CA/DOH/AIHL/SP-30

EXECUTIVE SUMMARY

TO

MONITORING OF MUTAGENS AND CARCINOGENS IN COMMUNITY AIR

Contract No. ARB A1-029-32
Final Report
May 1984

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SUMMARY OF FINDINGS

Analyses of mutagenicity, air quality and meteorological measurements made between 1979 and 1982 in Contra Costa County yielded the following conclusions:

1. *A major portion of the mutagenicity of Contra Costa aerosols that were collected during August and October 1981 pollution episodes could be accounted for by the variability in the fine-fraction lead concentration in these aerosols. This observation suggests that during the summer and fall episodes the majority of the mutagenicity was due to vehicular emissions. The correlation between mutagenicity and fine-fraction lead during the winter episode in January 1982 was lower than during the summer or fall episodes. This suggests that during the winter episode vehicular emissions contributed proportionally less to mutagenicity than during the summer and fall episodes.*

2. *During the three 1981-82 episodes, no evidence that refineries contributed to aerosol mutagens was found. Nickel is a tracer for fuel oil combustion and refinery operation. No significant statistical relationship was found between nickel and aerosol mutagenicity. The monitoring site at Martinez, which is in close proximity to several refineries, experienced the highest average concentrations of nickel and the lowest average aerosol mutagenic densities.*

3. *The source pattern during the January 1982 winter episode was the most complex, and the measurements indicated unidentified sources of wintertime mutagens. Qualitative results suggested possible contributions of residential wood combustion to polycyclic aromatic hydrocarbons (PAH) during the winter.*

4. *Genetic evidence suggests that nitroarenes (nitro-substituted PAH) may have accounted for one-half or more of the observed direct-acting mutagenic density during pollution episodes. This is based on the observation that mutagenicities of most aerosol extracts were at least a factor or two lower in a nitroreductase-deficient strain of Salmonella (TA98NR) than in the parent strain (TA98).*

5. *Mutagenicity and PAH concentrations in four-month composites showed marked seasonal variations. Between November 1979 and June 1982, levels measured in the winter (November-Febraury) were five to ten times higher than those measured in the spring (March-June). Levels during the summer (July-October) were intermediate.*

6. *Annual average concentrations of mutagenicity and PAH did not change significantly over the period between November 1979 and June 1982.*

I. OBJECTIVES OF THE STUDY

The objectives of ARB Agreement A1-029-32 are:

A. To refine and standardize chemical and microbiological methods for determining the concentrations of selected mutagens and carcinogens in ambient community air particulate material.

B. To better determine the sources and chemical identities of mutagens and carcinogens in Contra Costa County community air.

C. To expand the community air mutagen-carcinogen data base for further integration with the epidemiological cancer studies in Contra Costa County and elsewhere.

II. BACKGROUND: THE WORK IN PERSPECTIVE

Research conducted over the past four decades has revealed the presence of a variety of chemical carcinogens in solvent extracts of community air particles (1). However, the presence of these chemicals, at the concentrations typically found in ambient air, constitutes a public health risk of uncertain magnitude (2).

Carcinogens in Dust and Air Particles

As early as 1942, Leiter, Shimkin and Shear (3) reported the experimental production of tumors in animals using tars from city air dusts. Connective tissue

tumors (sarcomas) were induced in mice following the injection of extracts of atmospheric particles collected in several eastern U.S. cities. In the early 1950s, Kotin and co-workers (4) demonstrated that carcinogenic aromatic hydrocarbons, including benz(a)pyrene (BAP), were present in Los Angeles air and that atmospheric extracts were carcinogenic to animals in the laboratory. Soon after, Sawicki and co-workers (5) measured BAP and other carcinogenic polycyclic aromatic hydrocarbons (PAH) in the air of more than two dozen American cities. BAP concentrations as high as 30 ng/m^3 were found in air particulate samples collected in Los Angeles during 1958-59; the annual average for the same period was 3 ng/m^3. Since that time BAP concentrations have decreased significantly in California and elsewhere (2,6). Until recently, research on carcinogens in community air particles has focused primarily on BAP and certain other carcinogenic PAH; however these compounds do not account for most of the carcinogenic activity of aerosol extracts. There must be other compounds which account for the "excess carcinogenicity" of ambient air extracts (7). Therefore the decrease in BAP over the past twenty years does not necessarily represent a significant reduction in the potential cancer hazard.

Air Particulate Mutagens

The recent development by Ames et al (8) of the Salmonella mutagenicity test has revolutionized environmental carcinogen testing. Because most chemicals that are carcinogenic in animals are also mutagenic in bacteria, the Ames test in practice is a good predictor of carcinogenic potential (9). Soon after introduction of the test in 1975, Pitts et al (10), Talcott and Wei (11) and Tokiwa et al (12) successfully applied it to community air particles and demonstrated mutagenic

activity in aerosol extracts. Research in this field has accelerated rapidly since then and we now know that chemical mutagens, as well as carcinogens, are ubiquitous components of the urban atmosphere in California and elsewhere (13-19).

Compounds of particular concern are those found in particulate polycyclic organic matter (POM). Mutagens in POM include certain unsubstituted PAH such as BAP and benz(a)anthracene (BAA).[1] However these PAH constitute only a small fraction of the observed mutagenicity of POM. Furthermore a major proportion of the mutagenicity in ambient POM extracts is due to direct-acting mutagens which do not require metabolic activation; the PAH require prior metabolic transformation to become active mutagens. Recent studies have focused on highly mutagenic nitrosubstituted PAH (nitroarenes) such as nitropyrenes, which are direct acting mutagens. Nitroarenes have been detected in diesel exhaust (20) and urban air (21,22).

It is important to assess the total mutagenic and carcinogenic potential of ambient air POM, especially as new and expanded energy technologies are introduced in California. One way, at least in theory would be to measure all the mutagens and carcinogens in POM. However, chemical methods are not now available which can detect all such compounds in complex mixtures. Further, even such exhaustive compilations would neglect synergistic and antagonistic effects. Fortunately, the Ames test has made this assessment task more tractable because it is a good predictor of the carcinogenicity (9).

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1. In the present study PAH is defined as the sum of eight unsubstituted PAH and one carbonyl derivative benzanthrone.

