Executive Summary

Stationary Source Division

November 1993
EXECUTIVE SUMMARY

ACETALDEHYDE AS A TOXIC AIR CONTAMINANT

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

APPROVED BY THE SCIENTIFIC REVIEW PANEL MAY 1993

November 1993
Preface

This report was developed in response to the provisions of Health and Safety Code, sections 39650-39662, which became effective January 1984. This legislation requires a two-phase process which separates risk assessment from risk management. During the identification phase, a report is developed which considers whether there are adverse health effects of a substance which may be, or is, emitted in California. However, in January 1993, AB 2728 was enacted and the procedure for toxic air contaminant (TAC) identification of federal hazardous air pollutants (HAPs) was changed. Pursuant to the new legislation, the state board was required to identify, by regulation, any substance listed as a federal HAP as a TAC. Although this report was developed under AB 1807, acetaldehyde is a HAP, and was identified as a TAC on April 8, 1993, when the Board identified all 189 HAPs as TACs.

The title of this report has also changed. The original report, "Proposed Identification of Acetaldehyde as a Toxic Air Contaminant," was released for public comment in August 1992. The report was revised, and this final version, "Acetaldehyde as a Toxic Air Contaminant," was approved by the Scientific Review Panel on May 12, 1993, and includes a review of exposure, the cancer potency number, and the chronic reference exposure level.

No control measures are proposed in this report. The cancer potency number and chronic reference exposure level were approved by the Scientific Review Panel, and may be used in the development of control measures.

In preparing this report, staff reviewed pertinent literature through March 1992.
EXECUTIVE SUMMARY

Introduction

This report was developed in response to provisions of state law (Health and Safety Code sections 39650-39662), which became effective in January 1984. This legislation requires a two-phase process which separates risk assessment (identification) of toxic air contaminants (TACs) from risk management (control). During the identification phase, a report is developed which considers whether there are adverse health effects of a substance which may be, or is, emitted in California. After conducting a public hearing, the Board decides whether or not the substance should be identified as a TAC. If identified, the substance is listed by regulation as a TAC in the California Administrative Code and enters the control phase.

With the adoption of the AB 2728 legislation, which was signed by the Governor in September 1992 and became effective in January 1993, the procedure for identification of federal hazardous air pollutants (HAPs) as TACs was changed. Pursuant to the new legislation, the state board was required to identify, by regulation, all HAPs as TACs. Acetaldehyde is a HAP and, therefore, was identified as a TAC by regulation at a Board hearing on April 8, 1993.

What is contained in this report?

This report summarizes the emissions, exposure, and atmospheric persistence and fate of acetaldehyde in California. It also describes the health effects of acetaldehyde which includes an estimate of the cancer potency number and a chronic reference exposure level. The Scientific Review Panel (SRP) evaluated the entire report including a review of the data, and approved the cancer potency number and the chronic reference exposure level on May 12, 1993. The cancer potency number and the chronic reference exposure level may be used in the development of control measures for acetaldehyde.
This report contains several parts. Part A, developed by the ARB staff contains information on exposure to Acetaldehyde in California. Part B, developed by the Office of Environmental Health Hazard Assessment (OEHHA), contains information on the health effects of acetaldehyde. Part C contains public comments received during the first and second comment period (August 7 - October 1, 1992 and April 5 - May 5, 1993) and the ARB/OEHHA responses to these comments.

**What is acetaldehyde?**

Acetaldehyde is a colorless, flammable liquid, volatile at ambient temperature and pressure, with an irritating odor. Acetaldehyde can be used in a wide variety of chemical reactions, many of which are useful in commercial processes. The commercial uses of acetaldehyde include the manufacture of acetic acid, acetic anhydride, pyridines, peracetic acid, penteerthritol, ethyl acetate, alkylamines, lactic acid and crotonaldehyde. Acetaldehyde is also known by a variety of synonyms which include acetic aldehyde, ethanol, ethyl aldehyde and methyl formaldehyde. The molecular formula of Acetaldehyde is CH3CHO.
Why was acetaldehyde evaluated as a toxic air contaminant?

