DRAFT

Potential Health Risks of Ethanol in Gasoline

Office of Environmental Health Hazard Assessment

California Environmental Protection Agency

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TABLE OF CONTENTS

1.0 INTRODUCTION

On March 25, 1999, Governor Gray Davis issued Executive Order D-5-99 which stated that, while the gasoline additive, methyl tertiary-butyl ether (MTBE), has benefited California by decreasing air pollution, it also poses an environmental threat to groundwater and drinking water. Weighing all of the evidence, the Governor declared that there is significant risk to the environment from using MTBE in gasoline in California. As a result, the Governor assigned tasks, by way of the Executive Order, to various designated state agencies, regarding the reformulation of gasoline in the State. Task 10 requires the California Air Resources Board (CARB) and the State Water Resources Control Board (SWRCB) to conduct an environmental fate and transport analysis of ethanol in air, surface water, and ground water. It further requires the Office of Environmental Health Hazard Assessment (OEHHA) to prepare an analysis of the health risks of ethanol in gasoline, the products of incomplete combustion of ethanol in gasoline, and any resulting secondary transformation products. This draft report, prepared by OEHHA, is the analysis of the potential public health impacts of ethanol as an oxygenate in gasoline.

1.1 Objective

The objective of this document is to present an evaluation of the public health impacts of ethanol as an oxygenate in gasoline. In order to give the analysis a frame of reference, the potential health impacts of ethanol in gasoline have been evaluated in comparison to the current MTBE formulation, as well as to gasoline with no oxygenate. This evaluation includes an analysis of evaporative emissions, tailpipe (exhaust) emissions, as well as atmospheric transformation products that are produced as a result of the use of ethanol in gasoline. Four

gasoline formulations were selected by CARB for analysis, all formulations fully complying with existing air pollution regulations. The formulations are as follows:

- Current MTBE-based California Phase 2 Reformulated Gasoline (CaRFG),
- Ethanol-based (with oxygen content of 3.5%),
- Ethanol-based (with oxygen content of 2.0%), and
- A non-oxygenated fully complying fuel.

The CARB model produced estimates of the total concentrations of specific pollutants in ambient air from all sources (emissions from stationary and mobile sources, and atmospheric transformation products). Generation of these estimates is described in detail in the report contributed by CARB: the results are cited here without modification. Rather than evaluating the health impact of each fuel-related pollutant, this report focuses on the differences in combustion by-products, evaporative emissions, and atmospheric transformation products that occur from use of one fuel versus another. It is not intended to assess the overall impact of gasoline usage regardless of type, or the adequacy of current regulatory controls in limiting the impacts of this usage on public health.

In conducting a quantitative assessment of the relative health impacts of the different fuel formulations, we used estimates of total air concentrations modeled by CARB, and calculated the risk levels associated with these concentrations. However, our confidence in these lifetime cancer risk estimates, and acute and chronic hazard indices, is lower than our confidence in the relative differences in risk estimate or hazard index associated with the different fuel scenarios. This arises because of the intrinsic uncertainties in the CARB exposure modeling and the risk assessment processes.

While this assessment focuses primarily on the potential impacts from emissions into the ambient air, an evaluation of potential risks from groundwater contamination by fuel components is also included. This evaluation centers primarily on the differences between MTBE and ethanol in groundwater.

2.0 HAZARD IDENTIFICATION: CHEMICALS OF CONCERN

In examining the health risks of ethanol in gasoline, we considered the impacts from evaporative emissions, exhaust (tailpipe) emissions, as well as secondary transformation products formed in the atmosphere. Evaporative emissions of unburned gasoline occur primarily during refueling at the pump, from fuel spills, and directly from carburetors and other fuel system components of automobiles. Exhaust emissions include unburned fuel and other products of incomplete combustion. Many of these products, particularly emissions of hydrocarbons and nitrogen oxides (NO_X), together are critical precursors in the formation of ozone and other atmospheric transformation products.

CARB provided the speciation profiles for the air emissions and modeling to determine concentrations of key chemicals from the four fuels. OEHHA focused this analysis on key chemicals associated with fuel use and potential changes in air concentration of those chemicals. The chemicals determined to be the most important in terms of public health risks are: 1) the oxygenates methyl *tertiary*-butyl ether (MTBE), ethanol; 2) Combustion products butadiene, formaldehyde, acetaldehyde, carbon monoxide; 3) Evaporative emittents benzene, hexane, and toluene; and 4) Atmospheric transformation products peroxyacetyl nitrate (PAN) and ozone. Summaries of the toxicity of each of these compounds are included with this document as Appendix A.

3.0 DOSE-RESPONSE ASSESSMENT

In a risk assessment, health impacts are quantified using health assessment values. These values characterize the dose-response relationship, that is the relationship between exposure to an agent and the incidence of an adverse health effect in an exposed population. In this risk analysis, health assessment values used to quantify risks were those currently available from Cal/EPA and U.S. EPA as described below. In the absence of adopted health assessment values suitable for estimating potential health impacts from the chemicals of concern, OEHHA used 'proposed' numbers that have been developed under other California regulatory programs that are currently undergoing scientific peer review. In cases where 'proposed' numbers were not available, OEHHA calculated 'health protective concentrations' for the purpose of this report. The sources of adopted values and proposed values currently undergoing scientific and public peer review, as well as the methodology used to calculate draft 'health protective concentrations', are all described below. The health assessment values used in this report are shown in Tables 1-3. The derivation of all proposed numbers and draft numbers are included in the chemical summaries in Appendix A.

3.1. Values for Assessing Potential Health Impacts from Inhalation Exposures

3.1.1. Carcinogenic Endpoints

Risks from exposure to a carcinogen are quantified using potency values. A carcinogenic potency is an estimate of the slope of the dose-response curve at low exposure concentrations. Potency values are expressed in units of inverse dose, either as a cancer potency or *slope factor* (i.e., units of $(mg/kg\text{-}day)^{-1}$), or for inhalation exposures, as a *unit risk factor* (i.e., units of $(\mu g/m^3)^{-1}$). These values represent the theoretical probability of extra cancer cases occurring in

an exposed population assuming a 70-year lifetime exposure. Potencies may be derived from animal or human data, and their derivation takes into account the available information on pharmacokinetics, mechanism of carcinogenic action, and the effects of different models on low dose extrapolation. The unit risk or cancer potency factors are usually a 95% upper confidence limit of the slope of the dose-response curve, and thus represent an upper estimate of the risk.

For this risk assessment, unit risk factors used in inhalation risk calculations are those currently available from Cal/EPA and U.S. EPA. These values, and information on how they were derived, are documented in the "Air Toxics Hot Spots Program Risk Assessment Guidelines, Part II: Technical Support Document for Describing Available Cancer Potency Factors" (OEHHA, 1999b).

3.1.2. Noncancer Endpoints

3.1.2.1. Acute, One-Hour Inhalation Exposures

Potential health impacts from acute, one-hour exposures were estimated using acute reference exposure levels (RELs). An REL is a concentration in air at or below which no adverse health effects are anticipated for a specified exposure duration. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. They are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Acute RELs used in the present risk analysis were obtained from the "Air Toxics Hot Spots Program Risk Assessment Guidelines, Part I: The Determination of Acute Reference Exposure Levels for Airborne Toxicants" (OEHHA, 1999a). As mandated by state legislation, these guidelines underwent public and scientific peer review, prior to approval by the

Scientific Review Panel and adoption by OEHHA (Senate Bill 1731, Statutes of 1992, Ch. 1162 of the California Health and Safety Code).

In the absence of adopted acute RELs, OEHHA calculated draft 'health protective concentrations' following the adopted methodology for developing acute RELs. Details on the methodology are provided in OEHHA (1999a).

3.1.2.2. Annual Average Inhalation Exposure Concentrations

Potential health impacts from chronic inhalation exposures were estimated using several types of health assessment values.

a) U.S. EPA's Reference Concentrations (RfCs)

U.S. EPA has developed reference concentrations (RfC) for many airborne toxicants. An RfC is defined by U.S. EPA as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime."

b) Proposed Chronic Reference Exposure Levels

In the absence of a U.S. EPA RfC, OEHHA used the proposed chronic reference exposure levels currently being developed by the Air Toxics Hot Spots Program risk assessment guidelines process. These values, as well as the methodology used to develop them, are in the process of undergoing public and scientific peer review. The methodology and proposed values are described in the "Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III:

Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels" available on our web page (www.oehha.ca.gov).

c) Reference Exposure Levels for Toxic Air Contaminants

OEHHA has developed RELs during evaluation of some of the compounds as toxic air contaminants (TACs), mandated by Assembly Bill 1807 (California Health and Safety Code 39660 et seq.) These numbers underwent public and scientific peer review. One chemical in the present analysis, acetaldehyde, has an REL that was developed under the TAC process.

In the absence of a U.S. EPA RfC, a proposed chronic REL, or an REL developed under the TAC process, OEHHA calculated draft health protective concentrations using the chronic REL methodology.

