter 1968). Also in rat by Barr, 1973; Chernoff, 1973 and Ishizu et al, 1973.

h. Necrosis and blood clot formation of placenta with fetal death 24 hours after s.c. injection of cadmium salt into rats on one of days 17-21 of pregnancy (Parizek, 1964).

i. Exposure to very low doses of Cd by inhalation did not demonstrate teratogenesis.

B. Human Studies

a. Sex differences: multiparous post-menopausal women in Fuchu, Japan develop itai - itai disease (Tsuchiya, 1969) characterized by elevated cadmium blood levels and osteomalacia (proximity to cadmium mine).

b. Placental transfer: Lauwerys, Bachet, Rolls and Hubermont-a series of papers on cadmium levels in Belgim women in placenta, maternal and cord blood between 1975 - 1976. Results suggest a partial placental barrier to cadmium transfer. Cadmium crosses the placenta and levels in the fetus are generally slightly less than in maternal blood. Results in smokers suggest cadmium from smoking may induce metallothionein synthesis in the placenta which can bind cadmium and protect the fetus.

c. Study of Tsvetkova, 1970, showing no effect on the menstrual cycle in 106 women working with cadmium.

d. There is limited evidence that reported exposure to very high levels of cadmium may induce testicular damage in men. (Smith et al, 1960 and Favino et al, 1968).

V. Pharmacokinetics and Toxicity

A. Animal studies - toxicity

1. LC_{50} in dogs of cadmium chloride mist: 0.32 mg/cadmium/l of air for 30 minute exposure. (Harrison et al., 1947)

2. LD_{50} in mice, rats, guinea pigs, rabbits, dogs and monkeys of cadmium oxide fumes for 10-30 minutes were $<$ 700, 500, about 3500, about 2500, 4000 and 15,000 min-mg/m^3, respectively (Barrett et al., 1947). 11% retention of cadmium oxide by the lungs of these animals.

3. Snider et al. (1973) produced emphysema experimentally in male rats exposed to cadmium chloride aerosols (0.1% solution) for 1 hour per day for 5-15 days.

4. Cadmium damages testes of experimental animals (see section IV).

5. Several experiments in rats (Friberg et al., 1974, 1975) show kidney tubular damage when the concentration in the kidney cortex is about 200 ug/g wet weight.

6. Hypertension has been produced in rats given cadmium in drinking water (5 ug/ml Cd) for long periods of time (Kanisawa & Schroeder, 1969; Perry and Erlanger, 1974).

7. Cadmium may produce zinc deficiency (Petering et al., 1971)

8. Large doses of zinc prevent toxic action of cadmium on testes (Friberg et al., 1974)

B. Animal studies -pharmacokinetics

1. Biological half-life of a single oral dose of cadmium in female mice was estimated to be 200 days (Richmond et al., 1966).

2. Following inhalation 10 to 40% of Cadmium is absorbed, primarily in the lung and additionally by the gastrointestinal tract after mucocilliary clearance.

3. Absorbed cadmium in rats is first transported to the liver and slowly transferred to the kidney. Only 1 to 2% of absorbed cadmium is excreted via the urine or feces. Metallothionein (a low mol. wt. protein) found in the liver, red cells, plasma and duodenal mucosa of several species is thought to play an important role in transport of cadmium in the body. (Norberg, 1971; Friberg et al., 1974). Calcium and iron deficiencies enhance the absorption and tissue retention of cadmium.

C. Man: toxicity and pharmacokinetics

1. Long term exposure of man to large amounts of cadmium by inhalation or ingestion eventually causes renal tubular dysfunction (Holden, 1969; Piscator, 1962; Potts, 1965). Osteomalacia may result from disturbances in mineral metabolism (Adams et al, 1969; Friberg, 1974).

2. Cadmium's role in human hypertension causation is uncertain (Friberg et al., 1974)

3. About 5% of ingested cadmium is absorbed (Rahola et al., 1972; Yamagata et al., 1974) and remainder is excreted in feces. Non-exposed subjects excrete 10-60 ug daily in feces (Wester, 1974).

4. Cadmium is stored with metallothionein in the liver and kidneys. One-third of body burden occurs in the kidneys (Friberg et al., 1974).