The staffs of the ARB and OEHHA have reviewed the available scientific evidence on the presence of acetaldehyde in the atmosphere of California and its potential adverse effect on public health. The ARB staff has determined that acetaldehyde is the product of incomplete combustion, is emitted from a variety of sources, and can be detected in the ambient air throughout California. It can be formed in polluted ambient atmospheres from a variety of precursors. It is highly mobile in the environment, and is not naturally removed or detoxified at a rate that would significantly reduce public exposure. The International Agency for Research on Cancer (IARC) classified acetaldehyde as a possible human carcinogen (2B), based on sufficient evidence in animals and inadequate evidence in humans. The United States Environmental Protection Agency (U.S. EPA) has decided that acetaldehyde is a "probable human carcinogen" (B2). The OEHHA staff has concluded that at ambient concentrations acetaldehyde may cause or contribute to an increase in mortality or serious illness and may therefore pose a potential hazard to human health.

What are the sources of acetaldehyde emissions?

Acetaldehyde is not manufactured in California. It is both directly emitted into the atmosphere through incomplete combustion from such sources as stacks, tailpipe exhaust and fires, as well as formed in the atmosphere as a result of photochemical oxidation of hydrocarbons and free radical reactions involving hydroxyl radicals. In urban areas, emissions are primarily from mobile sources with some contribution from stationary sources. The largest sources statewide of directly emitted acetaldehyde are from combustion of fuels from mobile sources, agricultural burning, and wildfires. Direct sources are estimated to contribute approximately 44 percent of the acetaldehyde in the atmosphere.
Photochemical oxidation is estimated to contribute 56 percent of the ambient acetaldehyde as predicted by the Urban Airshed model. Concentrations via photochemical oxidation can vary significantly depending on the season, location, meteorological conditions, and time of day. Mobile and stationary sources emit reactive organic gases such as ethyl peroxide, and ethoxy radicals which are precursors of photochemically generated acetaldehyde. Reductions of these hydrocarbon precursors can be expected to reduce the contribution of acetaldehyde from photochemical oxidation.

Figure 1 shows the percentages of acetaldehyde emissions in California from all sources in 1987. The ARB staff estimates that approximately 14,000 - 30,000 tons, were produced from photochemical oxidation of organic precursors. Stationary point sources, stationary area sources, on-road mobile sources, and other transportation sources contribute approximately 11,000 - 24,000 tons/year. The estimate of photochemical production is based on information from the Emissions Data System (EDS) and could vary by 50 percent.

Indoor acetaldehyde sources are numerous and include such diverse combustion sources as cigarette smoke, fireplaces, woodstoves and cooking.
Total direct outdoor acetaldehyde emissions from mobile, stationary and area sources as based on ARB's 1987 emission inventory in California are estimated to be approximately 11,000 - 24,000 tons per year. Figure 2 is a breakdown of direct emissions only. Mobile sources, which include on-road motor vehicles and other transportation sources such as trains, ships, farm and utility equipment, emitted approximately 3,700 to 7,800 tons of acetaldehyde or 15 to 32 percent of direct emissions. Stationary point sources such as fuel combustion sources, refineries and coffee bean roasters emitted approximately 820 to 1,000 tons (3 to 4 percent) of acetaldehyde. Stationary area sources include wildfires, agricultural burning and management burning, and diesel combustion in internal combustion engines at oil and gas fields, etc. The staff estimates that these stationary area sources emitted approximately 7,000 to 15,000 tons (29 to 62 percent) of acetaldehyde.
What are the effects of the ARB motor vehicle control program on acetaldehyde and its precursors?

The emissions standards applied by the ARB to new vehicles over the past years have caused steady declines in vehicular reactive organic gases (ROG) including acetaldehyde and nitrogen oxides (NOx) emissions. Further, reductions in ROG and NOx are expected to result in a decline in secondary acetaldehyde due to vehicular emissions. Declines in this source of emissions is expected to continue because of the recently adopted low emission vehicle (LEV) emission standards. Additionally, the primary, directly emitted acetaldehyde, also, a ROG, is expected to decline.

The ARB adopted new gasoline specifications ("Phase 2 gasoline standards") which become effective in 1996. These standards also will reduce ROG and NOx emissions from all gasoline vehicles (vehicles currently on the road plus LEVs) made before 1996, thereby reducing the formation of both primary and secondary acetaldehyde.

