

**BLUE RIBBON PANEL FINDINGS ON MTBE**

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**HEARING**

BEFORE THE

SUBCOMMITTEE ON CLEAN AIR, WETLANDS,  
PRIVATE PROPERTY, AND NUCLEAR SAFETY

COMMITTEE ON  
ENVIRONMENT AND PUBLIC WORKS  
UNITED STATES SENATE

ONE HUNDRED SIXTH CONGRESS

FIRST SESSION

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OCTOBER 5, 1999  
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# C O N T E N T S

	Page
<b>OCTOBER 5, 1999</b>	
OPENING STATEMENTS	
Bennett, Hon. Robert F., U.S. Senator from the State of Utah .....	10
Boxer, Hon. Barbara, U.S. Senator from the State of California .....	3
Chafee, Hon. John H., U.S. Senator from the State of Rhode Island .....	2
Inhofe, Hon. James M., U.S. Senator from the State of Oklahoma .....	1
Lieberman, Hon. Joseph I., U.S. Senator from the State of Connecticut .....	39
Voinovich, Hon. George V., U.S. Senator from the State of Ohio .....	20
WITNESSES	
Campbell, Robert H., chairman and chief executive officer, Sunoco, Inc. ....	28
Letter, to Senators Inhofe and Graham .....	55
Prepared statement .....	53
Responses to additional questions from Senator Inhofe .....	56
Summary, Blue Ribbon Panel Recommendations on MTBE .....	41-47
Garn, Hon. Jake, vice chairman, Huntsman Corporation, Salt Lake City, UT .....	25
Prepared statement .....	71
Greenbaum, Daniel S., president, Health Effects Institute, Cambridge, MA, and former chair, Blue Ribbon Panel on the Use of Oxygenates in Gasoline ..	11
Prepared statement .....	40-48
Responses to additional questions from:	
Senator Boxer .....	50
Senator Inhofe .....	48
Senator Lieberman .....	52
Kenny, Michael P., executive officer, California Air Resources Board, Sacramento, CA .....	26
Letters:	
To EPA Assistant Administrator, Robert Perciasepe .....	63
To EPA Director of Mobile Sources, Margo Oge .....	70
Prepared statement .....	57
Responses to additional questions from Senator Inhofe .....	59
ADDITIONAL MATERIAL	
Articles:	
MTBE Groundwater Impacts in South Lake Tahoe, CA .....	87
Former Senator Garn Tells Senate Panel to Save Fuel Additive MTBE ....	85
Comments, National Research Council Report .....	77-81
Letters:	
To Margo T. Oge, Director, Office of Mobile Sources, from Michael P. Kenny .....	70
To Senators James M. Inhofe and Bob Graham, from Robert H. Campbell .....	55
To EPA Assistant Administrator Robert Perciasepe from Michael P. Kenny .....	63-69
To Senator Bob Graham from Robert Baer .....	87
Report, Public Health Goal for Methyl Tertiary Butyl Ether (MTBE) in Drinking Water .....	88-177

IV

	Page
Statements:	
Association of Metropolitan Water Agencies and American Water Works Association .....	81
Bergson, Ivo, South Tahoe Public Utility District .....	87
Hall, Steve, executive director of the Association of California Water Agencies .....	82
Lyondell Chemical Company—Summary of Dissenting Report .....	48
Santa Clara Valley Water District .....	85
Summary, The Blue Ribbon Panel on Oxygenates in Gasoline—Executive Summary and Recommendations .....	41–47

## **BLUE RIBBON PANEL FINDINGS ON MTBE**

**TUESDAY, OCTOBER 5, 1999**

U.S. SENATE,  
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,  
SUBCOMMITTEE ON CLEAN AIR, WETLANDS, PRIVATE  
PROPERTY, AND NUCLEAR SAFETY,  
*Washington, DC.*

The subcommittee met, pursuant to notice, at 9:30 a.m., in room 406, Senate Dirksen Building, Hon. James N. Inhofe (chairman of the subcommittee) presiding.

Present: Senators Inhofe, Bennett, Voinovich, Boxer, and Chafee [ex officio].

### **OPENING STATEMENT OF HON. JAMES M. INHOFE, U.S. SENATOR FROM THE STATE OF OKLAHOMA**

Senator INHOFE. The subcommittee will come to order.

At today's hearing we are going to examine the recommendations of the Environmental Protection Agency's Blue Ribbon Panel Advisory Committee on the use of Oxygenates in Gasoline on MTBE.

MTBE is a fuel additive used to add oxygen to gasoline. The Clean Air Act requires reformulated gasoline, RFG, to contain 2 percent oxygen by weight. MTBE is used in over 85 percent of the RFG, and ethanol is the second largest at 8 percent. The requirement for RFG began in 1995, as mandated by the 1990 Clean Air amendments.

In the last few years, MTBE has been found in drinking water sources, and it is my understanding that the great majority of the levels found are well below the public health concerns, although they create a problem with odor and taste. Because of these water-related concerns, the use of MTBE has been questioned. In March 1999, Governor Gray Davis issued an executive order that will eliminate MTBE from California's gasoline by the end of 2002.

There are various legislative options in Congress for dealing with MTBE. These range from an outright ban, to phase-out, to making oxygenates optional.

Over the last 2 years the full committee has held two hearings addressing the concerns in California; today is the first time for our subcommittee to consider the MTBE program nationwide. We will be hearing from members of the Blue Ribbon Panel and other representatives.

The EPA's Blue Ribbon Panel issued their report on July 29, 1999. The findings of the report have, in my opinion, been mischaracterized by both the press and the Senate. It is my under-

standing that the panel's recommendation for an orderly phase-down in the use of MTBE was dependent upon the repeal of the Federal oxygenate mandate. It is my hope that the ultimate goal of today's hearing is for the members of the committee to understand fully what the report says and does not say regarding MTBE.

Recently the Department of Energy identified several areas of concern for the U.S. refining industry, including the uncertainty of the role of oxygenates, particularly MTBE in gasoline. I think it is important that we do not jump to any rash conclusions out of unfounded fear or unjustified claims of fuel alternatives. We should not act in haste on the MTBE issue because the potential impacts to the consumer are significant. The safeguarding of the nationwide supply and distribution of gasoline must be a key consideration in any action that is taken to address MTBE.

I believe that one of the most important lessons to be learned from the current situation is that prescriptive mandates reduce flexibility and may lead to unintended consequences.

There are a number of issues and questions that I would like addressed, both today and in the coming weeks and months.

First, what are the health concerns of MTBE—not the talk, but the real health concerns?

Are there benefits to the air from MTBE or other oxygenates, and are they necessary?

Are there specific negative environmental effects from MTBE?

What is the impact of MTBE and other oxygenates on the fuel supply and delivery system?

Since MTBE was required under the Clean Air Act, if we ban or phase out, should we compensate for stranded cost of investment of the MTBE producers?

And last, what impact will States' efforts to address MTBE have on the gasoline and distribution system?

Senator INHOFE. We have an excellent slate of witnesses today, including the chairman of the Blue Ribbon Panel and former Senator Jake Garn. This is probably not the last time we will address the MTBE or oxygenates issue. We will probably be having a hearing on the environmental effects of ethanol.

Senator Chafee, do you have an opening statement you would like to make?

**OPENING STATEMENT OF HON. JOHN H. CHAFEE,  
U.S. SENATOR FROM THE STATE OF RHODE ISLAND**

Senator CHAFEE. I do.

I am delighted that you are having this hearing, Mr. Chairman.

I want to welcome our witnesses, especially our former colleague from Utah, Senator Jake Garn. It's so nice to see you here once again.

The full committee held a hearing on MTBE last year. Since then, as you mentioned, much has happened. In March, Governor Davis in California ordered the State to phase out MTBE use by the end of 2002.

In July, the Blue Ribbon Panel issued its report on the use of oxygenates in gasoline. Those findings have served to guide the debate about the future of MTBE, the 2 percent mandate, and the problems with leaking underground storage tanks.

In August, the future of MTBE was even debated on the Senate Floor. In that debate I urged the Senate to move forward cautiously, guided by the recommendations of the Blue Ribbon Panel. It is important that we not rush to judgment, as you indicated, Mr. Chairman, or make hasty decisions, but I believe we have to address this problem. The future of the program, the progress we've made on air quality and public confidence in our water supply, all depend upon resolving this question about the use of oxygenates in gasoline.

I want to stress that this is not a "California only" problem. MTBE has been found in water supplies in 26 States, including my State of Rhode Island. Much of it comes from leaking underground storage tanks, which were required by law to be upgraded or closed by December 22, 1998. MTBE contamination of water is most acute in the 17 States that use reformulated gasoline.

Air quality benefits of reformulated gasoline have been substantial. Toxics and ozone-forming compounds have been reduced dramatically.

Last year, in a full committee hearing, I called for the 2 percent mandate to be lifted. I am glad to see that the Blue Ribbon Panel agreed with me in that recent report. We look forward to hearing what they have to say.

I want to thank you again, Mr. Chairman, and I would submit this entire statement for the record.

Senator INHOFE. Thank you, Chairman Chafee.

Senator Boxer.

**OPENING STATEMENT OF HON. BARBARA BOXER,  
U.S. SENATOR FROM THE STATE OF CALIFORNIA**

Senator BOXER. Thank you so much to both my chairmen, my subcommittee chair and the full committee chair. I am really glad to have the opportunity to make some remarks.

It is a very serious issue. I think I have a slightly different view than my friends, and I want to lay it out, if I might.

After I asked Administrator Browner to phase out MTBE, she appointed the Blue Ribbon Panel, and I am very pleased that my reading of the panel's report suggests my view, that we would be better off without MTBE. Specifically, on the question of whether MTBE use should continue, the panel report states:

The panel agreed broadly that in order to minimize current and future threats to drinking water, the use of MTBE should be reduced substantially. Several members believe that the use of MTBE should be phased out completely.

I first formed a view that we should phase out MTBE in 1997, after the city of Santa Monica lost 71 percent of its drinking water supply due to MTBE contamination. And I want to say to both my Chairmen, I can assure you that if one of the cities in your State was faced with that situation, of losing 71 percent of their water supply, I think perhaps you would be closer to my view.

Now, on August 4, 1999, the majority of the U.S. Senate joined with me in expressing the view that we should phase out MTBE. We adopted my Sense of the Senate; it provided that "The United States should phase out MTBE in order to address the threats that MTBE poses to public health and the environment." And I would

like to place the text of this Sense of the Senate into the hearing record, Mr. Chairman, if I might.

Senator INHOFE. Without objection.

Senator BOXER. Thank you.

Senator BOXER. The Sense of the Senate counted Senator Crapo, Chairman of the Environment and Public Works Drinking Water Subcommittee, among its cosponsors, and for good reason. This issue is, first and foremost, a drinking water issue, because MTBE is contaminating drinking water.

I would like to place into the hearing record the testimony of the Association of California Water Agencies, the Santa Clara Valley Water District, and the South Tahoe Public Water Utility District.

Senator INHOFE. Without objection.

Senator BOXER. Thank you so much, Mr. Chairman.

Senator BOXER. These agencies wanted to testify today, but there wasn't time to have a panel on water quality, so I am putting their statements in the record.

Why does MTBE pose a threat to drinking water and public health? First, in 1997, MTBE was the second-most produced chemical in the United States, so it's out there, in the environment, in huge quantities.

Second, MTBE is classified by the EPA as a possible human carcinogen. The University of California has also concluded that MTBE is an animal carcinogen and has the potential to cause cancer in humans.

I would like to place a peer-reviewed MTBE report, prepared by the California Office of Environmental Health Hazard Assessment, in today's hearing record. I would like to place, Mr. Chairman, a peer-reviewed MTBE report prepared by the California Office of Environmental Health Hazard Assessment in today's hearing record.

Senator INHOFE. Without objection, so ordered.

Senator BOXER. Thank you.

This report details and summarizes the health studies underlying the agency's recommendation, that California adopt an MTBE public health drinking water standard of 13 parts per billion. This standard is more protective than EPA's current non-binding standard of up to 40 parts per billion. So California is way ahead, in my view, in terms of protecting the public health, way ahead of where we are at the EPA, and I am very disappointed about that.

To summarize what I've said so far, MTBE is dangerous and it is widely used. Another reason is that it is also very hard to control. When MTBE leaks from an underground storage tank, from a motorboat, or from a gas tank after a car accident, into the groundwater, it moves through that water very fast and very far. It extends well beyond the area of a typical gasoline groundwater plume.

Also, unlike the other constituents of gasoline, it resists degrading once it is in the water. Moreover, it only takes a very small amount of this widely-used chemical to contaminate a drinking water source. For example, in Maine—I'm not talking about California, in Maine—about 7 to 12 gallons of gas containing MTBE spilled during a car accident and contaminated 24 nearby private drinking water wells. Twelve of those wells showed contamination



above Maine's MTBE drinking water standard. And it takes even less MTBE to render water undrinkable.

MTBE causes water to take on the taste and smell of turpentine at very low levels. Consumers can taste MTBE in their water at as low as 5 parts per billion. That is equivalent to less than a tablespoon of MTBE in an Olympic-size pool.

I have an example of this from Santa Monica. This is what their water smells like. I thought it would be interesting to smell it.

[Laughter.]

Senator BOXER. If you could pass it on to Senator Bennett.

I think it's important, because—here's the point I'm trying to make—even at 5 parts per billion, this stuff ruins the water in terms of your perception of it. People will not drink it if it smells that way. It smells like turpentine.

What is the extent of MTBE contamination in the Nation? And, Mr. Chairman, I am winding down, you will be happy to know. Since the Santa Monica catastrophe, South Lake Tahoe, CA has lost 13 of its 34 drinking water wells to MTBE contamination. Santa Clara County in the Silicon Valley has detected MTBE at over 400 groundwater sites, many of which are near public water supply wells. In 1998, a study conducted by Lawrence Livermore determined that MTBE is leaking at 10,000 sites in California.

But MTBE contamination is not just a California problem, as I have said before. Maine has determined that between 1,000 and 4,300 private wells may contain MTBE. In New Hampshire, MTBE has been detected in more than 100 public wells and water supplies. Suffolk County Water Utility in New York, which serves 1.2 million customers entirely with groundwater, tells me that 80 percent of its wells show detectable levels of MTBE.

Overall, the panel report states that the USGS estimates that between 5 and 10 percent of drinking water supplies now show MTBE contamination. I believe that is a low-end estimate, and I can get into why I believe that, but I won't go into it in the statement.

So again, to summarize, the chemical is out there; it's out there in large quantities; it has the potential to cause cancer in humans; it can render drinking water undrinkable, as I think you would agree, at very low levels. And one more point. We know that the potential cleanup costs are already astronomical.

A University of California study in 1998 estimated cleanup costs could run as high as \$1.5 billion just in California. Mr. Chairman, we all care about balancing the budget. Why would we want to put more of this stuff out there when we already know the cleanup costs are \$1.5 billion?

Now, I had further discussions. The authors of the studies believe they have underestimated the cleanup costs. They believe they could be 20 to 30 percent higher than that estimate.

Some argue that replacing gasoline storage tanks is the answer, but even new tanks have problems. A July 22, 1999 study by Santa Clara Valley Water District, in fact, found that many of its new tanks are leaking. The study reviewed a total of 28 sites with fully upgraded storage tank systems, to observe whether MTBE had leaked. MTBE was detected in groundwater at 13 of these sites at

concentrations ranging from 1 part per billion to 200,000 parts per billion. I would like to place that study into the record.

Senator INHOFE. Without objection.

Senator BOXER. So upgrading the tanks isn't the full answer, and neither will legislation which would amend the Clean Air Act to eliminate the oxygen requirement, but not ban MTBE—that's not the answer.

The argument behind such legislation is that if we give oil companies the flexibility to make reformulated gas without an oxygenate, they will voluntarily stop using MTBE. A story from the San Francisco Bay area, however, shows we can't rely upon the oil companies to voluntarily stop using it.

Even though oxygenated gas is not required to be used in the San Francisco Bay area, in May of this year it was disclosed that Chevron and Tosco were adding large quantities of MTBE to their gasoline in order to stretch gasoline supplies. So they didn't have to do it; they know the controversy—Governor Davis had already acted to say he was going to phase it out, and they used it anyway.

And I would like to place an L.A. Times story detailing this incident into the record at this time.

Senator INHOFE. Without objection.

Senator BOXER. As a result of the action of Chevron and Tosco, an area of California we could have hoped would be spared MTBE contamination may now also face significant threat.

In conclusion, I believe there are two ways to end MTBE use.

First, Congress should pass a phase-out schedule; or second, Administrator Browner could use her emergency authority to phase it out, and I'm very sorry that she hasn't done so.

I have introduced legislation which would phase out MTBE in stages beginning January 1, 2000, and adopting equal interim reductions each year until the phase-out deadline is completed on January 1, 2003. The DOE predicts it would take approximately 4 years to allow refiners to re-tool their facilities and increase ethanol production in the United States in order to implement such a phase-out, so my bill is in the ball park in terms of its timeframe.

MTBE is destroying water supplies throughout the Nation. MTBE cleanup costs are astronomical. MTBE is harming our lakes. MTBE is dangerous to your health. MTBE should be phased out.

Clean air is crucial to the health of our citizens; so is a safe drinking water supply. We need to do both—not one, but both.

Thank you, Mr. Chairman.

[The prepared statement and article submitted by Senator Boxer follow:]

STATEMENT OF HON. BARBARA BOXER, U.S. SENATOR FROM THE  
STATE OF CALIFORNIA

Good morning, Mr. Chairman. Thank you for holding this hearing today.

After I asked Administrator Browner to phase out MTBE, she appointed the Blue Ribbon Panel. I am pleased that panel report supports my view that we would be better off without MTBE.

Specifically, on the question of whether MTBE use should continue, the panel report states that:

[t]he Panel agreed broadly that, in order to minimize current and future threats to drinking water, the use of MTBE should be reduced substantially. Several members believed that the use of MTBE should be phased out completely.

I first formed the view that we should phase out MTBE in 1997 after the City of Santa Monica lost 71 percent of its drinking water supply due to MTBE contamination.

On August 4, 1999, the majority of the Senate joined with me in expressing that view by adopting my Sense of the Senate on this issue. That Sense of the Senate provided that the United States should "phase out MTBE in order to address the threats MTBE poses to public health and the environment."

I would like to place the text of this Sense of the Senate into the hearing record.

The Sense of the Senate counted Senator Crapo, chairman of the Environment and Public Works' drinking water subcommittee, among its cosponsors.

And for good reason.

This issue is, first and foremost, a drinking water issue.

I would like to place into the hearing record the testimony of the Association of California Water Agencies, the Santa Clara Valley Water District and the South Tahoe Public Water Utility District.

These agencies requested to testify here today. They support phasing out MTBE completely.

Why does MTBE pose a threat to drinking water and public health?

First, in 1997, MTBE was the second most-produced chemical in the United States. It's out there in our environment in huge quantities.

Second, MTBE is classified by the EPA as a possible human carcinogen. The University of California has also concluded that MTBE is an animal carcinogen, and has the potential to cause cancers in humans.

I would like to place a peer reviewed MTBE report prepared by the California Office of Environmental Health Hazard Assessment in today's hearing record.

This report details and summarizes the health studies underlying the agency's recommendation that California adopt a MTBE public health drinking water standard of 13 parts per billion.

That standard is more protective than EPA's current nonbinding standard of up to 40 parts per billion.

So MTBE is dangerous and widely used.

It is also very hard to control.

When MTBE leaks from an underground storage tank, from a motor boat or from a gas tank after a car accident into groundwater, it moves through that water very fast and very far.

It extends well beyond the area of a typical gasoline groundwater plume.

Also, unlike the other constituents of gasoline, it resists degrading once in water. Moreover, it only takes a very small amount of this widely used chemical to contaminate a drinking water source.

For example, in Maine about 7 to 12 gallons of gasoline containing MTBE spilled during a car accident and contaminated 24 nearby private drinking water wells. Twelve of those wells showed contamination above Maine's MTBE drinking water standard.

And, it takes even less MTBE to render water undrinkable.

MTBE causes water to take on the taste and smell of turpentine at very low levels. Consumers can taste MTBE in their water at as low as five parts per billion.

That is equivalent to *less than a tablespoon* of MTBE in an *Olympic size pool*.

What is the extent of MTBE contamination in the nation?

Since the Santa Monica catastrophe, South Lake Tahoe, California has lost 13 of its 34 drinking water wells to MTBE contamination. Santa Clara County, in the Silicon Valley, has detected MTBE at over 400 groundwater sites, many of which are near public water supply wells.

A 1998 study conducted by Lawrence Livermore determined that MTBE is leaking at approximately 10,000 sites in California.

But MTBE contamination is not just a California problem.

Maine has determined that between 1,000 and 4,300 private wells may contain MTBE. In New Hampshire, MTBE has been detected in more than 100 public wells and water supplies. Suffolk County Public Water Utility in New York, which serves 1.2 million customers entirely with groundwater, tells me that 80 percent of its wells show detectable levels of MTBE.

Overall, the panel report states that the United States Geological Survey estimates that between 5 and 10 percent of drinking water supplies now show MTBE contamination. And, I believe that this is a low-end estimate.

So again, to summarize so far, the chemical is out there, it is out there in large quantities, it has the potential to cause cancer in humans, and it can render drinking water undrinkable at very low levels.

We also know that the potential cleanup costs are *already* astronomical.

In 1998, a University of California study estimated that cleanup costs could run as high as 1.5 billion in California alone.

Based upon further discussions, the authors of the study now believe that the cleanup costs are about 20 to 30 percent higher than that estimate.

Some argue that replacing gasoline storage tanks is the answer.

But even the new tanks have problems, a fact acknowledged by the panel.

A July 22, 1999 study by the Santa Clara Valley Water District, in fact, found that many of its new tanks are leaking. The study reviewed a total of 28 sites with fully upgraded storage tank systems to observe whether MTBE had leaked from those tanks.

MTBE was detected in groundwater at 13 of these sites at concentrations ranging from 1 part per billion to 200,000 part per billion.

I would like to place that study into the hearing record.

Upgrading the tanks won't solve the problem.

And neither will legislation which would amend the Clean Air Act (CAA) to eliminate the oxygen requirement, but not ban MTBE.

The argument behind such legislation is that if we give oil companies the flexibility to make reformulated gasoline without an oxygenate, they will voluntarily stop using MTBE.

A story from the San Francisco Bay area, however, shows why we can't rely upon the oil companies to voluntarily stop using MTBE.

Even though oxygenated gasoline is not required to be used in the Bay Area, in May of this year it was disclosed that Chevron and Tosco were adding large quantities of MTBE to their gasoline in order to stretch gasoline supplies.

I would like to place a *Los Angeles Times* story detailing this incident into the record.

As a result of Chevron and Tosco's action, an area of California we could have hoped would be spared MTBE contamination may also now face significant threat.

I believe that there are two ways to end MTBE use.

First, Congress should pass a phase out schedule. Second, Administrator Browner should use emergency authorities to phase it out.

I have introduced legislation which would phase out MTBE in stages beginning on January 1, 2000, and adopting equal interim reductions each year until the complete phase-out deadline of January 1, 2003.

The Department of Energy predicts that it would take approximately 4 years to allow refiners to retool their facilities and increase ethanol production in the United States in order to implement such a phase out—so my bill is in the ballpark.

MTBE is destroying water supplies throughout the nation. MTBE cleanup costs are astronomical. MTBE is harming our lakes. MTBE is dangerous to health. MTBE should be phased out.

Clean air is crucial to our health. So is a safe drinking water supply. We need both—not one, but both.

Thank you.

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[From the Los Angeles Times, May 7, 1999]

#### MTBE PUT IN GAS

(By Jennifer Warren)

SACRAMENTO—Just as Gov. Gray Davis Was declaring MTBE an environmental hazard and ordering it phased out of gasoline, two oil companies were increasing amounts of the controversial additive in gas sold in Northern California.

Officials at Chevron Corp. and Tosco Corp. confirmed the boost in MTBE, saying it was necessary to stretch their gasoline supply after refinery fires and marketplace factors reduced production.

The move enabled the companies to keep a high volume of their gasoline flowing to market in March and April, when pump prices spiked to more than \$2 a gallon in some parts of California.

Chevron and Tosco officials defended the move as a temporary measure to help them serve customers during a short-term emergency. And while MTBE—a possible carcinogen—is scheduled to be banned in California, adding more of it to gasoline now is not illegal.

Critics, including a state senator, condemned the tactic, accusing the companies of putting profits ahead of public fears of a chemical that has contaminated drinking water wells throughout the state.

They also call the move hypocritical because both oil companies have been leaders in making MTBE-free gasoline. Last month, Tosco held a press conference to publicize its delivery of MTBE-free gasoline to Union 76 stations in the Lake Tahoe area. It also sells MTBE-free gas in three Bay Area counties.

Chevron, meanwhile, had been supplying MTBE-free gasoline to much of Northern California. About half of the gasoline produced at its Richmond refinery is typically made without MTBE.

"These are companies that have been making MTBE-free gas for quite awhile, so why are they doing this?" said state Sen. Don Perata (D-Alameda). "It's pure economics. The price is high and they're stretching their supply by adding more MTBE. . . . It's hard not to be cynical about it."

Assembly Speaker Antonio Villaraigosa (D-Los Angeles) expressed similar concerns: "They seem to be sending mixed signals here. There's no formal MTBE ban yet, but this is obviously taking us in the wrong direction."

MTBE is a key component of "cleaner-burning gasoline," which has been used in most of California's 24 million vehicles since 1996. While credited with reducing auto emissions, MTBE has leaked from underground storage tanks to contaminate drinking water from Santa Monica to Lake Tahoe. It also taints lakes by entering the water from two-stroke engines such as those that power water scooters.

Although other components of gasoline also seep from subterranean tanks, MTBE is a particular peril because it travels into ground water so quickly. Its health effects on humans are poorly understood, but it has been shown to cause cancer in mice and rats.

Responding to a rising clamor, the Governor declared in late March that MTBE poses a "significant risk" to the environment and ordered it phased out in California by the end of 2002.

Davis was traveling Thursday and had no immediate comment on the new developments. A spokeswoman noted that Davis has publicly urged companies to voluntarily remove MTBE from gasoline before the deadline.

The boost in MTBE usage by Chevron and Tosco came to light after Perata—acting on a tip—asked an East Bay water district to take samples of gasoline at three service stations in early April. The samples showed that various grades of gas at the stations—in San Francisco and Oakland—contained levels of MTBE as high as 15 percent, the legal limit.

Company officials did not dispute the findings, and acknowledge that they represent an increase. MTBE typically makes up about 10 to 11 percent of Tosco's gasoline, while much of Chevron's Northern California gas had previously contained no MTBE, officials said. The exception to that is in Sacramento, where clean air rules mandate an 11 percent concentration of the smog-fighting additive.

Mixing in more MTBE was one of many steps the companies took in response to a gas supply shortage that hit in March, officials said. The shortage was caused in part by a Feb. 23 explosion that closed Tosco's Martinez refinery and a fire at Chevron's Richmond refinery on March 25.

Al Jessel, a Chevron fuels specialist, said the fire cut the refinery's capacity by 10 percent to 15 percent, forcing officials to hunt for ways to stretch supply. In addition to blending in more MTBE, Chevron "bought every gallon of gasoline we could find from anyone anywhere in the world."

If the company had not added more MTBE, the result would have been an even tighter supply and even higher prices, Jessel said.

"We had to make a balance in our minds between having, MTBE-free gas and running out of gas and being unable to supply our customers," said Jessel, adding that Chevron has since found new sources of gasoline and is no longer mixing in more MTBE.

At Tosco, spokesman Duane Bordvick said his company went to similar lengths to cope with the supply crunch. He took issue with critics who suggest that adding more MTBE was an environmental sin.

"Tosco is doing an awful lot to get MTBE out of gasoline. We've been a leader in the industry," Bordvick said. "But I don't think increasing it up to the legal limit over a period of days has any impact."

At Communities for a Better Environment, staff scientist Azibuike Akaba disagreed and called the companies' action "extremely irresponsible."

"We've already got a terrible contamination problem with MTBE," said Akaba, whose San Francisco-based nonprofit group has been a critic of MTBE since 1991. "The more they put in, the worse it gets."

Senator INHOFE. Thank you, Senator Boxer.

Senator Bennett.

**OPENING STATEMENT OF HON. ROBERT F. BENNETT,  
U.S. SENATOR FROM THE STATE OF UTAH**

Senator BENNETT. Thank you, Mr. Chairman. I am going to look forward to the testimony of the witnesses.

I hope when we finally come down to a decision here, it is based on sound science, based on a full understanding of all of the aspects of the issue. I cannot help but reflect on an experience that occurred before I came to the Congress but that nonetheless, I think, had lasting impact. This was the concern about Alar on apples, and the Congress received a great deal of testimony, some of it from Academy Award-winning actresses, about the terrible effects on human health from Alar being sprayed on apples. Congress reacted to that testimony, and Alar was banned. The apple crop for that particular season was ruined.

I talked with the individual in Utah who handles food for the homeless, and he said, "That was a great boon for us, because we received all the apples that could not be sold in the supermarkets that were perfectly wonderful food, that had no contamination to them at all, and had been taken off the market as a result of panic. And that was fine, in that we had food to distribute to the homeless, but once the science caught up with the rhetoric, we found out we had made a serious mistake."

I get very nervous anytime we get into any of these kinds of hearings, to make sure that the science catches up with the rhetoric. And if we come out with a sound scientific answer this morning as a result of the balanced witnesses we're going to hear, I will be very grateful and I will be happy to support an appropriately scientific answer to what is a very troubling and difficult kind of question.

Thank you, Mr. Chairman.

Senator BOXER. Would the Senator yield for one point?

I would not send this to a homeless shelter. I mean, you're talking about a different situation. You're talking about a city that has 71 percent of its water supply finished, closed off, shut down. You're not talking about theory here.

Also, I hope my friend would read the scientific reports that we have placed in the record.

Senator BENNETT. I will read those.

Senator BOXER. I think it's part of the balance that he's looking for.

Senator BENNETT. I will read the scientific report.

I have relatives who live in Santa Monica. They continue to drink the water, but I'll look forward to hearing from them, I'm sure, as this thing goes on.

Senator BOXER. Well, we do deliver clean water to those people. Those wells are closed, but the rest of it is fine.

Senator INHOFE. Our first witness was the chairman of the Blue Ribbon Panel. Mr. Greenbaum, we appreciate very much your being here today. As you can see, there are some clarifications that we are looking to you to take care of for us.

We're going to have four witnesses today. What we're going to do is ask the witnesses to try to confine their opening statements to 5 minutes, and we will use the lighting system here. However, your entire statement will be entered in as a part of the record.

We will also have 5-minute rounds, and we will try to hold ourselves to that same timeframe.

Mr. Greenbaum.

**STATEMENT OF DANIEL S. GREENBAUM, PRESIDENT, HEALTH EFFECTS INSTITUTE, CAMBRIDGE, MA, AND FORMER CHAIR, BLUE RIBBON PANEL ON THE USE OF OXYGENATES IN GASOLINE**

Mr. GREENBAUM. Mr. Chairman, thank you very much. To you, Mr. Chairman, and Chairman Chafee and other members of the committee, I am pleased to have the opportunity to be here. I have to say, sitting here and listening to your opening statements, I felt for a moment that I was back at the first meeting of the Blue Ribbon Panel because some of the same issues and questions were placed on the table, as you might guess. We made an effort to try to bring a group of people together to try to look at this issue, look at the facts of this issue, and hopefully I can share with you some of that in my testimony and then answer questions as we go through the session this morning.

In the wake of the detection of MTBE in drinking water supplies, as Senator Boxer said, in both Maine and California and elsewhere, Administrator Browner convened the Blue Ribbon Panel to investigate the facts of the situation and to recommend actions to achieve both clean air and clean water.