5. Newborn is practically free from cadmium (Schroeder et al.; 1961).

6. Normal levels in kidney cortex at age 50 ranges from 15-30 ug/g. (Friberg et al.; 1974)

7. Smokers have higher (5-100%) renal concentrations of cadmium than do non-smokers (Lewis et al., 1972)

8. Urinary excretion of cadmium is 1-3 ug/day in adults. Smokers excrete more (Piscator, 1976).

9. Biological half-life of cadmium in man is 10-30 years (Elinder, et al., 1976).

VI. Risk Assessment: (Review Articles: U.S. EPA Draft-[April, 1984] Updated Mutagenicity Assessment of Cadmium)

A. Quantitative estimation of risk

1. Unit risk for cadmium in air (lifetime-cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 ug/m^3 of the agent in the air they breathe)

2. Potency of cadmium relative to other carcinogens that CAG has evaluated.

3. Data for quantitative estimation of risk taken from:

a. Lifetime animal studies or

b. Human studies where excess cancer risk has been associated with exposure to the agent.

c. Mutagenicity studies of irreversible damage to DNA. (Quantal type of biologic response characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship).

B. Dose-response assessment based on:

1. Available animal data
2. Human epidemiology and monitoring
3. Workplace evidence

C. Range of potential risks

1. Unit risk estimate based on animal study of Takenaka et al. (1983) using male Wistar rats and cadmium chloride inhalation exposure over lifetime of rat with dose-response relationship shown.

2. Unit risk estimate based on a human study.

   Study of Thun et al. (1984). Cohort of cadmium smelter workers hired on or after January 1, 1926, and employed for at least 2 years in a production capacity in the same plant from January 1, 1940 to December 31, 1969 developed 16 cases of respiratory cancer deaths through December 31, 1978. (Only 6.99 would be expected).

ATTACHMENT II

Cadmium References (1/24/85)

1. Amacher, D. E. and Paillet, S. C. (1980) Induction of trifluorothymidine-resistant mutants by metal ions in L5178Y/TK+/- cells. Mutat Res . 78 (3):p279-88

2. Anderson, L. D.et al (1983) Updated mutagenicity and carcinogenicity assessment of cadmium: addendum to the health assessment document for cadmium (May 1981) EPA Report. EPA-600/8-83-025A:151pp.

3. Anonymous (1981) Role of metals in carcinogenesis. Problems of epidemiological evidence. Environ Health Perspect. 40:11-20.

4. Baranski, B., Opacka, J., Wronska-Nofer, T., Trzcinka-Ochocka, M., Sitarek, K. and Matczak, W. (1983) Effect of inhalation exposure to cadmium oxide on arterial blood pressure, lipid metabolism and tissue cadmium concentration in rats.. Med Pr. 34(1):11-9.

5. Baranski, B., Stetkiewicz, I., Trzcinka-Ochocka, M., Sitarek, K. and Szymczak, W. (1982) Teratogenicity,fetal toxicity and tissue concentration of cadmium administered to female rats during organogenesis.. J Appl Toxicol (UNITED STATES) . 2 (5) :p255-9.

6. Beaudoin, A. R. and Fisher, D. L. (1981) An in vivo/in vitro evaluation of teratogenic action . Teratology . 23 (1):p57-61.

7. Becking, G. C (1981) Recent advances in the toxicity of heavy metals--an overview. Fundam Appl Toxicol. 1(5):348-52.

8. Best, J. B. and Morita, M. (1982) Planarians as a model system for in vitro teratogenesis studies.. Teratogenesis Carcinog Mutagen. 2(3-4):277-91.

9. Bishop, J. B. and Kodell, R. L. (1981) The heritable translocation assay: its relationship to assessment of genetic risk for future generations. Teratog Carcinog Mutag. 1:305-332.

10. Bruce, W. R. and Heddle, J. A. (1979) The mutagenic activity of 61 agents as determined by the micronucleus, Salmonella, and sperm abnormality assays.. Can J Genet Cytol . 21 (3) :p319-34.