Table 1. Health Assessment Values and Draft "Health Protective Concentrations" (HPCs)

	Non-cancer		Cancer	
	1-hour ($\mu g/m^3$)	**Annual Average ($\mu g/m^3$)**	**Unit risk $(\mu g/m^3)^{-1}$**	**Air concentration corresponding to 10^{-6} lifetime risk ($\mu g/m^3$)**
acetaldehyde	**115 (65 ppb)** [draft HPC]	**9 (5 ppb)** [TAC]	**2.7 E-6 (4.8 E-6 ppb^{-1})** [TAC]	**3.7 E-1 (2.1 E-1 ppb)**
benzene	**1300 (400 ppb)** [acute REL]	**60 (20 ppb)** [proposed chronic REL]	**2.9 E-5 (9.3 E-5 ppb^{-1})** [TAC]	**3.5 E-2 (1.1 E-2 ppb)**
butadiene	**310 (140 ppb)** [draft HPC]	**8 (4 ppb)** [U.S. EPA RfC]	**1.7 E-4 (3.7 E-4 ppb^{-1})** [TAC]	**5.9 E-3 (2.7 E-3 ppb)**
ethanol	**100,000 (53,000 ppb)** [draft HPC]	**100,000 (53,000 ppb)** [draft HPC]	No evidence of carcinogenicity by inhalation	
formaldehyde	**94 (76 ppb)** [acute REL]	**3 (2 ppb)** [proposed chronic REL]	**6.0 E-6 (7.0 E-6 ppb^{-1})** [TAC]	**1.7 E-1 (1.4 E-1 ppb)**
MTBE	**25,000 (7000 ppb)** [draft HPC]	**3000 (800 ppb)** [U.S. EPA RfC]	**2.6 E-7 (9.3 E-7 ppb^{-1})** [draft TAC]	**3.9 E0 (1.1 ppb)**
PAN	**8.8 (1.8 ppb)** [draft HPC]	**3.2 (0.6 ppb)** [draft HPC]	No evidence of carcinogenicity / inadequate data	

Draft HPC: health protective concentration; In the absence of adopted health assessment values OEHHA developed draft numbers for use in this analysis. The basis of these numbers is described in Appendix A in the toxicity summaries. These numbers are not regulatory numbers.

TAC: toxic air contaminant; peer-reviewed value developed under the TAC program mandated by AB 1807

Acute REL: acute reference exposure level; peer-reviewed value developed as part of the legislatively mandated Air Toxics Risk Assessment Program risk assessment guidelines process (SB 1731; Statutes of 1992; California Health and Safety Code, Chapter 1162)

Proposed chronic REL: chronic reference exposure level; currently being developed as part of the legislatively mandated Air Toxics Risk Assessment Program risk assessment guidelines process (SB 1731; Statutes of 1992; California Health and Safety Code, Chapter 1162); these numbers are currently undergoing various stages of a scientific peer review and public comment process.

Table 2. Health Assessment Values for the Criteria Air Pollutants

	1 hour	*8 hour*	*24 hour*	*Annual Average*
carbon monoxide	**23000 $\mu g/m^3$ (20 ppm)** [acute REL; CA AAQS]	**10,000 $\mu g/m^3$ (9.0 ppm)** [CA and federal AAQS]		
NO_X	**470 $\mu g/m^3$ (0.25 ppm)** [acute REL; CA AAQS]			**100 $\mu g/m^3$ (0.053 ppm)** [federal AAQS]
Ozone	**180 $\mu g/m^3$ (0.09 ppm)** [acute REL; CA AAQS]	**157 $\mu g/m^3$ (0.08 ppm)** [federal AAQS]		
Particulate Matter > 10 microns (PM_{10})			**50 $\mu g/m^3$** [CA AAQS]	**30 $\mu g/m^3$** [CA AAQS]

Acute REL: Acute Reference Exposure Level
CA AAQS: California Ambient Air Quality Standard
Federal AAQS: Federal Ambient Air Quality Standard

3.2. Values for Assessing Potential Health Impacts from Exposure via Drinking Water

3.2.1. Public Health Goals (PHGs)

The PHG describes concentrations of contaminants in drinking water at which adverse health effects are not expected to occur, even over a lifetime of exposure. PHGs have been developed by OEHHA under the California Safe Drinking Water Act of 1996 (amended Health and Safety Code, Section 116365). PHGs are based solely on scientific and public health considerations, unlike many other state and federal drinking water standards which take into account non public health considerations (e.g., technological feasibility and economic factors).

Table 3. Health Assessment Values and Draft "Health Protective Concentrations" for Assessing Potential Health Impacts of Ethanol in Gasoline – Oral exposures from Drinking Water.

	Health Assessment Value	Endpoint	Source	Reference
benzene	1.4×10^{-4} mg/L (0.14 ppb)	cancer	draft public health goal	OEHHA (1999c)
ethanol	1100 mg/L (1.1×10^{6} ppb)	noncancer	draft HPC	---
methyl t-butyl ether (MTBE)	0.013 mg/L (13 ppb)	cancer	public health goal	OEHHA (1999d)
toluene	0.150 mg/L (150 ppb)	noncancer	public health goal	OEHHA (1999e)
xylenes	1.8 mg/L (1800 ppb)	noncancer	public health goal	OEHHA (1997)

Public health goal (PHG): developed under the California Safe Drinking Water Act of 1986 (amended Health and Safety Code, Section 116365)

draft HPC: health protective concentration; In the absence of adopted health assessment values OEHHA developed draft numbers for use in this analysis. The basis of these numbers is described in Appendix A in the toxicity summaries. These numbers are not regulatory numbers.

4.0 EXPOSURE ASSESSMENT: SOURCES OF EXPOSURE DATA

The CARB conducted modeling analyses of the air quality impacts of use of one fuel versus another. As noted above, the fuels modeled include the current MTBE formulation, two formulations containing ethanol, and a formulation containing no oxygenate. These were considered in exposure scenarios based on the predicted emissions inventory for the year 2003. A scenario of the 1997 emissions inventory and MTBE-containing fuel was used to calibrate the model against actual measured data for that year. The model predictions of one-hour and 24-hour peak concentrations of pollutants are worst case estimates, but the population-weighted annual averages are best estimates calibrated against actual measurements for 1997, the baseline year. CARB's modeling analysis is described in more detail in their report and the accompanying appendices of this document. CARB developed speciation profiles of volatile organic compounds (VOC) for each fuel under varying conditions (e.g., start exhaust, hot

stabilized exhaust, etc.). The modeling results provide one-hour peak concentrations for all compounds, eight-hour concentrations for ozone and CO, 24-hour average concentrations for PM_{10}, and population-weighted annual average concentrations for all compounds of interest. These modeled air concentrations are summarized in Table 4 and form the basis of the health impacts analysis. The concentrations are input into simple arithmetic equations to estimate health risks.

The SWRCB contracted with Lawrence Livermore National Laboratories to model a variety of scenarios related to release of ethanol-containing gasoline onto soil or into surface waters. The main purpose of the modeling is to estimate potential water quality impacts of using ethanol as an oxygenate in gasoline. The modeling focused on predicting the movement of ethanol through the soil and into groundwater as well as the effects ethanol may have on the movement of other gasoline constituents (primarily benzene, toluene, and xylenes or BTX) through soil and into groundwater. Although the modeling analyses are not yet complete, information to date indicates that ethanol degrades readily in the environment by microbial degradation. This is in contrast to MTBE which is resistant to microbial degradation. As a result, it appears unlikely that ethanol will contaminate groundwater to any degree approaching a public health concern. At this time, OEHHA is awaiting results of the modeling being conducted on the effects of ethanol on the movement of other gasoline constituents (BTX). The results of the modeling will be used to ascertain whether the probability of well-water contamination by BTX will increase, decrease, or stay the same relative to baseline MTBE-containing fuels. If possible, we may be able to obtain modeled concentrations of BTX and ethanol to compare to existing PHGs or estimated health-protective concentrations as described in the following section.

Table 4. Atmospheric Concentration Estimates: Range of Estimated Maximum Pollutant Levels for Various Scenarios in the South Coast Air Basin[a]

	1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 Non-Oxy
Acetaldehyde (ppb)					
Population-Weighted Annual Exposure					
Upper	1.8	1.5	1.6	1.8	1.5
Lower	1.8	1.5	1.5	1.7	1.5
Maximum Daily Average					
Upper	6.2	6.2	6.2	6.2	6.2
Lower	5.1	5.1	5.1	5.1	5.1
Maximum 1 Hour Average					
Upper	17.7	16.7	16.9	17.1	16.9
Lower	13.8	13.1	13.2	13.4	13.2
Benzene (ppb)					
Population-Weighted Annual Average					
Upper	1.19	0.87	0.86	0.88	0.85
Lower	1.07	0.78	0.72	0.73	0.76
Maximum Daily Average					
Upper	9.6	7.2	7.1	7.2	7.1
Lower	7.4	5.5	5.2	5.2	5.5
Maximum 1 Hour Average					
Upper	22.5	16.4	16.4	16.4	16.4
Lower	11.6	8.4	7.4	7.4	8.4
Butadiene (ppb)					
Population-Weighted Annual Average					
Upper	0.36	0.20	0.21	0.20	0.21
Lower	0.34	0.19	0.19	0.19	0.20
Maximum Daily Average					
Upper	2.9	2.9	2.9	2.9	2.9
Lower	2.0	2.0	2.0	2.0	2.0
Maximum 1 Hour Average					
Upper	6.8	6.8	6.8	6.8	6.8
Lower	3.1	3.1	3.1	3.1	3.1
Ethanol (ppb)					
Population-Weighted Annual Average					
Upper	5.4	5.1	8.2	11.3	5.1
Lower	5.4	5.1	7.0	8.8	5.1
Maximum Daily Average					
Upper	51	46	48	50	46
Lower	47	43	44	45	43
Maximum 1 Hour Average					
Upper	108	98	101	103	98
Lower	78	71	72	73	71

Table 4 (continued). Atmospheric Concentration Estimates: Range of Estimated Maximum Pollutant Levels for Various Scenarios in the South Coast Air Basin[a]