The Panel consisted of experts on air, water, and public health, as well as representatives of the oil, ethanol, and MTBE industries, and the environmental community. We began our work in January of this year and we conducted an in-depth investigation of the air quality, water quality, fuel supply, and price issues surrounding the use of oxygenates in gasoline. We held six meetings in 6 months, including field meetings in New England and California. We heard from experts, we reviewed dozens of both existing and new studies of oxygenates in gasoline.

Based on that review the Panel found, first, that RFG has provided substantial reductions in the emissions of a number of air pollutants from motor vehicles, in most cases resulting in emission reductions that exceed those required by law.

Second, we found that there have been growing detections of MTBE in drinking water across the country, with between 5 percent and 10 percent of drinking water supplies in RFG areas showing detectable amounts of MTBE. There have not, at the same time, been increases in detections of the other portions of gasoline which behave fundamentally differently than MTBE in groundwater.

The great majority of the MTBE detections have been below levels of public health concern, as you yourself said in your opening comments, Mr. Chairman. With approximately 1 percent rising to levels above 20 parts per billion, and some instances, such as Santa Monica—although rare—of levels of 100 parts per billion and higher.

Detections at these lower levels, however, have raised consumer taste and odor concerns, and they have caused water suppliers to stop using some water supplies and to incur the costs of treatment and remediation.

The third thing we found is that the major source of this contamination appears to be releases from underground gasoline storage systems. These systems have been upgraded in the past decade, and that has likely resulted in reduced risk of leaks. However, approximately 20 percent of the storage tanks have not yet been upgraded. As well, there continue to be reports of releases from some upgraded systems due to inadequate design, installation, maintenance, and operation.

In addition, under the law under which USEPA regulates these tanks, they do not currently have the authority to regulate many fuel storage systems beyond those we see in gasoline stations.

Based on these facts, the Panel evaluated a range of alternatives for addressing the problems, and we recommended that EPA work with you in Congress and the States to implement a four-part integrated—and that's an important term here—integrated package of reforms to ensure that water supplies are better protected, while the substantial reductions in air pollution that have resulted from RFG are maintained.

Specifically, the Panel recommended, No. 1, a comprehensive set of improvements to the Nation's water protection programs, including over 20 specific actions to enhance underground storage tanks, safe drinking water, and private well protection programs. The Panel considered these necessary to prevent future water contamination, but not sufficient in and of themselves to ensure that the problem will be solved.

We recommended further that we agreed broadly that the use of MTBE should be reduced substantially, with some members supporting its complete phase-out, and that Congress should act to provide clear Federal and State authority to regulate and/or eliminate the use of MTBE and other gasoline additives that might threaten drinking water supplies.

Third, recognizing that MTBE was a very important part of the Nation's fuel supply, we recommended that Congress act to remove the current Clean Air Act requirement that 2 percent of RFG by weight consist of oxygen, to ensure that adequate fuel supplies can be blended in a cost-effective and timely manner, while reducing the use of MTBE.

And fourth, we recommended that EPA seek mechanisms to ensure that there is no loss of the current air quality benefits as the use of MTBE declines.

Now, although the Panel agreed broadly in its recommendations, two members—while agreeing with most recommendations—did have concerns over specific provisions, and I feel it my duty as Chairman to share those with you here.

The MTBE industry representative on the Panel felt that the water protection reforms that we proposed were sufficient to protect water supplies, and was concerned that the Panel had not adequately considered the air quality benefits of oxygenates.

The ethanol industry representative was concerned that the Panel's recommendation to lift the oxygen requirement did not adequately reflect the benefits of using oxygenates.

Copies of their statements are attached to the executive summary and in the final report.



In sum, the Panel found that we have a successful, cleaner-burning gasoline program in place, but we need to take action to ensure that the detections of MTBE in drinking water that we have seen, and which fortunately in the great majority of cases have not yet been a public health concern, do not continue to grow.

We have provided to the committee the executive summary, as well as the full report of the Panel, as now available on the World Wide Web, and I thank you for this opportunity to testify. I would be glad to answer any questions.

Senator INHOFE. Thank you, Mr. Greenbaum. We will have rounds of questions.

You know, all of this started, I guess, when Senators Dole and Daschle, in the 1990 amendments to the Clean Air Act, put the requirement in for oxygenates. I think that's getting down to the core of the problem. That's Dole and Daschle from the beautiful corn States of Kansas and South Dakota.

What did the Panel find in relation to the air benefits from MTBE? I think this is getting down to the crux of the problem, because it's my understanding that your Panel called for the repeal of the Federal oxygen content mandate. When you talk about the phase-down in the use of MTBE, it was dependent upon the repeal of the Federal mandate, is that correct? Could you elaborate on that?

Mr. GREENBAUM. Yes, I will.

First of all, we on the Panel understood that the RFG program as enacted into law, including the oxygen mandate, has been a tremendous air quality success. And as a result, it has been one that has greatly aided a number of people in their air pollution exposure across the country.

The benefits of the oxygenates themselves, both MTBE and ethanol, in that have been substantial in getting that program up and running. I think it is fair to say that at the time, there were no other proposals on the table that would provide fuels as clean that could provide similar air quality benefits.

What has occurred, and what the Panel saw, was that today the refining industry has emerged with fuel formulations that would contain no oxygenate at all, and that would meet or exceed the current performance of RFG. I don't think that was in place, necessarily, in 1990; I think that that's something that we've seen emerge, and I think in the end it was a challenge for the Panel. We didn't totally agree on the air quality benefits of MTBE specifically, in part because although they have played a role, it seems clear that there are other formulations of fuel that are available that could provide the same benefits as the ones with MTBE.

We did feel, particularly in the area of air toxics reductions, that MTBE and the oxygenate presence—both MTBE and ethanol—had contributed in some way to that, but the Panel recommended that if you remove the oxygen mandate and reduce the amount of MTBE, and at the same time made sure that the air quality requirements stayed strong, that you could see the fuel supply provide fuel that was cost-effective and provide the same air quality benefits, but without the problems with high levels of MTBE.

That was a long answer, Senator, I'm sorry.

Senator INHOFE. No, it's a good answer. I just want to be sure that we're all clear, that if the MTBE is phased out, did the Panel have any recommendation to replace that? Or was it for a replacement of the 2 percent oxygenate mandate? In other words, did they have the idea of phasing this out and then replacing it with something else, or phasing it out and also phasing out the 2 percent mandate?

Mr. GREENBAUM. First of all, to clarify, the Panel as a whole called for phasing down the use of MTBE, not phasing it out, although there were members who thought we should phase it out, but that was not the majority opinion of the Panel. In other words, reduce its use substantially; that's what we called for.

We did not pick one alternative that was "the best" alternative. We felt that that would depend on a complex mixture of decisions, including decisions at each refinery, decisions about the availability of fuel blending stocks, and decisions about what else within the gasoline already that neither the Panel, we thought, or the Federal Government should necessarily dictate. We thought that one of the alternatives that would come in to meet this would be increased use of ethanol. We thought another one would be increased use of components of existing gasoline, particularly alkylates, which are in gasoline currently and would have to be increased in production. And the Panel did not rule out the possibility of continued lower-level use of MTBE, in part because we saw in areas of the country where it had been used at lower levels, we did not see the same level of contamination.

In order to provide the flexibility necessary for that range of alternatives to be chosen in a cost-effective fashion by the refining industry to meet the air quality needs, we felt it was essential that the 2 percent mandate be lifted that in some parts of the country it might still be 2 percent or even more, where ethanol was available, and could even grow in its use; in other parts of the country, other parts of crude oil would be used. In some components of it, smaller levels of MTBE might continue to be used.

Senator INHOFE. Did your Panel look into the idea that if you are replacing MTBE, you could be replacing it with something that is as toxic or more toxic than MTBE is—benzene?

Mr. GREENBAUM. Well, we were concerned with that, and called for immediate review of the health effects of all of the alternatives that might mean an increase in supply, including ethanol, including alkylates, and including aromatics like benzene, which might increase in supply.

First of all, we felt that benzene is already capped in its use in reformulated gasoline; and that second, if at the same time you were tightening the air quality requirements to ensure that we kept the same level of good performance that we've had from existing RFG, that that would in itself keep the lid on some of the levels of more toxic components of gasoline that might increase, like benzene.

Senator INHOFE. Yes.

Senator Chafee.

Senator CHAFEE. Thank you, Mr. Chairman.

Let's see if I understand what you're recommending. As I understand it, you are recommending that we give up the 2 percent oxygen by weight, is that correct?

Mr. GREENBAUM. That's right.

Senator CHAFEE. And leave it up to each State as to how they want to handle this situation, whether they want to have some kind of an oxygen requirement, is that correct?

Mr. GREENBAUM. Well, we actually called for each State's and EPA's authority to be clarified as to how they could regulate additives to gasoline that might cause a threat to groundwater. And in essence that might mean that a State could continue to require oxygen, per se, but more importantly the question was, did they want to regulate the use of additives that might affect groundwater, which was really the crux of our concern.

Senator CHAFEE. Now, it seems to me that an important part of all this is that we ought to get on with this upgrading of the tanks, the underground storage tanks, and the surface tanks, too. But then you mentioned somewhere in your testimony that older outboards on recreational boats are contributing to this, likewise.

Mr. GREENBAUM. Yes. I actually have that in my formal testimony; to keep within the 5 minutes, I didn't comment on that.

There's no question that the tank systems—we have to complete the upgrade that we already started back in 1988, but really have to go much further than that with the tank system than we have. First of all, to complete it, but second, for the tanks, the standards for those tanks were put in place in 1988, prior to any knowledge that there would be high levels of use of MTBE in the fuel. The oxygen requirement didn't pass until 1990.

MTBE, as we have heard and as our Panel heard in detail from hydrogeologists, behaves substantially differently in groundwater than the other components of gasoline. Therefore, the consequences of a leak from an underground tank are greater, and the standards almost certainly would have been looked at more tightly in 1988 if we knew MTBE was in place, and probably need to be revisited.

So even within the tank program, there are things that need to be done to really tighten those standards, which cannot be done overnight. They need to be done.

Second, there are tanks that have been upgraded that are leaking. That's not because they weren't properly designed, necessarily, although in some cases they weren't well designed; they may have been misinstalled, they may have been improperly used. But we have had evidence of cases of that in Maine, Delaware, and California already. Not every tank, but some of the tanks will continue to leak.

Third, and I think this gets more focused to the point of your question, Senator Chafee, once you put this material in gasoline, there are a number of ways it can get into the groundwater and into the surface water. It can happen if leaks occur, if a tanker truck turns over, which does happen on highways; I saw that when I was Commissioner in Massachusetts. If a car accident occurs or a truck accident occurs, it can happen in a number of situations. It also comes out of motorboats, and we saw evidence in a number of surface waters of seasonal peaks in MTBE levels in waters because of older boat engines.

So it gets into the water in a number of ways. The single largest source of that has been underground tanks, but we shouldn't neglect the fact that there are other possible ways.

Senator CHAFEE. What is your answer to this statement? I have a memo here, and it says as follows: "The oxygen requirement is a redundant mandate that costs consumers over \$1 billion a year."

Mr. GREENBAUM. Well, I'm not sure of the source of that. I can't go back to a specific economic analysis that would tell me that.

I think, as I said earlier, it is not clear to me, looking backwards with 20-20 hindsight, that in 1990 there was another way to get to the kind of clean fuel we have, other than by the way that law was put in place. I think where we are today, technology has changed and the options have changed, and there are more options available to us for producing very clean gasoline than were available then. Whether there is an extra cost that consumers have incurred over time or not, I can't guarantee. I do know that most of the estimates of the increased cost of RFG, which were not just because of the oxygenates, ended up being in the \$0.01 to \$0.02 per gallon range, once that was actually implemented.

So I don't think we were talking about large increases in cost.

Senator CHAFEE. Thank you, Mr. Chairman.

Senator INHOFE. Let me ask about the leakage in these newly-installed underground tanks, like what percentage of them do have leaks, and what could be done to stop these leaks. Was it from installation? Was any kind of a study done? Because if it gets down to a point where we're going to have to be looking at regulation for the installation of these tanks, it will be important to know that.

Mr. GREENBAUM. There were only the beginnings of such studies, since many of the tanks didn't get upgraded until the latter part of the last decade, with a deadline of 1998. We did have one review done by the California Water Resources Board of a series of tanks in trying to identify what percentage were leaking and what the nature of the problems was, and they had the same interest, to try to understand whether they could tighten the rules, whether they could make it better. And we can certainly refer you to that.

There was also this survey by the Santa Clara Water District that Senator Boxer mentioned. Because we've only had 3 to 5 years, really, of experience with most of these tanks being upgraded, we probably don't have the full range of experience necessary. One of the things the Panel called for was an immediate look to try to build that data base.

Senator INHOFE. Would you submit that for the record, the reports of the analysis in California?

Mr. GREENBAUM. We certainly will do that.

Senator INHOFE. Senator Boxer.

Senator BOXER. Thank you, Mr. Chairman. Thank you for pressing the issue of the tanks, because it is really stunning to realize that in Santa Clara they put in all these new tanks, and they still have the leakage. It's very troubling.

I want to thank you very much for all the work you did and the committee did, the Blue Ribbon Panel. I think the fact that you had a member from the MTBE industry on there and a member from the ethanol industry on there says that there was quite a tug

of war going on, and I know that makes it very, very difficult to come to some firm conclusion.

Again, I want to read the conclusion that you came to, because I think it is very clear:

The Panel agreed broadly that in order to minimize current and future threats to drinking water, the use of MTBE should be reduced substantially. Several members believed that the use of MTBE should be phased out completely.

That's pretty clear guidance for this committee, I hope.

I wanted to probe a little bit about this recommendation. Without identifying who, because that's not important, approximately how many felt it ought to be phased out? And I'm not counting the two—I don't think it's fair to count the two who had a special economic interest, because obviously the ethanol person would say, "phase it out," and the MTBE person would say, "don't."

So without that, how many of that 11 left would you say said, "phase it out"?

Mr. GREENBAUM. Well, first of all, I think it's fine not to count those two. Actually, as I said in my comments, the representative of the MTBE industry did not think that we needed to go to the next stage of reductions, that the upgrading of the tanks and the other recommendations were sufficient.

To my recollection, there were four or five members, all from California—no, not all from California, four or five members of the Panel who felt they were most comfortable with a phase-out. The remainder of the Panel felt that a substantial reduction was adequate.

Senator BOXER. OK.

Mr. GREENBAUM. But the whole panel could not agree on—

Senator BOXER. I understand.

But I think that's important, Mr. Chairman, to recognize that, if you take away the ethanol and the MTBE people, because frankly, I think they have a particular view on the point. I think that's pretty interesting, because that is a large number who would say, "phase it out."

Now, out of those who said there ought to be a substantial reduction, what is a "substantial" reduction? What does that mean? Does that mean reduce the amount by 25 percent, 50 percent, 60 percent, 70 percent? What does that mean? It's kind of a fuzzy word.

Mr. GREENBAUM. Well, we actually did—in our recommendations we did not set a specific number because we felt, I think, more broadly that the setting of specific numbers in this process has been part of the problem rather than part of the solution. But we did give an example of such a substantial reduction, which would be moving back to the historical levels in which MTBE was used as a lead and other additive replacement prior to the introduction of RFG. It was used in those situations, on average across the whole fuel supply, at about 2 percent of the fuel supply, although in some cases it was higher and in some cases it wasn't used at all. But on average—

Senator BOXER. So the average was about 2 percent. And what is it now?

Mr. GREENBAUM. Well, in the RFG areas it is required to be 11 percent—I'm speaking, by the way, by volume here, 2 percent by volume versus 11 percent by volume.

Senator BOXER. OK. Well, that's very important guidance, Mr. Chairman. That is a substantial reduction.

Mr. GREENBAUM. That would be a substantial reduction. We were not prepared, because of the nature of the issue in different parts of the country, to say that that's what should be happening in every single location, but it was one example that we could give.

Senator BOXER. OK.

Now, here's a point I want to get at. If you eliminate the 2 percent requirement for oxygenates, but you don't ban MTBE, what assurance is there that MTBE won't be used? Because I gave you the example of San Francisco, which was completely shocking to people, where we did not need, because we were meeting the clean air requirement, to have MTBE. The oil companies, after learning that Gray Davis wanted to phase this out and the legislature wanted to phase it out and all the rest, decided it was the cheapest way to expand their gasoline supplies. It was stunning to people that they did that.

So my question is, if you don't ban MTBE but you list the oxygenate requirement, there's no guarantee that this substantial reduction is going to take place, wouldn't you agree, given the facts of what happened in San Francisco?

Mr. GREENBAUM. Well, I don't want to suggest that I know all the details of the situation in San Francisco, although I will say that the Panel happened to be having its meeting in California on the day that two things occurred simultaneously, No. 1, that Governor Davis made his announcement, and No. 2, that there was a major fire, the second in a short period of time, in one of the refineries in California.

While I would agree that it was unusual that the use of MTBE went up within 2 months of Governor Davis calling for its reduction, I would also suggest, based on what we as the Panel understood, that the situation in California was unique in the sense that the supply of gasoline from crude oil was substantially reduced because of the incidents in these two refineries.

Having said that, I think the general evidence that the Panel saw and the analyses that the Department of Energy has done, which are in the record and are summarized in our final report and can be provided, suggested that if you solely removed the mandate, that economic forces probably would reduce the amount of MTBE but continue to use it at fairly high levels, because it is a relatively cost-effective blending component for gasoline, very high octane and very clean.

The factor that the Panel knew would be a factor in industry decisionmaking, but which we couldn't quantify, is the growing concerns that a variety of people in industry have raised about the future liability for cleanup costs for industry of using MTBE, and that in and of itself is often a driver to get industry to reduce its use. And I think some companies are already trying to figure out, without the 2 percent mandate, how to reduce the use.

Having said that, the Panel did not feel that just lifting the mandate was sufficient, and that's why we called for Congress to clarify both Federal and State authority to regulate and/or eliminate the use of MTBE and similar additives. In other words, we were not calling for a ban, but we were calling for clarity on what the au-

thority would be to ensure that you could get a reduction over time if the market didn't provide that.

Senator BOXER. I appreciate that.

Two more quick questions—

Senator INHOFE. I'm really sorry, I'm going to have to cut you off because you've gone over—

Senator BOXER. Other people went over. You went over it, and so did Senator Chafee.

Senator INHOFE. No, I'm really trying to be fair with everyone.

Senator BOXER. Well, then, can I ask this when you finish everybody else? I have two more questions.

Senator INHOFE. How about 1 more minute, all right?

Senator BOXER. Fair enough.

Here's the point. You're right about litigation. In Santa Monica—and this goes to the point raised by my good friend and colleague, Senator Bennett—in Santa Monica, they shut down 71 percent of the water supply. Do you know where they're getting the water from to serve your family or friends? From the Colorado River.

Now, we are already over our allocation. This is a real serious problem for us. That is not a solution. And by the way, they are in litigation, trying to get the oil companies to pay for this importation of water.

We all love local government here. I served in local government. This is putting the burden on them for some mistake we made here.

So the bottom line is, the cost of this cleanup is enormous and it leads to litigation, and therefore we should ban MTBE.

And the last question I have deals with the fact that this—I think it's good to focus on the leaking tanks; the chairman is right on that point. However, that's not the only way this stuff gets in the water. We already talked about the use of MTBE and the boats and it goes in the lakes. When we transfer the fuel at transfer stations, it leaks. There was a car accident in Maine that contaminated 24 wells.

So it isn't just a matter of the underground tanks. We have other ways for MTBE to get in, is that correct, into the water supply?

Mr. GREENBAUM. I think I said that in response to Senator Chafee's question. There clearly are other ways. Our best estimate was that the great majority of the problems have been tanks, but that there are a number of other ways in which it can get in, the major ones being spills, accidents, and boats.

Senator BOXER. All right.

Thank you very much, Mr. Chairman.

Senator INHOFE. Thank you.

Before going to Senator Bennett, did you have an opening statement to make, Senator Voinovich?

**OPENING STATEMENT OF HON. GEORGE V. VOINOVICH,  
U.S. SENATOR FROM THE STATE OF OHIO**

Senator VOINOVICH. I will just submit it for the record.

Senator INHOFE. All right.

[The prepared statement of Senator Voinovich follows:]

## STATEMENT OF HON. GEORGE V. VOINOVICH, U.S. SENATOR FROM THE STATE OF OHIO

Mr. Chairman, I am pleased you are conducting this hearing on the EPA's Blue Ribbon Panel findings on the use of oxygenates in gasoline.

Throughout my 33 years of public service, I have been committed to preserving our environment and the health and well-being of our citizens. While in the Ohio House of Representatives, I was responsible for creating the Environment and Natural Resources Committee and was honored to serve as vice chair of that committee.

I am proud that the State of Ohio realized significant improvements in air quality in recent years. When I first entered office as Governor in 1991, most of Ohio's urban areas were not attaining the 1-hour ozone standard. By the time I left office in 1998, all cities had attained the standards, except one. However, earlier this year EPA proposed a rule to revoke the 1-hour standard for the last nonattainment area.

Overall, the ozone level in Ohio has gone down by 25 percent. In many urban areas, it has gone down by more than 50 percent in the past 20 years. My point is that Ohio is doing its part to provide cleaner air and a healthier environment for its citizens. For instance, Ohio's public utilities spent \$3.7 billion on air pollution controls through 1995, more than the combined expenditures of all the Northeast states.

As I said, all of our urban areas but one have met the one-hour ozone standard. And one of the things we did in Ohio to achieve this was to implement an emission testing program. This was not an easy task and I took a lot of heat for it. As a matter of fact, I had to veto a bill passed by the state legislature which would have removed the E-Check program because it was so unpopular and the legislature did not want to take the heat for it. But my Administration thought this program would best help us attain the National Ambient Air Quality Standards.

Ohio could have chosen to opt into the reformulated gasoline program as one option to reach the NAAQS standards, but we were not mandated to use it. However, other areas of the country are required to participate in the reformulated gasoline program to help them comply with air standards.

I think most state and local governments are willing to take the necessary steps to make the air we breathe cleaner. However, we need to make sure that the right hand knows what the left hand is doing. We want to make sure that as we are trying to reduce pollution in one source, such as air, we aren't affecting other sources, such as drinking or ground water. We need to make sure there is proper analysis and sound science behind the decisions we make whether they are regulatory standards or legislative requirements.

Quite frankly, I am concerned we are here today. I am concerned that in 1990 Congress acted to put the 2 percent mandate in the reformulated gasoline program without showing the necessary scientific reason for doing so. I am concerned that there was no analysis of the costs, benefits or risks behind this provision before it was enacted into law.

However, I am not convinced that EPA's Blue Ribbon Panel provides us with the adequate cost, benefit or risk analysis behind their recommendations either. We need to know more information before we start off on a new course of action. And we need to know whether the same money should be spent in this area or on other priority environmental problems.

I'm not here to say whether these recommendations are wrong or right, but that we need more information to determine whether this is the right path to follow.

I think that something should be done. However, I propose that states should have the flexibility to determine how to handle this problem in their own states.

Today we have an example of where a mandate was made without adequately studying the potential risks that it could impose or the science behind it. However, before we jump forward with extensive suggestions on how to fix the problem, there needs to be careful analysis of the costs, benefits and risks that would be incurred by these proposals.

Thank you, Mr. Chairman. I look forward to today's testimony.

Senator INHOFE. Senator Bennett.

Senator BENNETT. Thank you, Mr. Chairman.

Mr. Greenbaum, I come to your final statement. You say, "In sum, the Panel found that we have a successful, cleaner-burning gasoline program in place, but need to take action to ensure that the detections of MTBE in drinking water that we have seen, and which fortunately in the great majority of cases have not been a public health concern, do not continue to grow."



Let's parse that statement. Let's go through that sentence very carefully, because that is your summary of everything else you say.

We must "take action" that "the detections of MTBE in drinking water that we have seen . . . do not continue to grow." I assume from that you're saying that MTBE, however noxious it may be, is not toxic? Is that a correct statement? If not, correct me. But that's what I read into what you're saying: this is unpleasant; it can cause people to not want to drink the water; it can cause great difficulty, but it's not killing anybody—at least, not yet.

Mr. GREENBAUM. The "not yet" is important, I think.

Senator BENNETT. OK.

Mr. GREENBAUM. I think that at the levels at which it has been seen in most water supplies, everyone would agree that it is not toxic. My institution, the Health Effects Institute, actually conducted a comprehensive review of the science of MTBE, asked for by the White House and Centers for Disease Control in 1996, and I think it is fair to say that while there are questions about the toxicity of MTBE, it does not rise to the same level of toxicity as things like benzene, which are already in gasoline.

Having said that, there are levels at which everybody would agree it would not be safe. The levels that were reached in Santa Monica were 600 parts per billion. The levels at one set of private wells in Delaware were several hundred parts per million. So we have seen only the tip of the iceberg in terms of health or toxicity effect concerns. That's fortunate, and that's good. I think the Panel felt that we could not be assured that we wouldn't see continuing problems with that, and growth of that number of wells, and that's why we felt that we needed to take action now.

Senator BENNETT. You do not call for a ban. You call for a reduction, which would further support the notion that only in high concentrations is it toxic, and that a certain level is tolerable. Am I correctly summarizing your science here?

Mr. GREENBAUM. I think that's correct. The health basis for banning a chemical normally requires considerably more clear-cut evidence of the health concerns relating to that chemical than we have for MTBE. And that was a conscious discussion of the Panel.

Just to give you one example of that, benzene, which is in gasoline, is identified by both national and international cancer agencies as a known human carcinogen. MTBE is neither a known human carcinogen, or even a probable human carcinogen. At this stage it is in the "possible" category. In other words, there are some animal tests that show that it causes cancer, but there are questions about those tests.

Senator BENNETT. OK. So I am interested that you did not call for a total ban on these reasons.

Now, let's go to the other side of your examination. You found that we do have cleaner air because of MTBE, and I would ask, if MTBE were banned, what alternatives would you recommend in order to achieve the level of clean air? Are we talking about more ethanol, so that the corn farmers can rejoice? Or do we have something else that we can turn to?

Mr. GREENBAUM. Well, we actually found that clearly, RFG as a whole has had substantial air quality benefits. MTBE has been one of the components of that, but on the Panel we could not ascribe

with agreement any particular amount of benefit to MTBE providing that benefit versus ethanol providing that benefit, and that's based on the availability of data. The data is not clear enough and clean enough to be able to do that.

But there's no question that the fuel that has been out there with the oxygen has provided substantial air quality benefits. The challenge for the Panel was to try to answer your question: so if we take this stuff out, what's going to happen? We felt there were several scenarios that could occur. One of them would be increased use of ethanol. One of them would be increased use of the alkylate component refined from crude oil, which has very high octane and is generally clean. One of the scenarios could be increased use of aromatics, like benzene, which are things that we have been trying to reduce the use of in gasoline.

The Panel did not feel that it could choose the best of those alternatives, because each has strengths and weaknesses, but rather felt that what we needed to do was make sure that the requirements for RFG are stringent enough that we are assured that as the fuel goes forward, no matter what a refiner decides to do—whether they decide to use ethanol, whether they decide to use alkylates, or whether they decide to continue to use lower amounts of MTBE plus some of these other things—that you continue to have the air quality benefits. And the Panel felt that that was possible, given what we had seen in evidence before us.

Senator BENNETT. Thank you, Mr. Chairman.

Senator INHOFE. Thank you, Senator Bennett.

Senator Voinovich.

Senator VOINOVICH. Following up on Senator Bennett's line of questioning, if you eliminated the 2 percent requirement and banned MTBE, and you have reformulated gasoline, is there any guarantee that in order to achieve the same benefits to the air, that you wouldn't substitute something else that would be just as harmful?

Mr. GREENBAUM. Well, I think that's a very key question, Senator, and I think—because one of the possibilities would be that you would see some degree of increase from refiners in some of the refineries of the use of aromatics in the fuel, particularly things like benzene, which is very high octane. That's the kind of thing we've been trying to reduce; in fact, the refiners have reduced benzene below those required by the Clean Air Act and by EPA regulations.

And I think that goes to the fourth key recommendation which the Panel made, which was that you could only do these things if you, at the same time, ensured that the air quality requirements that were originally put out in the act were tightened sufficiently to require continued benefits equal to those we've actually had in the fuel, and that's what our fourth recommendation was. If you don't do that, then the concern that you raised is very real. I think there is a chance that you wouldn't see a return to pre-RFG days because there is a limit on how much benzene can be in the fuel, but you would see an increase in some of the components that contribute to air toxics and other emissions.

Senator VOINOVICH. So the fact is, to maintain the same improvement in the air quality that you're getting from MTBE, you

are really not sure if you eliminated it what else you would have to do, and you're not sure whether that might have more harmful impact on the water than MTBE?

Mr. GREENBAUM. Well, the issue of water—actually, we did look at the alternatives and we looked at the question of what those alternatives might have, not only for air quality impacts, but also water impacts. It would have been crazy for us not to look at it, given the experience we have had with MTBE.

I think there are two things there. First, most of the other components of gasoline, including the aromatics and the alkylates, actually, when they get into groundwater, they are not as soluble in groundwater as either MTBE or ethanol, and biodegrade more readily than MTBE. So our impression was that you would not be worsening the situation if you used more of crude oil components for the gasoline as a replacement for MTBE. The water situation would not be any worse than it has been historically, with leaks of gasoline.

With ethanol, ethanol is highly soluble in water, more so than any of the other compounds we considered, but it is also highly biodegradable, meaning that the bacteria in soil prefer to drink ethanol than drink benzene; I guess that's probably one way of putting it.

[Laughter.]

Mr. GREENBAUM. There are laboratory studies that confirm that. There are no field studies that say, "Well, what does that mean when you get out in the field?" We saw estimates, projections that were made, that suggested that what would happen with large volumes of ethanol in the fuel, is that you would see very rapid biodegradation of the ethanol. The ethanol would never move very far away from wherever the spill or the leak was. But you might see other components of the gasoline, like benzene, move further than they would otherwise move, because they wouldn't be biodegraded right away, and they might go as far as 30 percent further, but that is not tested in the field at this stage. That was something that we put in our report, and we called for an immediate look at that question before you went to a very broad use of ethanol.

Now, to be clear, ethanol can grow in its use, but it would also need infrastructure investment. The ethanol industry appeared before us and it was clear that they were prepared to make that, but overnight you would not see more ethanol—you know, dramatic increases in ethanol. You would see some increases.

Senator VOINOVICH. OK.

The last question I have is this. I read your summary, and it looks like you are making all kinds of recommendations to the Federal level or the State level. It sounds to me like there was no mention in it about the cost to do everything that was recommended. The dissenting opinion at the end said that if this happened and they eliminated it, that it would increase the cost of gasoline by \$1 billion to \$3 billion. The money side of this wasn't involved.

Wouldn't the best solution be to give EPA the flexibility to work with State people where they did have a problem to try to come up with something that would best respond to the needs of the particular community, rather than having some new Federal administration getting into all of this, and so on? In other words, in your

report you also say that if we did a better job, for example, dealing with storage tanks, that the primary source of this is leaking storage tanks. Now, in my State we have a very aggressive program that we started to get these out of the ground and replace them with things that are getting the job done.

The point is that this problem is localized, isn't it, in certain places in the country? And rather than come up with some gigantic new program, why don't we give the EPA flexibility to deal with the problem in the areas that have problems, and let them look at the alternatives, let the States come back with recommendations, and let them approve it or disapprove it, having to weigh the issue of clean air versus the issue of water? And leave it at that.

Mr. GREENBAUM. Well, there are a number of ways that one can address the sets of issues that we recommended. I think that embodied at the core of our recommendations was the recommendation that Congress act to clarify the authority of both EPA and the States to deal with these problems, because we do think there are going to be needs for some State flexibility and some ability to address this on a localized basis, in some cases going further than in others. And if anything, the Panel was suggesting moving away from a broad-based Federal requirement, the 2 percent requirement, because it—in and of itself—is an imposition in some ways on the entire system.