11. Caperna, T. J. and Failla, M. L. (1984) Cadmium metabolism by rat liver endothelial and Kupffer cells.. Biochem J. 221(3):631-6.

12. Carmichael, N. G., Backhouse, B. L., Winder, C. and Lewis, P. D. (1982) Teratogenicity, toxicity and perinatal effects of cadmium.. Hum Toxicol (ENGLAND) . 1 (2) :p159-86.

13. Chang, L. W., Wade, P. R., Pounds, J. G. and Reuhl, K. R. (1980) Prenatal and neonatal toxicology and pathology of heavy metals.. Adv Pharmacol Chemother . 17:p195-231.

14. Cherian, M. G. (1980) Chelation of cadmium with BAL and DTPA in rats.. Nature . 287 (5785) :p871-2.

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ATTACHMENT III

Ambient Cadmium Concentrations and Emission Trends

Data from the U.S. Environmental Protection Agency's National Aerometric Data Bank for the 1964 to 1985 period show mean cadmium concentrations between 0 (below detectable limit) and 125 ng/m^3. At stations where more than 50 percent of the samples were above the detection limit for sampling periods between 1976 and 1984, mean cadmium concentrations ranged from 0.4 to 3.0 ng/m^3. These data represent total particulate cadmium 50 um and smaller collected from ambient air at sites throughout California.

Cadmium or its compounds are used in the production of pigments, alloys, and nickel-cadmium batteries, as stabilizers for plastics, and in cadmium plating. Potential sources of cadmium in air are emissions from secondary non-ferrous smelting, secondary steel production, fossil fuel combustion, refuse and sewage sludge incineration, cadmium electroplating, and manufacturing of cadmium-containing products. Investigations are under way to determine the magnitude of emissions in California from these and other types of sources.

United States demand for cadmium is expected to increase at an average annual rate of 1.8 percent through 1990.

ate of California

Department of Health Services

Memorandum

o : James Boyd
Executive Officer
Air Resources Board
1102 Q Street
Sacramento, CA 95814
B-4

Date : July 17, 1985

bject: Request for Extension of Cadmium Health Evaluation

RECEIVED

JUL 24 1985

om : Office of the Director
8/1253 5-1248

Due to the expected arrival of recently updated information on cadmium from the U.S. Environmental Protection Agency, we are requesting a 30-day extension to August 9, 1985 for submission of the cadmium document.

Kenneth W. Kizer, M.D., M.P.H.
Director

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JUL 24 1985

Stationary Source Division
Air Resources Board

tate of California

xc: PV

Department of Health Services

Memorandum

o : James Boyd, Executive Officer
Air Resources Board
1102 Q Street
Sacramento, CA

Date : August 14, 1985

Subject : Cadmium Document

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AUG 1

Stationary Source
Division
Air Resources Board

From : Office of the Director
714 P Street, Room 1253
Sacramento, CA

I have been informed the Department of Health Services will be unable to meet the August 9, 1985 due date for submission of the cadmium document because it has taken longer than anticipated to complete the document. It is expected to be completed by August 23, 1985. We are requesting an extension until August 26, 1985. Thank you for your patience.

Maridee Gregory MD

Maridee Gregory, M.D.
Acting Deputy Director
Public Health

cc: Kenneth W. Kizer, M.D., M.P.H.
Alex Kelter, M.D.
Donald Lyman, M.D.

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AUG 16 1985

Stationary Source
Division
Air Resources Board

State of California | Department of Health Services

Memorandum

To : James D. Boyd
Executive Officer
Air Resources Board
1102 Q Street
Sacramento
B-4

Date : October 10, 1985

Subject: Health Effects of Cadmium

From : Office of the Director
8/1253 5-1248

Attached is the document prepared in response to your memo requesting the assistance of the Department of Health Services in evaluating the health effects of cadmium.