	1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 Non-Oxy
Formaldehyde (ppb)					
Population-Weighted Annual Average					
Upper	4.7	3.7	3.7	3.7	3.7
Lower	4.7	3.6	3.6	3.6	3.6
Maximum Daily Average					
Upper	14.0	12.2	12.2	12.2	12.2
Lower	14.0	12.2	12.0	12.1	12.2
Maximum 1 Hour Average					
Upper	37.8	38.5	38.0	38.1	38.0
Lower	20.3	20.7	20.4	20.5	20.4
MTBE (ppb)					
Population-Weighted Annual Average					
Upper	3.9	2.7	0	0	0
Lower	3.6	2.5	0	0	0
Maximum Daily Average					
Upper	29	21	0	0	0
Lower	13	9	0	0	0
Maximum 1 Hour Average					
Upper	68	47	0	0	0
Lower	19	13	0	0	0
PAN (ppb)[b]					
Maximum Daily Average					
Model	2.1	2.1	2.1	2.1	2.1
Maximum 1 Hour Average					
Model	4.4	4.2	4.4	4.4	4.4

Table 4 (continued). Atmospheric Concentration Estimates: Range of Estimated Maximum Pollutant Levels for Various Scenarios in the South Coast Air Basin[a]

	1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 Non-Oxy
Carbon Monoxide (ppm)					
Maximum 8 Hour Average					
Best	17.5	14.3	14.3	13.4	14.7
Maximum 1 Hour Average					
Best	22.5	19.2	19.2	18.0	19.7
Nitrogen Dioxide (ppm)					
Maximum Annual Average					
Best	0.043	CARB did not estimate since no significant change in Max 1 hour			
Maximum Daily Average					
Best	0.117	0.098	0.097	0.097	0.097
Maximum 1 Hour Average					
Best	0.255	0.235	0.235	0.235	0.235
Ozone (ppm)					
Maximum 8 Hour Average					
Best	0.206	0.196	0.197	0.196	0.198
Maximum 1 Hour Average					
Best	0.244	0.231	0.232	0.229	0.234
Particulate Matter (10 microns or less) ($\mu g/m^3$)					
Maximum Annual Geometric Mean					
Best	56	CARB reported, "No significant change expected among 2003 scenarios"[c]			
Maximum Daily Average					
Best	227	CARB reported,"No significant change expected among 2003 scenario"[c]			

[a] Source: Table 6 of "Analysis of the Air Quality Impacts of the Use of Ethanol in Gasoline. September 28, 1999. Public Review Draft. California Air Resources Board, Cal/EPA"

[b] A population-weighted annual average for PAN was not determined because consistent long-term measurements of atmospheric PAN have not been performed. See CARB report for details.

[c] No significant change compared to 1997 MTBE-fuel scenario. See CARB report for details.

5.0. METHOLOGY USED FOR QUANTIFYING CANCER AND NONCANCER RISKS

Risk characterization is the process of integrating data on exposure with dose-response in order to estimate public health impacts. In quantifying risks of exposure, cancer and noncancer endpoints are considered separately.

5.1. Estimating Cancer Risk

Typically, carcinogenesis is treated as a "nonthreshold" toxicological phenomenon. In other words, there is some finite risk to any finite dose. It may be so small as to be indistinguishable from zero at very low doses. In order to estimate cancer risk, we multiply the unit risk factor by the modeled concentration in air to obtain a unitless probability of cancer occurring in a population thus exposed.

$$\text{Risk} = \text{concentration} \times (\text{URF or potency slope})$$

So, for example, if the unit risk factor is 1×10^{-6} for chemical "X", and the concentration of chemical "X" is 5 $\mu g/m^3$, then the risk is calculated as :

$$\text{Risk} = [(1 \times 10^{-6})\ (ug/m^3)^{-1}] \times 5\ \mu g/m^3 = 5 \times 10^{-6}$$

The cancer risks thus estimated reflect an upper estimate (usually a 95% upper confidence limit) of the potential cancer cases in a population exposed to chemical "X" at 5 $\mu g/m^3$ over a lifetime.

In other words, there is the potential for five extra cancer cases to occur over a 70 year period in a population of one million persons exposed to that level of chemical "X".

In cases where there is exposure to multiple carcinogens, the risk of each carcinogen is added. The assumption implicit in this is that the effect on cancer risk in the population exposed to multiple carcinogens is additive. It is possible that some carcinogens might be synergistic and some antagonistic, although at low exposure levels these mechanisms may be unimportant.

5.2. Estimating Noncancer Risk

Noncancer health endpoints are assumed to follow the concept of a threshold for effect. If the exposure is below the individual's threshold for effect, then no adverse impact would be expected. To quantify potential noncancer health impacts resulting from exposure to hazardous substances, a *hazard index* approach is used. In using this approach, measured or modeled exposure concentrations are compared to calculated health assessment values such as the Reference Exposure Level (REL). The Hazard Quotient (HQ) for a chemical is the ratio of the modeled concentration in air to the REL. If the HQ exceeds one, a 'red flag' is raised with regard to exposure to that chemical and possible adverse health impacts. Exceedance of an HQ of one does not necessarily mean that a health impact will in fact occur. It implies that the margin of safety built into the REL is being eroded. The higher the ratio, the closer the exposure is to an adverse level. It is impossible to calculate the exact concentration at which anyone in a diverse population would respond. Interindividual differences in response and generally limited information on the toxicant preclude such a determination. Uncertainty factors are included in the calculation of the RELs to protect sensitive members of the population.

To assess the cumulative impact of several chemicals present at the same time, it is important to consider the interaction of the effects of the toxicants. Unless specific information is available to the contrary, the interaction of two or more chemicals that affect the same target organ is assumed to be additive. An underlying issue in chemical interactions and additivity is the concept of threshold. Exposure to a single chemical may not result in a toxic response if it is below the threshold necessary to elicit a response. However, simultaneous exposure to two or more chemicals that impact the same target organ at sub-threshold concentrations may result in a toxic response. This is taken into account by adding the individual HQs for chemicals that impact the same target organ or system. Thus, if more than one toxicant is present that impacts the same target organ or system (e.g., respiratory system, cardiovascular system), then the HQ for individual compounds is added to produce the Hazard Index (HI). As such, the *hazard index* approach assumes that multiple sub-threshold exposures to chemicals acting on the same target organ could result in an overall risk of developing adverse effects. This may underestimate the effect in the cases in which interactions are synergistic, or overestimate it if the effects are not additive or are antagonistic.

6.0. RISK CHARACTERIZATION

6.1. Inhalation – Cancer and Noncancer Effects

The following section refers to data presented in Tables 5a-c.

6.1.1. Acetaldehyde

The lifetime cancer risk is the toxicological endpoint of concern for exposure to acetaldehyde in the South Coast Air Basin. Toxic endpoints may include nasal and pulmonary cancers, with a cancer unit risk value = 2.7 $x10^{-6}$ $(\mu g/m^3)^{-1}$ or 4.8 x 10^{-6} ppb^{-1}. Several sources of uncertainty result in a lower level of confidence in this estimate for acetaldehyde than for some of the other carcinogenic potency estimates, for example, the benzene and butadiene cancer risk estimates. The sources of uncertainty are similar to those facing other cancer potency estimates. They include reliance on animal studies due to the lack of human data, and the five orders of magnitude extrapolation from experimental animal exposure concentrations to current ambient levels. However, in the case of acetaldehyde, the extensive metabolism of the compound *in vivo* (and its occurrence as a normal intermediary metabolite) are additional sources of uncertainty with respect to the standard assumption in risk assessment that the dose response curve is linear down to the low ambient levels of the compound.

The upper bound estimate of the cancer risk resulting from exposure to the maximal predicted levels of acetaldehyde over a 70 year lifespan is seven to nine excess cancer cases per million people exposed. The real risk may in fact be considerably lower than this upper bound estimate. There are increased ambient concentrations of acetaldehyde from the ethanol-based fuel containing 3.5% oxygen, compared to the other formulations evaluated for the year 2003. This results in an increase of up to two in a million excess lifetime cancer cases in the upper bound estimate. However, in view of the uncertainties both in the emission and exposure predictions, and in the acetaldehyde lifetime cancer risk estimate, this predicted increase in risk may be regarded as of marginal significance when comparing the other consequences of the

different fuel formulations. None of the predicted ambient levels of acetaldehyde for the year 2003 exceeds the levels in the 1997 model.

The acute (one-hour maximal average) and chronic (maximum annual exposure) noncancer Hazard Quotients (HQ) for acetaldehyde generated by each of the fuel scenarios are well below one. In general, the air concentrations are three- to five-fold below the reference exposure levels and there is little difference in HQ values among the five fuel types. Toxicological endpoints considered include eye, skin, and respiratory tract irritation with acute exposure, and inflammation of the respiratory tract and degeneration of the olfactory epithelium with chronic exposure.

6.1.2. Benzene

The lifetime cancer risk is the toxicological endpoint of concern for exposure to benzene in the South Coast Air Basin. Health effects other than cancer are not expected to occur at maximal ambient levels. The primary toxic endpoint in humans is leukemia, with a cancer unit risk value = 2.9×10^{-5} $(\mu g/m^3)^{-1}$ or 9.3×10^{-5} ppb^{-1}. There is a moderate to high level of confidence in the estimate of this value. Principally, there exists sufficient evidence to consider benzene a carcinogen in both humans and experimental animals. The cancer risk value is the upper 95 percent confidence level estimate from the analysis of human data and falls within the range of estimates derived from numerous epidemiological and animal studies. However, the approximate five orders of magnitude extrapolation from human occupational exposure concentrations to current ambient levels represent a major source of uncertainty for the benzene cancer risk estimate. Although some experts have postulated that the mechanism by which benzene causes leukemia may have a threshold, there are also substantial reasons for considering

that benzene is acting as a nonthreshold genotoxic carcinogen. Based on this latter interpretation, benzene is treated as a substance without a carcinogenic threshold in humans.

Comparing the 1997 fuel scenario to the year 2003 formulations, the upper bound estimate of the cancer risk resulting from exposure to the maximal predicted levels of benzene over a 70 year lifespan is expected to drop from 110 to about 80 excess cancer cases per million people exposed. Given the uncertainty in the cancer risk estimate, the differences in cancer risks between the various year 2003 fuel scenarios are small and insignificant.