But having said that, I think the biggest concern counter to that that requires some careful interaction and thinking between the Federal Government and the States is the issue of not fractionating our fuel supply in so many different pieces that we end up with what some have called "boutique fuels" in different States and in different situations, where you would have—as you would know, Senator, if your constituents in Ohio have to pay one thing for fuel because you have one type of fuel, and they went across the border into another State and got much cheaper fuel, it would get very complicated very fast. And one of our strengths is having a national fuel supply.

So when we go to dealing with this, we have to be thinking about how we maintain that consistency while still giving States the authority and the flexibility to deal with their localized problems, and also while ensuring that the States have taken the actions to clean up their tanks as they should.

Senator INHOFE. Thank you, Mr. Greenbaum. We appreciate your being here and what you have contributed.

Senator INHOFE. We will now ask panel No. 2 to come forward. It will be the Honorable Jake Garn, our former colleague, who is vice chairman of Huntsman Corporation; Mr. Michael Kenny, executive officer, California Air Resources Board; and Mr. Bob Campbell, CEO of Sunoco, Inc.

Again, we will ask you to try to confine your opening remarks to 5 minutes, and then we will try to exercise the same discipline from this end of the table.

Senator Garn.

**STATEMENT OF HON. JAKE GARN, VICE CHAIRMAN,  
HUNTSMAN CORPORATION, SALT LAKE CITY, UT**

Mr. GARN. Thank you, Mr. Chairman. I am pleased that you have called these hearings and are willing to expand from the BRP and take additional testimony.

I am vice chairman of the Huntsman Corporation, which is the largest privately-owned chemical company in the United States, and we are a major producer of MTBE and a member of the Oxygenated Fuels Association. Huntsman's decision to get into the MTBE business was on the basis of clean air.

Huntsman is a unique company. One of the reasons we can do the things we do is because we are privately-held. John Huntsman has given \$150 million in cash to the University of Utah to create the Huntsman Cancer Institute. He is committed to solving the problem of cancer, and knowing John, I have no doubts he will probably over a number of years be able to accomplish that.

I bring that out because in all of our plants in the United States and around the world, I don't think you would find a company that is more socially responsible, has put their money where their mouth is in health and safety of their employees, the surrounding communities, and so on, and it really is a remarkable record. So I wanted you to understand the context in which I am speaking today.

We agree with much of what the Panel has found. For example, we agree that more research and monitoring is necessary concerning the health effects of not only MTBE, but also other constituents of gasoline. We agree that timely actions need to be taken to significantly enhance Federal and State gasoline storage programs. We also support the BRP finding that Congress must act to expand resources available to ensure safe drinking water supplies.

However, we have strong concerns about several of the BRP's conclusions. Most importantly, we disagree strongly that there is sufficient justification to recommend a substantial reduction in the use of MTBE. As described in greater detail in our written submission, we believe the BRP left many important questions unanswered. Unfortunately, the BRP is gone, and the responsibility to answer these questions falls to Congress, and to this subcommittee in particular. Until those questions are answered, we believe it is inappropriate to move forward with any effort to amend the Clean Air Act to reconfigure the reformulated gasoline program.

We appreciate this opportunity to contribute our thoughts on how Congress should endeavor to answer these remaining important questions.

Today I want to focus on a few of the many issues raised by the BRP in some greater detail.

We believe that the BRP's conclusion to phase down the use of MTBE is not supported by their own deliberative process. For example, the BRP made no finding with respect to the health effects due to MTBE exposure, and this result is not surprising, given that extensive research conducted over a number of years has indicated that MTBE exposure levels necessary to cause injury in animals are thousands and thousands of times greater than those humans could conceivably be exposed to. Therefore, an array of organizations has concluded that MTBE is not a human carcinogen. These

include the Department of Health and Human Services, the World Health Organization's International Agency for Research on Cancer, the National Academy of Sciences, and California's Office of Environmental Health Hazard Assessment.

Consistent with our own beliefs about cancer research, Huntsman supports the notion that much more research should be done, just as BRP recommended. However, with a clear consensus to date regarding the lack of adverse health consequences of MTBE exposure, and with substantial health benefits relating to clean air hanging in the balance, we cannot support BRP's conclusion regarding phase-down of the additive.

While we are on the subject of health benefits, Huntsman also believes that the BRP underestimated the air quality improvements attributable to the use of oxygenates like MTBE. EPA has written that oxygenates substantially reduce toxics and dilute or displace other fuel components, like sulfur, which in turn reduce emissions of the smog precursors. EPA has found that oxygenates like MTBE improve the performance of on-board automobile air pollution control devices. In short, the real world benefits of MTBE usage have exceeded even the most optimistic predicted results. By failing to give credit where credit is due for real world performance, BRP underestimated the environmental benefits of MTBE.

We also believe that BRP may have underestimated the effectiveness of enhanced underground storage protection as an appropriate response. Even Senator Feinstein observed during Floor consideration of an appropriations matter some 2 weeks ago, "The major way MTBE gets into groundwater is from defective underground tanks storing petroleum products." She has offered fixes to the UST program as a way to stop the contamination of drinking water by the gasoline additive MTBE.

Lastly, we are concerned that BRP simply paid too little attention to the potential consequences of shifting to alternative fuel additives to MTBE. As we all know, the primary alternative to MTBE is ethanol. We have several concerns about the viability of ethanol production constraints, and trouble with pipeline deliveries make ethanol a logical and logistical nightmare to use as a basis for the fuel supply of the United States.

Mr. Chairman, I notice the red light has come on, and I would simply refer to my detailed statement which you have already offered to put in the record. When I was chairman of the Senate Banking Committee I was very strict about time, and so I will be happy to respond to questions after the other witnesses, but when that red light comes on, I stop.

Senator INHOFE. Good for you, Senator.

[Laughter.]

Senator INHOFE. Thank you.

Mr. Kenny.

**STATEMENT OF MICHAEL KENNY, EXECUTIVE OFFICER,  
CALIFORNIA AIR RESOURCES BOARD, SACRAMENTO, CA**

Mr. KENNY. Thank you, Chairman Inhofe and members of the subcommittee. I am happy to be here today to present the California perspective on behalf of Governor Gray Davis, the California

Environmental Protection Agency, and the California Air Resources Board.

As the Blue Ribbon Panel report noted, California has its own reformulated gasoline program, which was established to deal with California's unique air quality problems. California's RFG program differs from the Federal program in a number of ways, and I think it's important to look at those.

The California program limits the sulfur and aromatic content of gasoline, while the Federal program does not. California's program also utilizes a predictive model that enables refiners to market innovative fuel formulations that vary from California's gasoline specifications, as long as refiners can demonstrate through the model that the formulations provide the required air quality benefits.

So far, the California RFG program has been immensely successful. Peak ozone levels in the State of California have been reduced by about 10 percent, and airborne benzene, a highly potent toxic, has been reduced by about 50 percent.

Unfortunately, the continuing controversy over MTBE has overshadowed the success of this program. Two California cities, Santa Monica and South Lake Tahoe, have seen their domestic water supplies decimated by MTBE contamination, and MTBE has been found in groundwater at several thousand leaking underground tank sites in California.

The Blue Ribbon Panel report documents that MTBE contamination is truly a national problem.

California took its own proactive steps last March. Governor Gray Davis declared that MTBE is an environmental risk, and he ordered it to be eliminated from California gasoline by the end of 2002. Perhaps the single most crucial factor affecting California's ability to eliminate MTBE use is the Federal 2 percent oxygenate requirement. The Blue Ribbon Panel recommendation on this was to eliminate that requirement. It is absolutely critical for California that that 2 percent requirement be eliminated.

California does not believe there is a technical or scientific basis for requiring the addition of oxygen to gasoline. It is possible to make California RFG without oxygen, and it is much more cost-effective to let each refiner decide for itself whether to use those oxygenates.

About 70 percent of the California gasoline market is subject to the Federal 2 percent oxygen rule, and in the other 30 percent of the market, three refiners have produced and sold non-oxygenated gasolines that provide all of the air quality benefits required by California reformulated gasoline.

California has shown that it can deliver the full benefits of its world-leading RFG program without an oxygen requirement. Once MTBE is eliminated in California, the only feasible oxygenate will be ethanol. If the 2 percent oxygen rule remains in effect, ethanol will be effectively mandated for 70 percent of California gasoline. California, in just 3 years, would need about half the amount of ethanol that is currently produced in the midwestern States.

The Blue Ribbon Panel report acknowledges the large investment in infrastructure that would be needed to meet this large demand. The California Energy Commission estimated that the elimination

of MTBE could add as much as \$0.06 to \$0.07 per gallon to gasoline costs if the oxygen requirement remains in effect. This would cost California motorists about \$840 million a year, without producing any additional air quality benefit. In contrast, elimination of the 2 percent requirement would allow gasoline costs to remain stable, and possibly decline.

Some have portrayed this as opposition to the use of ethanol. It's not. Even if the Federal oxygenate requirement is eliminated, we know that ethanol usage in California will increase exponentially; however, California should not trade its dependence on MTBE for a similar dependence on ethanol. Instead, we should strive for a diverse and stable RFG marketplace featuring a range of ethanol-based and nonoxygenated formulations. Such fuels will continue to achieve all the air quality benefits, but at less cost to the consumer.

I urge the committee to support the Blue Ribbon Panel's recommendation to eliminate the 2 percent requirement, and I especially urge you to support legislation by Senator Feinstein and by Representative Bilbray that would provide California with an early exemption from the requirement.

Action this year is crucial. Refiners need about 3 years to plan and complete the plant modifications that are needed to make non-MTBE gasoline by the end of 2002. To meet this challenging timeline, refiners need to know now whether they will have to continue to use 2 percent oxygen or have the flexibility to produce non-oxygenated formulations.

In closing, I would like to emphasize that California, as an arid State, is more dependent than most other States on our groundwater resources. Consequently, we crucially need the flexibility to produce RFG without oxygenates. Equally important, California RFG can be produced that maintains, and even improves upon, current air quality benefits, and we can do so at less cost if oxygenates are not required.

Thank you for agreeing to hear my testimony, and I would be happy to answer any questions.

Senator INHOFE. Thank you, Mr. Kenny.  
Mr. Campbell.

**STATEMENT OF ROBERT H. CAMPBELL, CHAIRMAN AND  
CHIEF EXECUTIVE OFFICER, SUNOCO, INC.**

Mr. CAMPBELL. Good morning, Mr. Chairman and members of the committee. My name is Bob Campbell, and I am chairman and CEO of Sunoco, Inc. My company is one of the largest refiners and marketers of gasoline on the east coast of the United States. In this region we produce and distribute more of the clean-burning RFG required by the Clean Air Act than by any other company, so consequently we have learned firsthand about the benefits and the burdens of the existing program.

We are also a manufacturer and consumer of MTBE, and we have been using it since 1980 for its high-octane qualities. After the Clean Air Act Amendments of 1990 were passed we constructed a world-class MTBE plant in Texas; consequently, we know about all there is to know about the use of that additive in gasoline.



In addition, we are also a major supplier of conventional gasoline in mid-America, and here we don't use MTBE, but we are a major buyer and blender of ethanol in gasoline. So we have extensive firsthand knowledge of both the benefits and limitations of ethanol in motor fuel.

Dr. Greenbaum has given an excellent summary of the deliberations and recommendations of the Panel, and those recommendations I wholeheartedly endorse. As you know, of course, we are now planning on implementation of those recommendations. Some of them require legislative action. Public concern is, of course, about the taste and smell of drinking water containing small amounts of MTBE.

Putting aside the complex question of MTBE as a health hazard, it should clearly not be getting into drinking water. But regardless of how much money is spent on tank replacement and inventory control, gasoline handled by 190 million drivers will inevitably be spilled, and we now know how persistent a contaminant MTBE can be in water.

California, as it so often has done, has led the way in defining a process for eliminating the problem. Critical to that, of course, is relief from the existing 2 percent oxygen mandate.

But one needs to remember that MTBE is principally used on both coasts, both the east and west coast of the United States. In fact, more MTBE is used in the 11 east coast States comprising the ozone transport region than in California—130,000 barrels a day versus 100,000 barrels. And I can assure you that people in Boston and Philadelphia are just as adamant about the quality of their drinking water as the people are in Sacramento and Santa Monica.

Consequently, my plea to you today is to help us solve the equally serious problem of MTBE in the Northeast, and I believe that to accomplish that we need a regional solution. If the proposed legislation deals only with California, I can assure you that several of the Northeastern States are poised to enact their own local solutions. The result will be a patchwork quilt of local initiatives and regulations, and that will be a nightmare for companies attempting to reliably supply low-cost, high-quality gasoline to consumers in the 11-State region.

The bottom line is that we can solve the problem in the Northeast in a manner similar to California only if we are also given relief from the 2 percent oxygen mandate. If you will do that, then we will be able to continue to supply RFG to those areas requiring it in an economic manner, in reliable quantities, with the same air quality benefits; and that reformulated gasoline will have substantially less amounts of MTBE.

I will tell you quite honestly that even with all our company's experience in blending ethanol in gasoline in mid-America, I don't know how to accomplish, in a real world practical manner, the same result in a northeast RFG system. Ethanol in RFG is successfully blended in the Chicago area because it's a relatively small proportion of the supply from the manufacturers in that region. In my opinion, if the 2 percent mandate remains and we are forced to directly substitute ethanol for MTBE in the large RFG volume areas in the northeast, we're going to have a disastrous scenario for both the supplier and the consumer. Obviously, there are two

very practical problems with ethanol as a blending component on the east and west coast. No. 1, of course, is the need for a reliable, adequate supply and the transportation issues between where it is currently manufactured today, and where it would be primarily used. Obviously, it has an affinity for water, and it can't be transported in common carriers, so you would have to put it in rail cars and trucks for both coasts.

Let me tell you exactly what I told the Blue Ribbon Panel this spring. Given enough time and money, an enterprising ethanol industry can expand production and create new logistics systems to address the problem. But the added cost will be immense and unnecessary.

Solving the logistics problem, however, will still not address ethanol's second and most critical defect, its high vapor pressure when blended into gasoline. The one thing we have learned in the past 10 years is that the most crucial characteristic of a successful RFG program is vapor pressure, or volatile organic compound—VOC—control. Higher vapor pressure means higher increased VOC emissions, which leads to more ozone pollution. It's as simple as that.

The next generation of RFG in January 2000 has even more stringent restrictions on vapor pressure than current. Consequently, blending ethanol into future RFG would severely compound both the environmental and the supply problems. It is my view that ethanol cannot be practically used on the east or west coast in the summertime period because of its low vapor pressure requirement and the high percentage of RFG that must be produced in those regions.

The solution? Legislation is needed to solve the oxygenate problem where it exists, in California and the ozone transport region of the east coast, because 75 percent of the RFG is there and 90 percent of the MTBE consumed is there. And we just ask you to give these regions three things: the authority to regulate the use of oxygenates when water quality impacts are substantiated; a waiver of the 2 percent oxygen mandate for RFG; and the requirement that no current clean air benefits be compromised as a result of these changes to the Federal fuel program.

I very much appreciate the opportunity to share these thoughts with you and look forward to any questions you ladies and gentlemen may have.

Senator INHOFE. Thank you, Mr. Campbell.

In deference to a scheduling problem that Senator Boxer has, I will go ahead and allow her to go first in her questioning, if she will agree to stay within 5 minutes.

[Laughter.]

Senator BOXER. Mr. Chairman, you have that promise.

I just have some statements to make, and I thank you so much for accommodating me. I know it's a "good news-bad news" thing for the chairman; he has to hear me first, but then I leave.

[Laughter.]

Senator BOXER. So it's a much happier situation to have four, versus no one on this side.

Senator INHOFE. Of course, we don't have any responsibility for the lack of interest on that side.

[Laughter.]

Senator BOXER. No, they have given me their mandate, so I speak with that.

Let me simply say a few things. Senator Voinovich made a very good point. He said, "You know, the problem is localized, isn't it true?" "Yes. It's where MTBE is used." So what I'm trying to do is do you a favor, tell you to avoid the heartache of what is happening to us.

And I want to thank Mr. Campbell for pointing out that the use of MTBE is really exploding in the Northeast. I want to spare them the problem. For me, you lift the oxygenate—Mr. Kenny, thank you for your clear explanation.

We are in good shape in California because our Governor has banned MTBE. We're OK, so I can relax on that point. But I do feel I want to spare the rest of the country the problem of shutting down water supplies. It's just dreadful, and then facing lawsuits and all the rest—it's very important.

Let me say to Senator Garn, thank you for your clear testimony. I know that you feel strongly about your product. I would say to you that your company giving money to cure cancer is laudable. I think that he ought to take a look, however, at this study that seems to be dismissed here, and I want to spend a minute just telling you about it, because when Senator Bennett says, "Isn't it true the Panel found that MTBE is not killing anybody," and to quote him, "at least not yet," that's far from a ringing endorsement, frankly. If somebody says, "It's not killing you, not yet," I'd say that's not an answer.

I would suggest—

Mr. GARN. Senator Boxer—

Senator BOXER. I have very little time and you will be able to respond. I won't even be here, so you can say anything you want and I won't be here.

Mr. GARN. No, I wanted you to hear what I have to say.

Senator BOXER. We'll meet in my office after. You just call and we'll talk.

But here's the situation. There was an Italian study that was made that shows that MTBE causes cancer in animals. It was very controversial, so it was peer-reviewed, and it was peer-reviewed by a very good group of people that was put together by the California Office of Health Hazard Assessment. And the people on there were very, very prominent people. They came from universities, the EPA, San Diego State, CALEPA, etc., the Air Board. And they essentially peer-reviewed that study and said it was right, and came to the conclusion that MTBE has the potential to cause cancer in humans. Now, look, you drink it now, and we're not sure, but it has the potential.

So I would say that if we can meet the Clean Air Act requirement without it, my goodness, let's do it. And as I said, I like the way this thing is moving. We've got the Blue Ribbon Panel here calling for a phase-out—excuse me, I would say a substantial reduction, with four or five of them calling for a phase-out. So I like what I'm hearing in terms of the direction that we're going. I appreciate what Governor Davis has done. We are on the cutting edge

in terms of reformulated gasoline; we're proving that a lot can be done without MTBE.

I just think that to me, as my kids would say, it's a "no-brainer." You have a very controversial chemical; it's showing up in the water supply; the new tanks are continuing to leak, so that's not the answer, and I would show you the study in Santa Clara.

So bottom line is, I think the road is clear. If we want to lift the oxygenate requirement, fine. I would add to that, banning MTBE, because I worry that if we don't clearly ban it, that it still will show up. I want to spare the rest of the country the agony we've gone through.

Again I want to say to Senator Garn, we will talk, we'll spend a half hour together going over whatever the issues are that you feel I am misinformed on. But I do feel comfortable with my position, and I do thank you for your graciousness, Mr. Chairman.

Senator INHOFE. Thank you, Senator Boxer.

Senator Garn, on my time if you would like to respond?

Mr. GARN. Well, the thing that I wanted to say, Senator Boxer, is that my political career started as the Water Commissioner of Salt Lake City. For 4 years I had the responsibility of delivering clean water to 375,000 people, so I know a great deal about water and water supplies, leaking tanks, and all of those problems. And I am not here just to defend MTBE. My position is simply that if I thought banning any one chemical would solve the problem, I would be for that—

Senator BOXER. Even though you work for a company that makes the chemical?

Mr. GARN. That's correct.

Senator BOXER. Well, that's very, very good.

Mr. GARN. But on the basis of adequate science, not opinions—

Senator BOXER. Of course.

Mr. GARN [continuing]. Because with gasoline you have benzene, you have toluene, you have alkylates, there are all sorts of things.

Senator BOXER. Of course.

Mr. GARN. It would not make me feel more comfortable to take MTBE out, and then have others of these chemicals leaking into the groundwater.

Senator BOXER. I understand.

Mr. GARN. We had multiple problems in our canyons and watersheds, of groundwater leakage and other difficulties. That's the only thing I wanted you to understand, where I am coming from personally. I voted against the 1990 Clean Air Act Amendments, for whatever that is worth. I wasn't sure we should be creating problems all over the country with uniform solutions. But until there is adequate science, we should not act. I'll give you an example. In Park City, UT, when I was still in the Senate, the EPA closed down a subdivision—no FHA loans, no new building construction, talking about moving people out of it—and I said, "Do you have the science to prove that lead is actually getting into these homes, and children are at risk?" So having been chairman of their appropriations subcommittee, I was in a position to say, "Stop. We will have scientific studies." We even blood-tested all of the children that lived in that entire subdivision. There was no lead contamination; EPA's case was absolutely wrong. We solved

the problem by putting 6 inches of topsoil in everybody's yard, instead of closing down the subdivision and causing great economic harm, the reduction in half of their housing prices, and so on and so forth.

That is essentially what I was trying to say in my statement. Let's not rush to judgment. Too many times in the 18 years I spent in this body we did. Let's get the science. You eliminate MTBE; you've still got a problem in California with leaking tanks.

So first of all I think we ought to make sure that we eliminate the source, to begin with, and have adequate science. I don't want to drink ethanol. I don't want to drink benzene. I don't want to drink MTBE.

Senator BOXER. I'm not suggesting you do.

Senator INHOFE. Reclaiming my time, Senator Boxer.

Mr. Kenny, I understand that the California Air Resources Board has raised air quality concerns about replacements for MTBE. I would like to ask you what your concerns are in terms of air quality changes that would take place if you did away with, or dramatically reduced, the MTBE and did not repeal the requirement for oxygenates.

Mr. KENNY. Thank you, Senator. The concern we have is that if you have a requirement to use oxygenates, and MTBE is not available, then what would happen more than likely is that the only alternative oxygenate that would be available would be ethanol. And our concern with regard to ethanol is that high uses of ethanol in the summertime would result in greater volatility of the gasoline, and the concern that we then see is potentially degraded air quality as a result of the evaporative emissions that would come from that gasoline. So we are very concerned about that.

There are ways to address that. The way it is commonly addressed is that you lower the vapor pressure of the base fuel that the ethanol is mixed into. When that occurs, then you can basically adjust and keep your RVPs down to a lower level. The difficulty with that, however, is that it is extremely expensive to lower that vapor pressure.

Senator INHOFE. Now, when you say "extremely expensive," I would like to get something in the record here as to what we're talking about, what type of framework in which we could characterize the expense.

Mr. KENNY. It's going to be in excess of \$0.06 per gallon of gasoline sold, at a minimum.

Senator INHOFE. Thank you.

Senator Chafee.

Senator CHAFEE. I'm all set, thank you.

Senator INHOFE. All right.

Senator Bennett.

Senator BENNETT. Thank you, Mr. Chairman.

I am interested in this discussion about removing the 2 percent requirement. Going back to my business career, I am always in favor of what I call "performance codes" as opposed to "specification codes." If I can describe it in an analogy, the old building codes required copper pipe, and they were written before anybody had invented PVC. You came along and said, "Well, we can now give you a tubing or piping into your house that is cheaper, lighter, better

in terms of its ability to withstand pressure per square foot, but the building code says you can't use it, because in the name of 'safety' we have to have copper pipe."

So if you go to a performance code that just says "you have to have this outcome," and let the market respond to the performance requirement instead of the specific requirement in a specification code, you get the best of all possible worlds. And I think had I been in the Senate, I probably would have voted against the specification code and would have said, "No, we just want clean air within these parameters," and allow Mr. Campbell and whoever else to come up with the ability to do that.

Now, I think that's what I'm hearing you say, Mr. Kenny, is that we want a performance code, not a specification code.

Mr. KENNY. That's correct, Senator Bennett. In fact, in California with our gasoline specifications, we do provide for a performance method as opposed to simply specifications.

Senator BENNETT. Yet at the same time, if a refiner like Mr. Campbell comes forward and says, "We can meet that performance code with the use of MTBE," you have just added a specification code component to your performance code, and you do sell on the basis that "MTBE is contaminating our groundwater." Is that correct?

Mr. KENNY. That is correct.

Senator BENNETT. OK.

We come back to the question of the Blue Ribbon Panel, my question to Mr. Greenbaum, and the clear statement in their summary position, which agrees with Senator Garn. He says we should "take action to ensure that the detections of MTBE in drinking water that we have seen do not continue to grow." And implicit in that and in his answer to me, he confirmed the same thing, the point Senator Garn has made, which is that there is no science to indicate that this—however unpleasant and noisome it may be—is toxic, and he agreed with that; at least I heard him agree with that in his comment here.

Now, I know Governor Davis has taken the position that he's taken, and you are here as his agent, and I wouldn't expect you to do anything but support that posture. But from the standpoint of the Congress, if we adopt a performance code and couple that with a performance code with respect to underground tanks, we may be achieving the requirement of the Blue Ribbon Panel.

Mr. Kenny, I know you don't think we are. I can tell that from your body language. But do any of the other witnesses have a comment?

Mr. Garn.

Mr. GARN. Senator Bennett, if I could just make a comment. When this issue first started to arise in California a couple of years ago, I had several conversations with Governor Wilson about the issue, and his position was quite different from Governor Davis in the fact that he—as I have tried to present today—wanted to "wait for the science," and that was his position. He said, "Jake, I don't know what action I will take; there is not sufficient evidence yet, and I am particularly waiting for the California Office of Environmental Health Hazard Assessment report before I make any deci-

sion," even though he was being pushed very hard in the summer of 1998 to do something.

Well, that office came up and voted in December 1998 that MTBE should not be considered a carcinogenic or developmental or reproductive toxicant. That happened in December, and that's the report he was waiting for. Of course, he was not in office. But again, I just have to keep making the point over and over again, I don't think you are in a position yet—meaning you, the Congress—to make a decision until there is a great deal more evidence, not only on MTBE, but the same scrutiny applied to all the other ingredients of gasoline that could be a problem. I just can't come to the conclusion that even if we banned MTBE, that we have solved the problem that we all agree is a problem.

Mr. KENNY. Mr. Chairman, if I could clarify one point.

Senator INHOFE. Surely.

Mr. KENNY. With regard to the Office of Environmental Health Hazard Assessment, there have been references to the fact that they voted that MTBE is not a human health carcinogen. I don't think that's quite accurate. I think it's probably more accurate to say that they took the matter up, and they voted 3 to 3, so they were unable to reach a decision.

Senator BENNETT. Thank you for that clarification.

Mr. Campbell.

Mr. CAMPBELL. Senator, a couple comments.

First of all, about tanks. There has been a leaking underground storage tank program in effect for 10 years; I think virtually every major oil company has replaced them. The liability of delivering to a tank which has not been replaced would be horrendous.

Part of today's problem is the fact that there have been over 20 percent of those tanks that are covered by the program that have not been replaced. They continue to ask for exemptions; that needs to be stopped.

Second thing, we've been told by EPA that there are more tanks that are exempted from the program than are currently in it—municipal, State, Federal, farms, small business. So completing that program is not going to be the answer. There is a tremendous number of exemptions out there.

Secondly, to those of us who have spent hundreds of millions of dollars replacing tanks, it's not a perfect system. Invariably there are some leaks. I hate to say this, but we are probably going to have continuing leaks of some amount. The Senator points out that the real answer would be to solve the leakage problem entirely. What I'm saying is that from practical reality, in all probability that's not possible.

So we have to deal with—if in fact there is a leak, what is done to remediate that? And we find that the remediation of gasoline based on crude oil occurs much more readily than when you put in some chemicals.

And I would like to mention one more thing about the health issue. The chemical companies, including my own, manufacture MTBE. The refiners use it because we're required to. The consumer gets upset because their drinking water smells or tastes funny. And trying to tell them that it's not a health effects issue is an impossible task. If it smells funny and tastes funny, as far as they

are concerned, your health effects study is incorrect. And continuing to supply that kind of product to the consumers is a liability that few companies are going to want to shoulder.

Senator BENNETT. I don't in any way to support water that smells and tastes funny, and I know the former Water Commissioner doesn't, either.

Thank you, Mr. Chairman.

Senator VOINOVICH. Just for the record, in response to Senator Boxer's statement that MTBE has not been listed as a carcinogen by either the National Institute of Environmental Health Sciences or the International Agency for Research on Cancer—and I understand, Mr. Kenny, that California has not listed it as a carcinogen under Proposition 65—I think we need to make that clear, because so often around here somebody gets a report, and before you know it, it's cancerous, and off we go, getting back to Senator Garn's good science.

If we eliminated the 2 percent and did not ban MTBE, and basically said, "You figure it out in California, and you figure it out on the east coast," if we came up with that result, would that cause chaos in the gasoline industry across this country?

Mr. KENNY. I don't think it would, Senator. I think the optimal situation here is one in which the 2 percent requirement is no longer in effect, and in California, MTBE is banned. So what would occur is that in California the performance standards would be in place.

I think with regard to the rest of the country, the issue would be how to maintain the air quality benefits that are currently being achieved from reformulated gasoline. In California we can maintain those air quality benefits because we have actually had fairly substantial investment by the refiners in upgrading the refineries, so that the cleaner gasoline can be produced.

I don't know if that could be said in exactly the same degree for the rest of the country.

Mr. CAMPBELL. Senator, in general the United States, with our 50 States, we have relatively few regions—we call "pads"—we have West Coast, Gulf Coast, mid-America, and eastern regions, so consequently that's why we talked about the need for a regional approach. Because refiners and marketers and distributors who supply regions would have tremendous difficulty if you had a patchwork quilt within a region—this State wanted that, this other State wanted something entirely different. That's why we are saying, in the Northeast—please remember, MTBE is predominantly a West Coast/East Coast issue. The West Coast is being addressed with California. In the case of the East Coast, what we have done is turn to the environmental directors in the States and some of the organizations, like NESCOM and MIRANA, and said, "Help us from the standpoint of establishing the standard so that we can have a regional fuel" so that companies like my own and other companies will be able to distribute it reliably and with relatively low cost. That's the intent.

Senator VOINOVICH. OK. If we didn't ban MTBE—if we eliminate the 2 percent and just say, "You work it out on a regional basis," or any way you could—do you think that's a practical approach?



Mr. CAMPBELL. Well, I don't, because I think there is so much focus on MTBE now in the individual States, the fact that it does cause odor and taste in water, that some of the States are almost ready to ban it themselves, much in the same manner as California.

Before the EPA Blue Ribbon Panel report came out, a number of States were ready to take action banning it themselves—

Senator VOINOVICH. The question is this. Do you think the Federal Government should ban MTBE?

Mr. CAMPBELL. No. I voted with the Panel to say that what we ought to do, first of all, is to have a substantial reduction—it was expected to be in the neighborhood of 75 percent less. The reason I say that was because for more than a decade, that was the level that was used generally throughout the industry as an octane-enhancer, and there were literally no complaints that anybody heard of. Only when we received the 2 percent oxygen mandate and the gasoline went from roughly 2 percent to 11 or 12 percent, or even higher, it seems to many of us that that's when the complaints began to surface.

So what we said was—and Dan Greenbaum has pointed out to us how difficult it is to totally ban a chemical—that what we ought to do is go from 11 to 15 percent down to 2 percent, which is where we were for many years—

Senator VOINOVICH. But if you eliminated the 2 percent, you could do that?

Mr. CAMPBELL. I'm talking 2 volume percent of MTBE, not 2 oxygen percent.

Senator VOINOVICH. OK. If you eliminated the 2 percent, you could still—

Mr. CAMPBELL. You would have to deal with the issue, though, Senator, of the States saying, "I don't care if you have a 2 percent mandate or not, I don't want MTBE in my State," and I think that's what we see occurring in kind of a patchwork quilt fashion on the east coast of the United States. So what we are trying to do is get them to put it together in a regional effect, and I think that you're going to have to deal with the issue of substantial reduction of MTBE. That's one person's opinion, in order to have the States satisfied.

Senator VOINOVICH. Well, if you eliminate the oxygenate requirement, then you can do what you want with it, can't you?

Mr. CAMPBELL. What I'm saying is—and I apologize if I'm not saying it clearly—if you eliminate the oxygenate requirement, and you still permit the use of MTBE, and you give no guidance as to the upper limit, my concern is that some of the States might end up saying that they want either zero, or begin to set their own limit on this.

