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Air Toxics and Health Risks in California: The Public Health Implications of Outdoor Concentrations

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Of the 188 hazardous air pollutants (HAPs) listed in the Clean Air Act, only a handful have information on human health effects, derived primarily from animal and occupational studies. Lack of consistent monitoring data on ambient air toxics makes it difficult to assess the extent of low-level, chronic, ambient exposures to HAPs that could affect human health, and limits attempts to prioritize and evaluate policy initiatives for emissions reduction. Modeled outdoor HAP concentration estimates from the U.S. Environmental Protection Agency's Cumulative Exposure Project were used to characterize the extent of the air toxics problem in California for the base year of 1990. These air toxics concentration estimates were used with chronic toxicity data to estimate cancer and noncancer hazards for individual HAPs and the risks posed by multiple pollutants. Although hazardous air pollutants are ubiquitous in the environment, potential cancer and noncancer health hazards posed by ambient exposures are geographically concentrated in three urbanized areas and in a few rural counties. This analysis estimated a median excess individual cancer risk of $2.7E^{-4}$ for all air toxics concentrations and 8600 excess lifetime cancer cases, 70% of which were attributable to four pollutants: polycyclic organic matter, 1,3 butadiene, formaldehyde, and benzene. For noncancer effects, the analysis estimated a total hazard index representing the combined effect of all HAPs considered. Each pollutant contributes to the index a ratio of estimated concentration to reference concentration. The median value of the index across census tracts was 17, due primarily to acrolein and chromium concentration estimates. On average, HAP concentrations and cancer and noncancer health risks originate mostly from area and mobile source emissions, although there are several locations in the state where point sources account for a large portion of estimated concentrations and health risks. Risk estimates from this study can provide guidance for prioritizing research, monitoring, and regulatory intervention activities to reduce potential hazards to the general population. Improved ambient monitoring efforts can help clarify uncertainties inherent in this analysis.

KEY WORDS: Air toxics; hazardous air pollutants; risk assessment; cancer; noncancer; dispersion modeling

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1. INTRODUCTION

Public health concern about the adverse effects of air pollution led to the enactment of regulatory and legislative actions, including the Clean Air Act of 1970 (CAA). The 1990 Clean Air Act Amendments focused regulatory attention on two classes of air pollutants: criteria pollutants and hazardous air pollutants (HAPs), also known as air toxics. Criteria air

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pollutants are common contaminants, including particulates, sulfur dioxide, nitrogen oxides, ozone, carbon monoxide, and lead.[1] Air toxics include 188 specific pollutants and chemical groups, many of which are associated with adverse health outcomes including cancer, neurological, respiratory, reproductive, and developmental effects.[2,3] Most known health effects of hazardous air pollutants are derived primarily from animal and occupational studies,[2,3] and there is a paucity of epidemiological studies evaluating the potential health risks of chronic, low-level exposures experienced by the general public.[4,5] Nevertheless, the U.S. Environmental Protection Agency (EPA) has estimated that as many as 2,500 cancer cases per year may be associated with exposure to outdoor concentrations of 45 of the 188 hazardous air pollutants[6] although this estimate has been criticized for being too high.[7] Nearly 50 million people live in locations where estimated ambient concentrations of one or more hazardous air pollutants exceed levels of concern for noncancer health effects in humans.[8]

Air pollution research on the health effects of air toxics has not been as extensive as for criteria air pollutants, which are consistently monitored at multiple sites in over 300 areas within the United States.[9] The extensive monitoring networks for criteria pollutants have provided comprehensive temporal and spatial data on ambient concentrations that have allowed for many studies on their health impacts.[10,11] In contrast, widespread ambient concentration data are available for only a few air toxics—such as benzene—and measurements are taken inconsistently and in only a few locations,[12] making it difficult to assess the extent of cumulative ambient exposures across all air toxics that could affect human health. The large number of contaminants listed under the Clean Air Act, their varied chemical nature, and the heterogeneity of their geographical distribution make comprehensive monitoring efforts exceedingly difficult. This lack of consistent data on ambient air toxics has undermined attempts to prioritize and evaluate policy initiatives for emissions reduction activities.

One way to address the lack of comprehensive measured data is to estimate outdoor air toxics concentrations using dispersion modeling techniques. A recent modeling analysis undertaken by the EPA's Cumulative Exposure Project (CEP) uses a Gaussian modeling approach to estimate long-term annual average outdoor concentrations of 148 hazardous air pollutants originating from myriad sources. Concentrations are estimated for every census tract in the continental United States for a base year of 1990.[13,14]

A recent national study compared modeled air toxics concentrations from this CEP database to previously defined benchmark concentrations for cancer and noncancer health effects. Results indicated that several hazardous air pollutants are ubiquitously high compared to benchmark concentrations.[15] This analysis utilizes the modeled outdoor HAP concentration estimates from the Cumulative Exposure Project to characterize the extent of the air toxics problem in California for the base year of 1990. The distribution of pollutant concentrations and their associated cancer and noncancer health risks are examined, and emission source contributions to HAP concentrations and health risk estimates are assessed.

To assess the public health significance of ambient hazardous air pollutants, this study evaluates whether concentrations are at levels that are of potential concern regarding cancer and noncancer toxicity. Modeled outdoor concentration levels are used as a proxy for exposures and compared to toxicity benchmarks using quantitative risk assessment measures to screen for potentially high risk pollutants and to estimate cumulative cancer and noncancer health risks. Cumulative HAP concentrations and health risk estimates are also disaggregated by source category in order to assess whether health risk patterns differ depending on the type of emissions source.

California has a unique regulatory history in terms of its ongoing efforts to solve some of the worst air pollution problems in the country, from both stationary and mobile sources. The cancer and noncancer risk estimates derived in this study provide a useful approach to screen those pollutants and emissions sources that are of public health significance, in order to provide guidance for prioritizing research and regulatory intervention activities.

2. METHODS

2.1. Modeled Estimates of Outdoor Air Toxics Concentrations

The pollutants selected for modeling were based on the list of 188 HAPs in Section 112 of the 1990 Clean Air Act Amendments. A baseline year of 1990 was chosen for modeling. Available emissions data were reviewed, and appropriate data were identified for 148 air toxics.

Outdoor concentrations were estimated using a Gaussian dispersion model.[13,14] The Assessment System for Population Exposure Nationwide (ASPEN) is a modification of EPA's Human Exposure Model

(HEM), which has been utilized as a standard modeling approach to support regulatory activity by modeling long-term concentrations over large spatial scales.[13,14,16] Annual average ambient concentrations of 148 air toxics were estimated for each census tract, based on emissions rates of the pollutants, meteorological data, and simulation of atmospheric processes such as reactive decay (breakdown of pollutants after their release into the atmosphere), secondary formation (chemical transformation of one pollutant into another), and deposition. The model estimates long-term HAP concentrations attributable to anthropogenic sources within 50 kilometers of each census tract centroid. Each modeled HAP concentration is a spatial average that approximates the population-weighted average of outdoor HAP concentrations experienced within a census tract over the course of a year. There are 5,858 census tracts in California, with each averaging between 4,000 and 5,000 residents. Thus, tracts vary in physical size, with urban tracts tending to be smaller and rural tracts larger. In this study, HAP concentrations by county were also examined. There are 58 counties in California.

Inputs to the model required the development of a national emissions inventory for both stationary and mobile sources. For large manufacturing sources, the EPA's Toxic Release Inventory was used, and emissions estimates for other sources, such as automobiles, combustion sources, and other smaller area sources such as dry cleaners, were estimated using air toxics speciation data in combination with EPA's national inventories of emissions of total volatile organic compounds (VOC) and particulate matter (PM).[17–19] Source-specific air toxics emissions are derived from these VOC and PM inventories by applying industry-specific and process-specific estimates of HAP levels in the VOC and PM emissions streams.[13,14] For 28 air toxics, estimated outdoor concentrations also included a background portion attributable to long-range transport, re-suspension of historical emissions, and natural sources derived from measurements taken at clean air locations remote from known emissions sources. These values were treated as a constant across all census tracts, and added to the modeled concentration estimates from mobile and stationary emissions sources.[13,14]

2.2. Application of Toxicity Information to Assess Health Risks

This analysis utilizes ambient concentration modeling data and toxicity information to characterize the distribution of cumulative cancer and chronic noncancer health risks in accordance with California's AB2588 "Hot Spots" Guidelines.[20] The guidelines provide procedures for the preparation of the health risk assessments required under California's Air Toxics "Hot Spots" Information and Assessment Act of 1987.[4] This law established a statewide program for the inventory of air toxics emissions from individual facilities as well as requirements for risk assessment and public notification of potential health risk.[20]

Cancer risks are assessed using inhalation unit risk (IUR) estimates in $(\mu g/m^3)^{-1}$ for each carcinogenic compound. Inhalation unit risk estimates are defined as the individual lifetime excess risk due to a chronic lifetime exposure to one unit of pollutant concentration.[21] Potency estimates generally assume a nonthreshold, low-dose linearity, unless there is compelling evidence to the contrary, and are derived from occupational or animal studies. The unit risk calculated from occupational studies is based on a maximum likelihood estimate of the dose–response data. Potencies derived from animal data represent a 95% upper bound estimate of the probability of contracting cancer.