The acute and chronic noncancer HQs generated for benzene by each of the five fuel scenarios are well below one. The upper bound concentrations in air are between 17- and 25-fold below the REL and there is essentially no difference in HI values among the year 2003 formulations. The most sensitive toxicological endpoint under acute exposures may include reduced birth weights in newborns. Chronic exposure and high acute exposure to benzene may result in hematotoxicity, including aplastic anemia.

6.1.3. Butadiene

The lifetime cancer risk is the toxicological endpoint of concern for exposure to butadiene in the South Coast Air Basin. Butadiene may induce cancers at multiple sites including, heart, lung, mammary gland, ovaries, liver, pancreas, Zymbal gland, thyroid, testes, and hematopoietic system. The cancer unit risk value is 1.7×10^{-4} $(\mu g/m^3)^{-1}$ or 3.7×10^{-4} ppb^{-1}. There is a moderate level of confidence in the estimate of this value. Recent epidemiological studies suggest a connection between excess cases of leukemia and lymphoma and butadiene exposure, although this provides only limited evidence to support the carcinogenic effects observed in experimental animals. Mice are known to develop lymphomas following butadiene

exposure, although this has not been observed in the rat. This interspecies difference is a significant source of uncertainty in the risk estimate, and may reflect differences seen between mice and rats regarding butadiene metabolism. Another uncertainty results from the necessity to extrapolate from experimental animal exposure concentrations to current ambient levels. However, the evidence on metabolism and carcinogenicity suggests that butadiene is a genotoxic carcinogen acting via metabolically generated reactive intermediates. Hence, no threshold is thought to exist for this substance.

Comparing the1997 fuel scenario to the year 2003 formulations, the upper end estimate of the cancer risk resulting from exposure to the maximal predicted levels of butadiene over a 70 year lifespan is expected to drop from 130 to about 74-78 excess cancer cases per million people exposed. The real risks may be lower than these upper bound estimates. Given the uncertainty in the cancer risk estimates, the differences in cancer risks between the various year 2003 fuel scenarios are small and likely to be of marginal significance.

The acute and chronic noncancer HQs generated for butadiene by each of the five fuel scenarios are well below one. There is essentially no difference in HQ values among the year 2003 formulations, though the year 2003 formulations have significantly lower chronic HQs than the 1997 formulation. The upper and lower bound one-hour average concentrations are between 20 and 50-fold below the acute REL. The most sensitive toxicological endpoint under acute exposures may include reduced birth weights in newborns. Chronic exposure to butadiene may result in ovarian atrophy.

6.1.4. Ethanol

Health effects due to ethanol exposure under any of the five fuel scenarios are not expected to occur at modeled ambient levels. There is no evidence that ethanol is carcinogenic via the inhalation route. Exposure to high concentrations of ethanol vapor may result in transient irritation to eyes and the respiratory system under either acute or chronic conditions. However, the acute and chronic noncancer HQs generated for ethanol by each of the five fuel scenarios are 0.002 or less, indicating that modeled concentrations are at least 500-fold below the RELs.

6.1.5. Formaldehyde

The lifetime cancer risk is the toxicological endpoint of concern for exposure to formaldehyde in the South Coast Air Basin. The primary toxic endpoint is nasal cancer, but may also include other respiratory tract cancers. The cancer unit risk is 6.0×10^{-6} $(\mu g/m^3)^{-1}$ or 7.0×10^{-6} ppb^{-1}. The sources of uncertainty in this estimate are similar to those facing other cancer potency estimates. They include reliance on animal studies due to insufficient human data, and three to four orders of magnitude extrapolation from experimental animal exposure concentrations to current annual average exposure levels. Another uncertainty involves cross-species scaling calculations. The evidence indicates formaldehyde is a contact carcinogen (i.e. formaldehyde generally reacts with the first cells it contacts) so that whole-body scaling factors may not be appropriate. Because the formaldehyde cancer unit risk is based on studies in rats, there is uncertainty in extrapolating to humans due to potentially significant differences in the anatomy and physiology of the respiratory systems between rats and humans.

Comparing the 1997 fuel scenario to the year 2003 scenarios, the upper bound estimate of the cancer risk resulting from exposure to the maximal predicted levels of formaldehyde over a

70 year lifespan is expected to drop from 33 to about 26 excess cancer cases per million people exposed by 2003. There is no apparent difference between year 2003 fuel formulations regarding cancer risk from formaldehyde.

The chronic (maximum annual exposure) noncancer HQ generated by the 1997 fuel scenario is 2.4. The 2003 fuel scenarios have lower HQs, but indicate that the concentrations of formaldehyde are almost two-fold above the REL. There is no apparent difference between fuel formulations for year 2003 of possible chronic health effects of formaldehyde. Toxicological endpoints include eye and respiratory system irritation. It is possible that some sensitive individuals may develop these chronic adverse effects at the maximal predicted annual exposure. Simultaneous exposure to other sensory irritants, such as acetaldehyde, may exacerbate the eye and respiratory irritation caused by formaldehyde (see section 6.4). However, it should be noted that the proposed chronic REL is undergoing revision and may change. The acute health effects from formaldehyde, primarily due to eye irritation, are not anticipated to occur at the predicted maximal ambient levels. The upper bound maximum one-hour average concentrations for all five fuel scenarios were two-fold below the acute REL.

Significant indoor exposures to formaldehyde are known to occur. However, the emission estimates determined in this report for all pollutants, including formaldehyde, do not address potential exposure to indoor sources of formaldehyde. Unlike the formaldehyde TAC document, the estimated cancer risk and noncancer hazards reported here do not reflect the potential for indoor exposure to formaldehyde.

6.1.6. Methyl t-Butyl Ether (MTBE)

The lifetime cancer risk is the toxicological endpoint of concern for exposure to MTBE in the South Coast Air Basin. Toxic endpoints in animal studies included leukemia, lymphoma, and testicular, kidney, and liver cancer. The cancer unit risk estimate is 2.6×10^{-7} $(\mu g/m^3)^{-1}$ or 9.3×10^{-7} ppb^{-1}. The sources of uncertainty include those facing other cancer potency estimates, such as reliance on animal studies due to the lack of human data, and the extrapolation from experimental animal exposure concentrations to current ambient levels. Some additional sources of uncertainty apply in this particular case since the mechanism of action of MTBE as a carcinogen is unknown. The estimate therefore relies on health protective default assumptions as to the applicability of the findings in animals to humans, and the shape of the dose response curve in the low-dose region.

The upper bound estimate of the cancer risk resulting from exposure to the annual average levels of MTBE predicted in the 1997 scenario, over a 70 year lifespan, is 3.6 excess cancer cases per million people exposed. In the 2003 scenario with the same MTBE-containing fuel, the predicted concentration of MTBE in air, and thus the associated cancer risk estimate, is approximately 30% lower (2.5 excess cancer cases). The cancer risk via inhalation of MTBE is zero for scenarios where the fuel formulation does not contain MTBE.

The acute and chronic noncancer HQs generated by each of the MTBE-containing fuel scenarios are at least 0.01, indicating that modeling concentrations are at least 100-fold below the RELs. Noncancer health effects due to acute or chronic MTBE exposure are not expected to occur at maximal ambient levels. The most sensitive toxicological endpoints for acute inhalation exposure may include eye irritation, respiratory irritation and CNS effects (headache, nausea,

vomiting, dizziness, and disorientation). The most sensitive toxicological endpoints for chronic exposure may include eye irritation, kidney damage, and CNS depression.

6.1.7. Peroxyacetyl Nitrate (PAN)

The acute noncancer HQs for PAN based on the results of the air modeling are above the threshold at which toxic effects may occur, and are also the toxicological endpoints of concern for this pollutant in the South Coast Air Basin. The most sensitive acute toxic endpoint is eye irritation. The one-hour maximum predicted average HQ is 2.4 under all fuel scenarios except the year 2003 MTBE fuel, for which the maximum predicted average HQ is 2.3. It appears that none of the scenarios for the year 2003 involve an exacerbation of the adverse health impact of PAN compared to the 1997 data (2.4). While these HQs are above one, it should be recognized that the air modeling results for short-term exposures reflect unfavorable meteorology. As mentioned above, the CARB notes that they have much more confidence in the relative values than the absolute values of concentrations of the modeled chemicals.

Of note, there has been a consistent downward trend in the observed average acute PAN concentrations in the South Coast Air Basin. Twenty-four hour average PAN concentrations have declined from 15-20 ppb in the late 1960's, to 5-12 ppb in 1985-1990 and 2-5 ppb in 1993. Therefore, it may be concluded that the irritant effects due to exposure to PAN have decreased proportionally in the South Coast Air Basin since the 1960's.

A population-weighted annual average exposure to PAN has not been determined because consistent long-term measurements of atmospheric PAN have not been measured. Therefore, a chronic HQ cannot be adequately determined. The most sensitive endpoint from

chronic exposure to PAN may include inflammation, epithelial metaplasia and hyperplasia in the respiratory tract.

Currently, there is inadequate evidence to determine if PAN is carcinogenic in either experimental animals or humans.