Epidemiological Studies with the Ames Test

Although not a quantitative test in the sense of having a well-established precision and accuracy, the Ames bioassay yields results which indicate relative mutagenicity. Thus, it has been considered appropriate for use in monitoring ambient air for relative mutagenicity and potential carcinogenicity. The results have been used as a key environmental measurement in epidemiological studies attempting to relate cancer and air pollution (16,17). Results obtained by AIHL using the Ames test were an integral part of the recent Contra Costa County Cancer Study (6). In the study, measurements of airborne mutagens, selected PAH and other chemical pollutants were integrated with lung cancer incidence data. The geographic distributions of mutagenicity and other air pollutants were not associated with the distribution of lung cancer, with one exception, viz. sulfate. However the correlation with $SO_4^{=}$ was significant only in males and disappeared when socio-economic status was factored in. Subsequently a case-control analysis established that smoking, not environmental or occupational hazards, was responsible for the high rate of lung cancer among male blue-collar workers in Contra Costa County (6). Thus, the tools of epidemiology did not detect an impact of community air pollution on the incidence of lung cancer. The lack of epidemiological sensitivity should not obscure the fact that many mutagens and carcinogens are present in community air particles.

The Excess Mutagenicity Problem

Another problem in applying the Ames test to ambient air mixtures is that organic extracts of air particulates are significantly more mutagenic than predicted on

the basis of the amounts of the known chemical mutagens present (23). Thus, there remains an "excess mutagenicity", as determined by the Ames test, which requires chemical description. The usefulness of the Ames test, as a predictor of potential health hazards will be enhanced if the disparity between observed and predicted mutagenicity can be lessened. Therefore, it is advantageous to use both the Ames test and chemical characterization together in attempting to assess the potential carcinogenic effects of ambient air particulate matter (16,17,23). Much current research is focused on identification of nitro-substituted PAH which may contribute significantly to the mutagenicity. Although easy to form, they are difficult to detect chemically (24,25). Recently new strains of Salmonella have been developed that are deficient in nitroreductase activity which allow them to be used as approximate "indicators" of mutagenic nitro-PAH in air samples (26). In the present work one of these strains TA98NR was incorporated into the Ames bioassay test in order to make the test not only a general predictor of genotoxicity but also an improved indicator of nitroorganics which might be causing some of the mutagenicity observed.

Sources of Airborne Mutagens

A fundamental problem concerns source identification. The measure of relatively high mutagenicity in a given geographical area is of limited value unless the major sources of the mutagenicity can be identified and therefore potentially controlled. The integrated use of chemical and biochemical data is of special value for this complex problem. There are indications that some of the elements (e.g., Pb,V) and PAH ratios (e.g., benzo(a)pyrene/benzo(ghi)perylene (BGP)) can be used as tracers for various pollution sources. For example, earlier studies (1,16)

have shown that power plant and petroleum refinery emissions have higher BAP/BGP ratios than auto emissions.

In the previous Contra Costa air pollution study, mutagenicity, organic and inorganic chemicals and gaseous pollutants were measured (16, 17). We sampled every sixth day for one year (1978-79) at 14 Contra Costa locations and concluded that mobile sources were undoubtedly significant contributors to carcinogenic PAH. However more research was needed to define the major sources of particulate mutagens. The present study attempts to address the problem of mutagenic sources.

III. EXPERIMENTAL APPROACH

This project was carried out in several concurrent and interconnected parts. One part was directed towards the refinement and standardization of chemical and microbiological methods for measuring selected carcinogens and mutagens in community air. A second part of the project consisted of three brief periods of intensive sampling and analysis to identify the sources and chemical nature of mutagenic activity and PAH in Contra Costa County community air. Sampling was carried out at four locations (Pittsburg, Richmond, Concord and Martinez) during seasonal pollution episodes in August and October 1981 and in January 1982 (Figure 1). A third part was the continuation, on a limited basis of the community air mutagen-carcinogen monitoring in Contra Costa County, initiated in 1978 under a grant from the EPA. This chronic phase consisted of measuring particulate mutagens and carcinogens in seasonal composites collected at the