A reference concentration (RfC) for chronic noncancer effects is defined as the amount of toxicant in $\mu g/m^3$ below which long-term exposure to the general population of humans, including sensitive subgroups, is not anticipated to result in any adverse effects.[22] A central assumption underlying the RfC is that a threshold exists below which no adverse effects will occur in the general population, although such a threshold is not observable and can only be estimated. In general, RfCs are derived from animal data through the application of extrapolation and uncertainty factors to the No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL).

Comprehensive risk assessment requires the collection of as much cancer and noncancer toxicity information as possible, while taking into account the potential differences in the level of uncertainty, peer review, and derivation of this data. Cancer and noncancer toxicity data were compiled from the EPA and the California Environmental Protection Agency. For purposes of this analysis, toxicity values were prioritized into three tiers based on assessments of data quality, consistency in derivation, and peer review. Details on the rationale and methodology for classifying hazard data are discussed extensively elsewhere.[23]

[4] Health and Safety Code, sec. 44360 *et seq.*

In short, cancer and noncancer toxicity information was prioritized in the following way:

- Tier I toxicity values, representing the highest level of data quality and peer review, included EPA inhalation unit risks for carcinogenicity and reference concentrations (RfC) for noncancer effects.
- Tier II toxicity data included EPA oral potency factors for carcinogenicity, converted to inhalation units, or California Environmental Protection Agency inhalation unit risk estimates and reference exposure levels developed for the California Hot Spots Program.
- Tier III values included HAPs identified as potential carcinogens—based on their weight of evidence classification by the EPA or the International Agency for Research on Cancer (IARC), or a positive study from the National Toxicology Program—but that did not have an assigned cancer potency.[23]

A science policy decision was made to include Tier III pollutants in the analysis rather than assuming that their potential cancer risk was zero. As a result, Tier III pollutants were assigned a default potency value equal to the median value of the potencies for all the Tier I and II carcinogens considered in this study [an IUR of $3.4E^{-5}$ $(\mu g/m^3)^{-1}$].[5] Of the 148 pollutants with modeled concentration estimates, 60 percent ($n = 89$ pollutants) had a cancer potency value (Tier I = 40, Tier II = 38, Tier III = 11) and 61 percent ($n = 90$ pollutants) had either an RfC or reference exposure level (REL; Tier I = 33, Tier II = 57).

Fourteen of the 148 HAPs assessed in this paper are chemical groups with several constituents that may have varying toxicity potential, such as mercury compounds. Toxicity values that could be assigned to an entire chemical group were included for this study, and those values that were applicable only to certain constituents of a group were excluded.[23] For example, the EPA potency estimate for total chromium (including chromium III and chromium VI) was derived from an occupational exposure study of total chromium, containing about 14% chromium VI. The modeled total chromium concentration estimates used in this analysis contain about 17% chromium VI. Thus, it is reasonable to apply the cancer potency estimate listed by the EPA for total chromium in this analysis. Finally, given that polycyclic organic matter (POM) is a chemical group of particular concern for cancer risk,[24] a potency estimate was derived for the modeled concentrations of polycyclic organic matter (POM) through the adaptation of three previously developed methodologies, which are discussed in detail elsewhere.[18,25–28]

[5] Pollutants with Tier III cancer potency values include: acrolein, cresol, diethyl sulfate, diethyl formamide, dimethyl sulfate, ethyl chloride, methyl iodide, 2-nitropropane, parathion, styrene, and vinyl acetate.

2.3. Assessment of Cancer Risks

The EPA and the International Agency for Research on Cancer (IARC) identify carcinogens based on the scientific weight of evidence for carcinogenicity, which is derived from human and animal data. The categories used by the two agencies are very similar. EPA classifies potential carcinogens based on the 1986 EPA Guidelines for Carcinogenic Risk Assessment,[21] into either Group A (known human carcinogen), Group B (probable human carcinogen) or Group C (possible human carcinogen). IARC categorizes potential carcinogens as Group 1 (known human carcinogen), Group 2A (probable human carcinogen), and Group 2B (possible human carcinogen). Air toxics classified as either Group A, B, or C or Group 1, 2A, or 2B, were evaluated in this analysis. Pollutants without either an EPA or IARC classification but which have a National Toxicology Program study indicating a clear carcinogenic response in animals were treated as potential carcinogens for this study, and have the greatest degree of uncertainty in assessment of potential cancer risks.[28]

Cancer risks were assessed using inhalation unit risk estimates in $(\mu g/m^3)^{-1}$ for each carcinogenic compound. Exposure units are in $\mu g/m^3$.[28] Estimated cancer risks for each pollutant in each census tract were derived with the formula

$$R_{ij} = C_{ij} \times IUR_j, \quad (1)$$

where R_{ij} is the estimate of individual lifetime cancer risk from pollutant j in census tract i, C_{ij} is the concentration of hazardous air pollutant j in $\mu g/m^3$ in census tract i, and IUR; is the inhalation unit risk estimate for pollutant j in $(\mu g/m^3)^{-1}$. The cancer risks of different air toxics were assumed to be additive and were summed together in each census tract to estimate a total individual lifetime cancer risk in each tract. To roughly estimate the number of cancer cases from lifetime exposures across California, the total cancer risk in each census tract was multiplied by the total tract population and summed across all census tracts

(N = 5,858 tracts). The potential for over- and underestimation of risks are qualitatively evaluated below.

2.4. Assessing Noncancer Risks

For noncancer health risks, pollutant concentration estimates were divided by their corresponding Reference Concentration (RfC) or Reference Exposure Level (REL) to derive a hazard ratio. Hazard ratios for each pollutant in each census tract were calculated using the formula

$$HR_{ij} = \frac{C_{ij}}{RFC_j}, \quad (2)$$

where HR_{ij} is the hazard ratio for pollutant j in tract i, C_{ij} is the concentration in $\mu g/m^3$ of pollutant j in census tract i, and RfC_j is the reference concentration or REL for pollutant j in ug/m^3. Hazard ratios greater than one indicate that outdoor HAP concentrations exceed their RfC and may be of concern for noncancer effects.

An indicator of total noncancer hazard was calculated by summing together the hazard ratios for each pollutant in order to derive a total hazard index,

$$HI_i = \Sigma_j HR_{ij}, \quad (3)$$

where HI_i is the sum of the hazard ratios for all pollutants (j) in census tract i. This measure assumes that multiple subthreshold exposures may result in an adverse health effect. Aggregate noncancer hazards for specific target organ systems were also evaluated by calculating a separate total hazard index for several chronic noncancer endpoints. These measures assume that in the absence of comprehensive information, the effects of each pollutant are additive for a given organ system.[29] California's AB2588 draft "Guidelines and Technical Support Document on Non-cancer Chronic Reference Exposure Levels" lists the pollutants to be considered in the total hazard index for each toxicological endpoint, and can be found in the Appendix.[20,30]

3. RESULTS

3.1. Geographic Distribution of Air Toxics Concentrations and Cumulative Health Risks by County

The maps in Figs. 1–3 provide a visual characterization of the air toxics problem in California by showing shaded quintiles of total pollutant concentrations and cancer and noncancer hazard by county (N = 58 counties). County-level analysis provides useful comparative information on HAP exposure and hazard and contains less uncertainty than census tract estimates, although sensitivity is somewhat diminished. Each number in the map identifies a county name in the legend.

In Fig. 1, county-level air toxics concentrations represent average total HAP concentrations across census tracts within each county. There is considerable variability in the concentrations among the census tracts within a county. The coefficient of variation of the total mean HAP concentrations across census tracts within each county ranged from 4% in Trinity County to 62% in Santa Clara County, with a median value of 35%. The average total HAP concentration across all 58 counties was 21.2 $\mu g/m^3$, with a median of 18.9 $\mu g/m^3$ and a standard deviation of 12.7 $\mu g/m^3$. There is wide geographical variation in air toxics distributions, with the highest concentrations found primarily in urban counties in the San Francisco Bay Area, Los Angeles Basin, and San Diego.