The Wintertime Oxygenate Program requires that refiners add oxygen to gasoline in the wintertime to reduce carbon monoxide. Depending upon the oxygenated additive used, acetaldehyde emissions from combustion of motor vehicle fuels may, or may not, increase. The addition of methanol and methy tert-butyl ether (MTBE) to fuel does not increase acetaldehyde emissions. Gasoline containing the oxygenate additives ethanol or ethyl tert-butyl ether (ETBE), upon combustion, may result in increased acetaldehyde emissions. However, it is not know to what extent ethanol or ETBE will be used as a winter oxygenate in California fuels and, therefore, the resulting effect on acetaldehyde emissions.

The overall effect of the vehicular emission standards and the gasoline specifications on acetaldehyde concentrations is a complex issue. Future acetaldehyde emissions will depend on the turnover rate of older vehicles to LEVs, the primary acetaldehyde contributions from oxygenated additives, such as ethanol and ethyl tert-butyl ether, the decrease of ROG from the combustion of Phase 2 reformulated gasoline, and possible use of ethanol.
What are the ambient outdoor air concentrations of acetaldehyde?

Acetaldehyde is routinely monitored by the statewide ARB tonics monitoring network. Basin-specific mean annual concentrations vary from a minimum of 1.63 parts per billion volume (2.93 micrograms per cubic meter) in the South Central Coast Air Basin to a maximum of 2.81 parts per billion volume (5.06 micrograms per cubic meter) in the South Coast Air Basin (based on 24 hour sample averages). The overall estimated mean statewide exposure, weighted by population, is estimated to be 2.33 parts per billion volume (4.19 micrograms per cubic meter). The population-weighted exposure is based on 20 million people represented by the tonics monitoring network (out of the 30 million total California populations).

Studies conducted in the South Coast Air Basin since 1980 have reported data for short-term (30 minute to 2 hour sample averages) ambient outdoor concentrations to vary from one part per billion volume (1.8 microgram per cubic meter) to 39 parts per billion volume (70.2 micrograms per cubic meter), with the most recent concentrations measured during the South Coast Air Quality Study (SCADS) (1987) with a range of 0.9 to 24.5 parts per billion volume (1.6 to 44.1 micrograms per cubic meter) in one hour samples. The SCADS was an integrated air quality study whose overall goal was to develop a comprehensive and properly archived air quality and meteorological data base for the South Coast Air Basin which can be used to test, evaluate, and improve elements of air quality simulation models for oxidants, NO2, PM10, fine particles, visibility, toxic air contaminants, and acid species.

Reports of ambient air sampling of two rural regions, Point Barrow, Alaska and Whiteface Mountain, New York provide examples of 'background' concentrations of 0 to 0.8 parts per billion volume (1.4 micrograms per cubic meter) of acetaldehyde.
Is there evidence of indoor air exposure to acetaldehyde?

Yes. In general, concentrations are higher indoors than outdoors due in part to the abundance of combustion sources such as cigarettes, fireplaces, and woodstoves. Acetaldehyde emissions have been reported from frying hamburger, suggesting that cooking may be an indoor source of acetaldehyde. Acetaldehyde can also be emitted from some building materials such as rigid polyurethane foams, and some consumer products such as adhesives, coatings, lubricants, inks, and nail polish remover. Other potential sources of indoor acetaldehyde concentrations are the infiltration of vehicle exhaust and, perhaps, the volatilization of acetaldehyde from certain foods.