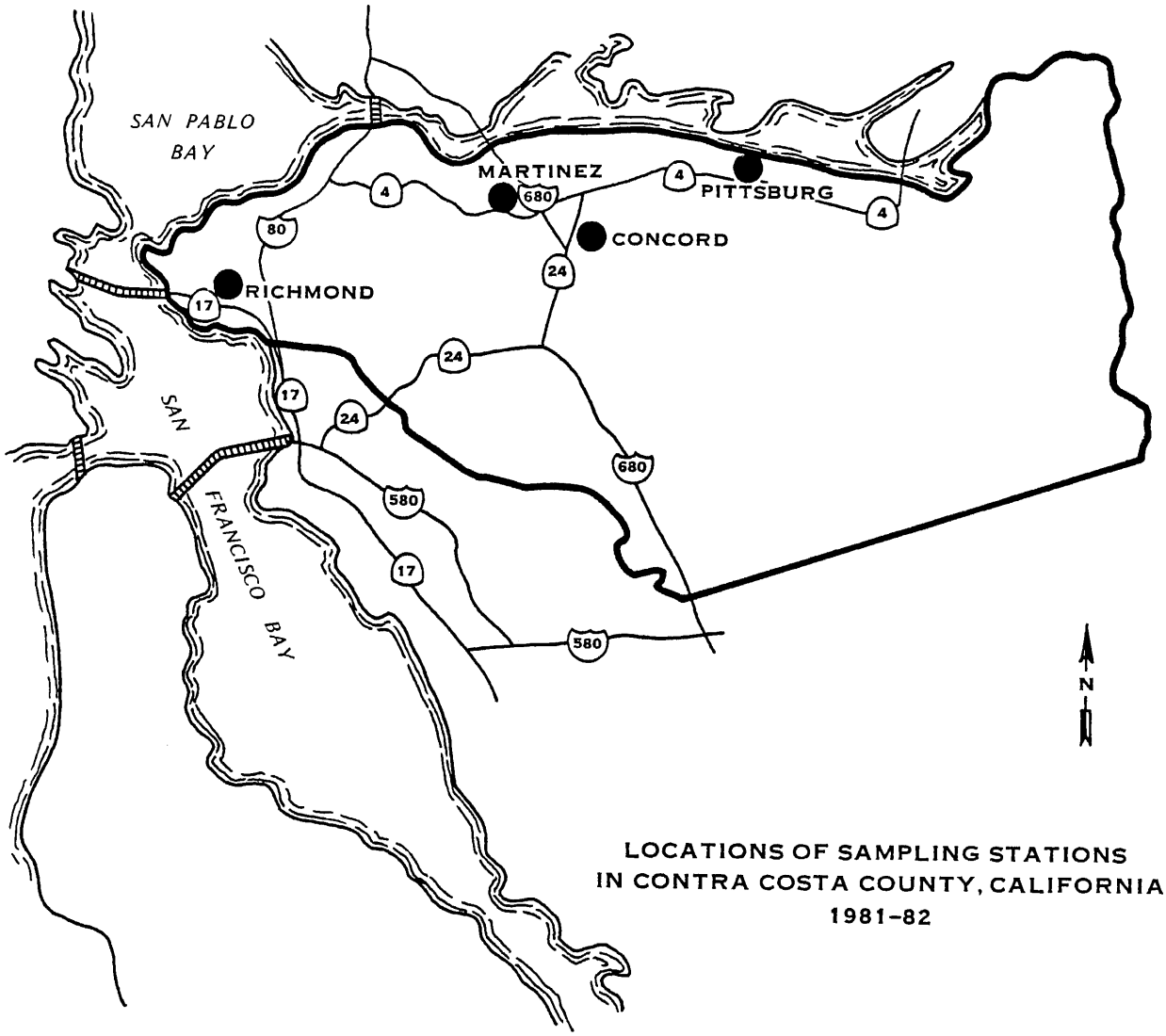
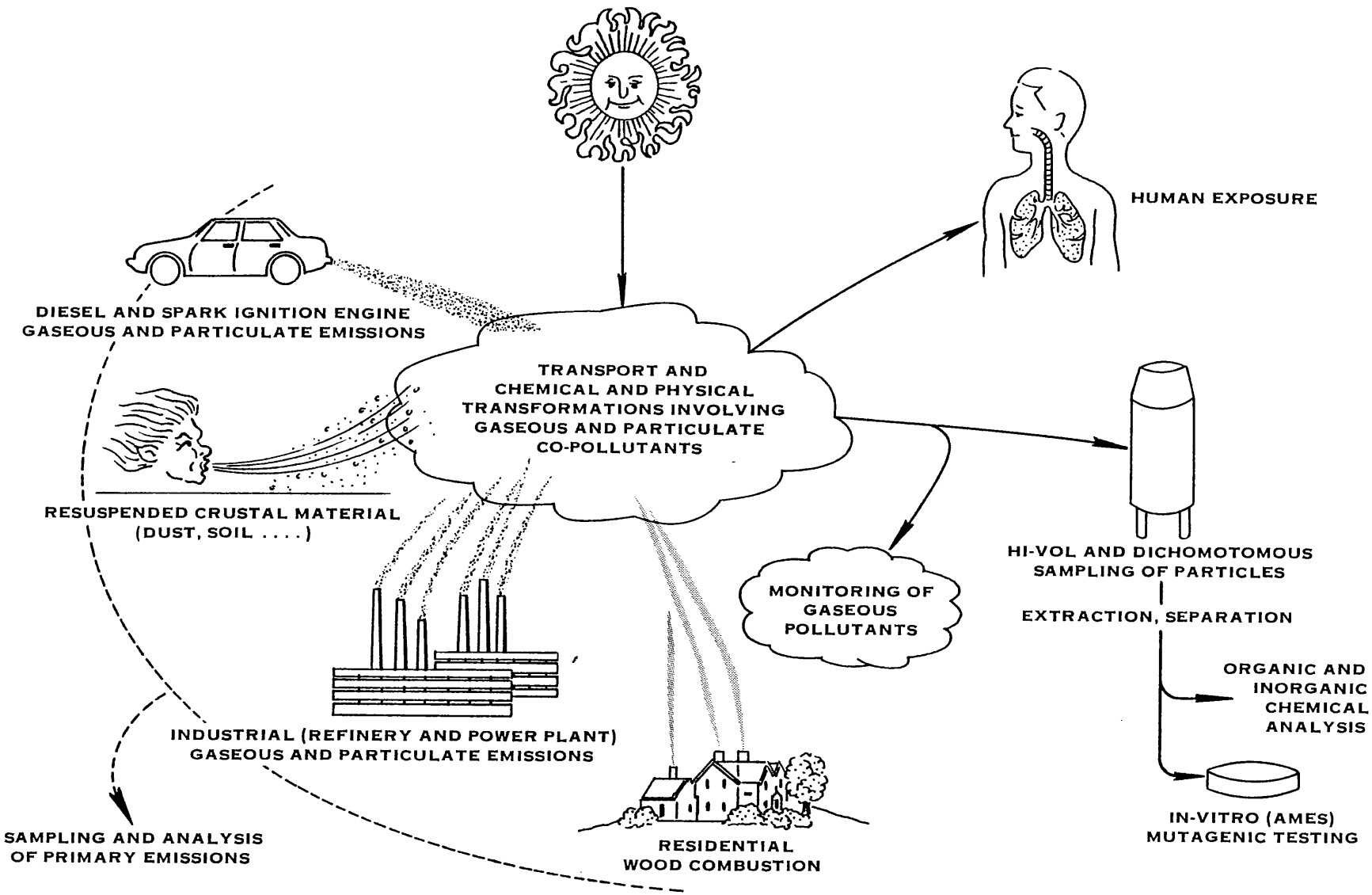

SAN PABLO
BAY
MARTINEZ
PITTSBURG
CONCORD
RICHMOND
SAN
FRANCISCO
BAY
4
680
80
24
17
580
N
LOCATIONS OF SAMPLING STATIONS
IN CONTRA COSTA COUNTY, CALIFORNIA
1981-82

FIGURE 1

permanent air monitoring stations of the Bay Area Quality Management District (BAAQMD) in Richmond, Concord and Pittsburg. Twenty-four hour hi-vol samples were collected every sixth day between November 1979 and June 1982, composited every four months and analyzed for mutagenic activity and PAH.