Figure 2 displays the distribution of total noncancer hazard indices by county. The average hazard index across all counties was 13, with a median of 10 and a standard deviation of 9. Counties with the highest noncancer hazard indices are predominantly urban, although some rural areas have high average hazard indices. Figure 3 shows the distribution of county averages for individual lifetime cancer risk. The average individual lifetime cancer risk across all counties was $1.5E^{-4}$ with a median of $1.3E^{-4}$, and a standard deviation of $9.0E^{-5}$. Metropolitan areas have the highest cancer risk estimates along with a few rural counties.

Figures 1–3 illustrate that although hazardous air pollutants are ubiquitous in the environment, the potential cancer and noncancer health hazards posed by these ambient exposures are geographically concentrated in three urbanized areas with a high population density, and in a few rural counties. County-level distributions of total HAP concentrations are highly correlated with cumulative cancer and noncancer hazard indicators. Spearman rank correlations indicate that there are significant and positive relationships between total average HAP concentrations and average individual lifetime cancer risk ($r = 0.92, p < 0.001$) and average hazard index ($r = 0.61, p < 0.001$).

3.2. Cumulative Cancer and Noncancer Hazard Estimates by Census Tract

At the census tract level (N = 5,858 tracts) for total noncancer hazards, the average total hazard index across all census tracts was equal to 21, with a me-

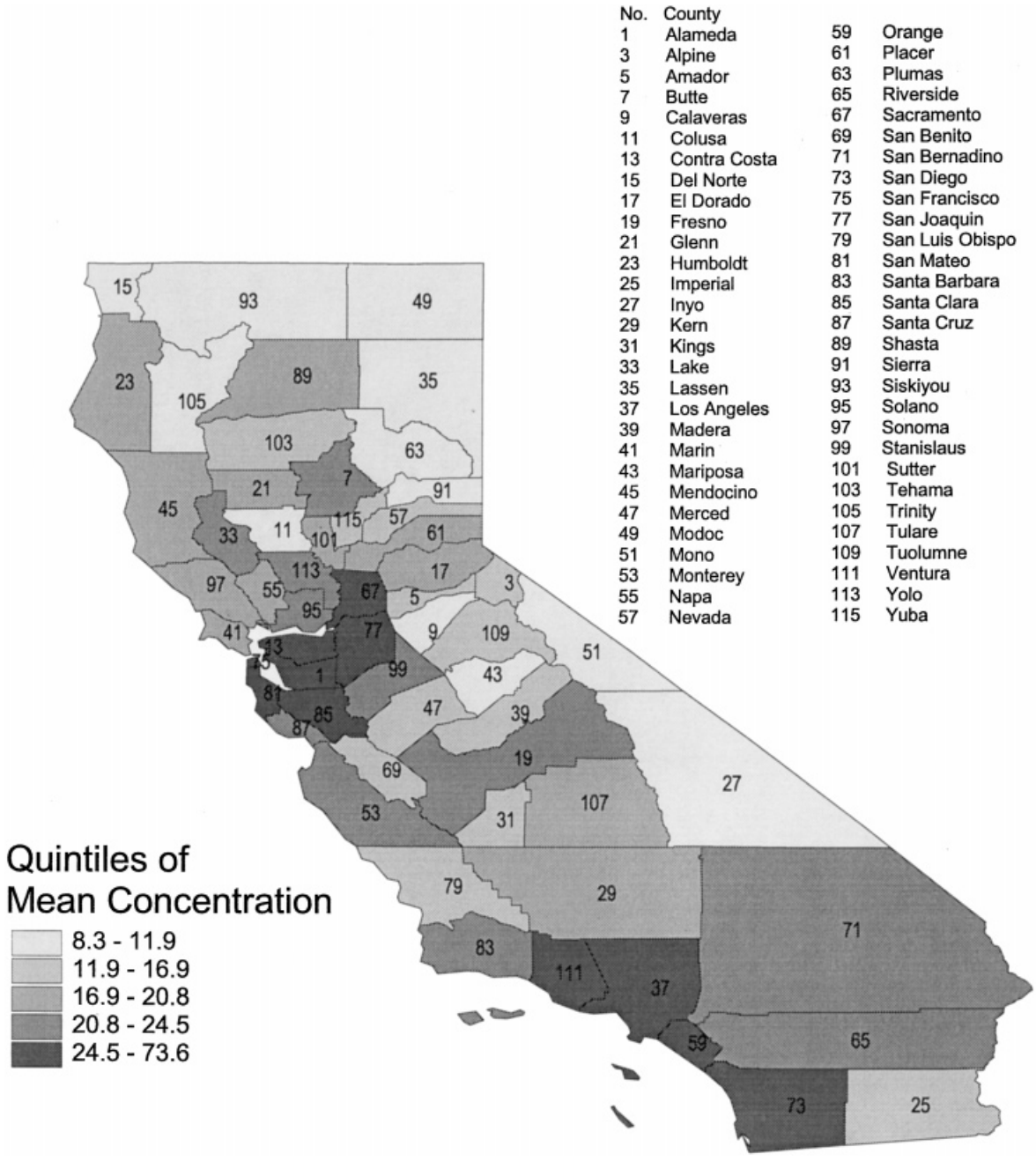

Fig. 1. Mean total air toxics concentration by California county ($\mu g/m^3$).

dian of 17, a minimum of 0.7, and a maximum of 382. Over 99% of all census tracts had total hazard indices exceeding 1, and over 85% of tracts had total hazard indices exceeding 10. Acrolein was the main driver of these total hazard indices.

Hazard indices were also calculated by specific health endpoints and target organ systems. Total hazard indices for respiratory effects exceeded 4 in 95% of all census tracts, with a mean of 20, a median of 17, a maximum of 381, and a minimum of 0.4. For central nervous system effects, slightly over 5% of all tracts had total hazard indices greater than 1, with a mean of 0.9, a median of 0.6, a maximum of 14, and a minimum of 0.4. For renal effects, 5% of all tracts had to-

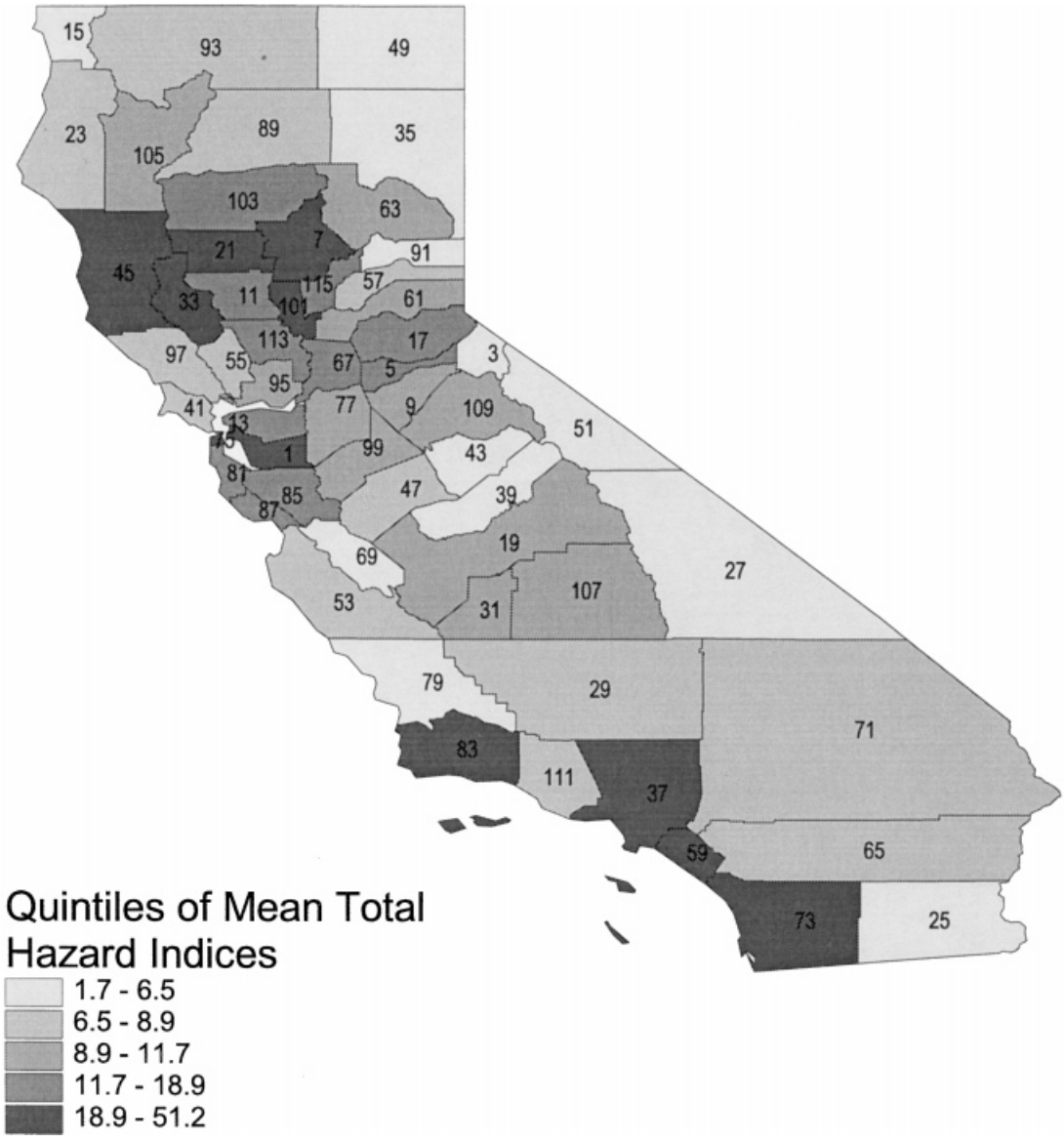

Fig. 2. Mean total noncancer hazard indices by California county.