Surveys have shown that indoor air concentrations of acetaldehyde in residences without smokers can be about two to eight times higher than the outdoor mean population-weighted statewide concentration of 2.3 parts per billion. The results of limited surveys allow a crude estimate of an average acetaldehyde concentration inside residences of about 3.0 parts per billion volume (5.4 micrograms per cubic meter) to 15 parts per billion volume (27.0 micrograms per cubic meter). Limited data suggest that the acetaldehyde concentrations in offices and public buildings are similar in magnitude to those inside residences. Higher levels may occur in some indoor environments; in the case of a tavern occupied by a number of smokers, levels of up to 113 parts per billion volume (203.4 micrograms per cubic meter) were reported. Average and maximum in-vehicle acetaldehyde concentrations measured in southern California [7.7 and 37.0 parts per billion volume (13.7 and 66.6 micrograms per cubic meter), respectively] appear to be similar in magnitude to those inside residences.
**Are there near source exposures to acetaldehyde in California?**

Some Californians may be exposed to near-source, or "Hot Spot" concentrations of acetaldehyde which are above the average ambient concentrations. "Hot spot" exposure may increase the potential cancer risk to individuals living near large combustion sources. Acetaldehyde emissions and risk information is currently under development by facilities under the AB 2588 Air Toxics "Hot Spots" emissions reporting program. This information will be used during the risk management phase to help determine priority and need for control of sources emitting acetaldehyde.

**Are there other routes of exposure to acetaldehyde?**

Yes. It is commonly ingested, but ingestion of acetaldehyde is not known to be a significant cause of respiratory tract cancers, the focus of the health assessment for this compound. Acetaldehyde is found as a natural constituent of a number of foods such as ripe fruits and alcoholic beverages. It is also used as a food preservative and a flavoring agent for a variety of foods including margarine, baked goods, and milk products. Ingestion of alcohol is a major source of acetaldehyde in the body; acetaldehyde is a metabolic intermediate in the oxidation of ethanol by liver enzymes. Acetaldehyde levels in water are relatively low. Dermal absorption of acetaldehyde is surmised to be negligible.

**What is the persistence of acetaldehyde in the atmosphere?**

Depending on atmospheric conditions, the overall half-life of acetaldehyde is calculated to be approximately 12 hours which under normal meteorological conditions is sufficient time to allow dispersion throughout an air basin. Stagnant air conditions which may occur in California
can impede this dispersion. The dominant atmospheric removal mechanism for acetaldehyde during daylight hours is by photolysis and oxidation by hydroxyl radicals. Peroxyacetyl nitrate (PAN), a component of photochemical smog and a strong eye and mucous membrane irritant, and formaldehyde are the major breakdown products. The episodic nature of precipitation events, the small degree of acetaldehyde hydration and a calculated low washout ratio indicate that wet deposition (rain/fog) is not a significant removal mechanism.

**What are the health effects of acetaldehyde exposure?**

The health effects of acetaldehyde exposure have been reviewed and evaluated to determine whether acetaldehyde meets the definition of a TAC. The following text summarizes OEHHA staff findings regarding the health effects of acetaldehyde exposure.

**What evidence exists that exposure to acetaldehyde poses a public health hazard?**

The OEHHA staff agrees with the U.S. EPA classification of acetaldehyde (EPA Group B2) as a probable human carcinogen. The U.S. EPA bases this on "adequate evidence for carcinogenicity in animals and inadequate evidence in humans." In California, acetaldehyde was classified on April 1, 1988, as a chemical known to the state to cause cancer (Safe Drinking Water and Toxic Enforcement Act of 1986, Proposition 65). Acetaldehyde causes cancer in rodents, producing squamous cell carcinomas and adenocarcinomas originating in the respiratory and olfactory epithelium, respectively, in the nasal passages of male and female rats. In hamsters, acetaldehyde-induced tumors were found predominantly in the larynx. Epidemiological evidence for human cancer from exposure to acetaldehyde is limited because it is based on one imperfect study. The study lacked age and sex distribution, all of the small number of subjects had previously smoked, and there was concurrent exposure to other chemicals.
Because acetaldehyde is a reactive molecule it is mostly bound to proteins and other blood components while in the bloodstream. Acetaldehyde has been shown to cause a number of genotoxic effects in a variety of cell culture and in vitro assays, including DNA-protein crosslinks, sister chromatic exchanges, gene mutations, single strand breaks and chromosomal aberrations.