Kenneth W. Kizer, M.D., M.P.H.
Director

Attachment

cc: C. Berryhill - w/o attachment
J. Sharpless - w/o attachment
Assemblymember Tanner - w/o attachment
P. Venturini - w/o attachment

10/10/85

APPENDIX C

DISCUSSION OF EMISSION ESTIMATES

APPENDIX C

I. STATIONARY SOURCES

1. Cadmium Plating:

There are 31 cadmium platers in the South Coast Air Basin which emitted 0.22 ton of cadmium in 1982. The ratio of cadmium platers to chrome platers in the South Coast Air Basin is approximately 0.20 to 1.0 (Zwiacher et al., 1983, pp. 207-214). It is estimated that there are 400 chrome platers in California (CARB, 1985). Assuming the ratio of cadmium to chrome platers in the state is the same as in the South Coast, approximately 80 cadmium platers are operating in California. Cadmium emissions from this source are estimated as follows:

EMS = (80 platers/31 platers) (0.22 ton/year) = 0.57 ton/year

2. Coal Combustion: (Sierra Energy & Risk Assessment, Inc., 1982).

a. Cement Manufacturing:

* Consumption in 1984 = 1,600,000 tons
* Emission factor = 2.03×10^{-4} lb. Cd/ton (Krishnan, et al., 1982)
* Ems = (1,600,000 tons/yr) (2.03×10^{-4} lb Cd/ton) (ton/2,000 lbs) = 0.16 ton/yr

b. Cogeneration:

* Consumption in 1981 = 240,000 tons
* Emission factor = 1.3×10^{-4} lb. Cd/ton (Krishnan, et al., 1982)
* Ems = (240,000 tons/yr) (1.3×10^{-4} lb Cd/ton) (ton/2,000 lbs) = 0.02 ton/yr

b. Sugar Beet Processing:

* Consumption in 1981 = 25,000 tons
* Emission factor = 9.1×10^{-4} lb. Cd/ton (Krishnan et al., 1982)
* Ems = (25,000 tons/yr.) (9.1×10^{-4} lb Cd/ton) (ton/2,000 lbs.) = 0.01 ton/yr.

Together, cadmium emissions from coal combustion amounted to 0.19 ton in 1981.

3. Residual Oil:

California burned approximately 1.44 billion gallons of residual oil in 1984 (CARB, 1986a; CARB, 1986b). Of this, utilities burned approximately 16 percent at their power plants. One utility, Southern California Edison (SCE) used only 2.2% of the residual oil burned (CARB, 1986a; CARB 1986b; CARB 1986c). Emissions for residual oil combustion were estimated using cadmium concentrations of fuel oil reported by SCE and Pacific Gas and Electric Company (PG&E) (SCE, 1986; PG&E, 1986), and the amount of residual oil burned in California (CARB, 1986a).

	SCE	PG&E
Cadmium concentration (ppm)	0.01	0.31-0.52

Assuming all cadmium in the residual oil is emitted into the atmosphere upon combustion, cadmium emissions were estimated as follows:

a. Utility Boilers:

California utilities used approximately 232.3 million gallons of residual oil in 1984 (CARB, 1986a). Cadmium emissions from residual oil burned at utilities are estimated using equation I.

$$EMS = PR * D * C_c * U_f^{-1} \qquad (1)$$

Where:

Ems = cadmium emissions, tons per year

PR = Process Rate, millions of gallons/year

D = Density of residual oil (8.2 lb./gal.), lb./gal.

C_c = Cadmium concentration of residual oil, ppm

U_f = Unit conversion factor (2000), lb./ton

	Lower (tpy)	Upper (tpy)
Ems	0.01	0.50

Note: Data on cadmium concentration from SCE were used to estimate the lower number while the highest cadmium concentration in fuel oil as reported by PG&E was used to estimate the upper number.

b. Other Sources:

Besides being used at utilities, residual oil is used in chemical manufacturing and oil and gas extraction activities and in ships. In 1984, approximately 1.21 billion gallons of residual and/or crude oil were used by such sources (CARB, 1986a; CARB, 1986b). Because the SCE fuel cadmium content data are only applicable to the South Coast utilities and refineries (probably only SCE, because SCE used foreign oils with very low sulfur content, 0.17% to 0.18% (Stepman, 1986), and because the South Coast industries only used approximately 0.6 percent (CARB, 1986a; CARB, 1986b; CARB, 1986d; CARB, 1986e) of total residual oil burned by sources in this category, only data from PG&E are used to estimate cadmium emissions. Equation I is also applicable for this category. Estimated cadmium emissions are as follows:

	Lower (tpy)	Upper (tpy)
Ems	1.5	2.6

Adding emissions for utility boilers to that for other sources, the statewide cadmium emissions from residual oil combustion are estimated to range from 1.5 to 3.1 tons in 1984.