tal hazard indices exceeding 2.6, with a median of 0.7, a maximum of 67, and a minimum less than 0.1. Analysis for gastrointestinal and liver effects revealed that 25% of all tracts had hazard indices greater than 1.5, with a median of 1.1, a maximum of 66.7, and minimum of 0.4. Total hazard indices for other health effects, such as cardiovascular, immunological, reproductive, and developmental endpoints did not exceed 1 in any of the census tracts.

For estimated lifetime individual cancer risks, the mean cumulative individual cancer risk was $3.0E^{-4}$ with a median of $2.7E^{-4}$, a maximum of $2.8E^{-3}$, and a minimum individual cancer risk of $3.7E^{-5}$. Over 75% of the census tracts had cumulative individual cancer risks of $1.0E^{-4}$ or less ($n = 4{,}393$ tracts), and fewer than 1% of tracts ($n < 50$ tracts) had ambient HAP concentrations posing a cumulative cancer risk of $1.0E^{-3}$ or greater.

3.3. Pollutant-Specific Health Risk Estimates

Air toxics were screened individually for their potential cancer risk. Figure 4 presents percentile plots of estimated lifetime cancer risk for 42 known

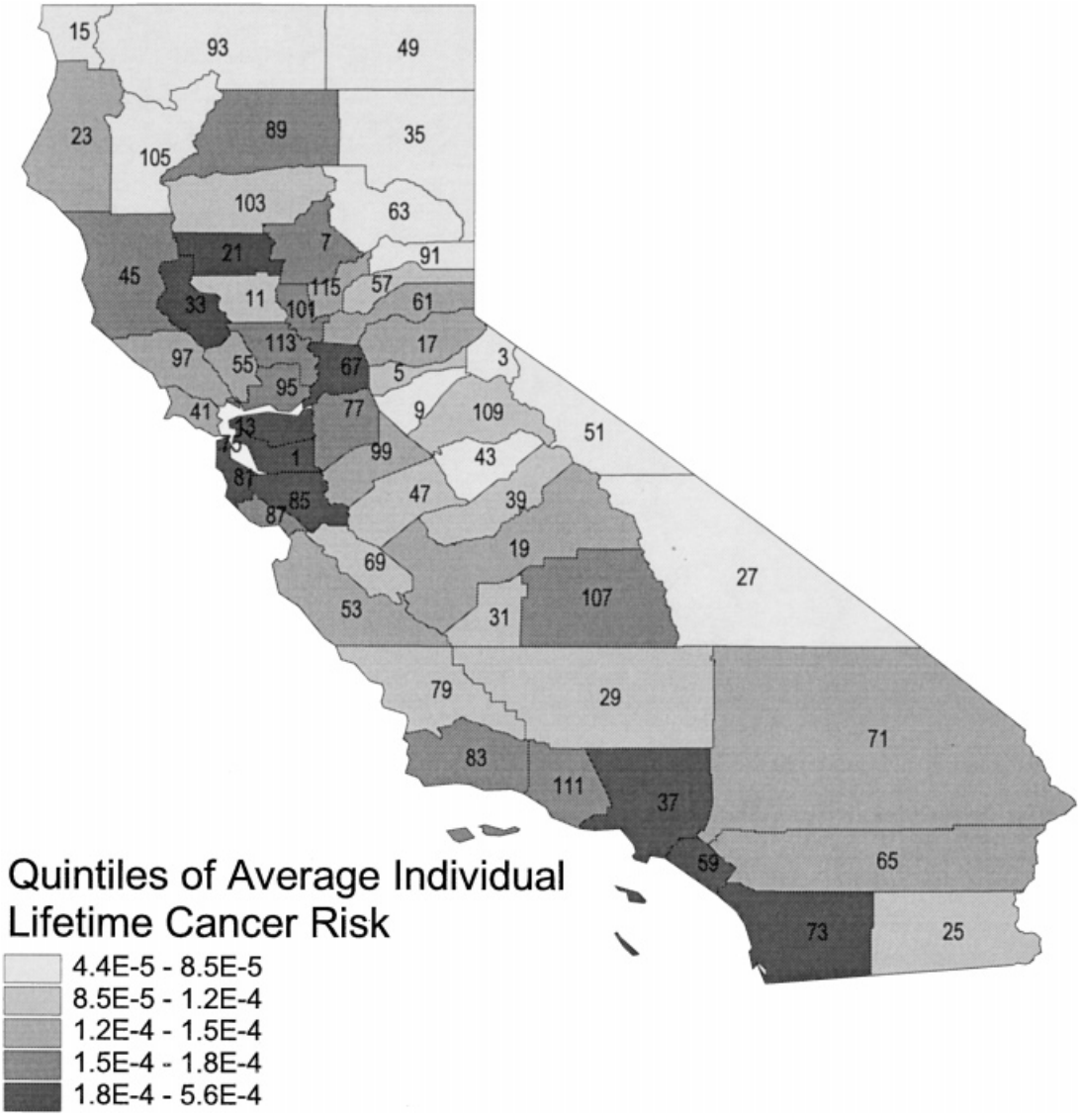

Fig. 3. Average individual lifetime cancer risk by California county.

or probable human carcinogens (EPA Group A and B, or IARC Group 1 and 2A). The distributions shown represent variation in concentration estimates across tracts, but do not include consideration of uncertainty or variability. Twenty-eight pollutants had at least one census tract with concentrations exceeding a 10^{-6} estimated cancer risk. Five pollutants (chromium, 1,3 butadiene, formaldehyde, POM, and benzene) had concentrations posing a potential cancer risk of 10^{-5} or greater in at least 75% of census tracts; two of these pollutants had maximum concentration estimates approximating a 10^{-3} risk (chromium, 1,3 butadiene). Chromium, 1,3 butadiene, formaldehyde, POM, benzene, and carbon tetrachloride all had median concentrations exceeding a 10^{-5} estimated cancer risk. There are several pollutants with modeled concentrations that included a constant background level: formaldehyde, benzene, ethylene dibromide, chloroform, and carbon tetrachloride. For some of these pollutants, the median is equal to the background and defines the lower bound of the percentile distribution. Six Group C (possible human) carcinogens were evaluated (not shown). Among these, only p-dichlorobenzene and methyl chloride had any census tracts with concentrations that exceeded an individual estimated cancer risk of 10^{-6}.