Senator VOINOVICH. Well, so your answer to the question is that you think that you ought to eliminate it, period?

Mr. CAMPBELL. Eliminate MTBE?

Senator VOINOVICH. Yes.

Mr. CAMPBELL. No—where I'm coming from is to say that what we ought to do is go back to where we were. I think we will be able to—

Senator VOINOVICH. Where we were before the 2 percent requirement for oxygen?

Mr. CAMPBELL. Before the 2 percent oxygen, yes, sir.

Senator VOINOVICH. OK. Fine. And then you would decide regionally how you would handle it?

Mr. CAMPBELL. That's right.

Senator VOINOVICH. And would you need the Federal Government to sit down and negotiate that in a region for you?

Mr. CAMPBELL. No, but we would need the Federal Government to deal with the 2 percent mandate. But I don't think you need the Federal Government to handle the gasoline formula regionally. I think you can look to the environmental organizations within those sections of the country to come up with what they desire for that region.

Senator VOINOVICH. OK.

Another question, just to finish up. We have this great issue of ethanol, because there are lots of States that are involved in it, my State and lots of others. If you eliminated the 2 percent oxygenate requirement, would gasoline still contain ethanol? Or would that disappear?

Mr. CAMPBELL. I believe that gasoline would still contain ethanol. In fact, I believe that ethanol will increase in gasoline in the future. The reason I say that—assuming some MTBE comes out of the system, and you get down to a lower level—we're also going to be reducing sulfur from gasoline, in all probability. You do that, and you're going to lower the octane pool in this country. The way the refiners will turn in order to correct that, more and more will be turning to ethanol.

So my expectation is, if you eliminate the 2 percent mandate, oxygenate mandate, ethanol consumption in fuel in the United States will go up.

Senator VOINOVICH. Probably immediately, because a lot of people wouldn't use the MTBE? Some would substitute it?

Mr. CAMPBELL. Some would substitute ethanol where you don't have the RFG program.

Senator VOINOVICH. Thank you.

Senator INHOFE. Our time has expired. I thought I had it pretty well sorted out in my mind until Senator Voinovich started asking these questions, so let me ask this just for my own clarification.

If we went back to the pre-1990 amendments, where they did not have the oxygenate requirement, we still had the additive in there at that time, but it was used as an octane enhancer, and there wasn't a problem with the "patchwork," as you have explained it.

Why would there be that problem now if there wasn't before, if they were to repeal what they did in 1990?

Mr. CAMPBELL. There would not. You are right, there would not. I misunderstood the Senator's question. What you're saying is that if you repeal the 2 percent mandate and you go back to where we were, back prior to the 1990 amendments, and you essentially permitted the companies to blend whatever they needed to blend to meet some requirement from the standpoint of performance, to go back to Senator Bennett, the chances are that you would be back at the 2 volume percent MTBE in gasoline, and back at—

Senator INHOFE. As opposed to 11?

Mr. CAMPBELL. As opposed to 11.

I think the concern is that, certainly in California and increasingly on the east coast, there is a call for the banning of MTBE. If you talk to the water people, the thought of saying, "Let's go from 11 percent to 2 percent, and the problem is solved," they will say to you that that just means it's going to take five times longer to get to the same problem level; you didn't solve it back in those earlier days.

I think you will end up having a cry for some maximum ceiling of MTBE in gasoline. But by eliminating the oxygenate mandate, trying to go back to the 1980 or 1990 period of time, I think you would solve much of the problem.

Senator INHOFE. All right.

Well, I thank you very much. I appreciate all of you coming and being present and testifying. You will be receiving questions in writing, which we will ask you to submit for the record.

Senator INHOFE. We are in recess.

[Whereupon, at 11:23 a.m., the subcommittee was adjourned, to reconvene at the call of the chair.]

[Additional statements submitted for the record follow:]

STATEMENT OF HON. JOSEPH I. LIEBERMAN, U.S. SENATOR FROM THE STATE OF CONNECTICUT

Thank you, Mr. Chairman, for holding today's hearing on the results of the EPA's Blue Ribbon Panel on Oxygenates in Gasoline. I look forward to hearing from today's witnesses on this very important issue.

The 2 percent oxygenate requirement was included as part of the reformulated gasoline program (RFG), an effort to address smog pollution from mobile sources. Nine areas with the worst smog problems—including parts of Connecticut—were required to use cleaner burning gasoline. Other areas opted in voluntarily. Overall, RFG has produced significant benefits—reducing volatile organic compounds, carbon monoxide, and mobile air toxics—in many cases exceeding the standards required by law.

Unfortunately, the oxygenate requirement has had some unforeseen consequences as well. MTBE, the most widely used oxygenate, has been found in the water supply in more than 20 states and in several states occurs in concentrations high enough to cause the shutdown of wells. In my home State, Connecticut, at least 200 wells have been identified as contaminated by MTBE, raising serious health concerns.

However, even as we contemplate federal action to address the serious issue of MTBE contamination of water quality, we must not sacrifice the clean air benefits gained through the use of oxygenates. Thus, the challenge that faces us today is one of preserving the advances we have made in air quality while acting to increase protection of our nation's water supplies.

Foremost among our responsibilities at the federal level should be allowing states to address concerns about MTBE within the confines of the RFG program. I therefore support a national approach to this issue, giving states flexibility in dealing with MTBE and other oxygenates in gasoline by removing the federal oxygenate requirement. However, because the Blue Ribbon Panel, NESCAUM, and others have confirmed that oxygenates sometimes help gasoline exceed current standards, I also feel strongly that any legislation we consider must maintain existing air quality benefits. This could be included in revised performance criteria for gasoline. Perhaps some of the witnesses will have suggestions as to how to accomplish this most effectively.

I would also like to point out that Connecticut has taken significant steps to address another source of MTBE pollution, leaking underground storage tanks. As of February 1999, 15,450 leaking tanks had been closed in Connecticut and 1,818 cleanups were initiated. In addition, the Connecticut Department of Environmental Protection just announced a state program to assist homeowners in identifying and remediating leaky tanks.

I look forward to hearing from the witnesses about how we can best use the Panel's findings to solve the problem of MTBE contamination of drinking water.

STATEMENT OF DANIEL S. GREENBAUM, CHAIR, BLUE RIBBON PANEL ON OXYGENATES  
IN GASOLINE AND PRESIDENT, HEALTH EFFECTS INSTITUTE

Mr. Chairman, and members of the Committee, thank you for the opportunity to appear before you today to provide you with the results of the work of the Blue Ribbon Panel on Oxygenates in Gasoline. I have attached a copy of the Executive Summary and Recommendations of the Panel, which were issued on July 27, 1999.

In the wake of the detection of the additive MTBE (Methyl Tertiary Butyl Ether) in drinking water supplies in Maine, California, and elsewhere, the Blue Ribbon Panel was convened by U.S. EPA Administrator Browner to investigate the facts of the situation and recommend actions to achieve both clean air and clean water. The Panel consisted of experts on air and water quality, as well as representatives of the oil, ethanol, and MTBE industry and the environmental community (see attached list).

The Panel, began its work in January of this year, and conducted an in-depth investigation of the air quality, water quality, fuel supply, and price issues surrounding the use of oxygenates in gasoline, holding six meetings in 6 months (including field meetings in both New England and California), hearing from experts, and reviewing dozens of existing and new studies of oxygenates in gasoline.

Based on that review the Panel found:

1. RFG has provided substantial reductions in the emissions of a number of air pollutants from motor vehicles, most notably volatile organic compounds (precursors of ozone), carbon monoxide, and mobile-source air toxics (benzene, 1,3-butadiene, and others), in most cases resulting in emissions reductions that exceed those required by law.

2. There have been growing detections of MTBE in drinking water, with between 5 percent and 10 percent of drinking water supplies in RFG areas showing detectable amounts of MTBE. The great majority of these detections to date have been below levels of public health concern, with approximately one percent rising to levels above 20 ppb and some instances, although rare, of levels above 100ppb. Detections at lower levels have raised consumer taste and odor concerns that have caused water suppliers to stop using some water supplies and to incur costs of treatment and remediation. The contaminated wells include private wells that are less well protected than public drinking water supplies and not monitored for chemical contamination. There is also evidence of contamination of surface waters, particularly during summer boating seasons.

3. The major source of groundwater contamination appears to be releases from underground gasoline storage systems (UST). These systems have been upgraded over the last decade, likely resulting in reduced risk of leaks. However, approximately 20 percent of the storage systems have not yet been upgraded. There continue, as well, to be reports of releases from some upgraded systems, due to inadequate design, installation, maintenance, and/or operation. In addition, U.S. EPA does not currently have the authority to regulate many fuel storage systems (e.g. farms, small above-ground tanks).

Beyond groundwater contamination from UST sources, the other major sources of water contamination appear to be small and large gasoline spills to ground and surface waters, and recreational water craft—particularly those with older motors—releasing unburned fuel to surface waters.

Following its investigation, the Panel evaluated a range of alternatives for addressing these problems, and recommended that U.S. EPA work with Congress and the states to implement a 4-part integrated package of reforms to ensure that water supplies are better protected while the substantial reductions in air pollution that have resulted from RFG are maintained. Specifically, the Panel:

- Recommended a comprehensive set of improvements to the nation's water protection programs, including over 20 specific actions to enhance Underground Storage Tank, Safe Drinking Water, and private well protection programs. The panel considered these necessary, but not sufficient in and of themselves, to prevent future water contamination.

- Agreed broadly that use of MTBE should be reduced substantially (with some members supporting its complete phase out), and that Congress should act to provide clear federal and state authority to regulate and/or eliminate the use of MTBE and other gasoline additives that threaten drinking water supplies;

- Recommended that Congress act to remove the current Clean Air Act requirement—that 2 percent of RFG, by weight, consist of oxygen—to ensure that adequate fuel supplies can be blended in a cost-effective manner while reducing usage of MTBE; and

- Recommended that EPA seek mechanisms to ensure that there is no loss of current air quality benefits as the use of MTBE declines.

The Panel also called for accelerated research into the air, water and health characteristics of all compounds whose use would likely increase as replacements for MTBE, including aromatics, alkylates, and ethanol.

Although the Panel agreed broadly on its recommendations, two members, while agreeing with most recommendations, had concerns with specific provisions: the MTBE industry representative felt that the water protection reforms proposed by the Panel were sufficient to protect water supplies and was concerned that the Panel had not adequately considered the air quality benefits of oxygenates, and the ethanol industry representative was concerned that the Panel's recommendation to lift the oxygen requirement did not adequately reflect the benefits of using oxygenates. (Their statements are attached to the Executive Summary and Recommendations).

In sum, the Panel found that we have a successful cleaner-burning gasoline program in place but need to take action to ensure that the detections of MTBE in drinking water that we have seen—and which fortunately in the great majority of cases have not been of public health concern—do not continue to grow.

The Panel's full report, including background issues summaries on all of the data the Panel reviewed, is now available on the World Wide Web at the Panel's home page: <http://www.epa.gov/oms/consumer/fuels/oxypanel/blueribb.htm>.

Thank you again for this opportunity to testify. I would be pleased to answer any of the Committee's questions.

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THE BLUE RIBBON PANEL ON OXYGENATES IN GASOLINE—EXECUTIVE SUMMARY  
AND RECOMMENDATIONS

INTRODUCTION

The Federal Reformulated Gasoline Program (RFG) established in the Clean Air Act Amendments of 1990, and implemented in 1995, has provided substantial reductions in the emissions of a number of air pollutants from motor vehicles, most notably volatile organic compounds (precursors of ozone), carbon monoxide, and mobile-source air toxics (benzene, 1,3-butadiene, and others), in most cases resulting in emissions reductions that exceed those required by law. To address its unique air pollution challenges, California has adopted similar but more stringent requirements for California RFG.

The Clean Air Act requires that RIO contain 2 percent oxygen, by weight. Over 85 percent of RFG contains the oxygenate methyl tertiary butyl ether (MTBE) and approximately 8 percent contains ethanol—a domestic fuel-blending stock made from grain and potentially from recycled biomass waste. There is disagreement about the precise role of oxygenates in attaining the RFG air quality benefits although there is evidence from the existing program that increased use of oxygenates results in reduced carbon monoxide emissions, and it appears that additives contribute to reductions in aromatics in fuels and related air benefits. It is possible to formulate gasoline without oxygenates that can attain similar air toxics reductions, but less certain that, given current federal RFG requirements, all fuel blends created without oxygenates could maintain the benefits provided today by oxygenated RFG.

At the same time, the use of MTBE in the program has resulted in growing detections of MTBE in drinking water, with between 5 percent and 10 percent of drinking water supplies in high oxygenate use areas<sup>1</sup> showing at least detectable amounts of MTBE. The great majority of these detections to date have been well below levels of public health concern, with approximately one percent rising to levels above 20 ppb. Detections at lower levels have, however, raised consumer taste and odor concerns that have caused water suppliers to stop using some water supplies and to incur costs of treatment and remediation. The contaminated wells include private wells that are less well protected than public drinking water supplies and not monitored for chemical contamination. There is also evidence of contamination of surface waters, particularly during summer boating seasons.

The major source of groundwater contamination appears to be releases from underground gasoline storage systems (UST). These systems have been upgraded over the last decade, likely resulting in reduced risk of leaks. However, approximately 20 percent of the storage systems have not yet been upgraded, and there continue to be reports of releases from some upgraded systems, due to inadequate design, installation, maintenance, and/or operation. In addition, many fuel storage systems (e.g., farms, small above-ground tanks) are not currently regulated by U.S. EPA. Be-

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<sup>1</sup>Areas using RFG (2 percent by weight oxygen) and/or Oxyfuel (2.7 percent by weight Oxygen)

yond groundwater contamination from UST sources, the other major sources of water contamination appear to be small and large gasoline spills to ground and surface waters, and recreational water craft—particularly those with older motors—releasing unburned fuel to surface waters.

#### THE BLUE RIBBON PANEL

In November, 1998, U.S. EPA Administrator Carol M. Browner appointed a Blue Ribbon Panel to investigate the air quality benefits and water quality concerns associated with oxygenates in gasoline, and to provide independent advice and recommendations on ways to maintain air quality while protecting water quality. The Panel, which met six times from January–June 1999, heard presentations in Washington, the Northeast, and California about the benefits and concerns related to RFG and the oxygenates; gathered the best available information on the program and its effects; identified key data gaps; and evaluated a series of alternative recommendations based on their effects on:

- air quality
- water quality
- stability of fuel supply and cost

#### THE FINDINGS AND RECOMMENDATIONS OF THE BLUE RIBBON PANEL

*Findings* Based on its review of the issues, the Panel made the following overall findings:

- The distribution, use, and combustion of gasoline poses risks to our environment and public health.
- RFG provides considerable air quality improvements and benefits for millions of U.S. citizens.
- The use of MTBE has raised the issue of the effects of both MTBE alone and MTBE in gasoline. This panel was not constituted to perform an independent comprehensive health assessment and has chosen to rely on recent reports by a number of state, national, and international health agencies. What seems clear, however, is that MTBE, due to its persistence and mobility in water, is more likely to contaminate ground and surface water than the other components of gasoline.
- MTBE has been found in a number of water supplies nationwide, primarily causing consumer odor and taste concerns that have led water suppliers to reduce use of those supplies. Incidents of MTBE in drinking water supplies at levels well above EPA and state guidelines and standards have occurred, but are rare. The Panel believes that the occurrence of MTBE in drinking water supplies can and should be substantially reduced.
- MTBE is currently an integral component of the U.S. gasoline supply both in terms of volume and octane. As such, changes in its use, with the attendant capital construction and infrastructure modifications, must be implemented with sufficient time, certainty, and flexibility to maintain the stability of both the complex U.S. fuel supply system and gasoline prices.

The following recommendations are intended to be implemented as a single package of actions designed to simultaneously maintain air quality benefits while enhancing water quality protection and assuring a stable fuel supply at reasonable cost. The majority of these recommendations could be implemented by federal and state environmental agencies without further legislative action, and we would urge their rapid implementation. We would, as well, urge all parties to work with Congress to implement those of our recommendations that require legislative action.

#### *Recommendations to Enhance Water Protection*

Based on its review of the existing federal, state and local programs to protect, treat, and remediate water supplies, the Blue Ribbon Panel makes the following recommendations to enhance, accelerate, and expand existing programs to improve protection of drinking water supplies from contamination.

#### *Prevention*

1. EPA, working with the states, should take the following actions to enhance significantly the Federal and State Underground Storage Tank programs:

a. Accelerate enforcement of the replacement of existing tank systems to conform with the federally-required December 22, 1998 deadline for upgrade, including, at a minimum, moving to have all states prohibit fuel deliveries to non-upgraded tanks, and adding enforcement and compliance resources to ensure prompt enforcement action, especially in areas using RIG and Wintertime Oxyfuel.

b. Evaluate the field performance of current system design requirements and technology and, based on that evaluation, improve system requirements to minimize

leaks/releases, particularly in vulnerable areas (see recommendations on Wellhead Protection Program in 2. below)

c. Strengthen release detection requirements to enhance early detection, particularly in vulnerable areas, and to ensure rapid repair and remediation

d. Require monitoring and reporting of MTBE and other ethers in groundwater at all UST release sites

e. Encourage states to require that the proximity to drinking water supplies, and the potential to impact those supplies, be considered in land-use planning and permitting decisions for siting of new UST facilities and petroleum pipelines.

f. Implement and/or expand programs to train and license UST system installers and maintenance personnel.

g. Work with Congress to examine and, if needed, expand the universe of regulated tanks to include underground and above ground fuel storage systems that are not currently regulated yet pose substantial risk to drinking water supplies.

2. EPA should work with its state and local water supply partners to enhance implementation of the Federal and State Safe Drinking Water Act programs to:

a. Accelerate, particularly in those areas where RFG or Oxygenated Fuel is used, the assessments of drinking water source protection areas required in Section 1453 of the 1996 Safe Drinking Water Act Amendments.

b. Coordinate the Source Water Assessment program in each state with federal and state Underground Storage Tank Programs using geographic information and other advanced data systems to determine the location of drinking water sources and to identify UST sites within source protection zones.

c. Accelerate currently-planned implementation of testing for and reporting of MTBE in public drinking water supplies to occur before 2001.

d. Increase ongoing federal, state, and local efforts in Wellhead Protection Areas including:

- enhanced permitting, design, and system installation requirements for USTs and pipelines in these areas;

- strengthened efforts to ensure that non-operating USTs are properly closed;

- enhanced UST release prevention and detection

- improved inventory management of fuels.

3. EPA should work with states and localities to enhance their efforts to protect lakes and reservoirs that serve as drinking water supplies by restricting use of recreational water craft, particularly those with older motors.

4. EPA should work with other federal agencies, the states, and private sector partners to implement expanded programs to protect private well users, including, but not limited to:

a. A nationwide assessment of the incidence of contamination of private wells by components of gasoline as well as by other common contaminants in shallow groundwater;

b. Broad-based outreach and public education programs for owners and users of private wells on preventing, detecting, and treating contamination;

c. Programs to encourage and facilitate regular water quality testing of private wells.

5. Implement, through public-private partnerships, expanded Public Education programs at the federal, state, and local levels on the proper handling and disposal of gasoline.

6. Develop and implement an integrated field research program into the groundwater behavior of gasoline and oxygenates, including:

a. Identifying and initiating research at a population of UST release sites and nearby drinking water supplies including sites with MTBE, sites with ethanol, and sites using no oxygenate;

b. Conducting broader, comparative studies of levels of MTBE, ethanol, benzene, and other gasoline compounds in drinking water supplies in areas using primarily MTBE, areas using primarily ethanol, and areas using no or lower levels of oxygenate.

#### *Treatment and Remediation*

7. EPA should work with Congress to expand resources available for the up-front funding of the treatment of drinking water supplies contaminated with MTBE and other gasoline components to ensure that affected supplies can be rapidly treated and returned to service, or that an alternative water supply can be provided. This could take a number of forms, including but not limited to:

a. Enhancing the existing Federal Leaking Underground Storage Tank Trust Fund by fully appropriating the annual available amount in the Fund, ensuring that treatment of contaminated drinking water supplies can be funded, and streamlining the procedures for obtaining funding.

b. Establishing another form of funding mechanism which ties the funding more directly to the source of contamination.

c. Encouraging states to consider targeting State Revolving Funds (SRF) to help accelerate treatment and remediation in high priority areas.

8. Given the different behavior of MTBE in groundwater when compared to other components of gasoline, states in RFG and Oxyfuel areas should reexamine and enhance state and federal “triage” procedures for prioritizing remediation efforts at UST sites based on their proximity to drinking water supplies.

9. Accelerate laboratory and field research, and pilot projects, for the development and implementation of cost-effective water supply treatment and remediation technology, and harmonize these efforts with other public/private efforts underway.

*Recommendations for Blending Fuel for Clean Air and Water*

Based on its review of the current water protection programs, and the likely progress that can be made in tightening and strengthening those programs by implementing Recommendations 1–9 above, the Panel agreed broadly, although not unanimously, that even enhanced protection programs will not give adequate assurance that water supplies will be protected, and that changes need to be made to the RFG program to reduce the amount of MTBE being used, while ensuring that the air quality benefits of RFG, and fuel supply and price stability, are maintained.

Given the complexity of the national fuel system, the advantages and disadvantages of each of the fuel blending options the Panel considered (see Appendix A), and the need to maintain the air quality benefits of the current program, the Panel recommends an integrated package of actions by both Congress and EPA that should be implemented as quickly as possible. The key elements of that package, described in more detail below, are:

- Action agreed to broadly by the Panel to reduce the use of MTBE substantially (with some members supporting its complete phase out), and action by Congress to clarify federal and state authority to regulate and/or eliminate the use of gasoline additives that threaten drinking water supplies;
- Action by Congress to remove the current 2 percent oxygen requirement to ensure that adequate fuel supplies can be blended in a cost-effective manner while quickly reducing usage of MTBE; and
- Action by EPA to ensure that there is no loss of current air quality benefits.

*The Oxygen Requirement*

10. The current Clean Air Act requirement to require 2 percent oxygen, by weight, in RFG must be removed in order to provide flexibility to blend adequate fuel supplies in a cost-effective manner while quickly reducing usage of MTBE and maintaining air quality benefits.

The panel recognizes that Congress, when adopting the oxygen requirement, sought to advance several national policy goals (energy security and diversity, agricultural policy, etc.) that are beyond the scope of our expertise and deliberations.

The panel further recognizes that if Congress acts on the recommendation to remove the requirement, Congress will likely seek other legislative mechanisms to fulfill these other national policy interests.

*Maintaining Air Benefits*

11. Present toxic emission performance of RFG can be attributed, to some degree, to a combination of three primary factors: (1) mass emission performance requirements, (2) the use of oxygenates, and (3) a necessary compliance margin with a per gallon standard. In Cal RFG, caps on specific components of fuel is an additional factor to which toxics emission reductions can be attributed.

Outside of California, lifting the oxygen requirement as recommended above may lead to fuel reformulations that achieve the minimum performance standards required under the 1990 Act, rather than the larger air quality benefits currently observed. In addition, changes in the RFG program could have adverse consequences for conventional gasoline as well.

Within California, lifting the oxygen requirement will result in greater flexibility to maintain and enhance emission reductions, particularly as California pursues new formulation requirements for gasoline.

In order to ensure that there is no loss of current air quality benefits, EPA should seek appropriate mechanisms for both the RFG Phase II and Conventional Gasoline programs to define and maintain in RFG II the real world performance observed in



RFG Phase I while preventing deterioration of the current air quality performance of conventional gasoline.<sup>2</sup>

There are several possible mechanisms to accomplish this. One obvious way is to enhance the mass-based performance requirements currently used in the program. At the same time, the panel recognizes that the different exhaust components pose differential risks to public health due in large degree to their variable potency. The panel urges EPA to explore and implement mechanisms to achieve equivalent or improved public health results that focus on reducing those compounds that pose the greatest risk.

*Reducing the Use of MTBE*

12. The Panel agreed broadly that, in order to minimize current and future threats to drinking water, the use of MTBE should be reduced substantially. Several members believed that the use of MTBE should be phased out completely. The Panel recommends that Congress act quickly to clarify federal and state authority to regulate and/or eliminate the Use of gasoline additives that pose a threat to drinking water supplies.<sup>3</sup>

Initial efforts to reduce should begin immediately, with substantial reductions to begin as Soon as Recommendation 10 above—the removal of the 2 percent oxygen requirement—is implemented.<sup>4</sup> Accomplishing any such major change in the gasoline supply without disruptions to fuel supply and price will require adequate lead time—Up to 4 years if the use of MTBE is eliminated, sooner in the case of a substantial reduction (e.g. returning to historical levels of MTBE use).

The Panel recommends, as well, that any reduction should be designed so as to not result in an increase in MTBE use in Conventional Gasoline areas.

13. The other ethers (e.g. ETBE, TAME, and DIPE) have been less widely used and less widely studied than MTBE. To the extent that they have been studied, they appear to have similar, but not identical, chemical and hydrogeologic characteristics. The Panel recommends accelerated study of the health effects and groundwater characteristics of these compounds before they are allowed to be placed in widespread use.

In addition, EPA and others should accelerate ongoing research efforts into the inhalation and ingestion health effects, air emission transformation byproducts, and environmental behavior of *all* oxygenates and other components likely to increase in the absence of MTBE. This should include research on ethanol, alkylates, and aromatics, as well as of gasoline compositions containing those components.

<sup>2</sup>The Panel is aware of the current proposal for further changes to the sulfur levels of gasoline and recognizes that implementation of any change resulting from the Panel's recommendations will, of necessity, need to be coordinated with implementation of these other changes. However, a majority of the panel considered the maintenance of current RFG air quality benefits as separate from any additional benefits that might accrue from the sulfur changes currently under consideration.

<sup>3</sup>Under §211 of the 1990 Clean Air Act, Congress provided EPA with authority to regulate fuel formulation to improve air quality. In addition to EPA's national authority, in §211(c)(4) Congress sought to balance the desire for maximum uniformity in our nation's fuel supply with the obligation to empower states to adopt measures necessary to meet national air quality standards. Under §211(c)(4), states may adopt regulations on the components of fuel, but must demonstrate that (1) their proposed regulations are needed to address a violation of the NAAQS and (2) it is not possible to achieve the desired outcome without such changes.

The panel recommends that Federal law be amended to clarify EPA and state authority to regulate and/or eliminate gasoline additives that threaten water supplies. It is expected that this would be done initially on a national level to maintain uniformity in the fuel supply. For funkier action by the states, the granting of such authority should be based upon a similar two-part test:

(1) states must demonstrate that their water resources are at risk from MTBE use, above and beyond the risk posed by other gasoline components at levels of MTBE use present at the time of the request.

(2) states have taken necessary measures to restrict/eliminate the presence of gasoline in the water resource. To maximize the uniformity with which any changes are implemented and minimize impacts on cost and fuel supply, the panel recommends that EPA establish criteria for state waiver requests including but not limited to:

a. Water quality metrics necessary to demonstrate the risk to water resources and air quality metrics to ensure no loss of benefits from the federal RFG program.

b. Compliance with federal requirements to prevent leaking and spilling of gasoline.

c. Programs for remediation and response.

d. A consistent schedule for state demonstrations, EPA review, and any resulting regulation of the volume of gasoline components in order to minimize disruption to the fuel supply system.

<sup>4</sup>Although a rapid, substantial reduction will require removal of the oxygen requirement, EPA should, in order to enable initial reductions to occur as soon as possible, review administrative flexibility under existing law to allow refiners who desire to make reductions to begin doing so.

14. To ensure that any reduction is adequate to protect water supplies, the Panel recommends that EPA, in conjunction with USGS, the Departments of Agriculture and Energy, industry, and water suppliers, should move quickly to:

- a. Conduct short-term modeling analyses and other research based on existing data to estimate current and likely future threats of contamination;
- b. Establish routine systems to collect and publish, at least annually, all available monitoring data on:
  - use of MTBE, other ethers, and Ethanol,
  - levels of MTBE, Ethanol, and petroleum hydrocarbons found in ground, surface and drinking water,
  - trends in detections and levels of MTBE, Ethanol, and petroleum hydrocarbons in ground and drinking water;
- c. Identify and begin to collect additional data necessary to adequately assist the current and potential future state of contamination.

*The Wintertime Oxyfuel Program*

The Wintertime Oxyfuel Program continues to provide a means for some areas of the country to come into, or maintain, compliance with the Carbon Monoxide standard. Only a few metropolitan areas continue to use MTBE in this program. In most areas today, ethanol can and is meeting these wintertime needs for oxygen without raising volatility concerns given the season.

15. The Panel recommends that the Wintertime Oxyfuel program be continued (a) for as long as it provides a useful compliance and/or maintenance tool for the affected states and metropolitan areas, and (b) assuming that the clarification of state and federal authority described above is enacted to enable states, where necessary, to regulate and/or eliminate the use of gasoline additives that threaten drinking water supplies.

*Recommendations for Evaluating and Learning From Experience*

The introduction of reformulated gasoline has had substantial air quality benefits, but has at the same time raised significant issues about the questions that should be asked before widespread introduction of a new, broadly-used product. The unanticipated effects of RFG on groundwater highlight the importance of exploring the potential for adverse effects in all media (air, soil, and water), and on human and ecosystem health, before widespread introduction of any new, broadly-used, product.

16. In order to prevent future such incidents, and to evaluate of the effectiveness and the impacts of the RFG program, EPA should:

- d. Conduct a full, multi-media assessment (of effects on air, soil, and water) of any major new additive to gasoline prior to its introduction.
- e. Establish routine and statistically valid methods for assessing the actual composition of RFG and its air quality benefits, including the development, to the maximum extent possible, of field monitoring and emissions characterization techniques to assess "real world" effects of different blends on emissions
- f. Establish a routine process; perhaps as a part of the Annual Air Quality trends reporting process, for reporting on the air quality results from the RFG program.
- g. Build on existing public health surveillance systems to measure the broader impact (both beneficial and adverse) of changes in gasoline formulations on public health and the environment.

*Appendix A*

In reviewing the RFG program, the panel identified three main options (MTBE and other ethers, ethanol, and a combination of alkylates and aromatics) for blending to meet air quality requirements. They identified strength and weaknesses of each option:

*MTBE/other ethers:* A cost-effective fuel blending component that provides high octane, carbon monoxide and exhaust VOCs emissions benefits, and appears to contribute to reduction of the use of aromatics with related toxics and other air quality benefits; has high solubility and low biodegradability in groundwater, leading to increased detections in drinking water, particularly in high MTBE use areas. Other ethers, such as ETBE, appear to have similar, but not identical, behavior in water, suggesting that more needs to be learned before widespread use

*Ethanol:* An effective fuel-blending component, made from domestic grain and potentially from recycled biomass, that provides high octane, carbon monoxide emission benefits, and appears to contribute to reduction of the use of aromatics with related toxics and other air quality benefits; can be blended to maintain low fuel volatility; could raise responsibility of increased ozone precursor emissions as a result of commingling in gas tanks if ethanol is not present in a majority of fuels; is produced currently primarily in Midwest, requiring enhancement of infrastructure to meet broader demand; because of high biodegradability, may retard biodegrada-

tion and increase movement of benzene and other hydrocarbons around leaking tanks.

*Blends of Alkylates and Aromatics:* Effective fuel blending components made from crude oil; alkylates provide lower octane than oxygenates; increased use of aromatics will likely result in higher air toxics emissions than current RFG; would require enhancement of infrastructure to meet increased demand; have groundwater characteristics similar, but not identical, to other components of gasoline (i.e. low solubility and intermediate biodegradability)

*Appendix B*

*Members of the Blue Ribbon Panel*

Dan Greenbaum, Health Effects Institute, Chair  
 Mark Buehler, Metropolitan Water District, So. California  
 Robert Campbell, Chairman and CEO, Sunoco Inc.  
 Patricia Ellis, Hydrogeologist, Delaware Department of Natural Resources and Environmental Conservation  
 Linda Greer, Natural Resources Defense Council  
 Jason Grumet, NESCAUM  
 Anne Happel, Lawrence Livermore Nat. Lab  
 Carol Henry, American Petroleum Institute  
 Michael Kenny, California Air Resources Board  
 Robert Sawyer, University of California, Berkeley  
 Todd Sneller, Nebraska Ethanol Board  
 Debbie Starnes, Lyondell Chemical  
 Ron White, American Lung Assoc.