This field and laboratory study was focused on the identification of the sources of particulate mutagens and carcinogenic PAH in ambient air collected in Contra Costa County, California. Intensive air sampling for source identification was carried out at the four locations (Pittsburg, Richmond, Concord and Martinez) during three 36-hour episodes in August and October 1981 and in January 1982. Organic extracts of air particulate matter were analyzed for mutagenic activity in the Ames Salmonella test (8) and for selected PAH by high pressure liquid chromatogaphy (HPLC) coupled with ultraviolet and fluorescence detection (17). Ames testing was performed in strain TA98 with and without added rat liver (S9) extract in order to measure both indirect (+S9) and direct-acting (-S9) mutagens. Strain TA98 responds primarily to frame-shift mutagens. Nine PAH were identified by HPLC and their sum used as a surrogate variable for total PAH. Air samples were also analyzed for trace metals (including Pb, Ni and Fe), secondary particulates (NO_3^- and $SO_4^=$) and pollutant gases (O_3, CO, NO, NO_2, SO_2). The complexity of the various emissions and atmospheric reactions is shown pictorially in Figure 2 (modified from reference 23). Multivariate statistical techniques were used in an attempt to provide insights as to sources of mutagens and PAH (27). Factor analysis was used to help identify types of emission sources and select source tracers. Using tracers for automobiles (Pb), industry (Ni), crustal material (Fe) and secondary aerosols (NO_3^-, $SO_4^=$), linear regression models were developed of the form Mutagenicity = a (Pb) + b(NO_3^-) + . . . where a and b



SOURCES OF GASEOUS AND PARTICULATE AIR POLLUTANTS:
CHEMICAL AND PHYSICAL TRANSFORMATIONS INVOLVING TRANSPORT IN THE ATMOSPHERE
AND DURING THE SAMPLING, MAKE SOURCE IDENTIFICATION DIFFICULT.

FIGURE 2

were the regression coefficients determined from data collected during intensive sampling episodes.

IV. MAJOR FINDINGS OF THIS STUDY

Progess was made in four areas of investigation: development of methods and standards for measuring mutagens and carcinogens; identification of sources of particulate mutagens and carcinogens; identification of mutagenic and carcinogenic compounds in air particle extracts; analysis of seasonal variations and trends in levels of mutagens and carcinogens in Contra Costa community air.

A. Methods and standards developments

1. A modification of the Ames bioassay (28) with increased sensitivity for mutagens was applied to the analysis of air particle extracts. Measurements of mutagenicity were obtained using air samples collected every two hours. The increased sensitivity will allow diurnal pattern measurements, an important technique for assessing sources of mutagens.

2. Novel nitroarene standards were synthesized and characterized (29). Unusually high mutagenicities of dinitrobenzo(ghi)perylenes and S-9 dependence for mononitrobenzo(ghi)perylene and mononitrocoronene were observed in the Ames Salmonella assay. These compounds may be found in vehicle exhaust and the atmosphere.

3. A method for routine separation, identification and quantitation of specific polycyclic organic molecules in ambient air extracts was perfected. The method does not require sample prefractionation or clean-up and employs high pressure liquid chromatography (HPLC) coupled with fluorescence detection.

B. Mutagen and carcinogen source identification.

1. It is feasible to use multivariate statistical techniques to identify types of air pollution sources and to apportion the contributions of these source-types to the mutagenicity and PAH in aerosols collected during pollution episodes.

2. Source patterns during pollution episodes were different. During summer and fall episodes, vehicular emissions accounted for most of the mutagenicity and PAH measured (Table 1, Figure 3). During a winter episode possible contributions of residential wood combustion to PAH were noted while mutagenic sources could not be quantitatively resolved. However, significant positive correlations were found between mutagenicity and fine lead (which is mostly released by vehicular traffic), fine zinc (which is released from vehicular traffic and industrial sources), and iron. Significant correlations between mutagenicity (TA9+S9) and nitrate were also observed; the correlation was positive in summer and negative in winter.

TABLE 1

SUMMARY ESTIMATES OF SOURCE CONTRIBUTIONS TO AMBIENT PARTICULATE MUTAGENICITY AND PAH[a]

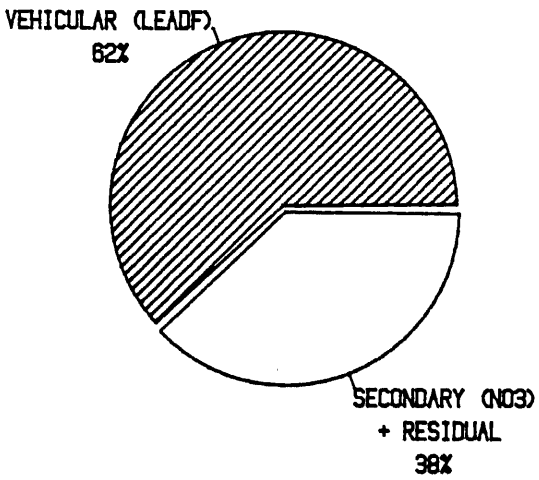
		CONTRIBUTIONS BY EPISODE					
		AUGUST 1981			OCTOBER 1981		
		MUTAGENICITY[b]		PAH[b]	MUTAGENICITY		PAH
SOURCE	TRACERS	+ S9	- S9		+ S9	- S9	
Transportation	LEADF	10.2 (62)	5.0 (69)	1.4 (58)	9.7 (95)	3.1 (48)	6.3 (129)
Industry	NICKELF	—	—	-0.8 (-33)	—	—	1.0 (20)
Secondary Aerosols	NO_3^-	6.9 (42)	2.7 (37)		—	—	-3.4 (-69)
	$SO_4^=$	—	—	0.6 (25)	—	3.5 (54)	—
Crustal Resuspension	IRON	—	—	—	-1.6 (-16)	—	—
Residual	Unknown	- 0.7 (-4)	- 0.4 (-6)	1.1 (48)	2.2 (22)	-0.1 (-2)	1.0 (20)
Total		16.4	7.2	2.4	10.2	6.5	4.9

[a]Estimates based on regression equations; percentage contributions are given in parenthesis.
[b]Concentrations of mutagenicity are in revertants/m^3; PAH in ng/m^3.