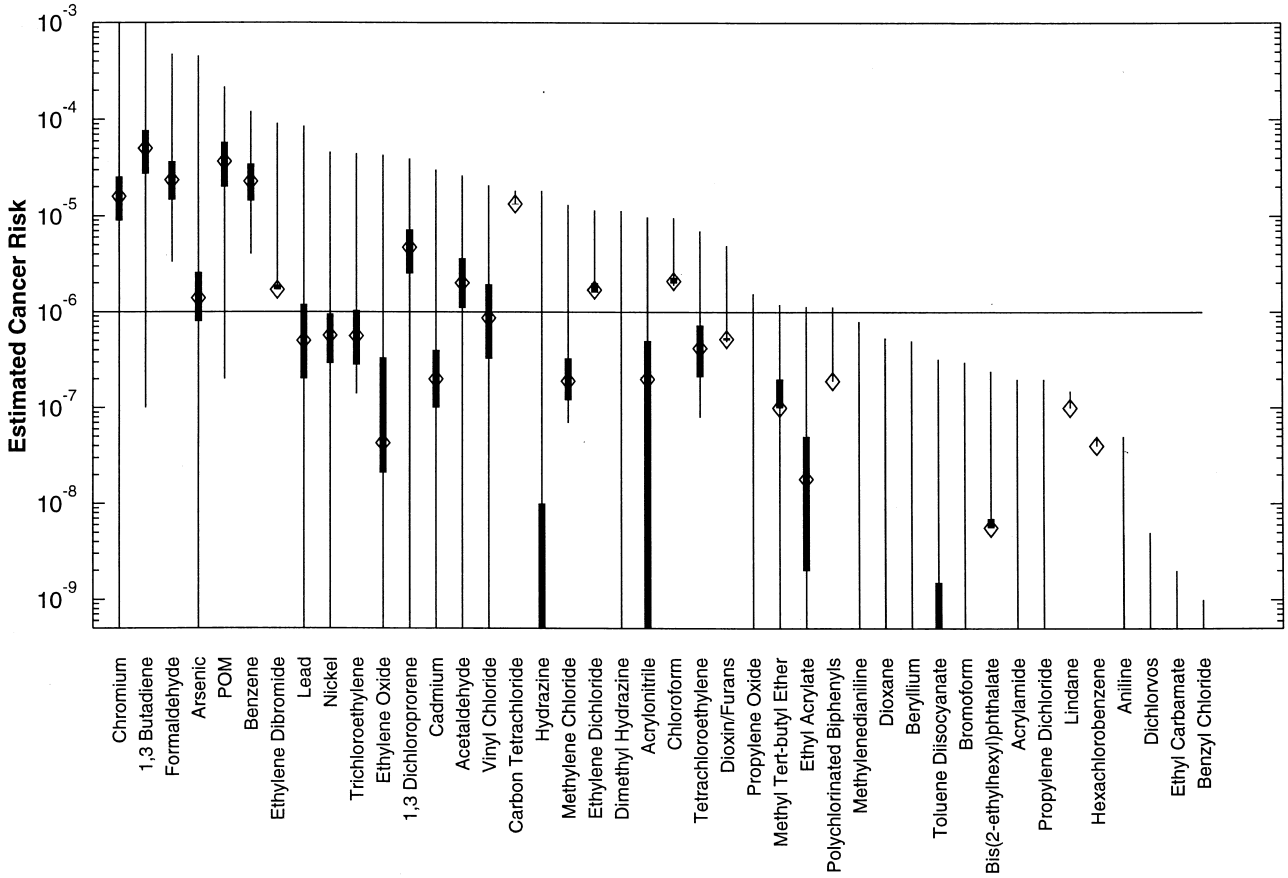

Fig. 4. Estimated cancer risk percentiles for Group A and B carcinogens. The thin line spans the range of data, the thick line spans the 25th–75th percentile, and the diamond marks the median.

Table I shows the individual HAP contributions to total estimated excess lifetime cancer incidence in California. Over 8,600 excess lifetime cancer cases were estimated based on cancer risk and population per tract. Sixteen of the 89 pollutants with a cancer potency value (18%) accounted for 97% of the estimated excess lifetime cancer incidence, and just four pollutants (polycyclic organic matter, 1,3 butadiene, formaldehyde, and benzene) accounted for over 70% of the estimated cases. Ninety-three percent of the estimated lifetime cases were associated with Group A or B carcinogens.

To compare the magnitude of estimated noncancer hazard among HAPs, Fig. 5 presents the percentile distributions for the hazard ratios of individual pollutants. Acrolein had hazard ratios exceeding 10 in over 75% of census tracts, while chromium, methylene diphenyl diisocyanate, manganese, and formaldehyde had hazard ratios exceeding 10 in 1% of census tracts. The remaining pollutants had distributions where hazard ratios were nearly equal to or less than one in all tracts.

Potential aggregate noncancer hazard is shown in Table II, which presents by HAP the number and percent of tracts where the hazard ratio exceeds one, and the relative contributions of each of these pollutants to the average hazard index. Acrolein and chromium had by far the highest number of tracts where hazard ratios exceeded one (99% and 28% of California census tracts, respectively) and together these pollutants contributed an average of 94% to tract level hazard index estimates. Formaldehyde hazard ratios exceeded one in 10% of census tracts, methylene diphenyl diisocyanate in 3%, and manganese in 1%. These three pollutants combined contributed less than 5% to the average hazard index.

Emission source allocations for the top five pollutants contributing to predicted cancer and noncancer hazard are presented in Table III. Average source contributions to estimated pollutant concentrations

Table I. HAP Contributions to Total Predicted Lifetime Cancer Incidence in California

Pollutant	WOE[a] classification	Tier[b]	Number of estimated cancer cases	Percent contribution to total predicted cancer cases	Cumulative percent
Polycyclic organic matter[c]	B	II	2,976	34	34
1,3 Butadiene	B	I	1,709	20	54
Formaldehyde	B	I	791	9	63
Benzene	A	I	749	9	72
Chromium	A	I	641	7	79
Carbon tetrachloride	B	II	395	5	84
Acrolein	C	III	335	4	88
1,3 Dichloropropene	B	I	170	2	90
Cresol	C	III	114	1	91
Styrene	C	III	108	1	92
Arsenic	A	I	85	1	93
Acetaldehyde	B	I	70	1	94
Methyl chloride	C	I	67	1	95
Chloroform	B	I	65	1	95
Ethylene dibromide	B	I	58	1	96
Ethylene dichloride	B	I	57	1	97
Other HAPS			276	3	100
Total estimated cancer incidence			8,667	100	

[a] Weight of evidence classification for carcinogenicity.
[b] Tier classification of cancer potency value.
[c] Classified as a B carcinogen based on benzo(a)pyrene.

are divided between point, area, and mobile sources. Point sources include manufacturing facilities reporting emissions to the Toxic Release Inventory, as well as nonmanufacturing facilities such as municipal waste combusters, hazardous waste disposal facilities, and electric utility generators. Area sources include small manufacturing and nonmanufacturing facilities, such as drycleaners and autobody paint shops. Mobile sources encompass both on-road and off-road sources, such as cars, trains, aircraft, and agricultural vehicles. Overall, area and mobile sources accounted for a major portion of the modeled concentrations of these pollutants, with the exception of methylene diphenyl diisocyanate concentrations, which are all attributable to large point sources. Mobile emissions constituted over half of the source contributions to outdoor concentrations of acrolein (59%), benzene (60%), 1,3 butadiene (81%), formaldehyde (51%), and POM (77%). Area sources were major contributors to outdoor manganese and chromium concentrations. Benzene and formaldehyde had background concentration data available that showed contributions of over 20% to estimated outdoor concentrations and about 2% to estimated excess lifetime cancer risk (not shown).

The bar chart in Fig. 6 shows source contributions to total air toxics concentrations, total estimated excess lifetime cancer incidence, and the average hazard index with the effects of background concentrations removed. All three bars indicate that mobile and area sources are the major contributors to average concentration and total cancer and noncancer health risk estimates, although the relative contributions of each source category vary somewhat between exposure and risk metric. Area sources contribute largely to total average air toxics concentrations (49%), whereas mobile sources account for the largest portion of source contributions to the total hazard index across all HAPs (56%) and to estimated excess lifetime cancer incidence (52%). Although point sources do not appear to contribute substantially to average modeled concentrations and predicted cancer and noncancer health risk measures in California, there are several tracts in the state where point source contributions are dominant. Counties such as Contra Costa, Los Angeles, Orange, Fresno, San Diego, and Humboldt have several tracts where point sources contribute 25% or more to estimated HAP concentrations.