*Federal representatives (Non-Voting)*

Robert Perciasepe, Air and Radiation, US EPA  
 Roger Conway, US Dept. of Agriculture  
 Cynthia Dougherty, Drinking Water, U.S. EPA  
 William Farland, Risk Assessment, US EPA  
 Barry McNutt, US DOE  
 Margo Oge, Mobile Sources, US EPA  
 Samuel Ng, Underground Tanks, US EPA  
 Mary White, ATSDR  
 John Zogorski, USGS

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TODD C. SNELLER, MEMBER, EPA BLUE RIBBON PANEL—SUMMARY OF  
 DISSENTING OPINION

In its report regarding the use of oxygenates in gasoline, a majority of the Blue Ribbon Panel on Oxygenates in Gasoline recommends that action be taken to eliminate the current oxygen standard for reformulated gasoline. Based on legislative history, public policy objectives, and information presented to the Panel, I do not concur with this specific recommendation. The basis for my position follows:

1. The Panel's report concludes that aromatics can be used as a safe and effective replacement for oxygenates without resulting in deterioration in VOC and toxic emissions. In fact, a review of the legislative history behind the passage of the *Clean Air Act Amendments of 1990* clearly shows that Congress found the increased use of aromatics to be harmful to human health and intended that their use in gasoline be reduced as much as technically feasible.

2. The Panel's report concludes that oxygenates fail to provide overwhelming air quality benefits associated with their required use in gasoline. The Panel recommendations, in my opinion, do not accurately reflect the benefits provided by the use of oxygenates in reformulated gasoline. Congress correctly saw a minimum oxygenate requirement as a cost effective means to both reduce levels of harmful aromatics and help rid the air we breathe of harmful pollutants.

3. The Panel's recommendation to urge removal of the oxygen standard does not fully take into account other public policy objectives specifically identified during Congressional debate on the *1990 Clean Air Act Amendments*. While projected benefits related to public health were a focal point during the debate in 1990, energy security, national security, the environment and economic impact of the *Amendments* were clearly part of the rationale for adopting such amendments. It is my belief that the rationale behind adoption of the *Amendments* in 1990 is equally valid, if not more so, today.

Congress thoughtfully considered and debated the benefits of reducing aromatics and requiring the use of oxygenates in reformulated gasoline before adopting the ox-

xygenate provisions in 1990. Based on the weight of evidence presented to the Panel, I remain convinced that maintenance of the oxygenate standard is necessary to ensure cleaner air and a healthier environment. I am also convinced that water quality must be better protected through significant improvements to gasoline storage tanks and containment facilities. Therefore, because it is directly counter to the weight of the vast majority of scientific and technical evidence and the clear intent of Congress, I respectfully disagree with the Panel recommendation that the oxygenate provisions of the federal reformulated gasoline program be removed from current law.

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LYONDELL CHEMICAL COMPANY—SUMMARY OF DISSENTING REPORT

While the Panel is to be commended on a number of good recommendations to improve the current underground storage tank regulations and reduce the improper use of gasoline the Panel's recommendations to limit the use of MTBE are not justified.

Firstly, the Panel was charged to review public health effects posed by the use of oxygenates, particularly with respect to water contamination. The Panel did not identify any increased public health risk associated with MTBE use in gasoline.

Secondly no quantifiable evidence was provided to show the environmental risk to drinking water from leaking underground storage tanks (LUST) will not be reduced to manageable levels once the 1998 LUST regulations are fully implemented and enforced. The water contamination data relied upon by the panel is largely misleading because it predates the implementation of the LUST regulations.

Thirdly, the recommendations fall short in preserving the air quality benefits achieved with oxygenate use in the existing RFG program. The air quality benefits achieved by the RFG program will be degraded because they fall outside the control of EPA's Complex Model used for RFG regulations and because the alternatives do not match all of MTBE's emission and gasoline quality improvements.

Lastly, the recommendations will impose an unnecessary additional cost of I to 3 billion dollars per year (3–7 c/gal. RFG) on consumers and society without quantifiable offsetting social benefits or avoided costs with respect to water quality in the future.

Unfortunately, there appears to be an emotional rush to judgment to limit the use of MTBE. For the forgoing reasons, Lyondell dissents from the Panel report regarding the following recommendations:

- The recommendation to reduce the use of MTBE substantially is unwarranted given that no increased public health risk associated with its use has been identified by the Panel.
- The recommendation to maintain air quality benefits of RFG is narrowly limited to the use of EPA's RFG Complex Model which does not reflect many of the vehicle emission benefits realized with oxygenates as identified in the supporting panel issue papers. Therefore, degradation of air quality will occur and the ability to meet the Nation's Clean Air Goals will suffer under these recommendations.

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RESPONSES BY DANIEL GREENBAUM TO ADDITIONAL QUESTIONS FROM  
SENATOR INHOFE

*Question 1.* How many members of the Blue Ribbon Panel individually endorsed removing the oxygenate mandate?

Response. Of the 13 members of the Blue Ribbon Panel, 11 explicitly endorsed the removal of the oxygen mandate.

*Question 2.* On August 12, Carol Browner held a public briefing on the Executive Order No. 13101 to promote the use of agricultural crops and forestry products in developing fuels, electricity, and industrial products. As part of this briefing, Administrator Browner said that the Administration would not support any reopening of the Clean Air Act that did not "preserve all of the opportunities that exist for ethanol." Is this also the position of the Blue Ribbon Panel? Is this EPA position consistent with the Panel's recommendations?

Response. The Panel did not have the expertise nor the charge to fully review all of the existing incentive programs for the use of ethanol and thus did not explicitly endorse any particular approach. We did, in recommending the lifting of the oxygen mandate (recommendation 10) state:

The panel recognizes that Congress, when adopting the oxygen requirement, sought to advance several national policy goals (energy security and diversity,

agricultural policy, etc) that are beyond the scope of our expertise and deliberations.

The panel further recognizes that if Congress acts on the recommendation to remove the requirement, Congress will likely seek other legislative mechanisms to fulfill these other national policy interests.

*Question 3.* The ethanol industry has stated that it could bring substantial new production capacity online in a short time frame. They estimate that new capacity could be built in 3 years to meet MTBE demand—nearly double current ethanol production. Did the Blue Ribbon Panel reach a consensus or evaluate the ability of the ethanol industry to replace MTBE demand within this ambitious 3 year window?

Response. The Panel did review the ability of the Ethanol industry to increase its capacity, and concurred that there could be a rapid increase in capacity (perhaps 20 percent) quite quickly because of existing permitted but unused capacity. The Panel could not agree on the timing for the industry to substantially increase its capacity (i.e. doubling) because of questions about the ability of existing and likely future rail and shipping infrastructure to transport the increased volumes in a timely and cost-effective manner to the East and West coasts.

*Question 4.* The report seems to imply that 20 parts per billion of MTBE in drinking water would be cause for “public health concern.” What scientific evidence did the Blue Ribbon Panel examine which indicated that 20 parts per billion of MTBE in drinking water, even if exposed for a lifetime, causes human beings to become sick?

Response. Actually the Panel did *not* make a determination that levels above 20 ppm would be a cause for public health concern. Rather, we used that level to indicate the lower level of the advisory issued by EPA (and also incorporated in the majority of standards in those states that have set standards). This range—20–40 ppm—was selected by U.S. EPA and the states based primarily on knowledge that at these levels taste and odor concerns would arise among users, and that based on existing evidence, adverse health effects are not likely to occur at or below these levels.

*Question 5.* The Blue Ribbon Panel Report indicates that several states have set guidelines for MTBE in drinking water under 100 parts per billion on the basis of health concern. To your knowledge, did any of the administrators in these states have better or more complete scientific information regarding MTBE health effects than does the federal EPA?

Response. The only health data which has been used by states that was not incorporated in the EPA advisory was that used by California to set its Water Quality Goal at 13 ppm. This was based on the study of Belpoggi, et al cited in the Panel’s report which found an increase in leukemia/lymphoma in laboratory rats that ingested relatively high levels of MTBE for a lifetime. Because of questions about the interpretation of this study, neither the International Agency for Research on Cancer, nor the National Institute of Environmental Health Sciences (in preparing its Biannual Report to Congress on carcinogens) felt that this study was adequate to be used for human risk assessment purposes.

*Question 6.* How much of the MTBE problem in California is caused by the recreational use of the water reservoirs?

Response. Although the Panel did not have a precise quantitative estimate of this, by far the vast majority of the contamination in California involved leaking underground fuel storage systems, with perhaps a rough estimate of 10 percent of the problems involving water craft.

*Question 7.* On July 27, 1999, the Blue Ribbon Panel issued its recommendations on the use of MTBE. In the Executive Summary, the Panel made several recommendations to enhance, accelerate, and expand existing programs to improve the protection of drinking water supplies. Recommendation No. 6 proposes the development of an integrated field research program to study the groundwater behavior of gasoline and oxygenates, including ethanol. Is it somewhat premature to promote substitutes for MIBE until this research on oxygenates is completed?

Response. It would definitely be premature to move to other oxygenates that are ethers (e.g. ETBE, TAME) and the Panel recommended explicitly in Recommendation 13 that these not be placed in wider use until fully tested. The Panel did have some water behavior information on other alternatives to MTBE—ethanol and other components refined from crude oil (e.g. alkylates). The other components from crude oil clearly have characteristics that would make them less likely to contaminate groundwater than even the existing components of gasoline. The Panel also saw some evidence that the high biodegradability of ethanol might cause somewhat

longer plumes of benzene and other components of gasoline—thus the recommendation for field studies—but did not identify likely problems comparable to those experienced with MTBE.

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RESPONSES BY DANIEL GREENBAUM TO ADDITIONAL QUESTIONS FROM  
SENATOR BOXER

*Question 1.* The Panel report concluded that MTBE presents a risk to drinking water and that its use should therefore be substantially reduced, with some members supporting a complete phase out. As a means of dealing with this drinking water threat, the Panel prescribes this solution:

The Panel recommends that Federal law be amended to clarify EPA and state authority to regulate and/or eliminate gasoline additives that threaten water supplies. It is expected that this would be done initially on a national level to maintain uniformity in the fuel supply. For further action by the states, the granting of such authority should be based upon a similar two part test: (1) states must demonstrate that their water resources are at risk from MTBE use, above and beyond the risk posed by other gasoline components at levels of MTBE use present at the time of the request. (2) states have taken necessary measures to restrict/eliminate the presence of gasoline in the water resource.

Isn't it true that merely providing EPA and/or the states with the authority to regulate MTBE use may lead to no change in the prevalence of MTBE in our fuel supply?

Doesn't the requirement that states prove that MTBE is causing a problem stand in the way of states stepping forward to take preventative measures to deal with what the Panel has acknowledged is a known threat?

If, as the Panel acknowledges in its report, MTBE poses a threat to drinking water requiring that its use be substantially curtailed or terminated, why didn't the Panel recommend that Congress take direct action to restrict MTBE use? Isn't this the only way to ensure that MTBE use is "substantially reduced?"

Response. There are several factors that will likely lead to a reduction in the use of MTBE: (1) the lifting of the oxygen mandate (thus allowing refiners to use readily available non-oxygenated, or less-oxygenated blends); (2) the increasing liability of refiners for the contamination caused by MTBE (and a desire to prevent the growth of these liabilities) and (3) active regulatory efforts by state and federal regulators to require reduced use of MTBE. Since a number of state have or are poised to take regulatory action, the Panel saw it as critical that they have the clear authority to regulate constituents of gasoline, and fully expect that with the authority they will move to take action.

As to the need for states to show that they have taken appropriate water protection actions before restricting use, it seemed to the Panel that states that have already taken appropriate action to prevent leaks should have no problem meeting these tests, while those that have not cleaned up their underground tanks should be expected to do that before being authorized to reduce or ban any particular part of fuel.

As to Congress taking direct action to reduce the use of MTBE, this is of course at the discretion of Congress to enact. The Panel felt that the details of any such reduction, because of the complex interactions among air quality, water protection, fuel supply and cost, and the fundamentally different groundwater situations in different states, would not best be mandated in a "one-size-fits-all" manner.

*Question 2.* You note in your testimony that you believe the San Francisco incident (where Tosco and Chevron were found to be adding large amounts of MTBE to their gasoline) was "unique." Do we know how much MTBE is used in attainment areas? It was my understanding that the only reason we discovered what was going on in San Francisco was because private parties tested the gasoline for MTBE. In other words, do we have a basis upon which to say whether the San Francisco incident was truly unique?

Response. The Panel did review existing data collected for the petroleum industry on levels of MTBE used in both RFG and non-RFG areas, and found that in general the use of MTBE in attainment areas is quite low, with some refiners using none, some refiners using as much as 5-8 percent in their premium blends, and overall perhaps 1 percent of the fuel by volume containing MTBE. The uniqueness of the San Francisco situation comes from the fact that the California fuel market, because of the tighter CalRFG fuel requirements, is served by only a limited number of refineries making only California-acceptable fuel. Because of its relative isolation, this market was particularly vulnerable to the two refinery incidents earlier this year,

resulting in lower available fuel, and higher demand for MTBE to make up for lost octane content.

*Question 3.* You note in your testimony that "I think the general evidence that the Panel saw and the analyses that the Department of Energy has done . . . suggested that if you solely removed the mandate, that economic forces probably would reduce the levels of MTBE but continue to use it at fairly high levels, because it is a relatively cost-effective blending component for gasoline, very high octane and very clean." (Transcript at 17) By this do you mean that substantial MTBE use may continue even if the oxygenate requirement were lifted?

Response. Yes, the analyses we saw would suggest that MTBE use would continue at relatively high levels if left only to direct market forces, although the DOE analysis did not factor in the growing liability concerns of refiners mentioned in 1. above, which would likely also drive down usage once the mandate is lifted. This is why the Panel also called for clarification of federal and state authority to regulate and/or eliminate MTBE use.

*Question 4.* You note in response to Senator Bennett's question about the health risks associated with MTBE that "MTBE is neither a known human carcinogen, or even a probable human carcinogen. At this stage it is in the 'possible' category. In other words, there are some animal tests that show that it causes cancer, but there are questions about those tests."

Your answer seems to imply that MTBE has been disproven to be a known human carcinogen. Isn't it accurate to say that MTBE has not been so designated because the scientific studies required to make that determination simply have not been performed?

You raise questions concerning the health tests that were performed which have found MTBE to be an animal carcinogen. Are you referring to the Belpoggi oral exposure study?

Are you aware that the California Office of Environmental Health Hazard Assessment reviewed that study in the context of establishing a drinking water public health standard for MTB and determined that the "study is valid, not critically flawed, and is consistent with reported results" and that the quality of the study was "as good or better than those typically available for chemical risk assessment." See Public Health Goal for Methyl Tertiary Butyl Ether (MTBE) in Drinking Water, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, March 1999, pp. 89-90.

Response. I was careful in my testimony to not say that MTBE has been disproven to be a carcinogen but rather to indicate that it is possible it is a carcinogen, but that the studies done to date have not been deemed adequate by state, national or international bodies to raise it into the probable or known human carcinogen category.

I was referring to two sets of questions about the animal tests of carcinogenicity: first, that the kidney tumors seen in some of the rat experiments may be unique to rats (and thus do not suggest human carcinogenicity), and second, that the combining of the leukemia and lymphoma cases in the Belpoggi study to arrive at a statistically significant increase in cancers may not have been appropriate. I am aware of the review of the Belpoggi study conducted by the California OEHHA, but also of the reviews of MTBE carcinogenicity conducted by both the International Agency for Research on Cancer (IARC), and the National Institute of Environmental Health Sciences (in preparing their Biannual Report to Congress on Carcinogens), which concluded that the current evidence, including the Belpoggi study, was not adequate to go beyond the "possible" carcinogen category.

*Question 5.* I am interested to hear about why some members of the Panel recommended "substantial reductions" in MTBE use, while other members recommended a complete elimination of use.

How many members of the panel recommended a complete MTBE phase out? What were the reasons they gave for that view?

Response. The majority of the Panel supported a substantial reduction in MTBE use and the granting of authority to EPA and the states to go beyond that if water protection justified the need. While I have not polled every member for their specific reasons, most cited a desire to reduce the risk, but continuing uncertainty as well from the groundwater data we had seen about whether MTBE at lower, historical levels, posed the same groundwater risks. Five of the Panel members indicated support for phasing out MTBE altogether, largely due to a concern that the data did not allow us to identify a safe level.

*Question 6.* You note in your testimony that one possible strategy for curing the MTBE problem would be to restrict MTBE use to pre-RFG levels (2 percent of vol-

ume). This recommendation, as I understand it, is based in part upon the theory that we only came to see MTBE contamination in the years following the implementation of the RFG program.

Isn't it possible, however, that we came to see MTBE contamination at this later time not because MTBE use had increased dramatically after the imposition of RFG program, but because we began to replace underground storage tanks during roughly the same time that MTBE use was increasing? It is my understanding that we often don't discover MTBE contamination until the tanks are removed.

Response. You are right that the interaction between the replacement of tanks and the increased use of MTBE makes it difficult to precisely identify what contamination was or still is occurring. My testimony, however, was based on the USGS data presented to the Panel and summarized in our report that showed that for the over 2,000 wells they monitor in non-RFG areas (where MTBE has been present but at lower levels) the rate of MTBE contamination has been identical to that of other components of gasoline (about 2 percent).

*Question 7.* In any event, if we know that a relatively small amount of MTBE can contaminate a drinking water source, how can we justify continuing to allow any of it to be used?

Response. We know for a fact that when MTBE use is high that there is a high probability of its contaminating water supplies. At the same time there are a number of substances, including benzene, trichloroethylene, and others, which are in everyday use in this country, and which contaminate a smaller number of water supplies, yet we have not banned their use. Rather, we have sought, when the risk is smaller, to prevent the contamination by tighter controls on placement and construction of underground storage systems, and by seeking, over the long term, substitutes that can reduce their use.

*Question 8.* How long and how extensive is the testing for MTBE contamination throughout the country?

Response. Contamination testing for MTBE in water supplies is about to be extended dramatically under rules recently promulgated by EPA under the Safe Drinking Water Act. I do not believe there is any time limit on these testing rules, although that should be confirmed by EPA.

*Question 9.* Can we solve the MTBE problem by simply upgrading all of our underground storage tanks?

Response. All but one member of the Panel felt that while it was important to continue to upgrade underground fuel storage systems, upgrading alone was not sufficient to protect water supplies, and thus the Panel called for the substantial reduction of the use of MTBE, the removal of the oxygen mandate, and the tightening of the air quality standards to ensure continuation of the current RFG benefits.

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RESPONSES BY DANIEL GREENBAUM TO ADDITIONAL QUESTIONS FROM  
SENATOR LIEBERMAN

*Question 1.* If Congress were to lift the requirement on oxygenate content, what options does the Panel propose for retaining current air quality *performance* of current reformulated gasoline blends? Specifically, if the oxygenate requirement were lifted, how can we best preserve the current performance level of air quality improvements as states move to adopt the haze 2 requirements for reformulated gasoline in the year 2000?

Response. While the Panel did not recommend any specific mechanism for retaining current air quality performance, it did note several approaches that could be used. Recommendation 11 notes:

There are several possible mechanisms to accomplish this. One obvious way is to enhance the mass-based performance requirements currently used in the program. At the same time, the panel recognizes that the different exhaust components pose differential risks to public health due in large degree to their variable potency. The panel urges EPA to explore and implement mechanisms to achieve equivalent or improved public health results that focus on reducing those compounds that pose the greatest risk.

*Question 2.* One dissenting opinion on the Blue Ribbon Panel Report raised concerns that lifting the oxygenate requirement could lead to increased use of aromatics. Because aromatics and alkylates can increase volatile organic compounds (VOC) and air toxic emissions, the dissenter suggested that the oxygenate standard is needed. What protections could be put in place to ensure that current RFG perform-



ance with regard to air toxic emissions levels is retained if the oxygenate requirement is lifted?

Response. Either of the mechanisms mentioned above could be used to ensure that the use of aromatics does not rise to a level that could reduce air quality benefits. That could take the form, as it does in CalRFG, of a cap on aromatics content, or of specific risk-based caps on specific aromatics (e.g. reducing the current 1 percent cap on benzene even further).

*Question 3.* Could you qualify the relative risk of MTBE compared to the many other hazardous constituents in gasoline?

Response. In general, the cancer potency of MTBE, if it is ultimately classified as a carcinogen, has been estimated to be lower than that of several substances in gasoline or in automotive emissions. Overall, the Northeast States for Coordinated Air Use Management (in a report cited in the Blue Ribbon panel's report), estimated that gasoline with MTBE would pose an approximately 12 percent lower cancer risk than gasoline without MTBE.

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STATEMENT OF ROBERT H. CAMPBELL, CHAIRMAN AND CEO OF SUNOCO, INC.

Good Morning Mr. Chairman and members of the committee. My name is Bob Campbell, and I am Chairman and CEO of Sunoco, Inc.—a company that is one of the largest refiners and marketers of gasoline on the East Coast of the U.S.

In this region, we produce and distribute more of the clean-burning reformulated gasoline required by the Clean Air Act than any other company. Consequently, we've learned firsthand about the benefits and burdens of the existing RFG Program.

My company is both a manufacturer and consumer of MTBE. We have used the chemical since the early 1980s for its high-octane properties in our gasoline. After the 1990 Clean Air Act Amendments were enacted, we, in partnership with others, invested nearly a quarter of a billion dollars to build a second plant—a world scale MTBE plant to supply our newly created additional needs for the oxygenate. Consequently, we know about all there is to know about the use of this additive in gasoline.

We are also a major supplier of conventional gasoline in the mid-America Region of the U.S.—Gasoline supplied from our Toledo, Ohio refinery system. Here we do not use MTBE, but rather we are a major buyer and blender of ethanol in gasoline. Therefore, we have extensive firsthand knowledge of both the benefits and limitations of ethanol in motor fuels.

Finally, through my membership on the EPA's Blue Ribbon Panel on oxygenates, I have been totally immersed over the past several months in the debate over the future of MTBE and ethanol in the RFG Program.

I offer this introduction so you will understand why I am very pleased to be given the opportunity to share my experiences and opinions with this committee.

Dr. Greenbaum has given an excellent summary of the deliberations and the recommendations of the Blue Ribbon Panel, and I'd like to salute Dan for his extraordinary accomplishment in moving a very diverse, fourteen member committee through a thicket of prickly issues to a remarkable consensus. Dan helped us develop an excellent database and a set of recommendations that I wholeheartedly endorse.

As you know, we are now embarking on the implementation of those recommendations—some of which require legislative action. Public concern about MTBE in drinking water is clearly the triggering event for the call for action. Putting aside the complex question of MTBE as a hazard to human health, it clearly should not be getting into drinking water. But regardless of how much money is spent on tank replacement and inventory control, gasoline handled by 190 million drivers will inevitably be spilled, and we now know how persistent a contaminant in water MTBE can be.

California—as it so often does—has led the way in defining the process for the elimination of this environmental problem. As you know, last March Governor Davis announced a 4-year program designed to eliminate MTBE from gasoline and yet preserve the air quality goals of the State. Critical to the achievement of that program is relief from the existing 2 percent oxygen mandate. I support Governor Davis' initiative for dealing quickly with a complex and often emotional problem.

But one needs to remember that MTBE is principally used on both the West and East Coasts of the United States. In fact more MTBE is used in the 11 East Coast states comprising the Ozone Transport Region than in California (130,000 vs 100,000 barrels per day). I can assure you that the citizens of Boston and Philadelphia are just as adamant about protecting their drinking water as the folks in Sac-

ramento and Santa Monica. Consequently, my plea to you today is to help us solve the equally serious problem of MTBE in the Northeast—and to do that we need a regional solution.

I know and understand that California's efforts are better coordinated than the group of East Coast States on this subject. But, if the current proposed legislation deals only with California, I can assure you that several of the northeastern States are poised to enact their own local solutions. The result will be a patchwork quilt of local initiatives or regulations. This will be a nightmare for companies attempting to reliably supply low cost, high quality gasoline to consumers in the 11-State region.

Before the EPA Panel published its findings and recommendations, several northeast States initiated their own legislative solution. We asked them to wait, and give us a chance to solve the problem collectively rather than individually. That is why I'm here today. I have no interest in doing anything that would delay or disrupt the Bilbray proposal. But we on the East Coast need to use that same legislative momentum to deal with the equally thorny problem in our region.

The bottom line is we can solve the problem in the Northeast in a manner similar to California only if we are also given relief from the 2 percent oxygenate mandate. If you will do that, we will be able to continue to supply RFG to those areas requiring it, in an economic manner, in reliable quantities, with the same air quality benefits. That reformulated gasoline will contain substantially reduced volumes of MTBE (the Panel called for "substantial reduction" not the elimination of MTBE).

I will tell you quite honestly, that even with all our experience in blending ethanol in gasoline in mid-America, I don't know how to accomplish in a real world, practical manner the same result in the northeast RFG system. Ethanol in RFG is successfully blended in the Chicago area, because it is a relatively small proportion of the supply from the manufacturers in that region. In my opinion, if the 2 percent mandate remains, and we are forced to directly substitute ethanol for MTBE in the large RFG volume area of the Northeast, we will have a disaster scenario for both the supplier and the consumer.

My reasoning for saying that is as follows: there are two very practical problems associated with ethanol as a blending component in East and West Coast reformulated gasolines. The first problem is the difficulty of adequate supply and economic transportation of ethanol from its point of manufacture (primarily the Midwest) to where it would be needed for blending (the East and West coasts). Because of its affinity for water, ethanol cannot be transported in common carrier pipelines and would have to be transported by rail or truck to both coasts. Let me repeat here exactly what I told the Blue Ribbon Panel this spring: Given enough time and money, an enterprising ethanol industry can expand production and create new logistics systems to address the problem. But the added cost will be immense and unnecessary.

Solving the logistics problem will still not address ethanol's second, and most critical, defect—it's high vapor pressure when blended into gasoline. The one thing we have learned in the past 10 years is that the most crucial characteristic of a successful RFG Program is vapor pressure or "Volatile Organic Compound (VOC)" control. Higher vapor pressure means increased VOC emissions which leads to more ozone pollution. The next generation of RFG—Beginning January 1, 2000—has even more stringent restrictions on vapor pressure than current RFG. Consequently, blending ethanol into future RFG would severely compound the environmental and supply problems. It is my view that ethanol cannot be practically used on the East or West Coast in the summertime period because of the low vapor pressure requirement and the high percentage of RFG that must be produced in those regions.

The solution—Legislation is needed to solve the oxygenate problem where it exists—in California, and in the ozone transport region of the East Coast. That is where 75 percent of all the RFG in the country is used, and where almost 90 percent of the MTBE is present in gasoline. That is also where water quality complaints from consumers have been most vocal. We need your help to fix what's broken. I ask you to give these two regions three things:

- The authority to regulate the use of oxygenates when water quality impacts are substantiated.
- A waiver of the 2 percent oxygenate mandate for RFG.
- The requirement that no current clean air benefits be compromised as a result of these changes to the Federal fuel program.

Congressman Jim Greenwood of Pennsylvania is attempting to advance this precise solution in the House Commerce Committee. Prompt, parallel action in your committee can help avoid the transportation fuel crisis that I see on the horizon, and I urge you to move quickly.

I appreciate the opportunity to share these thoughts with you, and I look forward to any questions you may have.

SUNOCO, INC.,  
Philadelphia PA, November 5, 1999.

Hon. JAMES M. INHOFE,  
Hon. BOB GRAHAM,  
U.S. Senate,  
Washington, DC.

DEAR SENATORS: I appreciate the opportunity you provided me to appear before your Subcommittee regarding the EPA's Blue Ribbon Panel on oxygenates.

As I testified, I support the recommendation of the Panel and consider the report to be the definitive study of the problems associated with the oxygenated fuel program. I also agree that congressional action is required for many of the corrections which are needed. This is a complex issue with many implications for energy and environmental policy, and it is certainly timely for your committee to begin to address it. Action is needed now.

I have offered answers to the questions you sent me on the attached sheet and I will be pleased to amplify these or address other concerns as you see fit.

I would also like to respond to a question that was posed to me at the hearing which I believe can be more clearly answered in a written response. Senator Voinovich asked whether simply granting states the authority to regulate oxygenates would solve the MTBE problem. If my answer seemed somewhat equivocal, it is only because our discussions on this topic during the Panel's deliberations left me believing that the states would like *some* policy guidance on how to proceed. No one—state regulators, refiners or consumers—want to create boutique, individual state fuel formulas. Consequently, the Panel actually recommended that what was needed was “. . . action by Congress to clarify federal *and* state authority . . .” in this area.

I believe that a legitimate Congressional function would be to codify the principle findings of the Panel as *policy guidelines* for state actions. Congressman Jim Greenwood has begun work on an amendment which captures this concept and clearly establishes the future roles for EPA and the states in regulating fuel oxygenates without risking a multiplicity of state fuel formulations. A copy of a recent draft of this amendment is attached for your review.

Thank you again for the opportunity to testify. Please feel free to contact me if further information is needed.

Sincerely,

ROBERT H. CAMPBELL.

## SEC. 2. STATE WAIVER OF OXYGEN REQUIREMENTS

Section 211 of the Clean Air Act (42 U.S.C. 7545) is amended by adding the following new subsection at the end thereof:

“(p) WAIVER OF OXYGEN REQUIREMENTS.—

“(1) IN GENERAL.—Upon the petition of any State referred to in section 184(a), the Administrator shall waive or reduce (in accordance with the State petition) any oxygen content requirement in effect under subsection (k) for that State.

“(2) ACTION BY ENVIRONMENTAL PROTECTION AGENCY.—Not later than 180 days after the date of receipt of a petition submitted under paragraph (1), the Administrator shall grant the petition. If, by the date that is 180 days after the date of receipt of a petition submitted under paragraph (1), the Administrator has not granted the petition, the petition shall be deemed to be granted.

“(3) FEDERAL CONTROL OF FUEL OXYGENATES.—The regulations under this section shall be revised by January 1, 2001, to provide a schedule for the reduction of the use of methyl tertiary butyl ether (MTBE) in reformulated gasoline (as defined in subsection (k)), introduced into commerce in any state for which a waiver or reduction is in effect and referred to in section 184(a). By January 1, 2005 the maximum content of MTBE by volume for all gasoline, shall be no more than 5 percent. Reductions below this amount, or on a schedule different than that set out by regulation, may only be authorized upon petition to the Administrator by a state for which a waiver or reduction is in effect under this subsection, and only upon a finding that further reduction of MTBE use is necessary to protect human health or the environment.

“(4) AIR TOXIC EMISSION CONTROL ENHANCEMENT REQUIREMENT.—No manufacturer or processor of any fuel or fuel additive may sell, or offer for sale, or introduce

into commerce any reformulated gasoline (as defined in subsection (k)) for resale in any State for which a waiver or reduction is in effect under this subsection unless the aggregate emissions of toxic air pollutants from baseline vehicles when using baseline gasoline shall be reduced by an annual average of at least 27 percent.

“(5) ASSURANCE OF ADEQUATE FUEL SUPPLY.—Any regulation for modification of fuel properties in this subsection shall be consistent with reasonable schedules for necessary refinery investment projects and appropriate fuel distribution system modifications to assure adequate supply for the states defined in section 184(a).

RESPONSES BY ROBERT H. CAMPBELL TO ADDITIONAL QUESTIONS FROM  
SENATOR INHOFE

*Question 1.* Are both the elimination of the 2 percent by weight oxygen mandate and a goal of no backsliding on current air quality levels achievable goals for the refining industry? How could this be accomplished?