Exposure of experimental animals to acetaldehyde in high doses may be related to reproductive and developmental effects. It crosses the placenta and can present an exposure to the fetus. In studies with rodents, acetaldehyde has been shown to cause growth retardation, to damage developing fetuses, and to kill embryos. Reproductive in vitro toxicity studies have shown that acetaldehyde is an inhibitor of testicular testosterone production. There are no data available to relate the adverse animal effects to possible human exposure. The mechanisms by which acetaldehyde causes these effects in vitro and in vivo are not known. It is not possible at present to determine if acetaldehyde poses a reproductive or developmental hazard to humans.

**What is the cancer risk assessment for exposure to acetaldehyde?**

The inhalation risk is the calculated, theoretical possibility of contracting cancer when exposed to acetaldehyde at a concentration of one part per billion volume or one microgram per cubic meter of air for a lifetime. Since several unit risks can be obtained depending on the different animal test groups considered and calculation procedures used, a range of values can be obtained. From the range a best value was selected which in the judgement of OEHHA has the strongest scientific support. The staff of the OEHHA recommends that the unit risk of $4.8 \times 10^{-6}$ per part per billion ($2.7 \times 10^{-6}$ per microgram per cubic meter) be considered the "best value of
the upper bound of risk." This is similar to the value calculated by the U.S. EPA to $2.2 \times 10^{-6}$ per microgram per cubic meter. This value is based on data from a recent bioassay in male rats. This unit risk, coupled with a lifetime exposure to one part per billion, would yield 4.8 excess potential cancer cases per million people. The use of this "best value" with California’s average ambient acetaldehyde exposure concentration of two parts per billion volume (3.6 microgram per cubic meter) yields 9.6 excess potential cancer cases per million people exposed throughout their lives. It can be estimated that up to 288 acetaldehyde-induced potential cancer cases would occur statewide among a population of 30 million people exposed to current ambient concentrations throughout their lives. The "best value" unit risk is a plausible upper bound estimate based on health-protective assumptions; the actual risk may be significantly lower. Table I shows a comparison of acetaldehyde potency with other compounds the Board has identified as TACs.

The upper limit estimate of the number of potential excess cancers due to outdoor airborne acetaldehyde exposure ranges from 2 to 54 per million people exposed throughout their lives, based on California’s present average ambient acetaldehyde exposure concentration of 2 parts per billion volume (4 microgram per cubic meter), and an excess cancer risk range of $0.97 \times 10^{-6}$ to $27.0 \times 10^{-6}$ per part per billion ($0.54 \times 10^{-6}$ to $15 \times 10^{-6}$ per microgram per cubic meter). This range of risk is based on data from studies in male and female rats. The upper limit estimate of the number of potential excess cancer cases for the South Coast Air Basin (with an average exposure concentration of 2.8 parts per billion volume or 5.0 micrograms per cubic meter) is 3 to 76 per million people exposed throughout their lives.

The range of risk values represents several sources of uncertainty, including statistical uncertainty due to the relatively small number of animals used in the bioassay (less than the usual 50 per group). Other general sources of uncertainty, include the choice of the animal-to-human
Table I

COMPOUNDS APPROVED BY THE SCIENTIFIC REVIEW PANEL FROM 1984 TO 1993

(in order of cancer potency)

<table>
<thead>
<tr>
<th>Compound</th>
<th>Unit Risk microgram per cubic meter</th>
<th>Unit Risk part per billion volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dioxins</td>
<td>$3.8 \times 10^1$</td>
<td>Particulate Matter</td>
</tr>
<tr>
<td>Chromium VI</td>
<td>$1.4 \times 10^1$</td>
<td>Particulate Matter</td>
</tr>
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<td>Cadmium</td>
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<td>Particulate Matter</td>
</tr>
<tr>
<td>Inorganic Arsenic</td>
<td>$3.3 \times 10^{-3}$</td>
<td>Particulate Matter</td>
</tr>
<tr>
<td>Nickel</td>
<td>$2.6 \times 10^{-4}$</td>
<td>Particulate Matter</td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>$1.7 \times 10^{-4}$</td>
<td>$3.7 \times 10^{-4}$</td>
</tr>
<tr>
<td>Ethylene Oxide</td>
<td>$8.8 \times 10^{-5}$</td>
<td>$1.6 \times 10^{-4}$</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td>$7.8 \times 10^{-5}$</td>
<td>$2.0 \times 10^{-4}$</td>
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</tr>
<tr>
<td>Carbon Tetrachloride</td>
<td>$4.2 \times 10^{-5}$</td>
<td>$2.6 \times 10^{-4}$</td>
</tr>
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<td>Benzene</td>
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<td>$9.3 \times 10^{-5}$</td>
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<td>Ethylene Dichloride</td>
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<td>Perchloroethylene</td>
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<td>$4.0 \times 10^{-5}$</td>
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<td>Acetaldehyde</td>
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</tr>
<tr>
<td>Trichloroethylene</td>
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<td>$1.1 \times 10^{-5}$</td>
</tr>
<tr>
<td>Methylene Chloride</td>
<td>$1.0 \times 10^{-6}$</td>
<td>$3.5 \times 10^{-6}$</td>
</tr>
<tr>
<td>Asbestos</td>
<td>$1.9 \times 10^{-4}$ per 100 fibers per cubic meter</td>
<td></td>
</tr>
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</table>
scaling factors, the choice of the extrapolation model, and the large range of extrapolation (five orders of magnitude) from the acetaldehyde concentrations used in the animal experiments to current ambient levels.