4. Secondary Smelters:

   a. Copper:

   The California Emission Data System identifies 71 copper smelters in California. Together, these facilities consumed 54,100 tons of copper scrap in 1981 (CARB, 1985c). An uncontrolled emission factor of 3 lbs. Cd/ton of copper scrap was reported (Coleman, R., 1979). Assuming 90 percent control, cadmium emissions from these copper smelters are:

   Ems = (1.0 - 0.90) * PR * Emfac

   = 0.10 * 54,100 tons/yr. * 3 lb. Cd/ton * ton/2,000 lbs

   = 8.1 tons/yr.

b. Zinc:

Process Rate = 51,900 tons of zinc were produced in 1981 (CARB, 1985c).

Emfac = 0.01 lb. Cd/ton of zinc produced (Coleman, R., 1979)

Ems = PR * Emfac

= 51,900 ton/yr. * 0.01 lb. Cd/ton * ton/2,000 lbs

= 0.26 ton/yr.

5. Cement Manufacturing:

In 1984, California cement manufacturing plants produced 8,722,000 tons of cement (U.S. DOI, 1985) and emitted approximately 3,030 tons of PM (CARB, 1985d) excluding PM emissions from fuel.

Assuming the cadmium concentration in the cement kiln dust removed from the rotary kiln baghouse or ESP equals the cadmium concentration in the particulate matter, cadmium emissions from cement manufacturing can be estimated from total PM emissions and the Cd concentration in cement kiln dust.

Cadmium concentration in cement kiln dust from 9 California cement plants ranged from 5 ppm to 352 ppm and averaged 79 ppm (Haynes and Cramer, 1982; CARB, 1985e). Using these data and the total PM emission, the 1984 cadmium emissions from California cement plants are estimated to be:

Lower Estimate	Upper Estimate	Estimate based on mean Cd concentration
(TPY)	(TPY)	(TPY)
0.015	1.1	0.24

6. Resource Recovery:

Cooper Engineers, Inc. reported an uncontrolled emission factor of 3.04×10^{-6} lb. Cd per million Btu of solid waste heat content (Cooper Engineers, Inc., 1984). The emission factor was obtained from test results conducted at the Gallatin municipal waste-to-energy facility in Tennessee. It was assumed an overall of 99% controlled by application of fabric filters (CARB, 1984). The controlled emission factor, assuming 99% controlled, is therefore 3.06×10^{-6} lb. Cd per million Btu.

Annual average waste burned at the North County Recycling and Energy Recovery Center is 46.5 tons/hr or 5.21×10^{8} Btu/hr., assuming waste has an energy of 5,600 Btu/lb. (CARB, 1984). If the Center operates 24 hrs/day, 7 days/week and 50 weeks/yr., cadmium emissions from this facility is calculated as follows:

Ems = PR * Emfac

$= 5.21 \times 10^{8}$ Btu/hr. * 8400 hrs/yr. * 3.04×10^{-6} lb/10^{6} Btu

= 13.3 lbs/yr.

7. Fertilizer:

* Superphosphate applied in 1983 = 6,000 tons (Cushman, R., 1984)
* Emission factor = 0.0002 lb. Cd/ton of superphosphate applied.

Ems = (6,000 tons/yr.) (0.0002 lb. Cd/ton)

= 1.2 lbs/yr.