Response. Refiners can adjust to the elimination of the 2 percent oxygenate mandate while maintaining current air quality benefits. The experience of the industry in California is instructive. CARB regulations for fuel composition have for years focused on emission standards, not on government-specified formulas for various fuel components. Given federal emission targets, refiners will individually adjust fuel components and plant processes to produce gasoline which can meet these requirements.

Regarding the concern about “backsliding” from current levels of air toxics improvements, we would prefer that the Congress set any new minimum standards rather than allow state-by-state results predicated on unaudited baseline data.

*Question 2.* The refining industry will be facing a number of fuel initiatives over the next few years including reducing the sulfur content of gasoline and diesel fuel. Congressional action to remove the 2 percent oxygen mandate for RFG would provide more flexibility for the industry to meet these challenges. In contrast, renewable fuels mandates would place further constraints on the industry. Should Congress and EPA provide more, not less flexibility to the refining industry?

Response. Without question, it is preferable to have more operational flexibility to achieve regulatory environmental goals. The domestic refining industry is presently challenged by two serious constraints: (1) limited capital for non-productive investment requirements; and (2) current refinery operations at nearly 100 percent of operating capacity. When you overlay the multiple impacts of mandated MTBE reduction on top of the imposition of severe sulfur reduction in gasoline, the stage is set for a “train wreck” in the domestic fuel supply system *absent some Congressional—required coordination of these schedules*. The introduction of yet another, simultaneous fuel composition change—a mandated ethanol content—will severely exacerbate this problem.

*Question 3.* The Northeast States for Coordinated Air Use Management (NESCAUM) recently released a strategy to reduce MTBE use in the Northeast. NESCAUM recommends that EPA propose regulations to cap the MTBE content in gasoline over a three-year period to minimize adverse impacts. NESCAUM is very concerned that changes to gasoline formulation be implemented with adequate lead-time to avoid supply instability and unacceptable increases in gasoline prices. What do you think of the proposal’s three-year lead-time?

Response. We support NESCAUM’s overall approach to addressing the MTBE issue and believe that crafting a regional solution for the Ozone Transport Region is a reasonable and achievable Congressional goal. When coupled with a mechanism to deal with the California MTBE problem, this two-region approach would address the geographic areas where 90 percent of all MTBE is used.

Congressman Greenwood’s proposed MTBE amendment has been prepared with ongoing input from NESCAUM. This proposal would allow for a four-year transition period to achieve a new maximum use cap on MTBE. We believe that 4 years may be a more realistic time frame and this view is shared by most of the refining industry. Again, with the simultaneous imposition of new sulfur reduction rules, more time to comply with both of these mandates is essential.

*Question 4.* I understand that there are vapor pressure issues and increased vehicle emissions associated with replacing MTBE volumes with ethanol. What adjustments and investments would be necessary at a refinery in order to produce gasoline for blending with ethanol?

Response. The most serious limitation with ethanol is its high vapor pressure when mixed with gasoline. Consequently, the vehicle emissions of smog precursors increase if MTBE is replaced by ethanol without making any refinery changes. In

order to blend ethanol into gasoline and meet the vapor pressure specification, a refiner must first reduce the vapor pressure of the base gasoline by distilling out the pentane fraction. This approach can allow the final ethanol blended gasoline to meet the vapor pressure specification, but according to testimony at the BRP, it also reduces the amount of gasoline by about 5 volume percent. This represents not only a major reduction in the U.S. gasoline pool, but it also eliminates the most environmentally beneficial fraction of the refining process.

While the cost for distillation columns within a refinery is quite significant, it only represents a portion of the total investment that is needed in order to replace MTBE with ethanol. Significant investment is needed at each terminal and some expenditure will be needed at each retail site because of the necessity of splash blending ethanol near ultimate distribution points.

By comparison however, the worst case scenario for refiners and for consumers would be to limit or ban MTBE usage while maintaining the 2 percent oxygenate mandate. Every regulatory body which has considered this option—EPA, CARB and NESCAUM—uniformly rejects this approach because of unavoidable impacts on the price and supply of gasoline.

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STATEMENT OF MICHAEL P. KENNY, EXECUTIVE OFFICER, CALIFORNIA AIR  
RESOURCES BOARD

Thank you, Chairman Inhofe and members of the subcommittee for holding today's hearing on The Report of U.S. EPA's Blue Ribbon Panel on Oxygenates in Gasoline. As the California state representative on the panel, I am pleased to be here on behalf of Governor Gray Davis, the California Environmental Protection Agency and the California Air Resources Board to discuss our state's perspective on the report and its findings.

As the report noted, California has its own reformulated gasoline program, which was established by the Air Resources Board to deal with California's unique air-quality problems. California's RFG program differs from the federal program in a number of ways.

Most notably, the California program contains limits on the sulfur and aromatic content of gasoline, while the federal program does not. California's program also utilizes a predictive model that enables refiners to market innovative fuel formulations that vary from California's gasoline specifications, as long as refiners can demonstrate using the model that the formulations provide the required air-quality benefits.

The California RFG program has been an unqualified success. Analyses of weather data and air pollution levels indicate that, following its introduction in 1996, California RFG reduced peak ozone levels in Los Angeles by about 10 percent. Airborne benzene levels throughout California decreased by 50 percent.

California RFG reduces smog-forming emissions from motor vehicles by 15 percent, and it reduces cancer risk from exposure to motor vehicle toxics by about 40 percent. These are about twice the air-quality benefits produced by Phase 1 Federal RFG, and they still exceed somewhat the benefits of Phase 2 Federal RFG, which will be introduced in much of the country in January 2000.

Unfortunately, the continuing controversy over MTBE has overshadowed the success of California RFG. Two California cities, Santa Monica and South Lake Tahoe, have seen their domestic water supplies decimated by MTBE contamination, and MTBE has been found in groundwater at several thousand leaking underground tank sites in California. But, as the Blue Ribbon Panel report emphasized, MTBE contamination is truly a national problem. The USGS/EPA Northeastern study found that MTBE is detected 10 times more often in community drinking-water systems in areas using oxygenated fuels than in areas using non-oxygenated fuels.

California took its own proactive steps to remedy its MTBE problem this past March, when Governor Davis declared MTBE to be an environmental risk and ordered its elimination from California gasoline by the end of 2002. Governor Davis followed the recommendation of a comprehensive assessment of MTBE by the University of California.

We are extremely pleased with the Blue Ribbon Panel's recommendations for a substantial reduction of MTBE use, and for a clarification of both federal and state authority to eliminate the use of additives that threaten drinking water supplies. Both recommendations back up Governor Davis' order. However, perhaps the single most crucial factor affecting California's ability to eliminate MTBE use is the federal 2 percent oxygen requirement. The Blue Ribbon Panel's recommendation for the elimination of that requirement is absolutely critical for both the environmental and

economic well-being of California. I would like to discuss this recommendation in more detail.

California does not believe that there is a technical or scientific basis for requiring the addition of oxygen to gasoline. Oxygenates are an important tool for making reformulated gasoline, and in general, oxygenates should remain an option that is available to refiners. But there is absolutely no reason to mandate them. It is possible to make both California and federal reformulated gasoline without oxygen, and it is much more cost-effective to let each refiner decide for itself whether to use oxygenates.

About 70 percent of the California gasoline market is subject to the federal 2 percent oxygen rule. In the other 30 percent of the market, at least three refiners have produced and sold non-oxygenated gasoline that provides all the air-quality benefits required of California RFG. In 1998, a substantial amount of the remaining gasoline in that market contained less than 2 percent oxygen. The Blue Ribbon Panel report pointed out that California's predictive model, along with its sulfur and aromatics requirements, ensure that non-oxygenated formulations developed by refiners provide the same air-quality benefits as standard California RFG formulations.

The same cannot be said of Phase 1 Federal RFG—the Blue Ribbon Panel report notes the concern that the elimination of oxygenates could cause a backsliding of benefits due to the higher use of aromatics. There is a need for U.S. EPA and the Congress to address this issue, but please understand: It does *not* apply to California. California has shown that it can deliver the full benefits of its world-leading RFG program without an oxygen requirement of any kind.

The federal oxygen rule has awkwardly bifurcated California into two states.

In San Francisco, which is not subject to the requirement, it is possible to buy non-oxygenated gasoline. This non-oxygenated gasoline is California RFG; it meets all our requirements. However, if an oil company were to try to sell that gasoline two hours up the highway in Sacramento or six hours away in Los Angeles, it would be in violation of federal law even though the non-oxygenated gasoline provides the same air-quality benefits as the oxygenated gasoline mandated in Sacramento and Los Angeles.

Once MTBE is eliminated in California, the only feasible oxygenate will be ethanol. If the 2 percent oxygen rule remains in effect, ethanol will be effectively mandated in 70 percent of California gasoline. California welcomes the prospect of increased ethanol use that will almost certainly occur even without a federal mandate. The continuance of an oxygen requirement in California, however, raises serious economic questions.

In just 3 years, California would need about half of the amount of ethanol as the amount currently produced in the midwestern states. The Blue Ribbon Panel report acknowledges the large investment in infrastructure that would be needed over the next 3 years to meet this large demand. There is a cost to this:

The California Energy Commission estimates that the elimination of MTBE would add 6 to 7 cents a gallon to gasoline costs if the oxygen requirement remains in effect. This would amount to an average cost of \$40 per year per California motorist, or \$840 million per year to California motorists as a whole. Elimination of the requirement would allow gasoline costs to remain stable and possibly decrease by one cent a gallon. It is patently unfair—and makes no economic sense—to saddle California motorists with this extra \$840 million cost, particularly because it would not even buy a single pound of additional air-quality benefits.

Let me be absolutely clear: This is not an ethanol issue. It is about the free marketplace. We expect ethanol to gain a new importance in California. But the market—not federal rules—should determine how much ethanol is used in California. In addition to the economics, it also is a matter of common sense. We have seen what happened when California and the nation in general became too dependent on a single additive, MTBE. Why should California simply trade its dependence on MTBE for an identical dependence on ethanol, when we can have a diverse and stable RFG marketplace featuring a range of ethanol-based and non-oxygenated formulations?

This past spring, California asked U.S. EPA for a waiver from the 2 percent oxygen requirement. We have exchanged technical correspondence with U.S. EPA on this issue and we are still awaiting their decision. At the same time, California continues to support legislation by Senator Feinstein and Representative Brian Bilbray (S. 266/H.R. 11) that, at the very least, would exempt California and possibly other states from the requirement.

I urge the committee to support the Blue Ribbon Panel's recommendation to eliminate the 2 percent requirement, and I especially urge you to support legislation that would provide California with an early exemption from that requirement. Refiners need to make decisions regarding plant modifications needed to produce non-MTBE

gasoline by the end of 2002. Bear in mind that refinery modifications can take 2½ to 3 years or longer to complete—environmental reviews and permitting typically take 12 to 18 months, engineering work takes 6 to 12 months, and construction can take 12 to 24 months. In order to complete these plant modifications within 3 years, refiners need to know now whether they will have to continue to use 2 percent oxygen or have the flexibility to produce non-oxygenated formulations.

In closing, I would like to emphasize that California has the need and the capability to produce RFG without an oxygen requirement.

As an arid state, we are more dependent than most other states on our ground-water resources, and we have an RFG program in place that can ensure the use of non-oxygenated fuel without sacrificing air-quality benefits.

Thank you once again for providing me with the opportunity to testify here today.

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RESPONSES OF MICHAEL KENNY TO ADDITIONAL QUESTIONS FROM SENATOR INHOFE

*Question 1.* I understand this past July the California Energy Commission recommended Governor Davis not to advance the removal of MTBE from California's gasoline any earlier than December 31, 2002. They also asked that this date not apply to downstream locations such as pipelines, terminals and service stations. These downstream locations should have a later MTBE-free compliance date. CEC listed several reasons, including time for refinery modifications and terminal modifications to add ethanol-blending facilities. What is your reaction to this schedule? Is it appropriate, unnecessarily long, and too short?

Response. We agree with the CEC report, which recommended the staged approach that was used for the implementation of California Phase 2 Reformulated Gasoline (CaRFG2) in 1996. That approach allowed an additional 90 days from the compliance date at the refinery to the compliance date at the service station. This is precisely what we are proposing in the California Phase 3 RFG (CaRFG3) regulations which will be considered for adoption by the ARB on December 9, 1999. (The staff report has been publicly available since October 22, 1999.)

*Question 2.* I understand that CARB has raised air quality concerns about replacements for MTBE. What are your concerns?

Response. The air quality concerns raised by the ARB staff were presented in a letter dated July 9, 1999 to Mr. Robert Perciasepe, Assistant Administrator for Air and Radiation, U.S. EPA. These concerns were further explained in a letter dated September 20, 1999 to Ms. Margo Oge, Director, Office of Mobile Sources, U.S. EPA. Both letters are attached. Basically, as explained in the letters, more air quality benefits can be attained after the elimination of the use of MTBE, if the two weight percent oxygen requirement for federal reformulated gasoline is removed.

Specifically, for the use of ethanol as a replacement for MTBE, the use of ethanol in RFG results in an increase in volatility that must be offset to avoid the loss of emission benefits. This would mean that all pentanes would have to be removed at a significant cost. In my testimony I had indicated that the cost would be about 6 cents per gallon. Upon further evaluation, we now estimate the cost to be no more than 3 cents per gallon.

*Question 3.* If EPA granted California's request for a waiver of the 2 percent by weight oxygen content requirement for federal RFG, would the state's concerns with MTBE be eliminated? What is the status of the waiver request at the Agency? Why is it taking so long?

Response. The concerns with MTBE do not change with a waiver from the federal RFG oxygen requirement. A waiver from the oxygen mandate in federal RFG sold in California would provide the most expeditious and least-costly phase-out of MTBE in California.

The regulatory mandate imposed by the U.S. EPA pursuant to the federal Clean Air Act requires that federal RFG contain at least 2.0 percent by weight oxygen year-round. About 70 percent of all gasoline sold in California is subject to the federal reformulated gasoline requirements.

The CaRFG2 requirements result in greater emission benefits than federal RFG, but do not require a minimum concentration of oxygen in all gasoline. Application of the current minimum oxygen content requirement serves no essential purpose in meeting California's air quality goals to reduce ozone and particulate matter precursors, and toxic pollutant emissions, from vehicles. The results of the University of California study, a National Research Council study, and a U.S. EPA Blue Ribbon Panel report all support the position that oxygen is not necessary for reformulated gasoline to provide the same or better ozone benefits as gasoline containing oxygen.

The request for a waiver is currently being evaluated by the U.S. EPA. Although it is crucial to hear as soon as possible, we have no information on when a decision will be made.

*Question 4.* Related to this issue, I understand that last Thursday an amendment was added to H.R. 11, a reformulated gasoline bill that is specific to California, in the House Health and Environment Subcommittee. This amendment purported to maintain the air quality benefits of reformulated gasoline if the oxygen content mandate is lifted. Does CARB believe that such an amendment was needed? Do you believe that this amendment improved CARB's ability to develop new gasoline formulations to improve air quality, or tied its hands by not only telling you what the goal would be for the gasoline formulation, but also prescriptive requirements on how to do it?

Response. The amendment would have little impact in California because Senate Bill 989 (Sher), signed by Governor Davis on October 10, 1999, already requires the ARB to ensure that the CaRFG3 regulations maintain or improve upon emissions and air quality benefits achieved by CaRFG2 as of January 1, 1999—both for ozone precursors identified in California's ozone SIP, and for air toxic compounds. These are the same emissions targeted by the federal RFG program.

*Question 5.* In your testimony, you mention that it is possible to make California reformulated gasoline without oxygen. Is it feasible to increase the production of such non-oxygenated RFG without providing refiners more regulatory flexibility?

Response. Currently, some gasoline is sold in California without oxygen in areas of the state not subject to the federal RFG requirements. In the short term, it is not feasible to substantially increase production of non-oxygenated gasoline if the federal oxygen mandate requires oxygen to be used in 70 percent of the state's gasoline. We expect that the production of non-oxygenated gasoline would increase substantially in the near term with a waiver from the federal RFG oxygen requirement. The proposed CaRFG3 regulations would also improve compliance flexibility and would significantly reduce the loss in production associated with the loss of MTBE as a blending component.

*Question 6.* Is it true that refiners have said that it is not cost-effective to expand production of non-oxygenated RFG without adding greater flexibility to the fuel regulations? Have you assessed the air quality impacts of affording refiners this greater flexibility?

Response. Eliminating MTBE from California gasoline will result in a loss in gasoline production, and additional flexibility would mitigate the loss in volume, therefore lowering the overall cost of compliance.

In developing its CaRFG3 proposal, the staff was sensitive to the loss in production volume, and some of the proposed changes to the CaRFG3 specifications were made to help refiners recover volume. Specifically, the proposed increase to the T90 and T50 specifications were made to provide refiners with flexibility to increase gasoline production. The staff was able to provide this flexibility while preserving the emission benefits of the current program because of the proposed tightening of the specifications for sulfur and benzene.

The emission benefits of the proposed CaRFG3 regulations are described in the responses to questions 7 and 9.

*Question 7.* More specifically, the ARB draft specifications propose relaxing five of eight categories of regulated fuel parameters. Does your analysis of non-oxygenated RFG, which concludes that such fuel can provide the same air quality benefits as oxygenated fuels include a consideration of the environmental impacts of relaxing these parameters?

Response. Our analysis fully considers the impacts of the proposed CaRFG changes. The following table provides a summary of the current CaRFG2 and proposed CaRFG3 standards.

Table 1.—Current and Proposed CaRFG Property Limits

Property	Flat Limits		Averaging Limits		Cap Limits	
	CaRFG 2	CaRFG 3	CaRFG 2	CaRFG 3	CaRFG 2	CaRFG 3
RVP, psi, max .....	7.0	7.0 <sup>(1)</sup>	none	none	7.0	6.4–7.2
Benzene, vol. %, max .....	1.00	0.80	0.80	0.70	1.20	1.10
Sulfur, ppmw, max .....	40	20	30	15	80	60/30 <sup>(2)</sup>
Aromatic HC, vol. %, max .....	25	25	22	22	30	35
Olefins, vol. %, max .....	6.0	6.0	4.0	4.0	10	10



Table 1.—Current and Proposed CaRFG Property Limits—Continued

Property	Flat Limits		Averaging Limits		Cap Limits	
	CaRFG 2	CaRFG 3	CaRFG 2	CaRFG 3	CaRFG 2	CaRFG 3
Oxygen, wt. % .....	1.8 to 2.2	1.8 to 2.2	none	none	0–3.5	0–3.5 <sup>(3)</sup>
T50 °F, max .....	210	211	200	201	220	225
T90 °F, max .....	300	305	290	295	330	335
Driveability Index .....	none	1225	none	none	none	none

<sup>1</sup>Equal to 6.9 psi if using the evaporative element of the Predictive Model.

<sup>2</sup>60 ppmw will apply 12/31/2002; 30 ppmw will apply 12/31/2004.

<sup>3</sup>3.7 wt% if gasoline contains no more than 10 vol % ethanol.

For each batch of gasoline being shipped from a refinery, a refiner can choose to meet the flat or averaging limits for each property shown in the table. But most refiners comply by using the California Predictive Model, which was developed from thousands of emissions data points generated in tests evaluating the emissions impacts of gasoline properties. A refiner using the Predictive Model selects a set of alternative specifications for the regulated properties, never exceeding a cap limit and indicating whether each alternative limit is a flat or averaging limit. The Predictive Model compares the emissions performance from these alternative limits with the emissions performance from the corresponding limits in the table above. The set of alternative specifications is only allowed if the Predictive Model shows there will be no increase in emissions of HC, of NO<sub>x</sub>, and of potency-weighted toxics compared to emissions from the limits specified in the regulations. For instance, a refiner might identify a less stringent aromatics limit in conjunction with a more stringent sulfur limit, so that the HC reductions associated with the sulfur limit offset the HC increase from the change in aromatics. Under the Predictive Model, gasoline formulations meeting the HC, NO<sub>x</sub> and toxics emissions criteria are allowed whether the oxygen is zero or 3.5 wt.%—except when oxygenates are mandated in greater Los Angeles in the winter.

As can be seen from the table, the CaRFG3 proposal would lower the flat, averaging and cap limits for two properties (sulfur and benzene) and raise the flat, averaging and cap limits for two other properties (T50 and T90). The aromatics cap would go up, but there would be no change in the aromatics flat and averaging limits. There would be no change for olefins, essentially no change for oxygen, refiners could vary RVP using the Predictive Model for the first time, and a new Driveability Index flat limit would be added.

The increases in stringency for sulfur and benzene in CaRFG3 would offset the relaxations for T50 and T90. The CaRFG3 Predictive Model can be used to directly compare the emissions impact of the CaRFG2 flat limits with the CaRFG3 flat limits. This comparison shows the CaRFG3 specifications reduce NO<sub>x</sub> by 3.3 percent, exhaust hydrocarbons by 0.9 percent, and potency-weighted toxics by 1.1 percent. This translates to additional reductions of 4 tons per day of hydrocarbon and 27 tons per day of NO<sub>x</sub>, compared to the CaRFG2 requirements. Furthermore, our analysis of effects on other media in our staff report concludes that there are no substantial adverse effects associated with the compounds expected to be used to replace MTBE.

*Question 8.* You stated in your testimony that the concerns about backsliding and aromatics do not apply to California. Does that mean that non-oxygenated RFG in California will not contain higher aromatic levels in comparison to oxygenated RFG? Could you explain in detail how the effects of non-oxygenated RFG are different for California?

Response. Unlike other states, California imposes a specific limit on aromatics content as part of the CaRFG standards. Following elimination of the federal RFG oxygen mandate, the CaRFG aromatics limits would continue to apply in California. As indicated in the response to the previous question, the proposed CaRFG3 flat and averaging limits for aromatics are the same as the current CaRFG2 limits. The ARB is proposing a limited relaxation of the cap limit for aromatics, and this means refiners using the Predictive Model could identify a higher alternative aromatics limit under CaRFG3 than they can now under CaRFG2. However, refiners can *only* identify the higher aromatics level if they simultaneously make sufficient reductions in the alternative limits for other properties for the Predictive Model to show there will not be an overall increase in HC, NO<sub>x</sub>, and potency-weighted toxics emissions. The question of potential backsliding for the gasoline actually sold in the state is addressed in the response to the next question.

*Question 9.* Are there backsliding issues for non-oxygenated RFG related to fuel parameters other than aromatics? For instance, what are the emissions impacts of the increased levels of toxics in non-oxygenated RFG? What are the emissions impacts of higher olefin levels in such fuel? What are the overall air quality impacts of increased CO emissions from non-oxygenated RFG? Are these issues included in your analysis?

Response. As discussed above, refiners using the California Predictive Model can only apply alternative specifications that the Model shows will achieve reductions in emissions of HC, NO<sub>x</sub> and toxics that are essentially equivalent to the emissions reductions from the flat and averaging limits in the regulation. Any specification changes that increase potency-weighted toxics will have to be offset by other changes that achieve an equivalent reduction. Additionally, the proposed CaRFG3 Predictive Model will account for the contribution of CO emissions make towards ozone formation.

In making sure that the CaRFG3 proposal is consistent with the anti-backsliding requirements of Senator Sher's SB 959, we compared emissions from 1998 average California gasoline to emissions from gasoline formulations we expect to be produced under the CaRFG3 proposal. These projected CaRFG3 formulations included gasoline with oxygen at different levels and with no oxygen, and reflected the same compliance margins as were seen in 1998 gasoline. Table 2 shows the results of the staff's analysis. Note that as oxygen is increased, NO<sub>x</sub> benefits decrease. The zero oxygen fuel provides almost 5 percent greater emissions reductions (about 38 additional tons per day) over a fuel with 3.5 percent oxygen.

For hydrocarbons, the difference between the fuels is similar when accounting for the CO reduction from the oxygenated fuel as equivalent evaporative hydrocarbon emissions. The data further demonstrate that the federal RFG minimum oxygen mandate precludes the use of fuels that do not contain oxygen and achieve greater emission benefits, principally in terms of NO<sub>x</sub> reductions.

Table 2.—Expected Change in Emissions (2005) 1998 Average In-Use Fuel Versus Three Fuels Based on Alternative Specifications Using the Proposed CaRFG3 Predictive Model

	1998 In-Use Fuel	Zero Oxygen [ln percent]	2.7 Percent Oxygen [ln percent]	3.5 Percent Oxygen [ln percent]
NO <sub>x</sub> .....	0.3%	- 5.4%	- 1.7%	- 0.7%
Hydrocarbons:				
Exhaust .....	- 3.6	- 1.7	- 6.0	- 6.0
Evaporative .....	- 6.6	- 12.6	0	0
Carbon Monoxide .....	0	0	- 4.2	- 8.9
Toxic <sup>1</sup> .....	- 7.9	- 14.7	- 15.6	- 15.7

<sup>1</sup> Potency weighted.

The reduction in NO<sub>x</sub> benefits as oxygen is increased stems from the fact that a refiner using the Predictive Model must simultaneously achieve the necessary emissions performance for HC, NO<sub>x</sub> and potency-weighted toxics. When oxygenates are not used, the greatest challenge presented by the CaRFG3 standards will typically be the need to achieve equivalent HC emissions reductions. The formulations that are most cost-effective in meeting HC equivalency will achieve somewhat greater reductions in NO<sub>x</sub> and toxics than are required under the Predictive Model. This is not the case when oxygenates are used, because of the NO<sub>x</sub> increases associated with oxygen.

Table 2 also shows that the oxygenated fuels have lower CO emissions than the non-oxygenated fuel. However, CO attainment is a problem in the winter only and the Clean Air Act addresses CO attainment in the wintertime oxygenate requirements of Section 211 (m) rather than in the Section 211 (k) federal RFG program. Both the CaRFG2 and the CaRFG3 regulations mandate oxygen in the wintertime in the southern California CO nonattainment areas, and these requirements cannot be eliminated without U.S. EPA approval of a SIP revision.

*Question 10.* ARB's analysis of the CEC study maintains that there is a considerable cost to consumers attributable to fuels oxygenated with ethanol (6-7 cents). Is that a short- or long-term price impact? How do the long-term scenarios compare for ethanol and non-oxygenated RFG?

Response. The CEC estimate of six to seven cents per gallon is based on an intermediate-term analysis (approximately 3 years). The CEC has also defined a long-term period to be six years. More importantly, this long-term period allows for the same supply and demand balances to be achieved as in the intermediate-term, but

allows refiners to make major process unit modifications such as equipment replacement or capacity expansions. The CEC estimates that the costs associated with the replacement of MTBE with ethanol during this period range from 2 to 3 cents per gallon. For the non-oxygenated gasoline case, the range is 0.9 to 3.7 cents per gallon. The long-term analysis is the most pertinent because this is what is happening with the elimination of MTBE in California under the proposed CaRFG3 program.

*Question 11.* The CEC report also says that “[i]f the scope of replacing MTBE were to be broadened to include the elimination of all oxygenates from gasoline, the cost impact for consumers would be the greatest, regardless of the length of time allowed for the transition.” How does this affect the ARB analysis for non-oxygenated RFG and pump prices? How can we be assured that refiners will use just enough ethanol and just enough non-oxygenated RFG to keep prices from spiking, particularly considering that it might not be in the refiners best interest to find that balance?

Response. The CEC report states that the cost would be greatest if all oxygenate were eliminated from gasoline, but following that statement, the report states that the long-term cost would be at the high end of the range (up to 3.7 cents per gallon). This is one cent higher than the high estimate for using ethanol to replace MTBE. The low end of the range is also one cent lower than the ethanol case.

There is a big difference between eliminating the oxygen mandate and imposition of a ban on all oxygenates. It is important for refiners to have the flexibility to use an oxygenate (expected to be ethanol) where it makes economic sense to do so. Providing flexibility will not guarantee that prices will not spike during times when supply shortages occur. Flexibility should lower the average cost of producing fuel, and enable supply shortages to be remedied more quickly.

If there were no oxygen requirement, our discussions with refiners have led to the conclusion that some gasoline would be made with ethanol and some would not. This would reduce California’s dependence on ethanol and would improve refiners’ ability to use other gasoline blendstocks.

In the event of a supply disruption of ethanol or other gasoline blendstocks, refiners would have more options to produce gasoline. With more options available, the likelihood of a price spike and the severity of the spike would be reduced.

*Question 12.* Could you address ARB’s recent Urban Airshed Modeling which demonstrates that non-oxygenated fuels result in higher ozone and CO emissions than oxygenated fuels, and will result in backsliding?

Response. There was an error in the draft analysis. The error was in the assumptions made for the compositions of the non-oxygenated fuel and the 5.7 percent ethanol fuel that affected the prediction of ozone, but did not substantially affect the performance relative to toxic compounds of interest.

The errors in the fuel composition have since been corrected and the results now show that there are no significant differences in ozone forming potential between oxygenated and non-oxygenated gasoline. The analysis was based on CaRFG2 fuels and not CaRFG3 fuels. These results are consistent with the findings of the U.S. EPA Blue Ribbon panel and the NRC that there are no statistically significant differences in ozone forming potential when comparing different reformulated gasolines.

It also shows, as we expect, that CO emissions are lower with an oxygenated fuel. As discussed earlier, the wintertime oxygen requirement would still apply in the South Coast as long as it remains in non-attainment of the ambient air quality standards for CO.

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AIR RESOURCES BOARD,  
Sacramento, CA, July 9, 1999.

MR. ROBERT PERCIASEPE,  
Assistant Administrator for Air and Radiation,  
U.S. Environmental Protection Agency,  
Washington, DC.

RE: SUPPORT MATERIALS FOR CALIFORNIA’S REQUEST FOR A WAIVER FROM THE REQUIREMENT THAT FEDERAL RFG CONTAIN AT LEAST 2 PERCENT OXYGEN YEAR-ROUND

Dear MR. PERCIASEPE: I am attaching a set of supplemental materials in support of California’s request for a waiver under Clean Air Act section 211 (k)(2)(B) from the requirement that federal reformulated gasoline contain at least 2.0 volume percent oxygen year-round. This waiver request was made in Governor Davis’s April 12, 1999 to Administrator Carol Browner. The materials I am now transmitting are

identical to the materials I gave you on June 21, 1999, except that Attachment 1 has been updated to reflect the emissions comparison based on the federal complex model.

I believe that our analysis presents a substantial and compelling justification for the requested waiver. Please call me at (916) 445-4383 if you have any questions. Your staff can address any questions to Dean Simeroth at (916) 322-6020 on technical issues, and to Tom Jennings at (916) 323-9608 on legal issues.

Sincerely,

MICHAEL P. KENNY.

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BASIS FOR A WAIVER FROM THE FEDERAL RFG 2.0 PERCENT OXYGEN REQUIREMENT FOR CALIFORNIA AS AUTHORIZED IN CAA § 211(k)(2)(B)

California believes that U.S. EPA can and should waive the year-round 2.0 percent by weight (wt.%) oxygen requirement for federal reformulated gasoline (RFG) in each of California's three federal RFG areas. This waiver is justified by the technical analysis of the California Air Resources Board (ARB) that maintaining the federal 2.0 wt. percent oxygen requirement after MTBE has been phased out of California gasoline will diminish the extent to which the California RFG regulations can achieve emission reductions over and above the reductions achieved by the federal program. This loss of additional benefits from the California program will interfere with attainment of the national ambient air quality standards for ozone, PM<sub>10</sub> and PM<sub>2.5</sub> in California's federal RFG areas.

Because California faces the most intractable air pollution problems in the nation, the ARB has designed the California RFG (CaRFG) program to achieve significantly greater overall emission reductions than those resulting from the federal RFG program. ARB is now developing its Phase 3 CaRFG rules. This is being done to eliminate the State's reliance on MTBE—which has been found to present an unacceptable threat to water supplies—and to enhance the emission reductions that the CaRFG program contributes to the State Implementation Plan (SIP). ARB's assessment shows that revised California rules accommodating a federal RFG requirement for 2.0 wt. percent oxygen in the fuel year-round will necessarily be less effective in reducing vehicular emissions than would be the case if the rules could be based on oxygen-content flexibility. This loss of additional potential emission reductions from CaRFG would delay attainment of the ozone standards in all three of California's federal RFG areas, and threaten eventual attainment of the ozone and PM<sub>2.5</sub> standard in the Los Angeles region.