Table 5a. Range of Estimated Maximum Noncancer Hazard Quotients (HQ) for Various Scenarios in the South Coast Air Basin

		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde						
Chronic HQ	Upper	0.4	0.3	0.3	0.4	0.3
	Lower	0.4	0.3	0.3	0.3	0.3
Acute HQ	Upper	0.3	0.3	0.3	0.3	0.3
	Lower	0.2	0.2	0.2	0.2	0.2
Benzene						
Chronic HQ	Upper	0.06	0.04	0.04	0.04	0.04
	Lower	0.05	0.04	0.04	0.04	0.04
Acute HQ	Upper	0.06	0.04	0.04	0.04	0.04
	Lower	0.03	0.02	0.02	0.02	0.02
Butadiene						
Chronic HQ	Upper	0.09	0.05	0.05	0.05	0.05
	Lower	0.09	0.05	0.05	0.05	0.05
Acute HQ	Upper	0.05	0.05	0.05	0.05	0.05
	Lower	0.02	0.02	0.02	0.02	0.02
Ethanol						
Chronic HQ	Upper	0.0001	0.0001	0.0002	0.0002	0.0001
	Lower	0.0001	0.0001	0.0001	0.0002	0.0001
Acute HQ	Upper	0.002	0.002	0.002	0.002	0.002
	Lower	0.001	0.001	0.001	0.001	0.001
Formaldehyde						
Chronic HQ	Upper	2.4	1.9	1.9	1.9	1.9
	Lower	2.4	1.8	1.8	1.8	1.8
Acute HQ	Upper	0.5	0.5	0.5	0.5	0.5
	Lower	0.3	0.3	0.3	0.3	0.3
MTBE						
Chronic HQ	Upper	0.005	0.003	0.0	0.0	0.0
	Lower	0.005	0.003	0.0	0.0	0.0
Acute HQ	Upper	0.01	0.007	0.0	0.0	0.0
	Lower	0.003	0.002	0.0	0.0	0.0
PAN*						
Acute HQ		2.4	2.3	2.4	2.4	2.4

* A population-weighted annual average for PAN was not determined because consistent long-term measurements of atmospheric PAN have not been performed. See CARB report for details.

Table 5b. Range of Estimated Maximum Noncancer Hazard Quotients (HQ) for Various Scenarios in the South Coast Air Basin – Criteria Air Pollutants

	1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Carbon Monoxide					
Acute 8 hour HQ	1.9	1.6	1.6	1.5	1.6
Acute 1 hour HQ	1.1	1.0	1.0	0.9	1.0
Nitrogen Dioxide					
Chronic HQ	0.8	concentrations not estimated by CARB since no significant change in Maximum 1-Hour*			
Acute 1 hour HQ	1.0	0.9	0.9	0.9	0.9
Ozone					
Acute 8 hour HQ	2.6	2.5	2.5	2.5	2.5
Acute 1 hour HQ	2.7	2.6	2.6	2.6	2.6
Particulate Matter (PM_{10})					
Chronic HQ	1.9	CARB reported, "No significant change expected among			
Acute 24 hour HQ	4.5	2003 scenarios" for both annual and daily concentrations*			

* compared to exposure estimates for the 1997 MTBE-fuel scenario (see CARB report for details)

Table 5c. Range of Estimated Maximum Lifetime Cancer Risk Values for VariousScenarios in the South Coast Air Basin

		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde						
Lifetime	Upper	8.6×10^{-6}	7.2×10^{-6}	7.6×10^{-6}	8.6×10^{-6}	7.2×10^{-6}
Cancer Risk	Lower	8.6×10^{-6}	7.2×10^{-6}	7.2×10^{-6}	8.1×10^{-6}	7.2×10^{-6}
Benzene						
Lifetime	Upper	1.1×10^{-4}	8.1×10^{-5}	8.0×10^{-5}	8.2×10^{-5}	7.9×10^{-5}
Cancer Risk	Lower	1.0×10^{-4}	7.3×10^{-5}	6.7×10^{-5}	6.8×10^{-5}	7.1×10^{-5}
Butadiene						
Lifetime	Upper	1.3×10^{-4}	7.4×10^{-5}	7.8×10^{-5}	7.4×10^{-5}	7.8×10^{-5}
Cancer Risk	Lower	1.3×10^{-4}	7.0×10^{-5}	7.0×10^{-5}	7.0×10^{-5}	7.4×10^{-5}
Formaldehyde						
Lifetime	Upper	3.3×10^{-5}	2.6×10^{-5}	2.6×10^{-5}	2.6×10^{-5}	2.6×10^{-5}
Cancer Risk	Lower	3.3×10^{-5}	2.5×10^{-5}	2.5×10^{-5}	2.5×10^{-5}	2.5×10^{-5}
MTBE						
Lifetime	Upper	3.6×10^{-6}	2.5×10^{-6}	0.0	0.0	0.0
Cancer Risk	Lower	3.3×10^{-6}	2.3×10^{-6}	0.0	0.0	0.0

6.2. Risk Characterization for Other Compounds of Concern:

Toluene, Xylenes, Isobutene, n-Hexane.

A number of compounds were evaluated for possible adverse health impacts, besides those for which detailed atmospheric model data were developed by the CARB. In particular, the toxicological properties of toluene, xylenes, isobutene and n-hexane were considered since they have been identified as substantial fuel components. CARB provided measured or modeled concentrations of these compounds in the South Coast airshed for the year 1997during which MTBE-containing fuel was used.

In each of these cases the annual average concentrations found or estimated were substantially below the chronic REL or other health protective concentrations, with a projected HQ of 0.1 or less (see Table 6 for details). None of these materials is a suspected human carcinogen. For acute exposures to toluene, m/p-xylene and o-xylene, the one-hour and 24-hour peak concentrations were also substantially below the corresponding health risk values. No acute health protective concentrations have been determined for isobutene and n-hexane, but since the predicted concentrations for acute episodes did not exceed the chronic protective levels for these compounds no adverse acute health consequences are anticipated.

The emissions data and atmospheric model outputs for these compounds were not developed using the 2003 scenario. However, it was anticipated that there would be little or no change between the different fuel types, and a modest improvement relative to the 1997 (MTBE) scenario. Since no plausible variations in the model output would alter the conclusion that these compounds present no significant risk, it was not considered necessary to predict the 2003 outcomes in greater detail.

Table 6. Comparison of Estimated Maximum Pollutant Levels in California (based on South Coast Air Basin Data for 1996-1997) and Health Assessment Values

	Toluene (ppbV)	m/p-Xylene (ppbV)	o-Xylene (ppbV)	Isobutene (ppbV)	n-Hexane (ppbV)
Annual Average *					
Maximum Measured Value **	5.1	2.2	0.77	---	---
Projected Range of Maximum based on CO levels	5.6-11.4	2.8-4.7	1.0 - 4.4	2.2 - 3.9	1.2 - 2.6
Chronic REL	100	170	170	1100	60
Maximum 1 hour Average					
Extrapolated from Measured 24-Hour Maximum **	29.7	14.3	5.5	---	---
Projected Range of Maximum based on CO levels	51 - 103	25 - 45	8.8 - 40	22 - 35	11 - 23
Acute REL	9800 (6h)	5000 (0.5h)	5000 (0.5h)	NA	NA

* Overall Statewide Population-Weighted Annual Exposure typically would be between ½ and ¾ of the Maximum
** There is currently some uncertainty in measurement techniques; actual values may be higher.

6.3. Cumulative Cancer Impact of Multiple Chemical Exposures

The cumulative impact due to exposure to multiple cancer-causing chemicals is determined by the addition of all the corresponding lifetime risks of the chemicals involved. The lifetime risk is expressed as the estimated excess risk that results from lifetime exposure (i.e. 70 years) to a specific air concentration of a cancer-causing chemical. Unlike the cumulative impact methodology for noncancer toxicological endpoints, the lifetime risk from exposure to multiple cancer-causing chemicals is assumed to be additive regardless of the toxicological endpoint or target organ. This is because chemically-induced cancer is considered predominantly a non-threshold event that is irreversible once initiated and because the target tissue may vary from species to species.

Table 7 displays lifetime cancer risk estimates based on the predicted exposure concentration of each chemical, and the aggregate lifetime risk attributable to exposures to all these chemicals, for each fuel scenario in the South Coast Air Basin. Risk estimates were calculated using the upper and lower atmospheric concentration estimates provided by CARB. These exposure estimates are best estimates of the population-weighted annual average exposures, with variations in model assumptions as described earlier by CARB. The last row displays the estimated range of excess cancer cases per million people that can be expected from lifetime exposure to the aggregate of cancer-causing pollutants.

Comparison among the individual pollutants shows that, regardless of the fuel scenario, benzene and butadiene are the major contributors to excess cancers due to airborne exposure to cancer-causing pollutants in the South Coast Air Basin. The contribution of excess cancer cases by acetaldehyde and MTBE by comparison is relatively minor.

Table 7. Lifetime Cancer Risk and Cumulative Cancer Risk For Each of the Five Fuel Scenarios.

Chemical		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde	Upper	8.6 E-6	7.2 E-6	7.6 E-6	8.6 E-6	7.2 E-6
	Lower	8.6 E-6	7.2 E-6	7.2 E-6	8.1 E-6	7.2 E-6
Benzene	Upper	1.1 E-4	8.1 E-5	8.0 E-5	8.2 E-5	7.9 E-5
	Lower	1.0 E-4	7.3 E-5	6.7 E-5	6.8 E-5	7.1 E-5
Butadiene	Upper	1.3 E-4	7.4 E-5	7.8 E-5	7.4 E-5	7.8 E-5
	Lower	1.3 E-4	7.0 E-5	7.0 E-5	7.0 E-5	7.4 E-5
Formaldehyde	Upper	3.3 E-5	2.6 E-5	2.6 E-5	2.6 E-5	2.6 E-5
	Lower	3.3 E-5	2.5 E-5	2.5 E-5	2.5 E-5	2.5 E-5
MTBE	Upper	3.6 E-6	2.5 E-6	0	0	0
	Lower	3.3 E-6	2.3 E-6	0	0	0
Cumulative Lifetime Risk	**Upper**	**2.9 E-4**	**1.9 E-4**	**1.9 E-4**	**1.9 E-4**	**1.9 E-4**
	Lower	**2.7 E-4**	**1.8 E-4**	**1.7 E-4**	**1.7 E-4**	**1.8 E-4**
Excess Cancer Cases Per Million Individuals	**Upper**	**290**	**190**	**190**	**190**	**190**
	Lower	**270**	**180**	**170**	**170**	**180**

Comparing the1997 fuel scenario to the year 2003 fuel scenarios, the upper bound estimate of the excess cancer cases per million individuals is expected to drop from 290 to 190 excess cancer cases by 2003. There is no apparent difference among the year 2003 fuel scenarios regarding cancer risk. The conclusion that can be drawn from the cumulative exposure to airborne cancer causing pollutants in the South Coast Air Basin is that the reduction in excess cancers, from the 1997 fuel scenario to the 2003 fuel scenarios, results from expected reductions in overall emissions, rather than a reduction in cancer causing emissions due to the use of any one fuel scenario.