4. DISCUSSION

This analysis of California's air toxics problem reveals that several hazardous air pollutants have es-

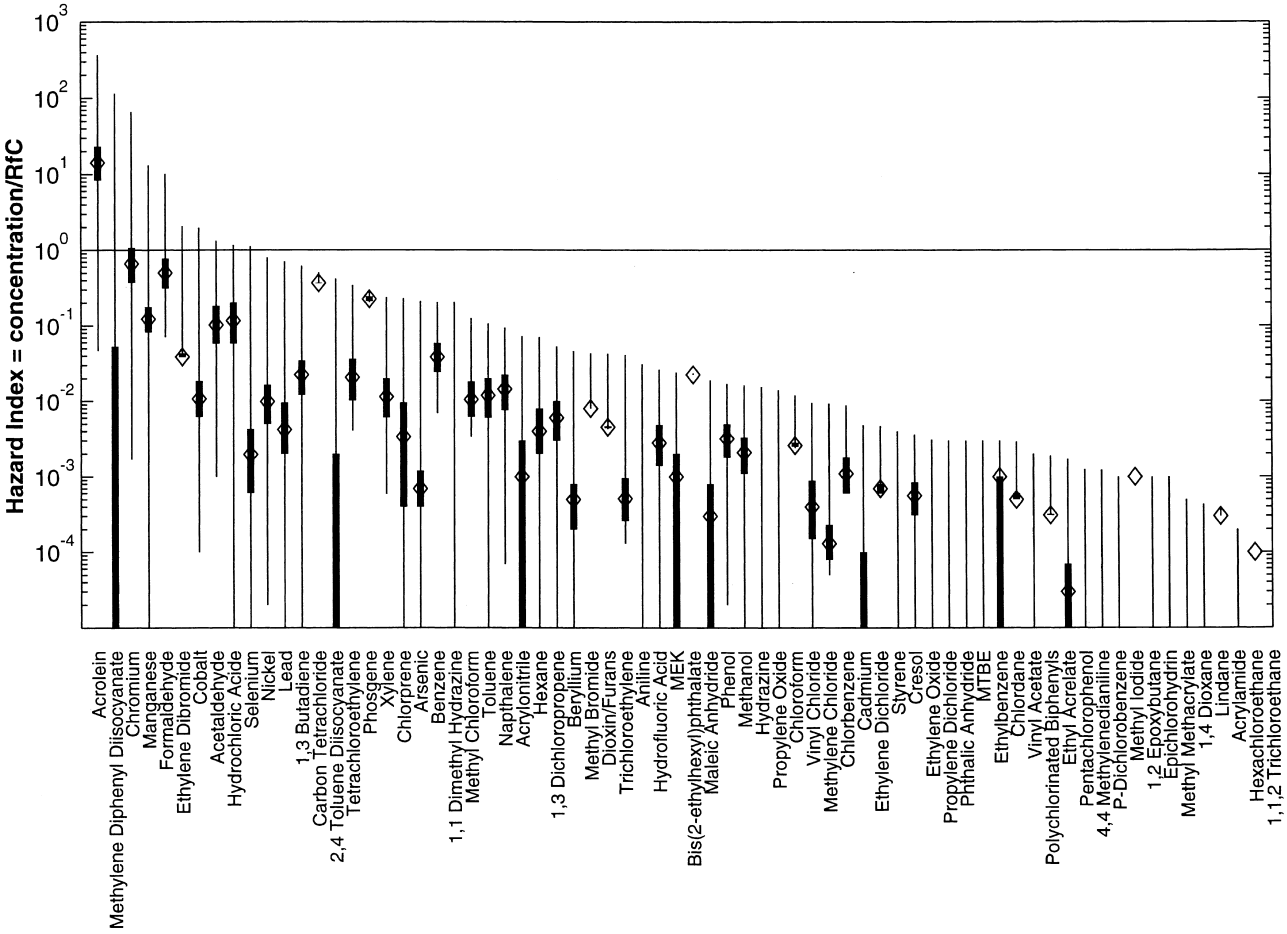

Fig. 5. Percentile distributions for hazard indices. The thin line spans the range of data, the thick line spans the 25th–75th percentile, and the diamond marks the median.

Table II. HAP Contributions to Estimated Noncancer Risk

Pollutant	Tier	Number (%) of tracts where hazard ratio[a] exceeds 1	Percent contribution to average hazard index[b]
Acrolein	I	5,818 (99)	89
Chromium	II	1,688 (28)	5
Formaldehyde	II	562 (10)	3
Methylene diphenyl diisocyanate	I	154 (3)	1
Manganese	I	70 (1)	1
Cobalt	II	7 (0.1)	0.1
Acetaldehyde	I	4 (0.1)	0.6
Hydrochloric acid	I	4 (0.1)	0.7
Ethylene dibromide	II	3 (0.1)	0.2

[a] Hazard ratio = individual pollutant concentration divided by its corresponding reference concentration.
[b] Hazard index = sum of the hazard ratios for each pollutant.

Table III. Emission Source Contributions to Average HAP Concentration Estimates for Top Five Pollutants With Highest Predicted Cancer Risk and Noncancer Risk

Pollutant	Top 5 for predicted cancer risk	Top 5 for cumulative noncancer hazard index	% Point[a]	% Area[b]	% Mobile[c]	% Background[d]	Average concentration ($\mu g/m^3$)
Acrolein	No	Yes	1	40	59	*	0.36
Benzene	Yes	No	1	16	60	23	3.03
1,3 Butadiene	Yes	No	1	18	81	*	0.22
Chromium	Yes	Yes	22	59	19	*	0.002
Formaldehyde	Yes	Yes	1	19	51	28	2.42
Manganese	No	Yes	5	66	29	*	0.008
Methylene diphenyl diisocyanate	No	Yes	100	0	0	*	0.003
POM	Yes	No	4	18	77	*	0.29

[a] Point sources include manufacturing facilitates reporting emissions to the Toxic Release Inventory, and nonmanufacturing facilities such as municipal waste combusters, hazardous waste disposal facilities, and electric utility generators.
[b] Area sources include area manufacturing and area nonmanufacturing sources (e.g., dry cleaners).
[c] Mobile sources include on-road and off-road sources.
[d] Background levels were identified for only 28 pollutants in the analysis and were treated as a constant across all tracts and added to modeled concentrations from anthropogenic sources.
*Background levels not modeled.

timated concentrations that raise important questions related to lifetime cancer and noncancer risks. Approximately 8,600 excess lifetime cancer cases and an average hazard index of 21 are associated with modeled 1990 HAP concentrations in census tracts across the state. These results are consistent with a previous national study using the same exposure data that found the number of cancer and noncancer toxicity benchmarks exceeded by modeled concentrations ranged from 8 to 32 per census tract.[15] Several uncertainties have ramifications for the risk estimates in this study, including: uncertainties in the cancer and noncancer toxicity information, and assumptions inherent in the risk assessment process; performance issues in the modeling algorithm; and emission source allocations.

4.1. Limitations of Toxicity Information and Uncertainties in Science-Policy Assumptions

Critiques of the relative strengths and weaknesses of current risk assessment tools suggest that crucial assumptions and science-policy decisions may lead to over- and underestimates of health risks.[31] For noncancer risk estimates, the total hazard index provides a useful screening-level tool for potential hazards for specific health endpoints or target organ systems, but does not provide an estimate of incidence or probability of effects. Several HAPs in this study have similar health endpoints seen at a variety of experimental conditions. Therefore, while providing a rationale for combining HAP exposures for a composite hazard index, the results should not be interpreted to mean that the common health effects would necessarily occur at ambient exposure levels. More research is needed to determine precisely what the noncancer risks are from total ambient HAP exposures. Little is known about how these pollutants interact to fully evaluate the health risks posed by cumulative air toxics exposures. Synergistic or antagonistic interactions among pollutants may mitigate risks in a way that has not been identified in this study. Nevertheless, this analysis clearly indicates that a relatively large proportion of California's population are experiencing concurrent HAP exposures that may pose potential noncancer risks for various endpoints.

Second, health risk estimates are somewhat limited by the availability of toxicity data, much of which is incomplete. Of the 148 pollutants analyzed, only 60% had cancer potency estimates, and 61% had chronic noncancer toxicity values, whereas 28% had no available toxicity information. Regulatory concern traditionally has emphasized cancer, which in part explains the lack of specific hazard information for other chronic outcomes such as developmental, reproductive, and neurological effects. For example, studies have indicated that benzene and 1,3 butadiene have potentially adverse chronic effects in addition to cancer, but there is no chronic toxicity infor-