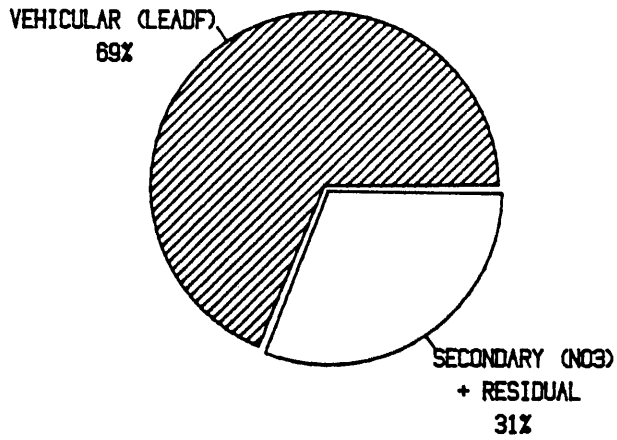
FIGURE 3

SOURCES OF AIR PARTICULATE MUTAGENS
AUGUST 1981 EPISODE

TOTAL INDIRECT ACTING [+S9]

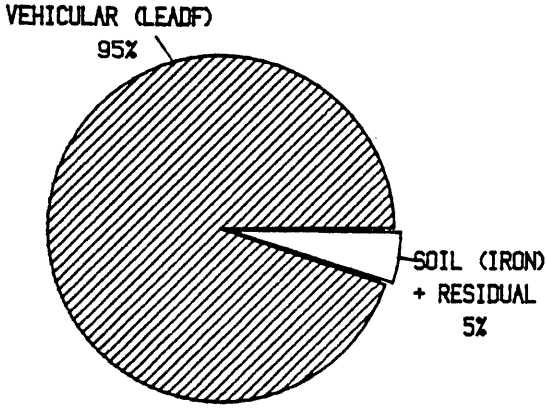


DIRECT ACTING [-S9]

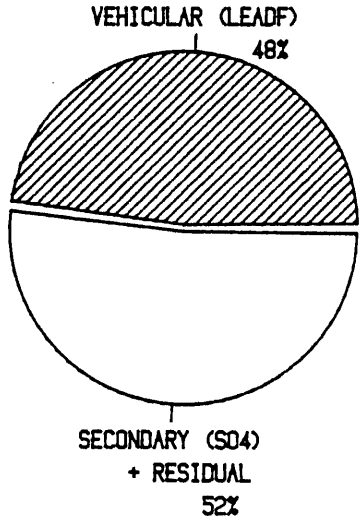


OCTOBER 1981 EPISODE

TOTAL INDIRECT ACTING [+S9]



DIRECT ACTING [-S9]



C. Chemical identification of mutagenic and carcinogenic compounds in air particle extracts.

1. Eight unsubstituted PAH, including BAP and other carcinogens, and one carbonyl-PAH, benzanthrone (BO), were identified and quantified in air particle extracts. These compounds accounted for only about one percent or less of the total indirect (S9-dependant) mutagenicity of extracts from pollution episodes.

2. Biochemical evidence suggests that nitroarenes may account for one-half or more of the observed direct-acting mutagenicity during pollution episodes. This is based on the observation that mutagenicities of most extracts were at least a factor of two lower in a nitroreductase-deficient strain of Salmonella (TA98NR) than in the parental strain (TA98). The nitroreductase is required for mutagenic activation of many nitroarenes.

D. Analysis of seasonal and annual trends.

1. Mutagenicity and PAH concentrations in 4-month composites showed dramatic seasonal variations. Levels measured in the winter (November -February) were five to ten times higher than those measured in the spring (March-June). Summer (July-October) concentrations were intermediate (Figures 4 and 5).