An inherent uncertainty resulting from addition of multiple lifetime cancer risks is that this may underestimate the cancer risk in cases where the interactions are synergistic, or overestimate the cancer risk in cases where the interactions are not additive or are antagonistic. Also, the aggregate risk prediction may exaggerate the confidence bounds on the estimate, since it is obtained by adding individual upper 95 percent confidence limits on the contributing risks. Since it is not known how the various risks and the uncertainties in their estimates interact, it has not been possible to allow for this effect.

The sources of uncertainty that are incorporated into the estimate of lifetime cancer risks, such as reliance on animal data and extrapolation from experimental exposure concentrations to ambient exposure concentrations, imply that the real aggregate risk may in fact be lower than the upper bound estimates. On the other hand, the exposure concentrations provided by CARB's model that serve as the basis for the cancer calculations are population-weighted annual averages. Certain individuals or communities located in areas where pollutant emissions are concentrated (such as those near freeways, or fuel storage and handling facilities) may experience greater increments in risk from some fuel-related pollutants, whereas the impacts in other areas may be less. As noted above, there is more confidence in the relative differences between fuels than the absolute magnitude of the risk faced by the exposed population under the various scenarios considered. Therefore, comparison of the aggregate cancer risks among the five fuel scenarios gives a reasonably good indicator of the relative impact of each fuel on the cancer risk from airborne pollutants in the South Coast Air Basin.

6.4. Cumulative Noncancer Impact of Multiple Chemical Exposures

Exposure to a single chemical in the air will likely not result in a toxic response if it is below the threshold necessary to elicit a response. However, simultaneous exposure to two similar chemicals at sub-threshold levels may result in a toxic response. Under the noncancer cumulative impact methodology, the combined impact of several chemicals present at the same time are assessed by assuming the interaction of the chemicals will be additive for a given toxicological endpoint, unless information is available to the contrary.

The cumulative impact is determined by simply adding the HQs for chemicals that impact the same target organ or system. If either the HQ for an individual chemical, or the cumulative hazard index (HI) for a particular toxicological endpoint exceeds one, the margin of safety implicit in the REL is eroded. As noted in the introduction, this does not automatically imply that adverse health effects will occur. Rather, it indicates that there is an increasing possibility that more sensitive individuals may be affected. For the airborne pollutants of concern in the South Coast Air Basin, cumulative HIs for a given toxicological endpoint can be determined under each fuel scenario and for each predicted exposure period (maximum one hour average and maximum population-weighted annual exposure).

For the maximum one-hour average exposure, the cumulative toxicological endpoints of concern are eye irritation and respiratory irritation. Table 8 displays the individual HQs for each chemical where eye irritation is a primary toxicological endpoint. PAN, ozone, and nitrogen dioxide are the major pollutants that cause the eye irritant effects. Under the cumulative impact methodology, sub-threshold pollutants such as acetaldehyde and formaldehyde may also participate by exacerbating the eye irritation primarily due to PAN, ozone, and nitrogen dioxide.

For acute respiratory irritation, ozone and nitrogen dioxide are the major pollutants of concern. Acetaldehyde may also exacerbate the respiratory irritation caused by ozone and nitrogen dioxide (Table 9). The ethanol and MTBE contribution to both eye and respiratory irritation tend to be so small that these pollutants are not likely to have a significant impact on these toxicological endpoints.

The primary pollutants involved in chronic respiratory irritation are formaldehyde and PM_{10}, both of which are individually above the threshold for this toxicological endpoint (Table 10). A limitation in using the cumulative impact approach for pollutants that cause either acute or chronic respiratory irritation is that their primary site of action within the respiratory system may differ. For example, formaldehyde is known to produce nasal and upper respiratory irritation while PM_{10} produces inflammation principally in the lower airways. Therefore, the cumulative impacts for these two pollutants may be less than additive.

Table 8. Maximum Acute Hazard Quotients (HQ) and Cumulative Acute Hazard Indices (HI) For Eye Irritation For Each of the Five Fuel Scenarios

Chemical		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde	Upper	0.3	0.3	0.3	0.3	0.3
	Lower	0.2	0.2	0.2	0.2	0.2
Ethanol	Upper	0.002	0.002	0.002	0.002	0.002
	Lower	0.001	0.001	0.001	0.001	0.001
Formaldehyde	Upper	0.5	0.5	0.5	0.5	0.5
	Lower	0.3	0.3	0.3	0.3	0.3
MTBE	Upper	0.01	0.007	0	0	0
	Lower	0.003	0.002	0	0	0
PAN	Model	2.4	2.3	2.4	2.4	2.4
Nitrogen dioxide	Best	1.0	0.9	0.9	0.9	0.9
Ozone	Best	2.7	2.6	2.6	2.5	2.6
Cumulative HI	**Upper**	**6.9**	**6.6**	**6.7**	**6.6**	**6.7**
	Lower	**6.6**	**6.3**	**6.4**	**6.3**	**6.4**

Table 9. Maximum Acute Hazard Quotients (HQ) and Cumulative Acute Hazard Indices (HI) for Respiratory Irritation For Each of the Five Fuel Scenarios

Chemical		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde	Upper	0.3	0.3	0.3	0.3	0.3
	Lower	0.2	0.2	0.2	0.2	0.2
Ethanol	Upper	0.002	0.002	0.002	0.002	0.002
	Lower	0.001	0.001	0.001	0.001	0.001
MTBE	Upper	0.01	0.007	0	0	0
	Lower	0.003	0.002	0	0	0
Nitrogen dioxide	Best	1.0	0.9	0.9	0.9	0.9
Ozone	Best	2.7	2.6	2.6	2.5	2.6
Cumulative HI	**Upper**	**4.0**	**3.8**	**3.8**	**3.7**	**3.8**
	Lower	**3.9**	**3.7**	**3.7**	**3.6**	**3.7**

Table 10. Maximum Chronic Hazard Quotients (HQ) and Cumulative Chronic Hazard Indices (HI) for Respiratory Irritation for Each of the Five Fuel Scenarios

Chemical		1997 MTBE	2003 MTBE	2003 Et2%	2003 Et3.5%	2003 NonOxy
Acetaldehyde	Upper	0.4	0.3	0.3	0.4	0.3
	Lower	0.4	0.3	0.3	0.3	0.3
Ethanol	Upper	0.0001	0.0001	0.0002	0.0002	0.0001
	Lower	0.0001	0.0001	0.0001	0.0002	0.0001
Formaldehyde	Upper	2.4	1.9	1.9	1.9	1.9
	Lower	2.4	1.8	1.8	1.8	1.8
Nitrogen dioxide	Best	0.8	0.8	0.8	0.8	0.8
PM_{10}	Best	1.9	1.9	1.9	1.9	1.9
Cumulative HI	**Upper**	**5.5**	**4.9**	**4.9**	**5.0**	**4.9**
	Lower	**5.5**	**4.8**	**4.8**	**4.8**	**4.8**

Other limitations may exist for determining the cumulative toxicological impacts of airborne pollutants. Combining HIs may underestimate the effect in the cases where interactions on a given target organ are synergistic, or overestimate the effect in the cases in which interactions are not additive or are antagonistic.

A limitation concerning the acute HIs is that the peak one-hour airborne concentrations for each of the chemicals may not have occurred in the same hour. However, given that the one-hour maximum average is a worst case scenario for an episodic event, it is appropriate to assume for the purposes of the cumulative impact analysis that peak one-hour concentrations occur during the same time period.

The modeling conducted by CARB to evaluate the differences in air quality impacts from using the various fuel formulations provided the concentrations in air that we have used in this analysis. The concentrations for acute exposures (e.g., 1, 8, 24 hour averages) reflect a scenario with relatively adverse meteorological conditions. In addition, the model is based on the South

Coast Airshed, in which pollutant concentrations from vehicular and other sources are typically somewhat higher than in some other areas of the State of California. Therefore, the absolute values of the annual average concentrations may not reflect an average in other parts of the State. There is more confidence assigned to the relative values of the concentrations representing the various fuel usage scenarios.

Given the above limitations in the cumulative impact methodology, as well as uncertainties in modeled exposure and toxicological risk methodology, the differences in the cumulative noncancer impacts among the year 2003 fuel scenarios are not significant enough to warrant a recommendation for any one fuel based solely on airborne exposure to eye and respiratory irritants.

6.5. Health Impacts of Drinking Water Contamination by Gasoline Components

Health protective concentrations for drinking water for various components of gasoline are shown in Table 4. Values for formaldehyde and tertiary butyl alcohol (TBA), which are probable breakdown products of MTBE, are also included.

The compounds of greatest concern from the point of view of potential low-level contamination of drinking water are benzene (a known human carcinogen), and MTBE. MTBE is a suspected carcinogen, and also has highly objectionable taste and odor which render drinking water unpalatable even at very low concentrations. Its breakdown product, TBA, is also a suspected carcinogen and has similarly objectionable organoleptic properties. Other compounds for which some adverse health effects might be anticipated are toluene, xylene, formaldehyde, and various aliphatic hydrocarbons. These are not considered carcinogenic by the oral route, but higher concentrations are toxic, and some may also adversely effect taste and odor. Ethanol, and

its oxidation products such as acetaldehyde, are toxic only at very high levels, and are also very rapidly biodegraded, so in general these are not expected to present major long-term contamination problems.