*The CAA § 211(k)(2)(B) waiver provision.*—CAA § 211(k)(2)(B) expressly authorizes U.S. EPA to waive the federal RFG year-round 2.0 wt. percent minimum oxygen requirement, in whole or in part,

. . . upon a determination by the Administrator that compliance with such requirement would prevent or interfere with the attainment by the area of a national ambient air quality standard.

*California's need for additional emission reductions in its three federal RFG areas.* The emission reductions from the CaRFG program are critical to attainment of the national ozone standards, and are essential to compliance with the PM<sub>10</sub> and PM<sub>2.5</sub> standards. California needs to add measures to its ozone SIP to assure attainment, and any loss of reductions of NO<sub>x</sub> or ozone-forming hydrocarbons will interfere with the timely attainment of both the ozone standards.

*Additional emission reductions achieved by the CaRFG rules.*—The current CaRFG rules, which have been applicable since 1996, require reductions in emissions of NO<sub>x</sub> and toxics that are substantially greater than the emissions reductions that will be required by the federal RFG Phase II rules that apply starting January 2000. Attachment 1 provides a comparison of the emission benefits of the two sets of rules, based on application of U.S. EPA's Complex Model. The NO<sub>x</sub> emissions reductions from the California program are more than twice the reductions required by federal RFG Phase II—the CaRFG rules achieve an additional overall NO<sub>x</sub> reduction of 8 percent. The toxics emissions reductions from the California program, on a potency-weighted basis, are about 20 percent greater than the corresponding emissions reductions from federal RFG Phase II. The VOC emission reductions required by the two programs are roughly equal.

ALTERNATIVE SCENARIOS FOR PHASE 3 CARFG

On March 26, 1999, Governor Davis issued Executive Order D-5-99, which outlines California's action plan for removing MTBE from all California gasoline by December 31, 2002 at the latest. California is phasing out MTBE because of the threat

it presents to the State's groundwater, surface water, and drinking water systems. ARB has initiated in Phase 3 CaRFG rulemaking with two fundamental objectives in mind—to make the total removal of MTBE from the State's gasoline feasible and practical, and to preserve or enhance the emission reductions achieved by the existing program after the phaseout of MTBE.

The Phase 3 CaRFG regulations will ultimately be implemented in one of two distinctly different regulatory environments. In one, the year-round 2.0 wt. percent oxygen requirement would continue to be mandated by the federal RFG regulations, applicable to about 70 percent of all of California's gasoline. In the other regulatory environment, affirmative action on California's waiver request by U.S. EPA—and/or action by Congress—would allow for oxygen flexibility. ARB technical staff have analyzed likely scenarios for a Phase 3 CaRFG program under the two environments and the results of this analysis are contained in Attachment 2.

If the federal RFG 2.0 wt. percent oxygen mandate is maintained after the phase-out of MTBE, it is clear that ethanol would be the only practical oxygenate. Three scenarios have been identified: (1) No use of MTBE and federal oxygen flexibility; (2) No use of MTBE and a federal RFG 2.0 wt. percent oxygen mandate met by 5.7 vol. percent ethanol; and (3) No use of MTBE and a federal RFG 2.0 wt. percent oxygen mandate met by 10 vol. percent ethanol. For each scenario, staff started with a hypothetical gasoline meeting all of the "flat" limits in the current CaRFG regulations. The staff next identified the changes in gasoline properties that refiners would necessarily have to make under the scenario, and identified the emissions impact of these changes. The staff then identified potential changes to the CaRFG standards that could be made to preserve the emissions benefits of the current program and to enhance those benefits to the extent feasible. Staff evaluated the feasibility of these changes to the CaRFG standards and their overall emissions impact. The underlying details supporting the analyses are attached.<sup>1</sup>

The analyses of the scenarios demonstrate that California's ability to have oxygen flexibility should result in technologically feasible increased reductions of NO<sub>x</sub> of 1.5 percent and toxics of 2.5 percent for CaRFG after the phase-out of MTBE. The scenarios for using ethanol to meet a federal RFG 2.0 wt. percent year-round oxygen mandate show that essentially all pentanes would have to be removed from gasoline just to preserve the existing hydrocarbon benefits. Also, taking sulfur down to zero—compared to 10 ppm for the oxygen flexibility scenario—still does not achieve the same NO<sub>x</sub> or toxics reductions. Additional changes to other CaRFG specifications would have to be made to provide these benefits. For 10 percent ethanol, it simply may not be possible at any cost to achieve the same benefits as the oxygen flexibility scenario. Finally, the zero sulfur requirement in both of the ethanol scenarios will make imports difficult if not possible.

The loss of NO<sub>x</sub> benefits that would result from maintenance of the federal RFG 2.0 wt. percent oxygen mandate would prevent or interfere with attainment of the federal ozone, PM<sub>10</sub> and PM<sub>2.5</sub> ambient standards in California's federal RFG areas. There is accordingly a sound technical and legal basis for U.S. EPA to waive the federal RFG year-round 2.0 wt. percent oxygen requirement for California's federal RFG areas. However, because the use of oxygen during the winter months does not threaten ozone attainment, it may be possible to retain a lesser oxygen averaging requirement. A waiver that retains an oxygen requirement of 2 wt. percent for the four winter months which is approximately 0.6 wt. percent, averaged over a year, and which allows any given fuel to contain zero and 3.5 wt. percent oxygen, would therefore be appropriate.

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<sup>1</sup>The California Predictive Model was used for projecting exhaust emissions impacts and the Complex Model was used for evaporative emissions. The Predictive Model is the tool in the CaRFG regulations for allowing alternative CaRFG formulations that achieve equivalent exhaust emissions reductions. It is more useful than the federal Complex Model in determining the future emissions impacts of California gasoline for purposes of CAA §211(k)(2)(B) waiver analysis, because the underlying fleet more closely represents the future California fleet. As required under CAA §211(k)(10)(A), the Complex Model is based on representative 1990 vehicle technology. This limitation is not present in the oxygen waiver provision. The Predictive Model does not have an evaporative emissions element because the CaRFG limit for RVP—the parameter affecting evaporative emissions—is not allowed to vary.

## ATTACHMENT 1

Model Predictions are Computed for the Following Fuel Property Values

Property	CCA Base-line	CA Phase 2 Avg. Limits	CA Mean Predictive Model Limits	Actual 1996 CA Mean Fuel Properties	EPA Phase II RFG	CA Phase I Limits
RVP .....	8.7	7.0	7.0	6.8	6.7	7.8
E200/T50 .....	41/218	50/200	49/01	51/197	49/202	44/212
E300/T90 .....	83/329	92/20	88/307	89/302	87/311	83/330
Aromatics .....	32	22	24	23	25	34
Olefins .....	9.2	4	5.8	3.9	11	11
Oxygen .....	0	2	1.9	2.07	2.1	0
Sulfur .....	339	30	28	20	150	151
Benzene .....	1.53	0.8	0.7	0.55	0.95	2.0

Model Predictions (Percent Change Relative to Clean Air Act Baseline Fuel)

Pollutant	EPA COMPLEX MODEL PREDICTIONS				ARB PREDICTIVE MODEL PREDICTION				
	CA Phase 2 Avg. Limits	CA Mean Predictive Model Limits	Actual 1996 CA Mean Fuel Properties	EPA Phase II RFG	CA Phase 2 Avg. Limits	CA Mean Predictive Model Limits	Actual 1996 CA Mean Fuel Properties	EPA Phase II RFG	CA Phase 2 Avg. Limits Relative to CA Phase 1 Limits
Exhaust VOC .....	-18.3 .....	-18.0 .....	-19.1 .....	-18.1 .....	-30.0 .....	-29.2 .....	-30.6 .....	-22.5 .....	-17.4
Evap VOC .....	-44.4 .....	-44.4 .....	-47.4 .....	-48.8 .....	-45.3 .....	-45.3 .....	-50.7 .....	-53.3 .....	-28.1
Total VOC .....	-28.2 .....	-28.1 .....	-29.9 .....	-29.8 .....	-35.4 .....	-34.9 .....	-37.7 .....	-33.5 .....	-21.2
NO <sub>x</sub> .....	-14.6 .....	-13.9 .....	-14.6 .....	- 6.8 .....	-11.6 .....	-11.0 .....	-12.1 .....	- 5.0 .....	- 7.0
Exhaust Benzene .....	-42.3 .....	-42.7 .....	-46.1 .....	-35.9 .....	-51.7 .....	-50.7 .....	-54.6 .....	-38.8 .....	-50.8
Evap. Benzene .....	-68.2 .....	-72.0 .....	-79.0 .....	-64.3 .....	-47.7 .....	-54.2 .....	-64.0 .....	-37.9 .....	-60.0
Acetaldehyde .....	-21.0 .....	-17.8 .....	-19.3 .....	-15.6 .....	4.5 .....	6.5 .....	5.1 .....	6.8 .....	8.2
Formaldehyde .....	18.1 .....	16.7 .....	21.5 .....	7.8 .....	43.1 .....	46.0 .....	46.6 .....	34.0 .....	29.7
1,3-Butadiene .....	-33.3 .....	-18.0 .....	-32.2 .....	- 8.8 .....	-34.1 .....	-26.2 .....	-36.3 .....	- 9.9 .....	-31.3
POM .....	-18.3 .....	-18.0 .....	-19.1 .....	-18.1 .....	Not Estimated	Not Estimated	Not Estimated	Not Estimated	Not Estimated
Total Toxics .....	-34.5 .....	-34.1 .....	-37.0 .....	-28.4 .....	Not Calculated	Not Calculated	Not Calculated	Not Calculated	Not Calculated
PWT .....	Not Calculated	Not Calculated	Not Calculated	Not Calculated	-43.2 .....	-39.6 .....	-44.1 .....	-26.6 .....	-41.8

## ATTACHMENT 2

SCENARIO 1: NO USE OF MTBE AND NO FEDERAL YEAR-ROUND 2.0 WT.%  
OXYGEN MANDATE

## Step 1. Initial impact

(a) Variations from current flat specifications: Reduce oxygen content from 2.0 to 0.0 (due to removal of MTBE)

(b) Initial impact, emissions and other:

NO<sub>x</sub>: -0.5%

THC: +3%

CO: +10%

Toxics: -0.5%

Loss of 11% volume.

## Step 2. Changes to CaRFG standards

Reduce RVP standard by 0.2 psi, from 7.0 to 6.8 psi.

Reduce sulfur standard by 30 ppm, from 40 ppm to 10 ppm.

## Step 3. Feasibility

Requires some capital investment and an increase in operating costs to reduce RVP by 0.2 psi and reduce sulfur to 10 ppm, but both are feasible.

The 11 percent lost volume will have to be made up by importing or increasing production of alkylates (blendstocks), or importing fully complying gasoline.

## Step 4. Cumulative emissions impact

NO<sub>x</sub>: -1.5%

THC: -0.3% (includes loss of reduction in ozone-forming potential from loss of CO emission reductions from 2.0 wt percent oxygen)

CO: +10 percent (doesn't apply when in CO winter nonattainment area)

Toxics: -2.5 percent

Winter oxygenates where required, using ethanol at 2.0 wt. percent oxygen:

CO: -0 percent

RVP: Summertime limits nor applicable

SCENARIO 2: NO USE OF MTBE BUT FEDERAL YEAR-ROUND 2.0 WT. PERCENT OXYGEN  
MANDATE MET WITH 5.7 VOL PERCENT ETHANOL

## Step 1. Initial impact

(a) Variations from current flat specifications:

RVP increases 1 psi from 7.0 to 8.0 psi (due to ethanol effect)

(b) Initial impact, emissions and other:

NO<sub>x</sub>: neutral

THC: +13 percent (from 1.0 psi increase in RVP)

CO: neutral

Toxics: +5.7 percent

Loss of 6 percent volume

## Step 2.A. Changes to CaRFG standards equivalent to changes for no oxygen mandate (Scenario 1)

Reduce RVP standard by 0.2 psi, from 8.0 to 7.8 psi.

Reduce sulfur standard by 30 ppm, from 40 ppm to 10 ppm.

## Step 2.B. Changes to CaRFG standards to achieve the same benefits as the no oxygen mandate (Scenario 1)

Further reduce RVP by 0.8 psi, from 7.8 to 7.0 psi.

Further reduce sulfur by 10 ppm, from 10 ppm to zero.

## Step 3. Feasibility

A. Feasibility of Step 2.A. changes is same as in Scenario 1

B. Reduction of RVP would necessitate removal of all pentanes. This is more expensive than in Scenario 1 and results in a loss of volume of about 4 percent. Reducing sulfur to zero is technically very difficult and would effectively preclude gasoline imports, as little or none available with zero sulfur. The overall 10 percent lost volume will have to be made up by importing or increasing production of alkylates (blendstocks), or importing fully complying gasoline.



## Step 4. Cumulative emissions impact

*Step 2.A*

NO<sub>x</sub>: -1 percent  
 THC: +8.8 percent  
 CO: neutral  
 Toxics: +3.3

*Step 2.B*

NO<sub>x</sub>: -1.3 percent  
 THC: -1 percent  
 CO: neutral  
 Toxics: -1 percent

Winter oxygenates where required, using ethanol:

CO: -0 percent  
 RVP: Summertime limits not applicable

SCENARIO 3: NO USE OF MTBE BUT FEDERAL YEAR-ROUND 2.0 WT. PERCENT OXYGEN  
 MANDATE MET WITH 10 VOL PERCENT ETHANOL

## Step 1. Initial impact

- (a) Variations from current flat specifications  
 RVP increases 1 psi from 7.0 to 8.0 psi (due to ethanol effect)
- (b) Initial impact, emissions and other  
 NO<sub>x</sub>: +2.6 percent  
 THC: +12 percent (from 1.0 psi increase in RVP)  
 CO: -5 percent  
 Toxics: +6.7 percent  
 Loss of 1 percent volume

## Step 2.A. Changes to CaRFG standards equivalent to changes for no oxygen mandate (Scenario 1)

Reduce RVP standard by 0.1 psi, from 7.9 to 7.8 psi (after allowing a 0.1 psi credit for impact of CO reduction on ozone)

Reduce sulfur standard by 30 ppm, from 40 ppm to 10 ppm.

## Step 2.B. Changes to CaRFG standards to achieve same benefits as the no oxygen mandate (Scenario 1)

Further reduce RVP by 0.6 psi, from 7.8 to 7.2 psi  
 Further reduce sulfur by 10 ppm, from 10 ppm to zero

## Step 3. Feasibility

- A. Feasibility of Step 2.A. changes is same as in Scenario 1
- B. Reduction of RVP by 0.7 psi would necessitate removal of all pentanes. This is more expensive than in Scenario 1 and results in a loss of volume of about 5 percent. Reducing sulfur to zero is technically difficult and would effectively preclude all gasoline imports, as little or none available with zero sulfur.

## Step 4. Cumulative emissions impact

*Step 2.A*

NO<sub>x</sub>: +1.6 percent  
 THC: +7.2 percent  
 CO: -5 percent  
 Toxics: +4.4 percent

*Step 2.B*

NO<sub>x</sub>: +1.3 percent  
 THC: neutral  
 CO: -5 percent  
 Toxics: +1.2 percent

AIR RESOURCES BOARD,  
Sacramento, CA, July 9, 1999.

MS. MARGO T. OGE,  
Director, Office of Mobile Sources,  
U.S. Environmental Protection Agency,  
Washington, DC.

DEAR MS. MARGO: This is in response to your August 6, 1999, letter posing several follow-up questions to my July 9, 1999, submission of supplemental data regarding our request for a waiver from the oxygen requirement of the federal RFG program.

The response provided below fully addresses each of your questions. We are hopeful that this supplemental information will allow you to expeditiously provide California the waiver it needs to remove methyl tertiary butyl ether (MTBE) from gasoline without impeding our ability to expeditiously attain federal national ambient air quality standards. For ease of reference, I am providing your original questions followed by our response.

*Question 1.* Based on our review we understand that the federal requirement of 2.0-wt percent oxygen can be met with 5.7-vol percent ethanol (your Scenario 2). For Scenario 2 you state that the reductions in NO<sub>x</sub> for this level of ethanol fall short of your NO<sub>x</sub> reduction goal of 1.5 percent by 0.2 percent even with reduction of sulfur to 0 ppm. Have you considered the potential impacts of other fuel parameters, such as aromatics and olefins?

Response. Our analysis demonstrated that maintaining the oxygen mandate reduced potential additional NO<sub>x</sub> emissions reductions that might otherwise be achieved in a cost-effective manner that preserved essential flexibility in meeting California reformulated gasoline regulations. We recognized that compliance with the specifications could be met by changing other properties. The demonstration was to show that the oxygen mandate restricts our ability to achieve the greatest possible NO<sub>x</sub> emissions reductions.

Our analysis stressed the effects of RVP and sulfur for setting new baseline fuel specifications because emissions are most sensitive to these parameters and when either is reduced, emissions of regulated pollutants tend to go down. If the other properties were changed, emissions of one or more pollutants would decrease (usually to a much smaller degree) but emissions of at least one other pollutant would increase. Therefore, these other parameters are much less useful in making complying fuels with the needed NO<sub>x</sub> reductions.

*Question 2.* For Scenario 2 the staff analysis states that all pentanes would need to be removed to reduce RVP from 7.8 to 7.0 psi to preserve existing hydrocarbon benefits. Yet the staff analysis indicates a reduction in hydrocarbons of 1.0 percent which is beyond the 0.3 percent reduction projected for your Scenario 1 in which there is 0.0 wt percent oxygen in the fuel. Are we correct in assuming that Scenario 2 would exceed hydrocarbon reduction goals? If so, could RVP be reduced by less than 0.8 psi for the 5.7-vol percent ethanol case?

Response. Yes, the hydrocarbon estimate in Scenario 2 is lower than the 0.3 percent reduction shown in Scenario 1. However, hydrocarbon emissions are very sensitive to changes in RVP. If RVP was increased by just 0.1 psi, then the current evaporative emissions model predicts there would be a 3.5 percent increase in the hydrocarbon evaporative emissions. Even such a small change in RVP would lead to an increase in hydrocarbon emissions and would not be practical because it would not preserve the emission benefits.

*Question 3.* Your letter states that ARB would consider appropriate a waiver of the 2.0 wt percent oxygen requirement based on averaging. That is, a minimum of 2.0-wt percent oxygen would be required for the four winter months, and for the remaining months any given fuel could contain from 0 to 3.5-wt percent oxygen. If the minimum oxygen requirement of 1.5 wt percent were eliminated, would that change the results and/or conclusions of your analysis?

Response. No, solely removing the 1.5 wt percent minimum oxygen requirement and keeping the 2.0 wt percent oxygen average would not change the conclusions of our analysis.

If the 2.0-wt percent average were required, with no minimum, a significant percentage of the summer gasoline would still require oxygen. If the oxygen level for the four winter months were at the 3.5 percent level, then to average 2.0 percent, the oxygen content in RFG for the remaining months would still have to average about 1.25 percent oxygen. In reality, given the California gasoline distribution system, such an approach would provide very little flexibility to produce non-oxygenated RFG. Thus, it would still be very difficult to achieve additional cost/effective NO<sub>x</sub> reductions during the summer.

*Question 4.* Your July 9 letter frequently cites concerns that the 2.0 wt percent oxygen mandate will create barriers to implementation of "Phase 3 CaRFG regulations". Please clarify, in light of the fact the ARB has not yet finalized the Phase 3 regulations, what assumptions were made about the Phase 3 fuel in the analysis.

Response. There was no need to assume anything for Phase 3 CaRFG other than there still exists a need for further reductions in emissions. The only assumptions in the analysis were that reductions of sulfur and RVP could provide additional emissions benefits in complying with our current or future regulations. No matter which scenario you consider or which properties you vary, the ability to reduce NO<sub>x</sub> and evaporative hydrocarbon emissions or maintain the existing emissions benefits is greater without oxygen.

*Question 5.* Please provide information of how CO and THC changes were calculated.

Response. The changes were calculated using the existing Predictive Model for exhaust, and the proposed evaporative model which is being developed as part of a revised Predictive Model. Both the current Predictive Model and the initial draft model for public comment are available on the ARB Cleaner Burning Gasoline web page. The evaporative hydrocarbon results from the evaporative portion of the initial draft model and the exhaust hydrocarbon results from the current Predictive Model were combined by using the ARB EMFAC7G inventory weightings of exhaust and evaporative emissions. Weights were calculated for the inventory years; 1996, 2000, and 2005. The weights were averaged to provide a composite weight. The NO<sub>x</sub> portion of the analysis was generated using the current Predictive Model.

For CO, we used the relationship that increasing fuel oxygen by 2 percent in results approximately a 10 percent reduction in exhaust CO. This is consistent with the estimates from the Auto/Oil research program. This is also consistent with estimates of the effectiveness in reducing ambient concentrations of CO for the winter-time oxygen program. The analyses of the ambient data for sites-primarily impacted by motor vehicles emissions estimated the reductions in CO to be between 7 percent and 12 percent.

*Question 6.* Has ARB considered the effect on ozone associated with reduction in CO emissions associated with oxygen levels above 2.0 wt percent? If so, please provide information on how such reductions were accounted for.

Response. We accounted for reductions in CO by converting tons of CO into tons of equivalent evaporative hydrocarbons emissions. We used the Maximum Incremental Reactivity (MIR) factors to adjust the ozone reactivity differences for CO and evaporative emissions to be on the same basis. The MIR factor for CO was 0.07 and the average MIR for evaporative emissions was about 2.2. This yields a conversion factor of approximately 31.4 to 1. Or, it takes about a reduction of 31.4 tons of CO to offset an increase of 1 ton of evaporative emissions. We used a revision of the Predictive Model, discussed in the response to Comment 5, that includes an evaporative emissions component to estimate the fuel property effects on THC. We then compared the reactivity weighted CO and THC to adjust the THC emissions accordingly.

Sincerely,

MICHAEL P. KENNY.

STATEMENT OF HON. JAKE GARN, VICE-CHAIRMAN, BOARD OF DIRECTORS, HUNTSMAN CORPORATION

#### I. INTRODUCTION AND SUMMARY

Good morning, Mr. Chairman, Senator Chafee, and other distinguished Members of the Committee. I am pleased to have this opportunity to appear before the Subcommittee this morning.

My name is Jake Garn. I am appearing on behalf of the Huntsman Corporation, headquartered in Salt Lake City, Utah. I am vice-chairman of the Board of Directors of Huntsman. Huntsman is the largest privately-owned chemical company in the United States. Huntsman is the one of the largest domestic manufacturer of MTBE for the merchant market and is a member of the Oxygenated Fuels Association which represents manufacturers of oxygenates used in fuels. Huntsman's decision to make additives that clean our air is consistent with this Corporation's long-standing commitment to socially responsible actions designed to improve the lives of all Americans. The Huntsman family has contributed over \$150 million to state-of-the-art cancer research facilities, home to over 300 of the world's top researchers. Our belief in protection of human health and the environment is no mere slogan; it is a promise we have made to our community.

On behalf of the Huntsman Corporation, I want to commend the Chairman Inhofe and Chairman Chafee for convening the Subcommittee to examine the findings and recommendations of the Blue Ribbon Panel. As the manufacturer of a significant amount of MTBE, we have an obvious interest in the BRP's findings and recommendations and, perhaps more importantly, in the actions Congress may take based on those recommendations.

We agree with much of what the Blue Ribbon Panel concluded. For example, we agree that more research and monitoring is necessary concerning the health effects of not only MTBE, but also other constituents of gasoline. However, we have strong concerns with several of the BRP's conclusions. Most importantly, we disagree strongly that there is sufficient justification to recommend a substantial reduction in the use of MTBE. As described in greater detail below, we believe that the BRP left many important questions unanswered. Unfortunately, the BRP is gone and the responsibility to answer these questions falls to the Congress, and to this Subcommittee in particular. Until those questions are answered, we believe it is inappropriate to move forward with any effort to amend the Clean Air Act to modify the reformulated gasoline program. We appreciate this opportunity to contribute our thoughts on how Congress should endeavor to answer these remaining important questions.

## II. HUNTSMAN AND MTBE

Huntsman Corporation is one of the largest producers of MTBE in the United States. It has been producing MTBE since early 1998 when it acquired Texaco's MTBE-producing facility in Beaumont, Texas. The company sells its MTBE product to refiners who, in turn, use it to meet the requirements of the Clean Air Act.

## III. THE BLUE RIBBON PANEL RECOMMENDATIONS

As you know, in 1998 EPA Administrator Browner appointed a Blue Ribbon Panel (BRP) to investigate the air quality benefits and water quality concerns associated with oxygenates in gasoline, and to provide independent advice and recommendations on ways to maintain air quality while protecting water quality. The BRP met on several occasions and issued its final report in July 1999. Mr. Greenbaum, the chairman of the BRP, is better qualified to describe the work of the Panel and to summarize its findings and recommendations. I would like to take this opportunity to explain why Huntsman agrees with some—but not all—of those findings and recommendations.

### A. *Points of Agreement*

The BRP made a number of findings and recommendations with which Huntsman Corporation agrees. They are, in significant part, the following:

- that MTBE has been detected in a number of water supplies nationwide, primarily causing consumer odor and taste concerns that have led water suppliers to reduce use of those supplies. The Panel further found that incidents of MTBE in drinking water supplies at levels well above EPA and State guidelines have occurred, but are rare;
- that MTBE is currently an integral component of the U.S. gasoline supply both in terms of volume and octane, and as such, changes in its use, with the attendant capital construction and infrastructure modifications, must be implemented with sufficient time, certainty, and flexibility to maintain the stability of both the complex U.S. fuel supply system and gasoline prices;
- that the BRP's recommendations were intended to "simultaneously" maintain air quality benefits while enhancing water quality protection and assuring a stable supply at reasonable cost;
- that EPA should take actions to enhance significantly the Federal and State Underground Storage Tank programs;
- that EPA should work with its State and local water supply partners to enhance implementation of the Federal and State Safe Drinking Water Act programs;
- that EPA should work with States and localities to enhance their efforts to promote lakes and reservoirs that serve as drinking water supplies by restricting use of recreational water craft, particularly those with older motors;
- that EPA should work with other Federal agencies, the States, and private sector partners to implement expanded programs to protect private well users;
- that we should expand public education programs at the Federal, State, and local levels on the proper handling and disposal of gasoline;
- that we should develop and implement an integral field research program into the groundwater behavior of gasoline and oxygenates;

- that EPA should work with Congress to expand resources available for the up-front funding of the treatment of drinking water supplies contaminated with MTBE and other gasoline components to ensure that affected supplies can be rapidly treated and returned to service, or that an alternative water supply can be provided;
- that States should reexamine and enhance State and Federal "triage" procedures for prioritizing remediation efforts at UST sites based on their proximity to drinking water supplies;
- that we should accelerate laboratory and field research, and pilot projects, for the development and implementation of cost-effective water supply treatment and remediation technology, and harmonize these efforts with other public/private efforts underway; and
- that we should identify and begin to collect additional data necessary to adequately assess the current and potential future State of contamination.

#### *B. Points of Disagreement*

However, there is one important recommendation of the BRP with which we emphatically do not agree. The BRP recommended that "in order to minimize current and future threats to drinking water, the use of MTBE should be reduced substantially." The BRP also recommended that the current Clean Air Act mandate requiring 2 percent oxygen, by weight, in RFG must be removed in order to provide flexibility to blend adequate fuel supplies in a cost-effective manner while quickly reducing usage of MTBE and maintaining air quality benefits. As a member of the Oxygenated Fuels Association, Huntsman Corporation has supported refiner flexibility through removal of the oxygen standard as long as adequate assurances of no air quality backsliding are provided. Concurrently, we have encouraged EPA to review its authority under existing law to provide such flexibility. However, we must object strongly to the suggestion that there is a sufficient basis of knowledge upon which to base a recommendation to limit the amount of use of MTBE, an effective tool to reduce air pollution.

#### *C. Comments on the BRP's Recommendation to Reduce Substantially the Use of MTBE*

We believe several comments are in order concerning the BRP's recommendation to reduce substantially the use of MTBE. I hope what is evident from the following discussion is that Huntsman Corporation does not challenge the mandate of the BRP or the great majority of its findings and recommendations. Instead, Huntsman believes that for a variety of reasons, the BRP was unable to finish the job, and it now falls to Congress to answer the remaining questions, including both questions of fact and questions of policy.

##### *1. The BRP Made No Finding Concerning Health Effects of Exposure to MTBE*

It is important to note that the BRP did not make any finding concerning the health effects of exposure to MTBE. The Panel acknowledged that was not constituted to perform an independent comprehensive health assessment. Of course, it could not perform such an assessment and report to EPA in the limited time available to it. Instead, the Panel chose to rely on "recent reports by a number of state, national, and international health agencies."

We now understand that there is negligible risk associated with exposure to levels of MTBE being reported in drinking water supplies. It is instructive to review the status of reports by state, national and international health agencies.

There is currently no regulated standard for MTBE in drinking water under the Federal Safe Drinking Water Act. EPA has published an Advisory document on MTBE which recommends that keeping levels of contamination in the range 20 to 40 micrograms per liter or below "to protect consumer acceptance of the water resource would also provide a large margin of exposure (safety) from toxic effects."<sup>1</sup> By its authority under the Federal Safe Drinking Water Act, EPA recently issued a regulation requiring most public water systems to monitor levels of a number of unregulated contaminants, including MTBE.<sup>2</sup> EPA will use this information, together with the results of research on the human health effects of MTBE, to determine whether it should regulate the amount of MTBE permissible in drinking water.

For over a decade, scientists have studied MTBE to identify its toxic properties and to determine whether they might be manifest in people exposed to small con-

<sup>1</sup>Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Methyl Tertiary-Butyl Ether (MTBE), U.S. Environmental Protection Agency (December 1997) at 2.

<sup>2</sup>64 Fed. Reg. 50556 (September 17, 1999)(to be codified at 40 C.F.R. §§9.1, 141.35, 141.40, 142.16 and 142.15).

centrations in air and water. MTBE, like all other chemicals, has the ability to cause some injury at sufficiently high dosages. Extensive research has indicated that the MTBE doses required to produce illness in laboratory animals are thousands of times greater than those humans could conceivably be exposed to. Furthermore, MTBE has been shown to be incapable of impairing fertility, or of damaging the developing fetus. Also, based on numerous tests, MTBE is incapable of damaging the genetic structure of cells, greatly reducing the chance that it might affect numerous bodily processes controlled by a person's DNA.

As explained earlier in this testimony, Huntsman agrees that there should be more research on the health effects of exposure to all the constituents of gasoline, including MTBE. Indeed, wherever MTBE is detected, there are likely to be other, potentially more harmful constituents of gasoline present. However, there is not yet sufficient evidence of harmful health effects from MTBE. The BRP accurately reflects this absence of such evidence. Without such evidence, and in light of the overwhelming evidence of benefits from the use of MTBE in gasoline, it is not appropriate to recommend that the use of MTBE be reduced substantially.

The latest scientific evidence concerning the health effects of MTBE must be considered together with the most recent information concerning the scope of MTBE contamination in drinking water. There is evidence to indicate that MTBE contamination of drinking water sources is limited to geographic pockets within the United States, and that the number of gasoline effected wells that exceed national guidelines and State primary and secondary drinking water standards is small. The Water Contamination Issue Summary shows primary and secondary drinking water standards and action levels for States that have them. The average secondary standard (aesthetically based) is over 20 ppb and the average primary standard (health based) is over 50 ppb. Yet, Table 1 of the same report shows that only 1 percent-2 percent of all wells tested so far exceed 5 ppb of MTBE. Therefore, the number of wells that exceed State standards is small enough to be manageable.