2. Winter composites were up to 3 times more mutagenic with added

FIGURE 4

SEASONAL COMPOSITES

MUTAGENICITY, TA98 +S9, AVERAGE OF THREE STATIONS

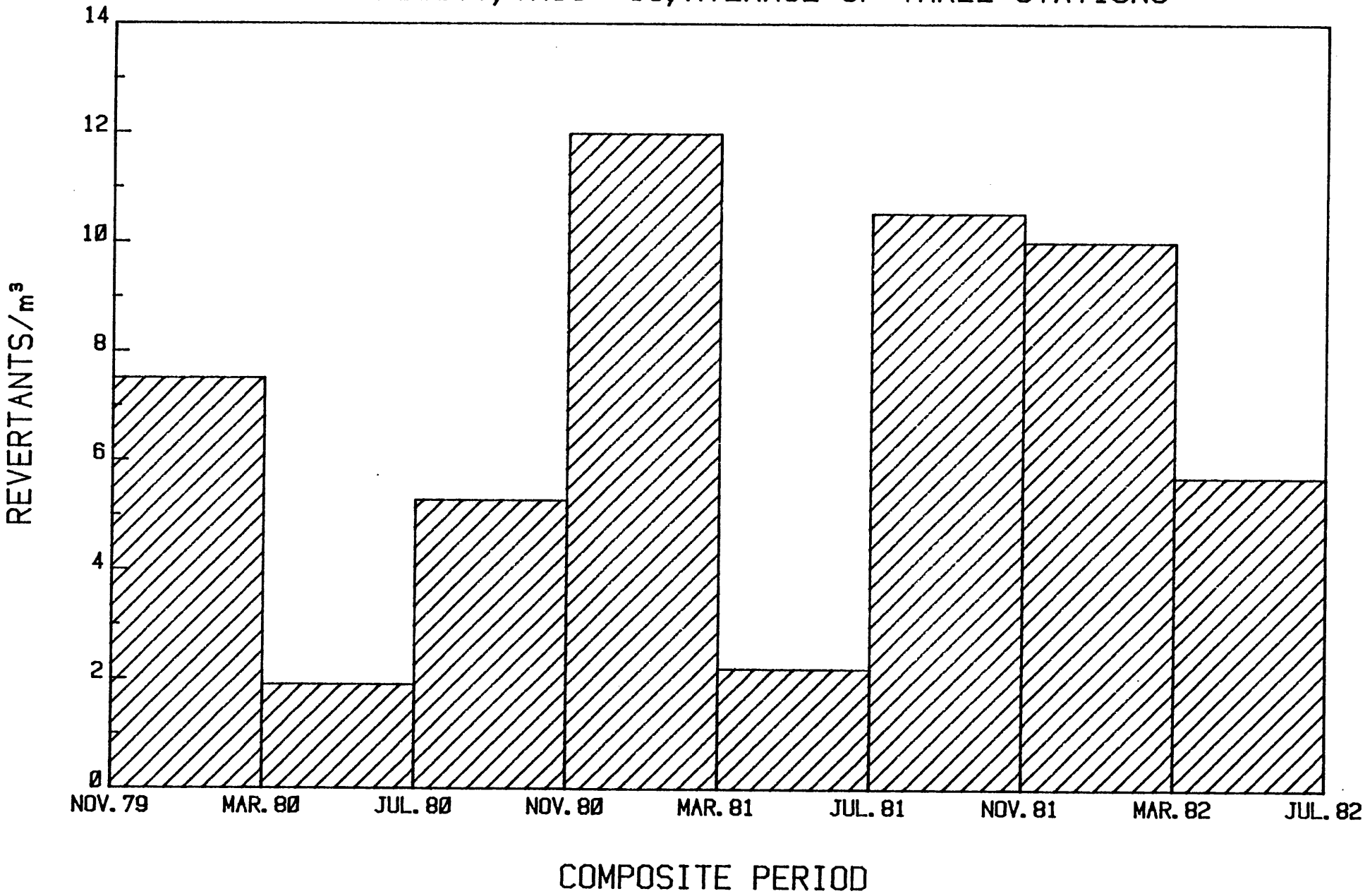
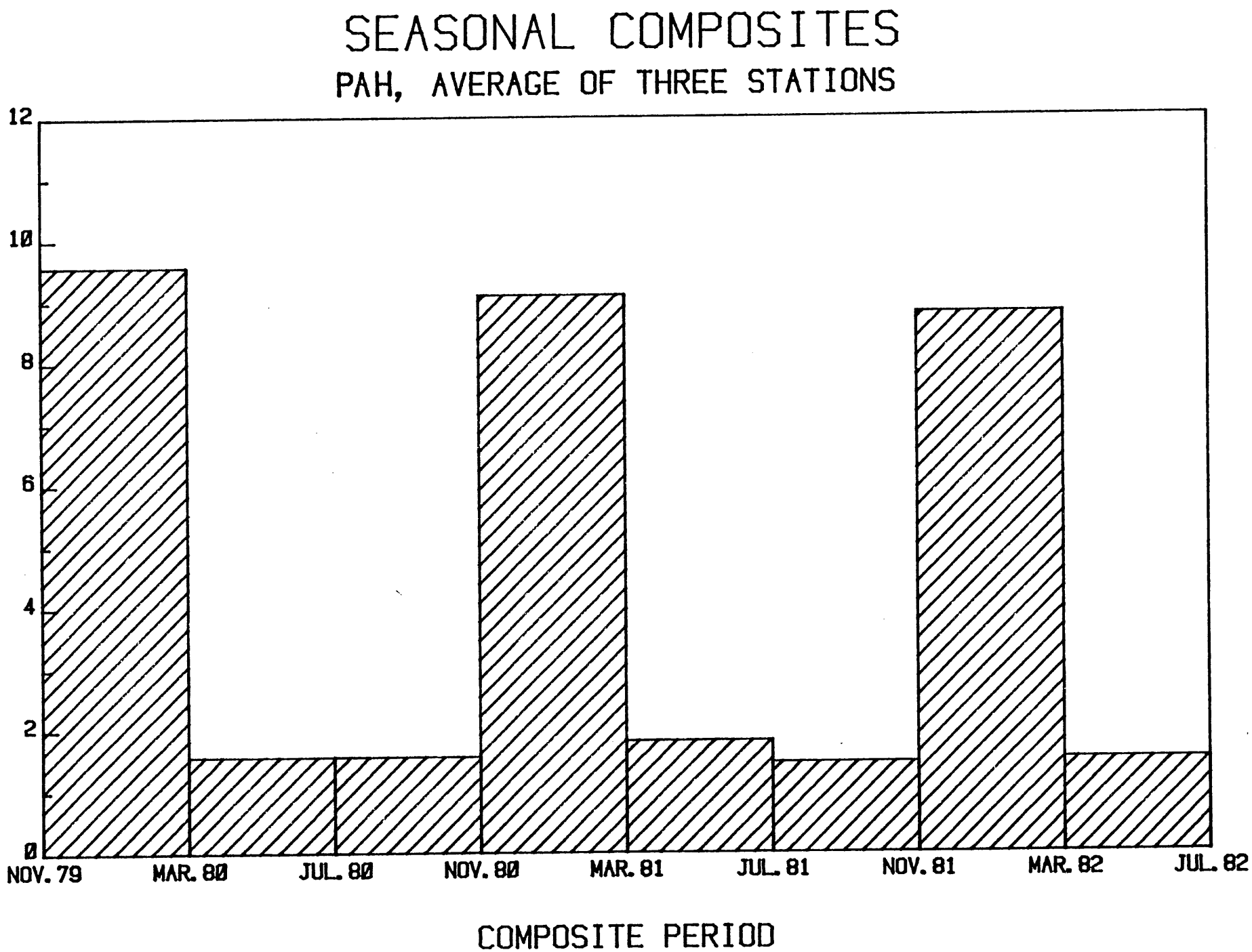


FIGURE 5


SEASONAL COMPOSITES
PAH, AVERAGE OF THREE STATIONS
NG/m³
12
10
8
6
4
2
0
NOV. 79
MAR. 80
JUL. 80
NOV. 80
MAR. 81
JUL. 81
NOV. 81
MAR. 82
JUL. 82
COMPOSITE PERIOD

metabolic activation (+S9) than without (-S9) while activities of spring composites were not significantly changed by metabolic activation. This implies that both direct-acting (e.g., NO_2pyrene-like) and indirect-acting (e.g., BAP-like) mutagens are present in winter while in spring direct-acting mutagens predominate.

3. Annual average concentrations of mutagenicity and PAH did not change significantly over the period between November 1979 and June 1982.

V. GENERAL CONCLUSIONS AND IMPLICATIONS

A. Sources of Mutagens and Carcinogens During Pollution Episodes

The study has demonstrated the feasibility of integrating mutagenic, chemical and multivariate statistical methods for mutagen and carcinogen source identification. We have shown that the source patterns during the three episodes were different and sources could be at least partially apportioned. Vehicular transportation sources were the predominate mutagenic contributors during the August and October 1981 episodes (Table 1, Figure 3). In addition, at least half of the PAH was also derived from automotive sources during summer and fall episodes. Industrial emissions contributed about one-fifth of the PAH in the fall. Contribution from secondary aerosols were also noted. During the summer episode, about one-third of the mutagenicity was attributed to nitrate associated secondary aerosols; however this conclusion is based on uncertain NO_3^- measurements and is therefore not very firm. During the fall, approximately one-half of the direct-acting

(-S9) mutagenicity was attributed to sulfate-associated secondary aerosols. The source pattern during the January 1982 episode was the most complex and quantitative source apportionment failed. However qualitative results of factor analysis suggested possible contributions of residental wood combustion to PAH during the winter episode.