Contamination of ground and surface waters by gasoline components, as a result of leakage, spills and transportation accidents is an established fact, and likely to continue in spite of efforts to prevent such occurrences. However, the organizations responsible for providing public drinking water supplies have monitoring and control measures in place for contaminants with potentially adverse impacts on public health. OEHHA has been advised by the State Water Resources Control Board (SWRCB) that it is the policy of the California Department of Health Services' (DHS) Drinking Water Program to avoid contamination of any public water supply by gasoline components in excess of the health protective levels. This may include closing down wells or surface water sources that show signs of contamination, . They also monitor the movement of known plumes from gasoline spills and leaks. These measures are intended by the SWRCB to ensure that public drinking water supplies remain free of contamination by gasoline components, and thus prevent adverse public health consequences for consumers of the public drinking water supply. This would be the case either with continued use of MTBE, or with its replacement by ethanol or non-oxygenated gasoline. Evidently, there may be extensive economic consequences and resource availability problems if well closures are widespread. These consequences, and their differential impact in scenarios with use of different fuel compositions, are the subject of a separate report by SWRCB.

There is a possible concern for public health impacts of different fuel formulations for those using private wells or other sources of drinking water not subject to the monitoring and regulatory oversight of the DHS Drinking Water Program. OEHHA has so far been unable to

determine the number and location of such sources that may be threatened by gasoline spills, or the number of people using them as their drinking water sources. There appears also to be little quantitative information on the differential impact of alternative fuel formulations on contamination levels in affected wells. It has not therefore been possible to provide a quantitative risk assessment for this situation. Qualitatively, it would appear that MTBE is already a problem for groundwater users, and its removal would be an unqualified benefit. Direct effects of ethanol would appear to be minimal even in cases of severe contamination, although the adverse consequences of contamination by the hydrocarbon fraction of the gasoline would remain. Research is currently being undertaken to determine whether any secondary effects of ethanol, such as enhancement of migration through soil, or acceleration or inhibition of biodegradation, would alter the concentrations of compounds of concern (such as benzene) in impacted wells.

No reports of systemic investigations of the effects of oral exposure to very low levels of ethanol, such as might be anticipated if groundwater contamination were to occur, were identified in the literature. It is known that ethanol is rapidly biodegraded, and expected exposures are low. The issue is the subject of more detailed investigations currently being undertaken for the SWRCB. Since the results of this investigation are not yet available, as a preliminary approach the CalTox model was used for a typical situation of contaminated soil, with literature values for ethanol degradation, etc. Starting with 10,000 ppm in soil and 5,000 ppm in the vadose zone, the predicted values in ground water and tap water at one year were 2.3×10^{-6} and 1.9×10^{-6} ppm, respectively. Human exposure was 8% by inhalation, 91% by oral and 1% by dermal routes. As an extreme worst-case scenario, the values for half-lives of ethanol in soil and water were increased by an order of magnitude and the exposure time shortened to

begin at 50 days instead of one year. In this case, the water concentrations were 0.24 and 0.20 ppm, respectively. Human exposure via tap water was assumed to be 10% by inhalation, 89% by oral, and 1% by dermal routes. These concentrations are well below the health protective concentrations developed in the Appendix and are also well below concentrations normally found in foods.

6.6 Uncertainties and Data Gaps

6.6.1 Uncertainties in Dose-Response Assessment

Risk assessment involves a number of assumptions. Due to data limitations, it is not possible to ascertain all the uncertainties inherent in any cancer potency or unit risk factor. As a result of a number of uncertainties (e.g., in the cases where human data were inadequate for risk assessment, applicability of animal data to humans, variability in response in the general population, presence of susceptible subpopulations, etc), the unit risk factor represents generally the 95% upper confidence limit of the slope of the dose-response curve. As such it may be considered a high-end estimate of the risks. The RELs for non-carcinogenic effects also have similar associated uncertainties. In developing RELs, uncertainty factors are applied to animal or human data to arrive at a concentration of the chemical in question that we are reasonably confident will not be associated with adverse health effects from long-term exposure. Thus, there is a built in margin for health-protection in the REL. These types of uncertainties are not readily quantifiable and in some instances, not quantifiable at all.

Most of the health values (unit risk factors, cancer potency factors, reference exposure levels) used in the evaluation were peer-reviewed numbers currently in use in a number of programs. In several instances, there was no available regulatory number. For example, there

was no acute REL for butadiene, and OEHHA has provided an interim value for a one hour averaging time. Likewise, there are no regulatory values for ethanol in either air or water. OEHHA has evaluated secondary literature and the key primary studies to develop interim values for use in this assessment as reference exposure levels in air and water.

A similar situation exists for other important chemicals of interest. The atmospheric transformation product, peroxyacetyl nitrate or PAN, is an irritant gas due to its oxidant properties. Despite the fact that PAN was identified as an irritating component of smog decades ago, no peer-reviewed regulatory numbers exist to use in a health effects assessment. We developed interim health protective concentrations for PAN for one hour and annual averaging times. A related compound, peroxyproprionyl nitrate or PPN, is also an irritant gas formed via atmospheric reactions. However, we could not locate adequate toxicity information on this chemical and so have not included formation of PPN in this evaluation.

Other chemicals of interest lack key toxicity data. Nonoxygenate formulas of gasoline will likely have increased levels of alkylates relative to fuels with an oxygenate including 2-methylpentane, 3-methylpentane, methylcyclopentane, 2,3-dimethylpentane, 2,4-dimethylpentane, 2,2,4-trimethylpentane, 2,3,4-trimethylpentane, 2-methylhexane, and 3-methylhexane. These branched chain hydrocarbons function much the same as an oxygenate by increasing the efficiency of combustion. There is almost no toxicological data on these compounds. We therefore are unable to estimate potential public health risks from increasing the concentrations of these motor fuel components in the non-oxygenate fuels. However, the concentrations of these compounds modeled for the South Coast Air Basin for existing fuel speciation profiles are in the low ppb range as an annual average and in the tens of ppb range as one-hour peaks. Thus the alkylates would need to be fairly potent toxicants (e.g., be about

equivalent to the cancer potency of benzene, have much greater acute noncancer toxic potential than benzene, and have greater chronic toxic potential than benzene) in order for these concentrations to be of concern. Our inability to estimate public health impacts of alkylates due to the almost complete lack of toxicological data is an uncertainty in this evaluation.

Finally, this evaluation focused on the key differences resulting from use of four different fuels. We used available evidence to decide on which chemicals are important in assessing the air quality impacts differences. Although we believe we have focused on the key primary and secondary pollutants that impact air quality as a result of fuel usage, there is a slight possibility that the air quality impacts analysis from use of different fuels omitted a significant chemical.

6.6.2 Uncertainties in Exposure Assessment

The concentrations in air modeled by the CARB modeling effort also have inherent uncertainties. The uncertainty in speciation profiles of the various VOCs in the different fuels carries into the modeling results. The modeled concentrations are thus subject to uncertainties due to potential inaccuracies in the species profiles as well as inherent model uncertainties. It is evident from comparing the 1997 and 2003 scenarios with the same MTBE-containing fuel that the predicted emissions inventory has a substantial dependence on the expected numbers and types of vehicles. Since the types of vehicles in service, and the mileage driven, in 2003 are estimates with a significant level of uncertainty, this uncertainty will carry through to the exposure estimates for key pollutants. As in the dose-response assessment, modeling uncertainties may be difficult to quantify. However, they are likely considerably less than the uncertainties inherent in dose-response assessment. All of the models used by CARB in this exercise have had some validation studies to characterize the accuracy of the models and guide

their improvement. Whatever uncertainty exists in the assessment of exposure carries into the assessment of risk.

Aside from the air modeling, there are exposure assumptions implicit in some of the health values used in assessing risk. For instance, the unit risk factors and some of the reference exposure levels generally assume that the average 70 kg person breathes 20 cubic meters per day. A recent analysis of breathing rate distribution conducted by OEHHA under the Air Toxics Hot Spots program indicates that the value of 20 cubic meters for a 70 kg person is about the 85th percentile of the distribution of breathing rates. As such it represents an above-average breathing rate. On the other hand, if the basis of the unit risk factor is a human inhalation study, this assumption results in a lower estimate of the potency if in fact the subjects were breathing less air and thus less chemical to produce the observed effect.

There is a very considerable degree of uncertainty over the level of exposure to fuel components and breakdown products occurring as water contaminants. A primary concern is exposure via drinking water. (In scenarios involving water as the pathway of exposure, it is generally assumed that people consume about two liters of water per day. For compounds that are volatile, they have inhalation exposures equivalent to drinking at least another liter by virtue of household water use.) We have been unable to perform a quantitative analysis of the risk from drinking water since estimates of contamination of the sources most likely to be affected, *i.e.* private wells, are not available. It isassumed that the DHS Drinking Water Program's regulatory and monitoring activities are sufficient to prevent actual delivery of contaminated water via public distribution systems, in which case there will not be health impacts from this source. We do not have access to a quantitative evaluation or failure analysis for this

expectation. Some of these uncertainties may be addressed by research currently being undertaken by the SWRCB.

There may be scenarios for contamination of water that are low probability and we have not evaluated those impacts. For example, since ethanol will be transported by truck, train, or barge, the possibility exists that a transportation accident might contaminate a surface-water drinking water supply. However, while the aquatic life immediately at the site of the spill might be affected by the ethanol, it is unlikely that such a scenario would impact public health due to the biodegradability and relatively low toxicity of ethanol. Similar accidents might also occur with a vessel transporting already-blended fuel. However, it does not appear useful to focus on that scenario for the comparative evaluation of ethanol-containing gasoline with other fuels since other components of fuels of interest (e.g. benzene, toluene, hexane, xylenes) are more toxic and more slowly degraded in the environment than ethanol and would become water contaminants in a blended-fuel spill into surface water.