Based on the information available on acetaldehyde-induced carcinogenicity and the results of the risk assessment, the OEHHA staff conclude that acetaldehyde is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

**What is the potential for acute or chronic non-carcinogenic health effects from exposure to ambient concentrations of acetaldehyde?**

Acute exposure to acetaldehyde vapor leads to eye, skin and respiratory tract irritation. Prolonged exposure of the skin to liquid acetaldehyde causes erythema and burns; repeated contact may lead to dermatitis due to primary irritation or sensitization. Long-term or chronic exposure has been shown to damage the respiratory tract in rats. Applying an uncertainty factor of 1,000 using standard methodology produces a daily Reference Exposure Level of five parts per billion (9 micrograms per cubic meter).

The OEHHA staff concluded that it is unlikely that noncarcinogenic adverse health effects would be caused at the average levels of acetaldehyde currently found in the ambient air. However, acetaldehyde concentrations could be significantly higher near emission sources.

**Is there a threshold level for acetaldehyde?**

There is compelling evidence of acetaldehyde genotoxicity because of binding to DNA and mutagenicity and there is experimental evidence for acetaldehyde acting as an initiator of tumorigenesis. Therefore, the OEHHA staff considers acetaldehyde-induced carcinogenesis as a genotoxic event and were unable to determine a threshold for the phenomenon.
Pursuant to Health-and Safety Code-section 39661, the Scientific Review Panel (SRP) has reviewed the report "Acetaldehyde as a Toxic Air Contaminant" prepared by the staffs of the Air Resources Board (ARB) and the Office of Environmental Health Hazard Assessment (OEHHA) on the public exposure to, and health effects of acetaldehyde. The Panel also reviewed the public comments received on this report. Based on this review, the SRP makes the following findings also pursuant to Health and Safety Code section 39661:

1. Acetaldehyde was declared a toxic air contaminant by the Air Resources Board on April 8, 1993.

2. There is evidence that exposure to acetaldehyde results in animal carcinogenicity and therefore has the potential for human carcinogenicity. The International Agency for Research on Cancer (IARC) classified "acetaldehyde as a possible human carcinogen (2B) based on sufficient evidence in animals and inadequate evidence in humans." The United States Environmental Protection Agency (U.S. EPA) classified acetaldehyde "as a probable human carcinogen (B2) on the basis of sufficient evidence for carcinogenicity in animals and inadequate evidence in humans."

3. Based on available scientific information, a level of acetaldehyde exposure below which no carcinogenic effects are anticipated cannot be identified.

4. Based on a health protective interpretation of available scientific evidence, the upper 95 percent confidence limits on the potential lifetime risk of cancer from acetaldehyde at ambient concentrations range from 0.97 to 27.0 x 10⁻⁶ per part per billion (0.54 to 15.0 x 10⁻⁶ per microgram per cubic meter). Furthermore, based on available Scientific evidence, 4.8 x 10⁻⁶ per part per billion (2.7 x 10 per microgram per cubic meter) is the best value of the upper confidence limit of risk. Table I compares the best value of the upper bound acetaldehyde potential unit cancer risk with those of other compounds reviewed by the SRP. These 95 percent upper confidence limits for excess lifetime risks are health-protective estimates; the actual risk may be significantly lower.
5. The range of risk values represents several sources of uncertainty. They include uncertainty due to the small number of animals used in the bioassay, the choice of the animal to human scaling factors, and the choice of the extrapolation model.