II. MOBILE SOURCES

1. Combustion:

a. Gasoline:

Motor gasoline consumption in 1984 = 1.224×10^{10} gallons (Morgester, J., 1985). An average cadmium content of gasoline is 0.02 mg/l or 1.67×10^{-7} lbs/gal. (Lee et al., 1973). Assuming all cadmium in gasoline is emitted from vehicular exhaust upon combustion, cadmium emissions from gasoline combustion are calculated as follows:

Ems = 1.224×10^{10} gals/yr. * 1.7×10^{-7} lbs/gal. * ton/2,000 lbs. = 1.0 ton/yr.

b. Diesel Fuel:

Diesel fuel consumed in 1983 by California motor vehicles was estimated to be 1.46×10^{9} gallons (CARB, 1983). Cadmium content of diesel fuel is 0.08 g/m^3 or 6.7×10^{-7} lbs/gal. (Tierney, et al., 1979). Based on estimates made in 1983 of consumption of diesel fuel in 1984, and assuming all cadmium in diesel fuel is emitted, cadmium emissions from diesel-powered vehicles are estimated as follows:

Ems = 1.46×10^{9} gal/yr. * 6.7×10^{-7} lb. Cd/gal. * ton/2,000 lbs. = 0.5 ton/yr.

c. Motor Oil:

In 1984, VMT driven by motor vehicles in California were estimated to be 1.67×10^{11} (CARB, 1983). An estimated emission factor of 0.6 gram of cadmium emitted per million kilometers driven, or 2.12 lbs. of cadmium emitted per billion

VMT driven, was reported (Tierney, et al., 1979). Cadmium emissions from this source are estimated as follows:

$$Ems = 1.67 \times 10^{11} \text{ VMT/yr.} * 2.12 \times 10^{-9} \text{ lb. Cd/VMT}$$
$$\text{ton/2,000 lbs.} = 0.2 \text{ ton/yr.}$$

Together, cadmium emissions from combustion of fuel and oil in motor vehicles are estimated as 1.7 tons in 1984.

2. Tire Wear:

For every million kilometers driven, 3 grams of cadmium are emitted (0.11 lb. of cadmium per billion VMT driven) (Tierney, et al., 1979). Cadmium emissions from wear-and tear of vehicle tires are estimated as follows:

$$Ems = 1.67 \times 10^{11} \text{ VMT/yr.} * 0.11\ 10^{-9} \text{ lb. Cd/VMT} * \text{ton/2,000 lbs.}$$
$$= 0.9 \text{ ton/yr.}$$

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APPENDIX D

ARB ANALYTICAL METHODS FOR SAMPLING AND ANALYSIS OF ATMOSPHERIC CADMIUM

Method for Sampling and Analysis of Atmospheric
Cadmium
Method 109

1. Method

1.1 Ambient air suspended particulate is collected on a glass fiber filter for 24 hours using a high-volume sampler.

1.2 The cadmium in the particulate sample is solubilized by extraction with nitric acid, facilitated by ultrasonication.

1.3 The cadmium content in ambient particulate samples is analyzed by flame atomic absorption spectrometry, using an electrodeless discharge lamp, a wavelength of 228.8 nm, a continuum source background correction, and the optimum instrumental conditions recommended by the manufacturer.

1.4 If the cadmium content of the sample is below the detection limit of the flame atomic absorption spectrophotometer, the heated graphite furnace (flameless AA) is used!

2. Apparatus & Supplies

2.1 The hi-volume sampler used to collect the sampler is described in Appendix D, "Procedure for Use of a High-Volume Sampler" (Air Resources Board Procedures Sampling and Analysis of Atmospheric Toxics).

2.2 Heated ultrasonic water bath.

2.3 Atomic Absorption (AA) Spectrophotometer equipped for automated flame and flameless analyses (graphite furnace, and Cd electrodeless discharge lamp.

2.4 Zero air - for flame analysis.

2.5 Acetylene - for flame analysis.

2.6 Argon - for flameless (graphite furnace) analysis.

2.7 Associated glassware: volumetric flasks, pipettes, 100 mL test tubes, mixing cylinder or centrifuge tubes with caps, funnels (for filtering samples, if desired).

2.8 Polyethylene bottles. For storage of samples.

2.9 Centrifuge - if desired (in lieu of filtration).

3. Reagents

3.1 Concentrated (16 M) HNO_3 ACS reagent grade HNO_3 and commercially available redistilled HNO_3 has been found to have sufficiently low metal concentrations.

3.2 Distilled or deionized water (metal free).

3.3 3 M HNO_3 - Add 182 mL of concentrated HNO_3 to D.I. water in a 1 L volumetric flask. Mix well, cool, and dilute to volume with D.I. water. CAUTION: Nitric acid fumes are toxic. Prepare in a well-ventilated hood.