Improvements could be made in the source apportionment method by introducing more complete and quantitative meteorological data than were available in these experiments into the multivariate statistical techniques. For example, Daisey and Kneip (27) used dispersion normalized concentrations with success in multiple regression modeling. It would also improve the technique if sampling were done at more stations.

B. Seasonal and Chronic Human Exposures

It is significant that concentrations of both carcinogenic and mutagenic pollutants vary widely as a function of season. Mutagenicity and PAH concentrations were measured to be at least five times higher in winter than in spring due mostly to reduced ventilation in the Bay Area in winter. Thus in terms of human exposure, the winter is clearly the major seasonal contributor to the mutagenic and carcinogenic burdens of ambient air particles. In a typical recent year, Contra Costa residents inhaled more mutagens and PAH during the four-month winter season (November-February) than during the other two seasons combined because concentrations are so much higher in winter.

For purposes of discussion, it is useful to provide some estimate of the possible human risks associated with exposure to airborne mutagens and carcinogens at these current levels. In this study, composite air samples had an annual average mutagenic density (+S9) of ca. 7 rev/m^3 between March 1980 and March 1982. This level may be compared with the mutagenicity in cigarette smoke condensates. The smoke condensate from one commercial cigarette gives approximately 17,500 revertants in the Ames test (30). Assuming that the average person breathes 20 m^3 per day, the number of "cigarette equivalents" per day is ca. 0.01 or less than 4 cigarettes per year. A second type of risk estimate was made by Pike and Henderson (2) who used BAP as a surrogate for cancer risk and compared amounts of BAP in cigarettes with excess lung cancer in smokers. These authors calculated that daily breathing of community air containing 15 ng/m^3 BAP poses the same life-time lung cancer risk as smoking 1 cigarette per day. In the present study, annual levels of BAP averaged 0.3 ng/m^3 between March 1980 and March 1982. Thus in terms of cancer risk, daily breathing of Contra Costa winter air may be considered equivalent to smoking about 0.3/15 = 0.02 cigarettes per day or less than 10 cigarettes per year. Considering the uncertainties in the <u>in vitro</u> bioassay and epidemiological data, and the assumptions and simplifications implicit in the calculations, the two-fold difference in the estimates derived from mutagenicity and BAP measurements is surprising small. Pike and Henderson conclude from their analysis that even at a BAP level as low as 1 ng/m^3, the life-time lung cancer risk is "slightly greater than 1/1500. Environmental regulations are usually made to keep such a risk to $1/10^5$ or even $1/10^6$" (2). These risk-estimates neglect contributions from indoor air pollution. Also, the

excess risk attributable to Contra Costa community air pollution (ca. 1/1500) is less than one percent of the observed incidence of lung cancer from all causes in Contra Costa County (between 1/20 and 1/10) (6). This is a number much too small to be identified by epidemiological tools, principally because smokers keep the background so high.

Presumably these possible excess risks will be less in the future if the recent downward trends in Bay Area air pollution levels continue. Air quality in the Bay Area has improved significantly over the past decade (31) as controls on stationary sources and vehicles have steadily reduced emissions. This has resulted in major reductions in concentrations of gaseous pollutants (notably ozone), total particulates and lead. Similar downward trends in polycyclic hydrocarbon concentrations are suggested by results of the present study. In San Francisco during the winter months of 1958-59, BAP concentrations ranged from 2.3 to 7.5 ng/m^3 (5) while in the winters of 1979-82, the average BAP concentration in Contra Costa County was significantly lower (0.7 ng/m^3). As discussed above, no downward trends in BAP, PAH or mutagenicity levels were observed within the brief 32-month period of this study. However the duration of our analysis was too short to have detected anything but major changes.

C. Chemical Nature of Particulate Mutagens

Aerosol extracts are extremely complex mixtures and much research on their chemical contents remains to be done. At present we know that Contra Costa aerosols contain predominantly direct-acting mutagens during

warm-weather months and both direct- and indirect-acting mutagens during cold weather months. This conclusion is based on mutagenic testing of seasonal composites. However, both direct- and indirect-mutagens are clearly present during the hot August episode as well as during the cool October and cold January episodes. Thus sources and/or atmospheric conditions for production of both direct- and indirect-mutagens are present all year around.

As expected, PAH are among the indirect-acting mutagens found in Contra Costa aerosols. However the eight PAH and benzanthrone species measured in this study made a very small contribution to the observed mutagenicity of air particle extracts. This was the case even during sampling periods when polycyclic hydrocarbon concentrations reached their highest levels (i.e., during the January 1982 pollution episode, when the concentrations of BAP and BO averaged approximately 1 ng/m^3 and 4.5 ng/m^3 respectively). A mixture containing the nine PAH at their concentrations measured during the January episode was prepared and subjected to mutagenic testing. The simple mixture of pure chemicals showed activity in TA98+S9 but the amount was only about 1% of the indirect mutagenic acivity observed in the complex mixtures extracted from the January episode air samples.

The question of NO_2 PAH in Contra Costa aerosols remains open. It seems likely that direct-acting nitroarenes are present in some urban aerosol extracts. However, the evidence in Contra Costa County is indirect and based on the behavior of extracts in the nitroreductase-deficient mutant, TA98NR, which lacks the ability to activate many nitro-compounds. Direct-mutagenicities of most Contra Costa samples were indeed much lower

in TA98NR than in TA98. Decreases of about a factor of two or more were observed in at least half of the composite samples and more than three-quarters of the episode samples. Activities in TA98NR relative to TA98 were especially low during the summer intensive episode, when the most reactive atmospheric conditions prevailed. This makes it probable that direct-acting nitroarenes are present in the atmosphere (or formed on filters after collection via mechanisms such as proposed by Pitts and co-workers (24)). Further research is required to chemically identify the postulated nitroarene species in air extracts. Based on the indirect evidence provided by testing in TA98NR, we conclude that most of the Contra Costa samples analyzed contain compounds with a reducible NO_2-group, like 1-NO_2pyrene, which are directly active in the Ames test. Such compounds may account for half or more of the direct mutagenicity in air particulate extracts, especially in warm weather months.