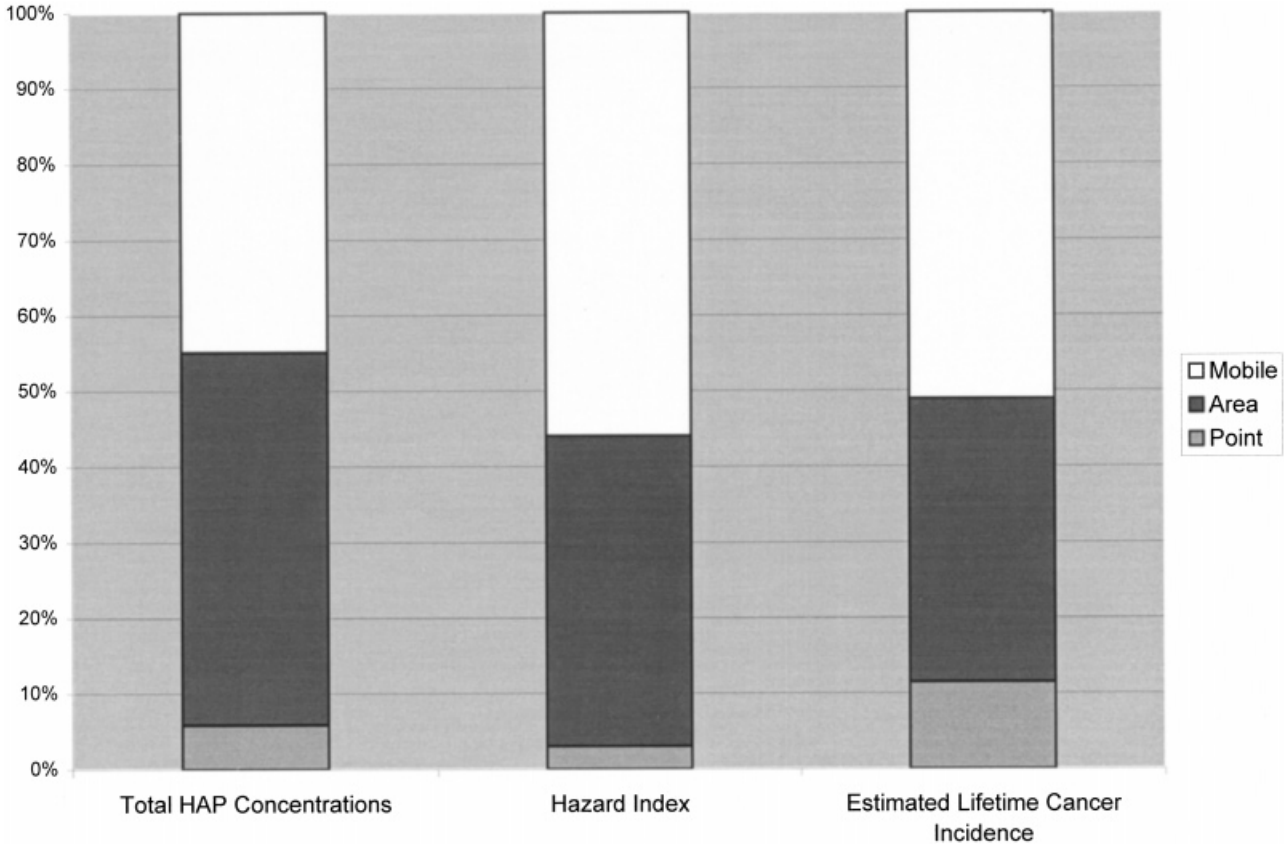

Fig. 6. Emission source contributions to air toxics concentrations, hazard index, and estimated lifetime cancer incidence in California (excludes background level effects).

mation available to compare with concentration estimates.[2]

More pollutants were associated with potential cancer risks than noncancer effects. Although concentrations for several carcinogenic air toxics were high, other factors may also help explain the predominance of cancer risk estimates. Cancer potencies are derived from occupational studies in humans—typically adult males—when available, and otherwise from toxicological studies in animals. A potency based on human data is typically defined as the maximum likelihood estimate derived from the available dose-response data. Yet, because potency estimates derived from human data are generally based on occupational cohorts of healthy males, they may not adequately reflect population variability in susceptibility, including sensitive populations—such as children, who may have increased exposures and are physiologically different from adults in how they metabolize chemicals in the body.[32,33] It is important to note that potency estimates for two of the pollutants in this study, benzene and chromium, are maximum likelihood estimates based on occupational studies and contribute significantly to overall risk estimates. Another important risk driver, POM, contains a myriad of constituents, many of which are toxicologically uncharacterized, which creates uncertainties when assigning this pollutant group a potency estimate.[25,26,34] Moreover, the toxic potential of diesel exhaust, which contains several HAPs and POM constituents, may not be adequately characterized in this study. The modeled concentrations used for this study estimate POM concentrations for a limited number of constituents and do not consider all the constituents of diesel exhaust. POM concentration estimates include 36 polycyclic aromatic hydrocarbons (PAHs), but there are other PAH species not included in the emissions inventory, including several that have carcinogenic potencies that are higher than those included in this analysis.[27] Thus, the characterization of the cancer hazard of the POM in the ambient air may be underestimated.

It is also important to point out that potential overestimation of total cancer risk can result by combining upper-bound cancer potency estimates for several pollutants. A recent study found that, in general, combining the upper-bound cancer risk for several pollutants can lead to an estimate of cancer risk that is about twice as high than the true 95% upper confidence bound of all the pollutants combined.[35] Moreover, potencies derived from animal studies have additional uncertainties that can lead to over- and underestimation due to some of the following factors: conducting studies on a homogeneous animal population that may not be reflective of the responses of a heterogeneous human population; the dosing of animals at levels that are much higher than concentrations typically experienced by humans; the process of extrapolating from these high dose levels to lower doses typically experienced by the general population; and the process of extrapolating results from animals to humans, based on adult males.[31]

4.2. Performance of Dispersion Model

Estimated concentrations and health risks may also be affected by potential bias in the modeled air toxics concentrations used in this study. Modeled estimates represent long-term average concentrations and do not capture spatial or temporal peak concentrations. Moreover, comparison of this study's modeled concentrations with available measured air toxics concentrations, shows a general tendency for the model to underestimate actual concentrations.[13,14] For pollutants of particular significance to this analysis for cancer and noncancer risk estimates, model-to-monitor comparisons for 1,3 butadiene and benzene suggested a degree of underestimation (by factors of 3 and 2, respectively), while formaldehyde and carbon tetrachloride comparisons showed good agreement between modeled and monitored concentrations. Unfortunately, no monitored information was available to assess the concentration estimates during 1990 for acrolein, which drives a major portion of the estimated total noncancer index. Subsequent monitoring that is more geographically comprehensive and encompasses a wider range of pollutants is necessary for further evaluating dispersion modeling estimates.

4.3. Emission Source Allocations

Emission source allocations indicate that on average, total air toxics concentrations and their associated cancer and noncancer health risks originate mostly from manufacturing and nonmanufacturing area sources, and mobile sources. Area and mobile source emissions also account for a substantial portion of the estimated concentrations of the top five pollutants driving total cancer and noncancer risk estimates, such as benzene, formaldehyde, acrolein, POM, manganese, 1,3 butadiene, methylene diphenyl diisocyanate, and chromium. Background levels that originate from re-suspension and transport of historical emissions and nonanthropogenic sources were major contributors to estimated concentrations of carbon tetrachloride, formaldehyde, and benzene. Thus, point sources such as large manufacturing facilities, refineries, and treatment, storage, and disposal facilities, appear to contribute considerably less on average to ambient air toxics concentrations in California than mobile or area sources. Nevertheless, there are specific tracts in several counties where point sources account for a large portion (at least 25%) of estimated air toxics concentrations and associated health risks. In general, the source apportionment pattern found in this analysis differs substantially from source allocations based on release data in the national air toxics emissions inventory used to generate the modeled concentrations. Nationally, point sources contribute a large percentage of air toxics releases (20%), although this remains less than releases from area and mobile sources (42% and 38%, respectively).[14] This discrepancy between the proportion of total releases and the proportion of health risks attributable to point sources, could be due to several factors: (1) missing toxicity values for pollutants that are emitted largely from point sources, which could lead to underestimates of the cancer and noncancer health risks of concentrations originating from this source category; (2) lower toxicity of high mass release air toxics from point sources, such as xylene and toluene, relative to other air toxics; (3) lower pollutant concentrations per unit of emissions from point sources due to elevated release points (e.g., smokestacks); (4) possible location of many point sources in less populated areas.

This study uses emissions data for 1990 to generate modeled HAP concentrations, yet changes in pollutant levels may have occurred since that time. Nationally, total volatile organic compounds (VOCs) emissions have reportedly decreased by 3% between 1990 and 1995.[36] Emissions and concentrations of the HAPs examined in this study may have also declined during this time period, but these changes will probably vary geographically and across source categories.

For example, on-road emissions of VOCs declined by 11% due to regulatory efforts such as the introduction of reformulated gasoline in polluted urban areas, while emissions for area and point sources and nonroad mobile sources have increased somewhat.[36] Monitoring data from California shows declines in some of the pollutants that have the highest contribution to overall estimated cancer risk. For example, between 1990 and 1995, benzene concentrations declined between 55% and 78%, and 1,3-butadiene concentrations declined 10–50%.