3.4 1 M HNO_3 - add 60.7 mL of concentrated HNO_3 to D.I. water in a 1 L volumetric flask. Mix well, cool, and dilute to volume with D.I. water.

3.5 Cadmium, 1000 ppm atomic absorption standard - commercially available.

4. Sample Preparation

4.1 The filter on which the sample is collected is prepared for analysis by ultrasonic extraction. Prepare a clean filter (with no sample collected) to serve as the sample filter blank.

4.2 Cut one quarter of the filter sample into pieces of approximately 1 cm. square and place in a 100 mL centrifuge or test tube.

4.3 Add 33.3 mL of 3M HNO_3 using a pre-set calibrated automatic dispensing pipette (the acid should cover the cut filter pieces completely).

4.4 Cap the centrifuge or test tubes loosely to prevent pressure build-up during the ultrasonication.

4.5 Place tubes in a test tube rack.

4.6 Put enough water in a clean ultrasonic bath so that the water level is slightly above the acid level of the tubes in the rack. Heat the water in the bath to around $180^{o}F$.

4.7 Set the rack in the ultrasonic bath.

4.8 Ultrasonicate the samples for 50 minutes.

4.9 Remove the tubes from the bath and add 66.7 mL water to each of the tubes.

4.10 Cap tubes loosely and ultrasonicate for another 15 minutes.

4.11 Filter or centrifuge the contents of the tubes. If the tubes are centrifuged, decant the supernatant. Use the filtrate (or supernatant liquid) for analysis.

4.12 The final concentration of nitric acid in the samples is 1 M.

5. Instrument Conditions

5.1 Prepare the instrument for flame or flameless operation (follow manufacturers recommended operating conditions). Set the wavelength of the atomic absorption spectrophotometer at 228.8 nm.

6. Calibration

6.1 Stock standard solution - 1000 ppm cadmium solution. Available commercially as atomic absorption standard.

6.2 Intermediate standard - 100 µg Cd/mL. Prepare by diluting 10 mL of stock standard to 100 mL with 1 M HNO_3.

6.3 Calibration standard - 0.50 µg Cd/mL (for flame). Prepare by diluting 1.0 mL of the intermediate standard to 200 mL with 1 M HNO_3. For flameless AA, prepare a calibration standard of .01 µg Cd/mL.

6.4 Aspirate the reagent blank (1 M HNO_3) to zero the instrument.

6.5 Aspirate the calibration standard to calibrate the instrument.

6.6 Plot the absorbance vs. µg Cd/mL to give the calibration curve if automatic calibration is not available in the instrument. PE 3030 AA calibrates automatically.

7. Sample Analysis

7.1 Aspirate the samples and filter blanks. Record their absorbances.

7.2 Determine the cadmium concentration in µg Cd/mL from the calibration curve (PE 3030 can do this automatically). Subtract the amount of Cd found in the filter blanks from those found in the samples.

7.3 Samples that exceed the linear concentration range should be diluted with 1 M HNO_3 then reanalyzed.

8. Calculations

8.1 Determine from the calibration curve the concentration of Cd found in the samples and blanks in µg/mL. The PE 3030 AA does this automatically

8.2 Calculate the concentration of Cd in the particulate sample as follows:

$$\frac{\mu g\ Cd}{m^3} = \frac{\mu g\ Cd^*}{mL} \times 100\ mL\ ^{**} \times \text{diln factor (if any)} \times 4\ ^{***}$$

*µg Cd/mL = (µg CD/mL found in sample -µg Cd/mL found in filter blank)

** final volume of the extract solution

*** if 1/4 of filter is used.

9. Precision and Accuracy

Single laboratory, single operator data were collected for Cd using automated flame atomic absorption technique with background correction. 2 and 5 µg Cd were spiked on 1/4 EPM Whatman 2000 glass fibre filters and extracted according to the procedure given above . Recovery values (7) from several replicates are given below.

µgm/ml Cd	# replicates	% Recovery
0.02	6	100 ± 15
0.15	7	100 ± 10

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