D. Implication for ARB Regulatory Programs

Results of this study may be applied to ARB regulatory functions related to control of toxic air contaminants. Hopefully identification of sources can assist in the development of control strategies for mutagens and carcinogens in community air. This is an area of significant long range public health concern.

In the present study, multivariate statistical methods were used to identify sources of mutagens and polycyclic aromatic carcinogens and to estimate their contributions to the ambient aerosol. It is important to recognize the

limitations of these source apportionment efforts. As with any application of statistics, there is no assurance that the observations and conclusions represent cause and effect. In addition, the number of observations is small. Therefore, all conclusions are subject to revision as additional data become available. However, one salient conclusion does seem apparent. A major proportion of the mutagenicity of Contra Costa County aerosols collected during the August and October 1981 episodes can be accounted for by the variability in the fine-fraction lead concentration in these aerosols. This observation suggests that during the summer and fall pollution episodes, the majority of the mutagenicity in Contra Costa aerosols was due to vehicular emissions. The contribution of diesel exhaust emissions to mutagenic aerosols should be considered in future research. Furthermore, nitrate-associated aerosols may have contributed to the mutagenicity of samples collected during the summer episode and sulfate-associated aerosols may have contributed to direct-acting mutagens in the fall. The first implication of these conclusions for ARB regulatory programs is that emission standards and controls on vehicles are probably the most efficacious means of controlling ambient levels of particulate mutagens. The possible contribution of secondary aerosols to mutagenicity in summer and fall suggests that regulation of secondary pollutant formation may have some impact on atmospheric levels of mutagenic compound, but at present this is speculation.

However, it is clear that the correlation between mutagenicity and nitrate is significantly positive in the summer and negative in the winter episode. One possible interpretation of the mutagen vs nitrate correlations can be provided (B. Appel, personal communication). Let us assume that the

active mutagens are nitro PAH, formed either in the atmosphere or as artifacts on filters, and that the rate of their formation is proportional to the HNO_3 concentration. The concentration of HNO_3 is controlled by the equilibriuim $NH_3 + HNO_3 \rightleftarrows NH_4NO_3$, so that conditions favoring high particle NO_3^- (i.e. low temperature, high relative humidity) lead to low HNO_3. Low HNO_3 would in turn lead to low nitro PAH formation and low mutagenicity. Perhaps this is relevant to the episode data. In winter, the observed NO_3^- is approximately equal to the true particulate NO_3^-, with little gas phase HNO_3 present. However during the summer, the observed NO_3^- equals the sum of the true particulate NO_3^-plus the gas phase HNO_3, which may account for half or more of the observed NO_3^-. Thus in summer the observed NO_3^- is probably correlated with HNO_3 and therefore a positive correlation between observed NO_3^- and mutagenicity may be expected. This is a topic for future research.

Another topic of possible interest for ARB regulatory programs concerns evidence that wood burning is a source of carcinogenic polycyclic hydrocarbons in Contra Costa air during winter. Several lines of evidence are presented in this report. First, diurnal patterns of selected PAH measured in the winter episode are consistant with night emissions from fireplaces. Because of meteorological factors, nighttime levels of most particulate pollutants measured in January were higher than daytime levels, but diurnal variations in certain PAH were the most dramatic. Specifically, concentrations of certain carcinogens (BAP, CHR, BAA) were three to five times higher by night than by day, especially in Concord and Martinez, the sampling stations located in the most residential environments. In a recent study of

wood-burning in Waterbury, Vermont, Sexton et al (32) observed dramatic diurnal variations in concentrations of respirable particulates, with peak values at night exceeding afternoon levels by 5- to 10-fold. They concluded that wood burning was the major source of airborne particles in residential sections of the town. A second line of evidence in the present study employed a simple ratio technique to obtain information about PAH sources. As shown in Table 2, various investigators have measured the ratio of BAP to BGP for a number of combustion sources (1,33,34). Automobiles tend to have the lowest ratios, 0.2 to 0.5 while industrial sources tend to be ≥ 1. The BAP/BGP ratios reported for wood combustion were 0.4 to 0.5. In this study, the average BAP/BGP ratios in the summer, fall and winter episodes were 0.17, 0.28 and 0.52 respectively. Clearly the ratios found in the summer and fall were characteristic of auto emissions whereas those in winter were more similar to the values reported for wood combustion. This is consistant with residential wood combustion being a major contributor of these PAH in winter. At present the conclusions drawn on the basis of BAP/BGP ratios must be viewed as speculation for the following reasons:

(a) The data used for comparison are from different references, dating back to 1972.

(b) Temperature differences probably influence, to an unknown extent, the observed ratios of BAP/BGP.

(c) Even on the basis of the ratios used (Table 2), no clear cut distinction is possible between vehicular and wood burning emissions.

TABLE 2

RATIO OF BENZ(A)PYRENE TO BENZ(GHI)PERYLENE FOR SELECTED AIR EMISSION SOURCES

Source-Type	BAP/BGP	Reference
Vehicular	0.2 - 0.5	1
Industrial		
Petroleum refineries	0.65 - 1.7	"
Oil-burning powerplants	2 - 3	"
Coal-burning powerplants	0.9 - 6.6	
Wood Combustion		
Stoves	0.42	42
Fireplaces	0.52	42
Forest-fire	0.47	43
Contra Costa Community Air Pollution episodes:		
-Summer	0.17	This study
-Fall	0.28	" "
-Winter	0.52	" "

A third type of evidence implicating wood combustion was obtained by factor analysis. During the winter episode, the factor analysis technique revealed a novel pollution factor containing both organic variables, PAH and BSO, and which explained 25 percent of the variance in the levels of particulate pollutants. However, this novel organic pollution factor did not contain any of the source-related tracers (LEAD, NO_3^-, NICKEL, $SO_4^=$, IRON). Furthermore, the factor was not present in the summer or fall episodes. Finally, the organic factor was only recognized in the pollution patterns at Concord and Martinez, the locations most subject to residential emissions. From these results, we conclude that residential wood combustion contributes seasonally to ambient PAH levels in Contra Costa County. If correct, this conclusion implies that a new control strategy may be needed.

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