It is also important to point out that the definitions of point sources and area sources used in this study did not correspond precisely to the Clean Air Act Section 112 definitions for "major sources" and "area sources." Different definitions were necessary in this study due to the definitions in some of the emissions databases used. As a result, some of the emissions sources characterized as area sources in this study may in fact be regulated as major sources under the Clean Air Act, and similarly, some emissions treated as point sources in this study may be regarded as area sources under the Act. Nevertheless, the results of this study for the three types of sources are instructive, as the differences in definition are expected to relate to a small portion of estimated emissions.

Finally, this analysis implicitly treats outdoor concentration estimates as equivalent to actual personal exposure to the population. However, human exposures to air pollution depend on concentrations in both indoor and outdoor environments and the amount of time people spend in various locations, including their homes, workplaces, and commuting.[37] Nevertheless, outdoor pollutant levels remain an important contributor to human exposures, even though most individuals spend between 80% and 90% of their time indoors.[38] A sampling of indoor and outdoor concentrations of VOCs found that most of these pollutants readily penetrate from outdoor to indoor air, even when air exchange rates are low.[39] Penetration of outdoor air toxics in particulate form was also fairly high.[40] Thus, while individuals are exposed to significant pollutant levels emanating from indoor sources—such as tobacco smoke—outdoor air toxics levels are a component of both indoor and outdoor exposures for all individuals, and represent a useful approximation of long-term exposures to air toxics in the absence of indoor sources.

5. CONCLUSIONS

This analysis demonstrates that potential cancer and noncancer health risks posed by outdoor air toxics in California may be of public health concern. Moreover, it identifies those pollutants and emissions sources that appear to be contributing heavily to estimated concentrations and cumulative health risks for the year 1990, when the Clean Air Act was amended and targeted emissions reduction efforts toward HAPs. These study results provide a baseline to assess subsequent progress in regulatory efforts to reduce the human health risks of air toxics in California. Given the challenge of assessing the cumulative health impacts of air toxics, further research is needed to better characterize chronic noncancer health risks by endpoint and to assess the exposures and risks posed by these pollutants in other media. From a regulatory perspective, the significant area and mobile source contributions to the air toxics problem tend to be more difficult to control in terms of emissions reduction activities, as compared to larger point sources which have traditionally been the focus of so-called "command and control" efforts. The proliferation of mobile sources combined with the wide dispersion of small area sources that are diverse in terms of their emissions and production characteristics, make the implementation of effective emissions reduction strategies more challenging. Future regulatory approaches to developing effective emissions reduction strategies must better address mobile and area emissions, with a particular emphasis on how changing land-use patterns, suburbanization, and the development of major transportation corridors, can affect pollution streams and the distribution of cancer and noncancer health risks.

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APPENDIX. Toxicological Endpoints to be Considered in a Hazard Index (Chronic Toxicity)

		Target organ or system								
	Pollutant	CV/BL	CNS/PNS	IMMUN	KIDNY	GI/LIVER	REPRO/ DEV	RESP	SKIN	ENDO
1	acetaldehyde							X		
2	acetamide									
3	acetonitrile									
4	acetophenone									
5	acrolein							X	X	
6	acrylamide		X							
7	acrylic acid							X		
8	acrylonitrile							X		
9	allyl chloride		X							
10	aniline	X								
11	anisidine									
12	antimony									
13	arsenic	X	X				X		X	
14	benzene	X	X	X			X			
15	benzotrichloride									
16	benzyl chloride							X		
17	beryllium							X		
18	biphenyl									
19	bis(2-ethylhexyl)phthalate					X		X		
20	bis(chloromethyl)−ether									
21	bromoform									
22	1,3 butadiene						X			
23	cadmium				X			X		
24	calcium cyanamide									
25	Captan									
26	Carbaryl									
27	carbon disulfide		X				X			
28	carbon tetrachloride		X			X	X			
29	carbonyl sulfide									
30	catechol									
31	chloramben									
32	chlordane									
33	chloroacetic acid									
34	chlorobenzene				X	X	X		X	
35	chloroform				X	X	X			
36	chloromethyl methyl ether									
37	chloroprene		X							
38	chromium				X	X		X		
39	cobalt							X		
40	cresol	X	X							
41	cumene									
42	cyanide compounds									
43	2,4 D									
44	dibutylphthalate									
45	3,3 dichlorobenzidine									
46	dichloroethyl ether									
47	1,3 dichloropropene									
48	Dichlorvos									
49	diethanolamine									
50	diethyle sulfate									
51	3,3 dimethoxybenzidine									
52	dimethyl formamide					X				
53	1,1 dimethyl hydrazine									
54	dimethyl phthalate									

(*Continued*)

APPENDIX. (*continued*)

Pollutant	Target organ or system								
	CV/BL	CNS/PNS	IMMUN	KIDNY	GI/LIVER	REPRO/ DEV	RESP	SKIN	ENDO
55 dimethyl sulfate									
56 4,6 dinitro-o-cresol									
57 2,4 dinitrophenol									
58 2,4 dinitrotoluene		X			X				
59 1,4 dioxane	X			X	X		X		
60 epichlorohydrin				X			X		
61 1,2 epoxybutane	X						X		
62 ethyl acrylate				X	X		X		
63 ethyl carbamate									
64 ethyl chloride					X	X	X		
65 ethylbenzene				X	X	X			
66 ethylene dibromide							X		
67 ethylene dichloride		X	X	X	X				
68 ethylene glycol									
69 ethylene oxide	X	X				X	X		
70 ethylene thiourea					X				X
71 ethylidene dichloride									
72 formaldehyde							X		
73 glycol ethers									
74 Heptachlor									
75 hexachlorobenzene					X				
76 hexachlorobutadiene				X	X				
77 hexachlorocyclopentadiene					X		X		
78 hexachloroethane		X		X	X				
79 hexane		X							
80 hydrazine					X		X	X	X
81 hydrochloric acid							X	X	
82 hydrofluoric acid							X	X	
83 hydroquinone									
84 lead	X	X	X	X		X			
85 Lindane									
86 maleic anhydride							X		
87 manganese		X					X		
88 Methyl Ethyl Ketone						X			
89 mercury compounds									
90 methanol		X							
91 methoxychlor									
92 methyl bromide		X			X	X	X		
93 methyl chloride									
94 methyl chloroform		X			X	X			
95 methyl hydrazine									
96 methyl iodide									
97 methyl isobutyl ketone									
98 methyl isocyanate						X	X	X	
99 methyl methacrylate		X				X			
100 methyl tert-butyl ether				X	X				
101 methylene bis(2-chloroaniline)									
102 methylene chloride	X	X			X				
103 methylene diphenyl diisocyanate							X		
104 4,4 methylenedianiline					X				
105 N,N-diethyl/dimethylaniline									
106 naphthalene	X						X		
107 nickel			X	X			X		
108 nitrobenzene					X	X	X		

(*Continued*)

APPENDIX. (*continued*)

Pollutant	Target organ or system								
	CV/BL	CNS/PNS	IMMUN	KIDNY	GI/LIVER	REPRO/ DEV	RESP	SKIN	ENDO
109 4 nitrophenol									
110 2 nitropropane					X				
111 o-toluidine									
112 p-dichlorobenzene					X				
113 p-phenylenediamine									
114 Parathion									
115 PCDD/PCDFs	X		X		X	X	X	X	X
116 pentachloronitrobenzene									
117 pentachlorophenol				X	X	X			
118 phenol	X	X		X	X		X		
119 phosgene							X		
120 phthalic anhydride				X	X		X		
121 polychlorinated biphenyls			X		X	X			
122 polycyclic organic matter									
123 propionaldehyde total									
124 Propoxur									
125 propylene dichloride									
126 propylene oxide							X		
127 1,2 propylenimine									
128 quinoline									
129 quinone									
130 selenium							X		
131 styrene		X			X				
132 styrene oxide							X		
133 1,1,2,2 tetrachloroethane									
134 tetrachloroethylene		X			X				
135 toluene		X			X	X			
136 2,4 toluene diamine									
137 2,4 toluene diisocyanate							X		
138 1,2,4 trichlorobenzene									
139 1,1,2 trichloroethane	X	X		X	X				
140 trichloroethylene		X			X				
141 2,4,6 trichlorophenol									
142 trifluralin									
143 2,2,4 trimethylpentane									
144 vinyl acetate					X	X			
145 vinyl bromide					X				
146 vinyl chloride		X			X	X			
147 vinylidene chloride					X				
148 xylene		X				X	X		

Adapted from References 27 and 35. CV/BL = cardiovascular or blood system; CNS/PNS = central or peripheral nervous system; IMMUN = immune system; KIDNY = kidney; GI/LIVER = gastrointestinal system and liver; RESP = respiratory system; REPRO/DEV = reproductive and developmental effects; SKIN = skin irritation; ENDO = endocrine disruption.

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