Finally, it is not yet determined which denaturants will be used to denature the ethanol, as required by law. Initial proposals include the use of naphtha or similar gasoline-like materials, so these are unlikely to have a substantial effect on the health impacts of the combined fuel. However, since both the actual composition of such additives and their toxicological properties are unknown to us at present, we have not evaluated potential health risks of denaturants in the ethanol used for gasohol.

7.0. RESEARCH NEEDS

As noted earlier in this report there are several issues which cannot be addressed, or for which our assessment is subject to very substantial uncertainties, due to lack of information

which would be required to better define expected risks. Whereas some of the uncertainties are intrinsic to the current process of risk assessment, some could be substantially reduced by further research. Potential areas for further research may be identified related to both the toxicological properties of presently identified pollutants, and the assessment of exposure to these materials (and perhaps to others as yet unidentified). These are summarized in Tables 11 and 12.

Table 11. Research Needs for More Complete Understanding of the Potential Health Effects of Ethanol in Gasoline.

Basic Toxicologic Information Needed:

alkylates, including but not limited to

2-methyl pentane	2,2,4-trimethylpentane
3-methylpentane	2,3,4-trimethylpentane
methylcyclopentane	2-methylhexane
2,3-dimethylpentane	3-methylhexane
2,4-dimethylpentane	

ethanol (at low concentrations)

isobutene

peroxyacetyl nitrate (PAN)

peroxyproprionyl nitrate (PPN)

Development of Health Assessment Values Needed:

acetaldehyde (acute exposures)

butadiene (acute exposures)

ethanol (acute and chronic exposures)

MTBE (acute exposures)

PAN (acute and chronic exposures)

PPN (acute and chronic exposures)

Table 12. Key Issues to be Resolved in Order to Further Our Understanding of the Potential Health Effects of Ethanol in Gasoline

Water contamination issues:

- what are the gasoline breakdown products?
- what is the likelihood of contamination of public / private wells?
- what are the impacts of transportation accidents?
- what are the impacts of watercraft use?

General risk assessment issues:

- what denaturants will be used in the new formulations?
- what are the risks posed by, and interactions of, complex mixtures associated with motor fuels?
- need to conduct life-cycle analysis to determine overall exposure from production, use and disposal of motor fuels; this will include air emissions as well as contamination of water and soil
- need more information on localized 'hot spots'
- address remaining uncertainties in emissions and atmospheric chemistry

The components of existing and proposed gasoline formulations include several compounds for which there is relatively little toxicological information available. This applies particularly to the branched-chain alkanes and alkenes classified as “alkylates”. Whereas these compounds occur to some extent in all of the fuel formulations considered in this report, they are specifically increased in the proposed non-oxygenated RFG3 fuel. There appears to be only very limited acute toxicological information on a few of these compounds, and none at all on many. Further investigation of those specific chemicals identified as major “alkylate” components of the new fuels is warranted, to include investigation of both short-term and long-term effects. Studies need to be performed on specific isomers, rather than on generic fractions such as pentanes, hexanes, octanes etc., because the toxicological properties of different isomers may differ substantially. There are some reports on the toxicological properties of generic mixtures, including previous formulations of unleaded gasoline (U.S. EPA, 1987). While these have assisted in quantifying the risk from fuels in general, they do not provide sufficient information on individual components to allow analysis of the differential impact of alternative fuel formulations.

In spite of the very large literature on the effects of consumption of alcoholic beverages, there is also a surprising lack of information on the toxicity of ethanol by inhalation, and on the effects of low level oral ingestion. This may reflect a consensus that ethanol occurs in many foods and the toxicity of ethanol is not considered a substantial problem under these circumstances. Nevertheless, it would be preferable to have more complete studies of acute and chronic effects, performed according to modern experimental design principles.

The toxicological information available on the photochemical reaction product PAN is limited. Although there are data on acute effects there has been no evaluation for carcinogenic

effects. Some genotoxic effects has been observed, and studies of up to six months duration identified squamous metaplasia in the respiratory tract of mice. While these latter findings were not considered evidence of neoplasia, they do raise the concern for possible carcinogenicity of this compound, suggesting a more exhaustive investigation using a lifetime bioassay protocol would be desirable. We were unable to locate any toxicological information on the related compound PPN. Since both these photochemical reaction products appear to have the potential for significant health impacts after both acute and chronic exposures, more information on their effects would be highly desirable.

Actual exposures to chemicals associated with fuels most commonly occur as exposures to complex chemical mixtures, rather than to isolated chemicals; thus, it is important that the health effects of interactions between individual components of these mixtures be characterized, in addition to the health effects of the individual components. There are presently large gaps in our knowledge of the health effects of exposures to complex mixtures associated with gasoline, as well as the health effects associated with some individual compounds. There is need to conduct original research and to further develop and evaluate existing epidemiological data on the human health effects from complex mixtures associated with fuel components.

Additional research is also needed to address uncertainties in the exposure assessment. While the emissions and atmospheric chemistry have already been the subjects of extensive study, and a sophisticated model is available, significant uncertainties remain. It is important that monitoring of the actual atmospheric pollutant levels be continued, to observe the outcome of changes in vehicle type and usage and fuel composition, and thereby to confirm the accuracy of the model predictions.

It is also evident that at present our knowledge of the possible exposures via drinking water is limited. This needs to be augmented in several respects. An analysis of the likelihood of contamination of the public drinking water supply in spite of the regulatory and monitoring efforts in place to avoid this should be undertaken. This needs to reflect the number of sources potentially affected, the frequency of monitoring of these sources and the size of the potentially impacted populations. This analysis also needs to provide indications of the possible concentration ranges and duration of exposures that might arise in the event of such a failure of the control measures. An equally pressing concern is the lack of information of the likelihood and severity of exposures to gasoline components due to contamination of private wells at risk from contamination.

Life-cycle analysis integrates the multi-media risks associated with production, use and disposal of substances. This is a resource intensive proposition; none-the-less it should be attempted for reformulated fuels. Life-cycle analysis would look at such issues as contamination of the environment from production, transportation, use, dissemination (e.g., at gasoline stations) and disposal. It is understood that work is at present being undertaken by SWRCB to address the likelihood of contamination from watercraft engine emissions, leaks, spills and transportation accidents. The conclusions of these current efforts are clearly important. In addition, there is a need to investigate the concentrations of pollutants (gasoline components and their breakdown products) that might occur in drinking water as a result of such events. While it may not be possible to predict actual outcomes from likely sources of contamination, it would be useful to have some information as to the severity of plausible incidents.

8.0 SUMMARY / CONCLUSIONS

Predicted levels of atmospheric pollutants for different fuel composition scenarios were provided by the CARB. Fuel compositions represented currently available MTBE-containing oxygenated fuel, two formulations of ethanol-containing fuel with either 2 or 3.5 percent oxygen content, and a non-oxygenated fuel formulated to comply with the proposed RFG3 requirements. These were considered in exposure scenarios based on the predicted emissions inventory for the year 2003. A scenario of the 1997 emissions inventory and MTBE-containing fuel was used to calibrate the model against actual measured data for that year.

Health protective concentrations in air and water for compounds of concern in the gasoline formulations, primary exhaust emissions or transformation products were selected from current California or United States regulatory standards where these were available. In the absence of suitable regulatory levels, draft levels currently under development for California programs were used, or else draft health protective levels were developed for this report using standard methodology.

The health protective levels for air contaminants were compared to the model predictions for these compounds provided by CARB, and risk characterizations developed for individual compound impacts. Risk characterizations of the cumulative impacts of carcinogens and irritant compounds were also developed.

It appears that there are no substantial differences in the public health impacts of the different non-MTBE fuel formulations considered in the scenarios for the year 2003. MTBE-containing fuels still pose a risk to water resources due to the high water solubility coupled with slow environmental degradation of MTBE. For all of these fuels the concentrations of irritants (including both air toxics and criteria pollutants) may achieve levels at which the safety margins

for short-term and long-term exposures are reduced. At these levels, adverse health effects are not necessarily expected, but more sensitive members of the population may be affected. The 2003 scenarios are based on relatively adverse meteorological conditions in a region (the South Coast Airshed) of California severely impacted by vehicle-generated pollution, so effects in other parts of the State and under different meteorological conditions will likely be less severe. Due to the reduction in overall emissions, all the scenarios for year 2003 show a significant improvement over the predicted averages for 1997. The pattern for airborne carcinogens is similar in that the overall estimated risks do not differ between fuel formulations, but show some improvement for 2003 relative to 1997. The absolute values of the risk estimates are not regarded as reliable indicators of the actual risks faced by the population in the South Coast region, but are regarded as useful in indicating the relative impacts of the different scenarios.

Due to the lack of quantitative information on possible public exposures as a result of fuel-related groundwater, surface water or drinking water pollution, it was not possible to provide risk estimates for public health impacts of water contamination. However, consideration of the relative toxicity of ethanol, MTBE and their degradation products suggests that the direct effects of ethanol (if any public exposure were to occur) would be substantially less severe than the effects of MTBE. Secondary effects, including alterations in distribution and biodegradation of other fuel components, are currently being evaluated by the SWRCB. They are also examining the possible impacts of various contamination scenarios, such as spills, leakage, transportation accidents and the use of gasoline-powered watercraft, on water contamination by fuel components. Further analysis of the relative effects on water of the different fuel formulations may be possible once these studies are complete.

Our analysis of the health effects of ethanol in gasoline is dependent on the modeled concentrations provided by the CARB. As CARB updates their atmospheric concentration estimates, this report will be updated to reflect any possible new findings and conclusions.

9.0 REFERENCES

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