6. The major identified sources of ambient outdoor acetaldehyde are direct emissions from mobile sources, fuel combustion, burning, wildfires, oil refineries, and secondary formation by photochemical reactions.

7. The addition of methanol and methyl tert-butyl ether (MTBE) to fuel does not increase acetaldehyde emissions. Alternate fuels containing the oxygenate additives ethanol or ethyl tert-butyl ether (ETBE), upon combustion, result in acetaldehyde emissions. However, it is not known to what extent ethanol or ETBE will be used as a winter oxygenate in California fuels, and therefore the resulting affect on acetaldehyde emissions.

8. One of the main products of the photooxidation of acetaldehyde is peroxyacetyl nitrate (PAN). Airborne acetaldehyde in the presence of nitrogen oxides can be converted to PAN which is a strong eye irritant and plant toxicant.

9. Based on data collected by the ARB's ambient toxic air contaminant monitoring network, the estimated mean annual population-weighted outdoor ambient exposure for approximately 20 million Californians is 2.3 parts per billion in volume (4.1 micrograms per cubic meter).

10. "Hot spot" exposure may increase the potential cancer risk to individuals living near large combustion sources. Acetaldehyde emissions and risk information are being submitted by facilities subject to the AB 2588 Air Toxics “Hot Spots” reporting program (Chapter 1252, Statutes of 1987, Health and Safety Code section 44300 et seq.). This information will be used during the risk management phase to help determine priority and need for control of sources emitting acetaldehyde.

11. Based on its gas-phase reactivity from photolysis and oxidation by the hydroxyl radical, acetaldehyde's estimated tropospheric lifetime is calculated to be approximately 12 hours under average meteorological conditions.

12. Results from indoor monitoring of acetaldehyde in California's homes, offices, and public buildings indicate that people are exposed frequently to higher indoor concentrations than outdoor acetaldehyde concentrations due to the abundance of combustion sources and consumer products in buildings that emit acetaldehyde. The results of surveys indicate that acetaldehyde concentrations inside California residences generally range from less than...
1 part per billion (1.8 micrograms per cubic meter) to 35 parts per billion (63 micrograms per cubic meter). Mean concentrations can range from 1 part per billion in office and public buildings to 113 parts per billion (203 micrograms per cubic meter) for a smoke filled tavern, with approximately 10 parts per billion (18 micrograms per cubic meter) found in conventional homes.

13. A number of acute adverse health effects are associated with acetaldehyde exposure. Such effects include irritation of the skin, eyes and mucous membranes, as well as nausea and headaches. Skin contact with acetaldehyde can induce long-term allergic dermal sensitization, and limited evidence suggests that inhalation of high concentrations of acetaldehyde can cause respiratory tract sensitization. Adverse health effects other than cancer are not expected to occur at mean statewide outdoor ambient concentrations. However, there is evidence that adverse acute health effects may result from exposure to levels found in indoor environments for those sensitive to acetaldehyde.

14. Long-term or chronic exposure has been shown to damage the respiratory tracts of rats. Using standard methodology to calculate a Reference Exposure Level (REL) produces a daily chronic REL of 5 parts per billion (9 micrograms per cubic meter).

15. The staffs of the ARB and OEHHA have developed a risk assessment based on relative exposure to outdoor concentrations. Using the OEHHA staff's best value for unit cancer risk of $4.8 \times 10^{-6}$ parts per billion volume, and the corresponding concentrations found in outdoor environments, the number of excess cancer cases due to outdoor exposure to acetaldehyde is estimated to be approximately 10 per million, for a 70 year lifetime. This corresponds to an excess cancer burden of 288 for outdoor exposures for a California population of 30 million.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on May 12, 1993

/s/
Dr. James N. Pitts, Jr.
Chairman, SRP