## *2. The BRP Underestimated the Air Quality Benefits of Oxygenates*

Huntsman Corporation is also concerned that the BRP underestimated the air quality benefits of oxygenates. The BRP recommendations are predicated on the regulatory requirements established in EPA's existing RFG rules. They fail to recognize that the RFG program (with MTBE as the oxygenate of choice in 80 percent of the market) has exceeded by nearly double the requirements of EPA's regulations. By underestimating such benefits, it is easier for the BRP to assume that other fuel formulations can achieve the same, or better, air quality benefits.

According to EPA's May 24, 1999 RFG Fact Sheet, oxygenates such as MTBE substantially reduce toxics such as benzene and other aromatics. Oxygenates also dilute or displace other fuel components like sulfur, which in turn reduce emissions of the smog precursors VOC and NO<sub>x</sub>. They also provide additional reductions in the distillation temperatures of gasoline. These improvements are important in reducing vehicle exhaust emission, particularly during the first few minutes of cold engine operation when the catalytic converter is not fully operational.

Unfortunately, the BRP underestimated the real-world air quality benefits of MTBE (and oxygenates, in general) in its narrow application of emission prediction models. Air quality predictions using these models ignore many of the remaining benefits that were identified during the Panel's meetings and presented in the Air Quality Issue Summary.

Throughout the recent debate, it has been convenient to ignore a large portion of oxygenate air quality benefits, to ascribe them to other fuel parameters, or to discount them altogether in favor of automotive technology advances. Real world impacts, such as the contribution of oxygenates to improved combustion before a vehicle's catalytic converter achieves normal operating efficiency, have been largely ignored. Similarly, the "leaning-out" benefits of off-road gasoline engines without catalytic converters have remained unaccounted for even though their percent share of the total emissions picture continues to rise. The benefits of lower combustion deposit formation and associated decrease in particulate emissions are also not quantified. Indirect oxygenate dilution benefits of undesirables such as olefins, sulfur and aromatics are typically discounted by the suggestion that refiners will find some other way to achieve them. The same is true for positive drivability impacts associated with improved oxygenated fuel midrange volatility. In their eagerness to obtain oxygenate flexibility, refiners have clearly misrepresented the degree of difficulty involved in replacing MTBE.

The risk of backsliding on the air quality gains of the last decade looms large in the horizon. This fact is demonstrated by recent claims by California refiners that they face great difficulty in achieving the actual air quality benefits of that state's clean burning gasoline (CBG) without MTBE, according to an August 20, 1999 "In-

side *CalEPA*" article. Regulators are not clear on how to preserve the air quality benefits of CBG without MTBE.

Several years ago, EPA asked the National Research Council (NRC) to (1) assess whether the existing scientific information allows a comparison of the ozone forming potential of automotive emissions obtained with different reformulated gasolines, and (2) evaluate the impact of applying the "ozone forming potential" approach to air quality on the overall assessment of oxygenate benefits within the RFG program.

The NRC's recent report on *Ozone-Forming Potential of Reformulated Gasoline* raises serious questions regarding the contribution of cleaner burning facts to the nation's air quality program in general, and the specific contribution of oxygenates in cleaner burning gasoline formulation. The NRC report correctly captures the impact of the substantial advances in automotive emissions controls over the past decades. However, it diminishes the value of fuel controls by ignoring real-world impacts and focusing exclusively on direct oxygenate impacts on ozone. A more detailed discussion of the NRC report was prepared by the Oxygenated Fuels Association, of which Huntsman is a member, and is attached as Appendix A.

### 3. *The BRP Did Not Have the Most Up-to-date Information on the Underground Storage Tank Program*

The BRP presented detailed recommendations aimed at making the Underground Storage Tank (UST) program more effective. Huntsman Corporation agrees with those recommendations. Unfortunately, when the BRP made its recommendations, it did not have the most up-to-date information on the effectiveness of the UST program. Had the BRP had this information, its recommendation would still have been appropriate, but it would have had even less basis upon which to recommend a substantial reduction in the use of MTBE.

Most MTBE detections in groundwater were found prior to the UST regulation implementation deadline (December 1998). The MTBE contamination data presented by the USGS and reviewed by the BRP was collected between 1988 and 1998 when underground storage tanks were only 25 percent to 50 percent in compliance with EPA's regulations. Data presented by the Association of State and Territorial Solid Waste Management Officials (ASTSWMO) show that less than 50 percent of all USTs were in compliance prior to 1998 and that as recently as 1996 only 30 percent were in compliance.<sup>4</sup>

There is also evidence that the risk of drinking water contamination by MTBE and other gasoline constituents has been greatly reduced with the onset of UST regulation compliance. The University of California at Davis study<sup>5</sup>, part of which was presented to the BRP March 25 & 26, 1999, showed that tank failure rates decrease by over 95 percent (from 2.6 percent failures per year for non-upgraded tanks to 0.07 percent per year for upgraded tanks) once tanks were upgraded to the current UST regulations. Also, with the required installation of early leak detection monitors, the time between when a leak occurs and when it is detected should be significantly reduced. As a result, the amount of gasoline released from a site before it has been remediated is minimized. Both of these effects combined should lead to substantial reductions in the amount of MTBE and other gasoline components that escape undetected.

### 4. *The BRP Failed to Adequately Assess Alternatives to MTBE*

One of the most disturbing shortcomings of the BRP's report is its failure to provide an analysis of the alternatives to MTBE. The BRP's recommendation to reduce the use of MTBE is of little use to policymakers if there is no credible alternative. It is our view that potential alternatives should be evaluated according to the same criteria by which MTBE is judged, to wit:

- whether the alternative yields the same real air quality benefits as MTBE;
- whether the alternative presents no significant risk to human health and the environment; and
- whether the alternative is preferable from the standpoint of cost and availability.

Huntsman Corporation agrees with the BRP recommendation to more fully investigate any major new additives to gasoline prior to their introduction. We would expect that this process should apply to the alternatives already identified by the panel, namely ethanol, alkylates, and aromatics. We should be hesitant to accept

<sup>4</sup>Sausville, Paul, Dale Marx and Steve Crimauco: *A Preliminary State Survey with Estimates Based on a Survey of 17 State Databases of Early 1999*, ASTSWMO UST Task Force, 11th Annual EPA UST/LUST National Conference, March 15-17, 1999, Daytona Beach, Florida.

<sup>5</sup>Keller, Arruro, et. al *Health and Environmental Assessment of MTBE, Report to the Governor and Legislature of the State of California as Sponsored by SB 521*, November 1998.

expanded use of these alternatives without more rigorous analyses of their respective impacts on human health, air and water quality, as well as gasoline supply and price.

It is especially disturbing that the most often suggested alternative to MTBE—ethanol—has not been subjected to a more rigorous analysis under the criteria set out above. For example, there are serious questions as to whether ethanol yields the same real/air quality benefits as reformulated gasoline using MTBE. Ethanol has a higher volatility; it evaporates more readily, creating more air pollution. EPA has acknowledged that the increased use of ethanol will result in increased emissions of nitrous oxides (NO<sub>x</sub>). And in addition to contributing to ozone exceedences, emissions of NO<sub>x</sub> contribute to elevated ambient levels of nitrogen dioxide and fine particulate matter, both of which are criteria pollutants for which EPA has established national ambient air quality standards.

Even assuming that there are some air quality benefits from the use of ethanol, those benefits as likely to be outweighed by the environmental costs of growing more corn and other ethanol feedstocks. The production of corn in the United States involves substantial applications of fertilizers, herbicides and pesticides. It would be interesting to compare the current incidence of MTBE contamination in drinking water supplies with the incidence of drinking water supplies contaminated with atrazine and other farm chemicals if corn production were to expand to the level necessary to produce enough ethanol to replace MTBE as an oxygenate in reformulated gasoline.

The BRP also failed to consider important issues related to the cost and availability of alternatives to MTBE, especially ethanol. First, ethanol production results in a net negative energy yield; it has been proven that it takes more energy to make a gallon of ethanol than you get from that gallon of ethanol. According to the Department of Agriculture, it takes 75,000 to 95,000 Btu's for a gallon of ethanol, and yet the gallon of ethanol yields only 76,000 Btu's.

Second, each gallon of ethanol receives a tax subsidy of 54 cents. In a March 1997 letter report, the U.S. General Accounting Office estimated that the subsidy for alcohol fuels reduced motor fuels excise tax revenues by about \$7.1 billion from fiscal years 1979 to 1995. Congressman Bill Archer, Chairman of the House Ways and Means Committee, has estimated that the ethanol tax credit will cost American taxpayers approximately \$2.4 billion between 1997 and 2000.

In addition, expanded ethanol production will increase the cost of gasoline at the pump, and will add to consumers' grocery bills. The cost of gasoline at the pump will increase because there will be less competition among fuel additives. The cost of food products will increase because as the demand for corn increases, the cost of corn used as animal feed will increase. Thus, the price of pork, beef and chicken in the supermarket will increase.

Finally, there is no guarantee that ethanol can replace MTBE as the oxygenate of choice without creating serious supply disruptions and, as a result, price increases. Because of its physical characteristics, ethanol cannot be blended with gasoline at the refinery and shipped by pipeline or barge to the marketplace. It must be transported separately and blended with gasoline near the location where it is to be sold to consumers. This limitation on the ability to transport gasoline with ethanol presents additional risks of supply disruptions and price dislocations. Right now, ethanol is being supplied to metropolitan areas in the vicinity of the ethanol producing facility with reasonable reliability. It remains to be seen whether ethanol could be transported nationally in a reliable and cost-effective fashion.

Ultimately, the net impact of whatever decision is taken will be reflected in the economics of the marketplace. There can be no doubt that the mandated use of oxygenates, primarily achieved by MTBE use, extends the nation's fuel supply. This helps keep prices in check and helps overcome localized spot shortages when they do occur. The recent California experience of increased MTBE use during gasoline shortages brought about by refinery hardware problems serves as a clear reminder of that fact. California Energy Commission studies showed a cost of 3-7 cents per gallon to remove MTBE from California's gasoline, assuming essentially unlimited and reasonably priced supplies of clean burning replacement blendstocks of ethanol and alkylate.

The true economic impact of a national phase down (or phase-out) of MTBE is likely to be dramatically higher, depending on the specifics of the action taken. MTBE makes up 10 to 15 percent of gasoline volume in RFG markets. With refinery capacity utilization currently running at 98 to 99 percent in the U.S., it is not difficult to see that drastic action could have catastrophic consequences. Members of Congress should insist on fully defining this price and supply risk to the American motoring public before it considers modification of the Clean Air Act.



## IV. CONCLUSION

Again, on behalf of the Huntsman Corporation, I would like to thank the Subcommittee for this opportunity to present our views on this important issue. We respect the work that the Blue Ribbon Panel has conducted and agree with many of its recommendations. At the same time, we feel strongly that the Blue Ribbon Panel's report is only the first—albeit important—step toward addressing the problem of contamination of drinking water supplies. We hope we have identified some of the important questions that the BRP has highlighted and which remain to be fully answered. Until those questions are answered, we believe there is no sound basis upon which to limit the use of a chemical—MTBE—which has helped to achieve important air quality goals.

## APPENDIX A.—COMMENTS ON: NATIONAL RESEARCH COUNCIL REPORT

## AN ANALYSIS OF THE NATIONAL RESEARCH COUNCIL'S REPORT ON OZONE-FORMING POTENTIAL OF REFORMULATED GASOLINE

## SUMMARY

The recently issued report by the NRC's Committee on Ozone-Forming Potential of Reformulated Gasoline, raises serious questions regarding the contribution of cleaner burning fuels to the nation's air quality improvement programs. The study questions the effectiveness of the reformulated gasoline program in general, and the specific contribution of oxygenates (such as MTBE and ethanol) in cleaner burning gasoline formulations. Several of the study's conclusions appear to contradict real-world air quality monitoring results and are inconsistent with previously held beliefs that the use of RFG significantly improves air quality.

The NRC Committee searches for certainty in the complex field of fuel contributions to atmospheric impacts, where few simple, direct cause-and-effect answers exist. The NRC report correctly captures the impact of the substantial advances in automotive emissions controls over the past decades. However, it improperly diminishes the value of fuel controls by ignoring real-world impacts and focusing exclusively on direct oxygenate impacts on ozone. More specifically, the report suffers from several fundamental drawbacks:

- It ignores real world air quality shifts associated with cleaner burning oxygenated fuels. EPA data based on actual RFG air quality surveys clearly indicate that, since 1995, the reformulated gasoline program has delivered emissions benefits substantially exceeding the minimum anticipated requirements. While vehicle technology advances have contributed the lion's share of the ambient air quality gains recorded since the 1960's, it is difficult to see how vehicle controls have changed substantially since the introduction of RFG. Furthermore, although the NRC report briefly alludes to real-world conditions when the vehicle emissions controls may not be active (i.e., cold start) or are not operational (high emitters), it largely bases its conclusions on laboratory controlled engine testing on low emitters (newer, well-maintained vehicles) under equilibrium conditions, where fuel contributions are appreciably diminished.

- It attempts to identify an exclusive, direct oxygen contribution to ozone abatement, similar to the one seen for carbon monoxide. Such an impact can not be supported on newer vehicles featuring advanced emission controls. However, understanding the indirect pathways by which oxygenates impact ozone, is essential to the *overall* assessment of oxygenate benefits. The report does not credit oxygenates for indirect impacts on other key fuel parameters that do, in turn, impact VOC and NO<sub>x</sub> emissions. By diluting gasoline sulfur, olefins, aromatics and benzene, and lowering gasoline mid-range volatility, oxygenates substantially (albeit indirectly) impact ozone precursor formation. Furthermore, oxygenates allow refiners octane flexibility to implement operating changes that reduce gasoline benzene and aromatics content. When these indirect VOC and NO<sub>x</sub> ozone precursor reductions are considered along with Carbon Monoxide reductions, NRC's focus solely on direct ozone impacts appears oversimplified and misleading.

- The Committee clearly exceeded its primary task (i.e., assess RFG ozone impacts) to evaluate impacts on other pollutants such as carbon monoxide, air toxics, etc. A comprehensive review of RFG and oxygenate benefits is ordinarily welcomed; however, the NRC report is largely superficial in presenting oxygenate impacts on these other key pollutants. The report does grant that "the most significant advantage of oxygenates in RFG appears to be a displacement of some toxics (e.g., benzene) from the RFG blend, which results in a decrease in toxic emissions." This substantially understates the facts: according to EPA, oxygenates are responsible for

approximately two thirds of the large overcompliance reported in air toxics since the introduction of the RFG program. Similarly, oxygenates are directly responsible for a 10–15 percent reduction in CO. As a result of focusing only on direct ozone impacts and the inadequate treatment of non-ozone pollutants, the NRC report leads to the improper conclusion that oxygenates have no air quality benefits.

- Lastly, the report leads to the erroneous conclusion that fuel controls may not play a key role in future air quality strategies. A key premise of the current RFG program is that reformulated fuel and vehicle controls contribute to emissions reductions in a mutually complementary way, i.e., they have been designed to function as a closely interactive system. In very simple terms, poorly performing vehicles are likely to pollute more and the fuel's role in optimizing vehicle performance is critical. The need for lower sulfur, improved distillation index controls and reduced combustion chamber deposits (CCD) are all testaments of the close coupling between fuel controls and vehicle technologies. Furthermore, in dismissing fuel contributions as potentially "small," the report ignores the regulatory dilemma of identifying additional and/or alternative air quality controls in the face of ever increasing vehicle miles traveled and continued pressure to improve ambient air quality. Ozone control strategies often depend on small incremental reductions in VOC or NO<sub>x</sub> emissions, which should be evaluated in terms of their magnitude and cost effectiveness versus remaining candidate controls, and not already implemented strategies.

#### BACKGROUND

At the request of Congress, the U.S. Environmental Protection Agency (EPA) asked the National Research Council (NRC) to:

- Assess whether the existing scientific information allows a comparison of the ozone forming potential of automotive emissions obtained with different reformulated gasolines,

- Evaluate the impact of applying the "ozone forming potential" approach to air quality on the overall assessment of oxygenate (i.e., methyl tertiary-butyl ether and ethanol) benefits within the RFG program.

The NRC report's conclusions were unexpected. While the study concedes that ground level ozone has declined by more than 10 percent since 1995, it claims that it is not possible to attribute a significant portion of these benefits to the introduction of reformulated gasoline during this period. Instead, the NRC study concludes that overall emissions of ozone precursors have substantially decreased in recent decades, largely as a result of better emissions control systems on vehicles. Furthermore, given the declining contribution of fuel formulations to air quality, the study concludes that, the *direct* contribution of oxygenates to ozone reduction is very small. Last, the NRC finds that evaluating fuel formulations based on their ozone reactivity potential (rather than strictly comparing mass emissions) does not alter the study's conclusions, *even though* it acknowledges that the ozone forming potential of carbon monoxide in exhaust emissions is large and has not been comprehended in existing fuel evaluation tools.

#### DISCUSSION

##### *Real World Emissions Performance*

The NRC study acknowledged that ambient monitoring data demonstrate that RFG successfully helps reduce ozone levels, and lowers overall ambient air toxics levels. According to EPA data, Phase I RFG areas have performed better than planned:

- VOC reductions average 36 percent in the south and 17 percent in the north (goal is 15 percent);
- Air toxics reductions average 22 percent (goal is 16.5 percent when averaging);
- NO<sub>x</sub> reductions average 3 percent (goal is 1.5 percent when averaging);
- Ambient benzene levels have decreased by 43 percent.

EPA's RFG Survey Group has estimated the average vehicle emission reductions by using fuel surveys for each of the cities in the Federal RFG Phase I program:

- Reductions in toxics from vehicles for all RFG cities far exceed the Performance Standard and that the average reduction is about double the requirement. More specifically, reductions in cities with RFG/MTBE blends exceed 35 percent in most cases while the average reduction for the four cities using RFG/ethanol blends is only about 27 percent. It would appear that the 2 percent oxygen standard and the benzene standard in RFG combine to provide a reduction in toxic emissions that is much greater than the Phase 1 Performance Standard as well as the Phase 2 Performance Standard for the year 2000 (22 percent minimum average reduction). Without the oxygen requirement, the toxic reductions with RFG would be expected to decrease to near the performance standard.

- NO<sub>x</sub> reductions with RFG are more than double (in many cases more than triple) the Performance Standard required by CAA regulations (1.5 percent reduction). In most cases, the NO<sub>x</sub> reductions with the MTBE/RFG blends are a few percentage points greater than that observed with the cities using ethanol blends. Low RVP fuels actually increase NO<sub>x</sub> emissions and are, thus, ineffective program for reducing peak ozone.

- VOC reductions for RFG cities generally exceed the corresponding performance standard by 1.5 to 8 percent. The performance standard for VOC reduction is more severe for southern cities as compared to northern cities. The cities using RFG with ethanol are all located in the north while low RVP cities (Atlanta, St. Louis, Phoenix, etc.) are mostly located in the south. There is little difference between RFG made with ethanol and MTBE in reducing VOC emissions. However, low RVP gasoline only provides about two-thirds of the VOC reduction as that observed for southern RFG cities.

Based on ambient monitoring data, RFG areas have performed better than conventional gasoline areas (including lower vapor pressure areas) in lowering the frequency of ozone exceedances as shown in Figure 1. By focusing on percent change, the effects of fleet turnover, weather, economic activity and related factors cancel out in the comparison. The control data set used was conventional gasoline areas because fuel standards did not change in those areas.

## Compare Ozone Exceedances by Type of Gasoline Used

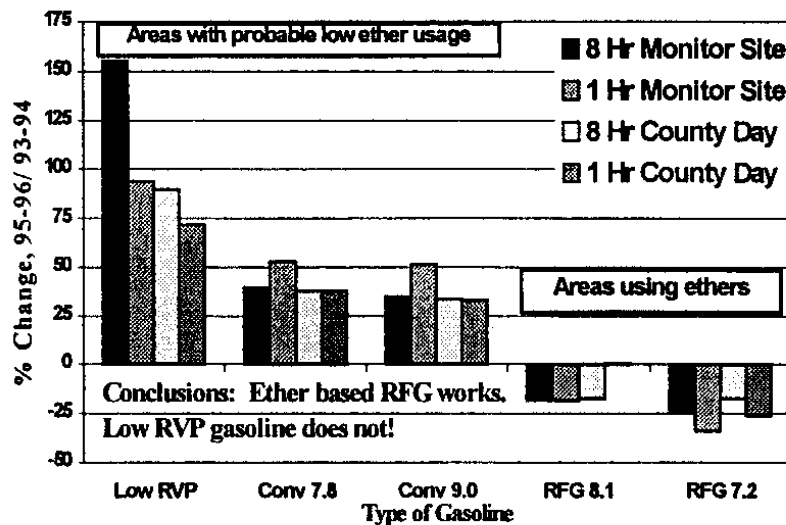
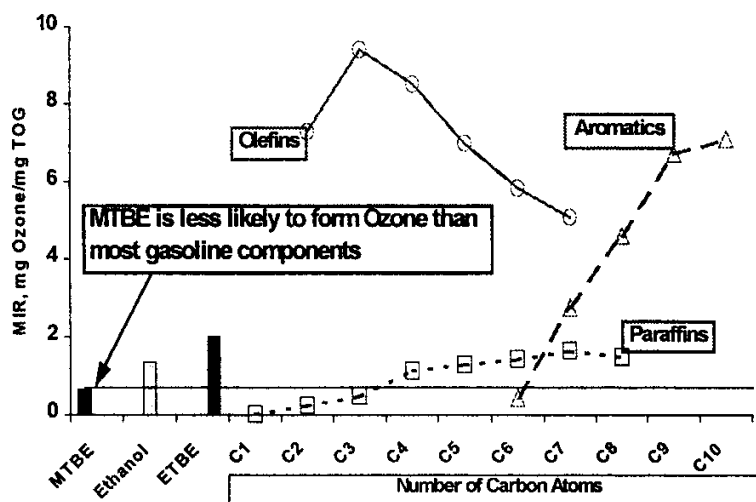


Figure 1

Figure 2 shows that MTBE is less likely to form ozone than most gasoline components. Only benzene, which is reduced in RFG, has a lower potential. Limited Auto/Oil results comparing "matched" oxygenated and a non-oxygenated reformulated gasoline blends showed that the oxygenated fuel had a 5-7 percent lower ozone forming tendency. It is noted that the results of that study likely underpredict the ozone reactivity impact, since despite efforts to control the experimental design, the oxygenated fuel had an octane value of 2.4 numbers above that of the non-oxygenated fuel. If the fuels had been octane balanced, the differences in emissions would have been even greater in favor of the cleaner burning oxygenated gasoline.

Figure 2

## Ozone Potential: Oxygenates Vs Average Paraffins, Olefins & Aromatics



### Indirect Performance Benefits of Oxygenates

The NRC study examined the direct impacts of oxygen on VOC and NO<sub>x</sub>. The Committee did not attempt to identify the significant *indirect* benefits of oxygenate blending to RFG. The Table below provides a simplified roadmap to understanding the relative direct and indirect contributions of oxygenate blending to reduced gasoline emissions.

### Air Pollutant

Oxygenate Action Pathway	CO	VOC	NO <sub>x</sub>	Air Toxics
Direct Impact	++	?	?	+
Indirect Impact(s)				
- Dilution	+	+	+	+
- Reduced Severity		+		++
- Distillation	+	++	+	+

The NRC's main focus was on the direct impact of oxygenates on VOC and NO<sub>x</sub>. As shown on the table above, this is correctly judged to be a rather insignificant impact. The primary fuel impacts on VOC are vapor pressure (RVP), midrange distillation (T50) and sulfur content. For NO<sub>x</sub>, the primary impact variables are olefin content and sulfur. The assumed oxygen level in this Table is 2 weight percent; at this level the NO<sub>x</sub> impact is negligible. However, as the NRC report points out, at

3.5 weight percent oxygen (i.e., maximum ethanol) can lead to increased NO<sub>x</sub> emissions.

The problem with the NRC analysis is that it fails to recognize the large indirect impacts that oxygenate addition would have on a typical conventional fuel:

- 10–15 percent dilution impact in sulfur, olefins, aromatics and benzene
- 15–20 degree Fahrenheit depression in T50
- 4–7 degree Fahrenheit reduction in T90
- 6–8 volume percent reduction in aromatics content
- 0.2–0.3 volume percent reduction in benzene

Oxygenates do not contain sulfur, olefins aromatics or benzene; they dilute their presence in the RFG blend. Dilution is very important to allow refiners to achieve the RFG air standards while maintaining fuel quality. High oxygenate octane values contributes to aromatics reduction by permitting lower severity in the key catalytic reforming step. Without oxygenate blending, most refiners would find it extremely difficult to produce satisfactory RFG in the premium grade, and probably in the mid-range grade. Such blending advantages for MTBE were unaccounted for in the NRC study.

It should be noted that of the four pollutants listed above, only CO is directly impacted in a very large way by oxygenates. However, this diminish our view of the value of oxygenates in generating overall pollution reduction benefits:

While there is no direct impact on VOC, the indirect effects of lowering midrange distillation, diluting and replacing aromatics, and reducing sulfur combine to yield a sizable VOC reduction benefit, estimated at approximately 10–15 percent of the total RFG VOC reduction.

Similarly, the combined impact of indirect aromatics and benzene reductions resulting from dilution and refinery operating adjustments is equally large at approximately 20 percent of the overall RFG air toxics benefit.

The large direct contribution of oxygenates to CO reduction is amplified by indirect benefits accruing as a result of the reduction in sulfur and T50. As a result, the portion of RFG's CO reduction benefits attributable to oxygenate use exceeds 30 percent.

Of the four pollutants listed, only oxygenate contributions to NO<sub>x</sub> could be described as "small." This is because the oxygenate benefits are primarily accruing as a result of the 10–15 dilution expected in sulfur and olefins content via dilution.

The discussion above is not aimed at fully comprehending all the indirect benefits of oxygenates. For example, reduction in fuel aromatics content should result in lower fuel combustion chamber deposit forming tendency, which will, in turn, result in additional air quality benefits. Furthermore, while the NRC acknowledges that "as VOC emissions from mobile sources continue to decrease in the future, CO will become proportionately an even greater contributor to ozone formation," it fails to credit the large direct contribution of oxygenates in this area.

In conclusion, it would appear that the assessment of oxygenates in cleaner burning gasolines is largely dependent on the reviewer's definition of the action pathways or mechanisms included in the accounting of air quality impacts. While there can be little doubt that oxygenates have a substantial favorable impact on the remaining fuel properties, the NRC study (like the University of California study before it) does not credit oxygenates with any of the emissions shifts associated with these secondary fuel impacts. While it can be argued that such oxygenate benefits can be replaced and thus should not be credited entirely to oxygenate use, there exists no basis to completely discount indirect oxygenate impacts. Moreover, by failing to focus on real world performance results, reviewers such as the NRC have tended to underestimate the fuel's contribution to the air quality gains of the recent past. This, in turn, risks projecting the erroneous view that there is limited value in the fuel component of what has heretofore been a very successful partnership between reformulated gasoline and vehicle emissions controls.

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STATEMENT OF ASSOCIATION OF METROPOLITAN WATER AGENCIES AND AMERICAN  
WATER WORKS ASSOCIATION

The Association of Metropolitan Water Agencies (AMWA) and the American Water Works Association (AWWA), on behalf of the nation's drinking water suppliers and their consumers, offer this statement regarding methyl tertiary butyl ether (MTBE).

AMWA is a nonprofit organization comprised of the nation's largest publicly-owned drinking water suppliers, represented by their city water commissioners and chief executive officers. Together, AMWA members serve clean, safe drinking water to over 120 million people.

AWWA is the world's largest and oldest scientific and educational association representing drinking water supply professionals. The association's 56,000 members are comprised of administrators, utility operators, professional engineers, contractors, manufacturers, scientists, professors and health professionals. The association's membership includes over 4,200 utilities that provide over 80 percent of the nation's drinking water. Since our founding in 1881, AWWA and its members have been dedicated to providing safe drinking water.

AWWA and AWWA subscribe to the recommendations set forth by the Blue Ribbon Panel on Oxygenates in Gasoline in their July 1997 report, with one very significant exception: the associations feel strongly that MTBE should be completely phased out.

MTBE is a known animal carcinogen and potential human carcinogen. Little else is known about its health effects. What we do know is that it has been found in 5 to 10 percent of drinking water supplies in high oxygenate use areas. MTBE has been found in city water supplies in California, New York, New Jersey, Massachusetts, Maine, and other states.

MTBE occurs mostly at low levels, and even at extremely low levels MTBE produces taste and odor concerns among consumers. Most consumers perceive drinking water with an unpleasant taste or odor as being unhealthy, and in some cases the water may very well be unsafe to drink. The bottom line is that consumers will not tolerate MTBE in their water.

The effect of these perceptions, accurate or not, is that consumers purchase bottled water costing 500 to 1,000 times more than tap water. And cities like Santa Monica and South Tahoe have abandoned otherwise usable ground water and surface supplies and turned to other sources usually at enormous expense. An alternative is to treat MTBE-contaminated water, but this comes with extraordinary cost. What's more, MTBE contaminates private wells, which are less well-protected by Federal or State regulations, if at all.

In addition to the complete phase out of MTBE use, AMWA and AWWA support the recommendations of the Blue Ribbon Panel, particularly:

- Enhancing Federal and State underground storage tank programs, including:
  - having all states prohibit fuel deliveries to non-upgraded tanks,
  - adding enforcement and compliance resources to ensure prompt action,
  - strengthening early detection and remediation mechanisms,
  - requiring monitoring and reporting of MTBE at all storage tank release sites, and
  - encouraging states to require that land-use planning consider the impact of underground storage tanks on water supplies;
- Enhancing the focus on MTBE in the Safe Drinking Water Act's source water assessment program, contaminant monitoring initiatives, and wellhead protection program?
  - Encouraging State and local governments to restrict the use of gasoline-powered water craft in lakes and reservoirs that serve as drinking water supplies;
  - Expanding programs to protect private well users;
  - Expanding public education programs on the proper handling and disposal of gasoline,
  - Developing and implementing a research program into the groundwater behavior of gasoline and oxygenates;
  - Expanding Federal resources for the treatment of drinking water supplies contaminated with MTBE and other gasoline and for securing an alternative source of water, if necessary;
  - Researching or increasing research on other ethers and oxygenates to determine their health effects and environmental behaviors; and
  - Estimating the current and likely future threats of MTBE contamination and establishing a system of collecting data on MTBE, other ethers, Ethanol, and petroleum hydrocarbons.

The nation's drinking water suppliers are deeply concerned about MTBE contamination. The additive poses a very significant threat to health, the environment, and the continued provision of affordable, safe drinking water.

STATEMENT OF STEVE HALL, EXECUTIVE DIRECTOR OF THE ASSOCIATION OF CALIFORNIA WATER AGENCIES

Mr. Chairman, members of the subcommittee, my name is Steve Hall and I am the Executive Director of the Association of California Water Agencies (ACWA). I am pleased to submit this statement to share the California Water community's concerns with the continued mandated use of MTBE in reformulated gasoline in Cali-

fornia. ACWA represents over 440 urban and agricultural water utilities throughout the State of California, which deliver more than 90 percent of the water distributed in California.

MTBE is a known animal carcinogen and potential human carcinogen. Existing health studies are inadequate to determine the risk posed by MTBE in drinking water. Yet, it has become the third most common chemical manufactured in the United States. It constitutes about 11 percent of the gasoline in areas such as Los Angeles, San Diego, and Sacramento. The University of California estimated that as many as 10,000 wells may be contaminated with MTBE in California. This could have a huge impact on the State's water resources and on the cost to consumers of providing alternate drinking water supplies.

Our consumers can taste MTBE in their water at extremely low concentrations, in the range of 5 parts per billion (this is equivalent to less than a tablespoon of MTBE in an Olympic-sized pool). If our consumers taste a chemical that is a known animal carcinogen and potential human carcinogen, they very often choose to buy bottled water at a cost of 500 to 1,000 times more than the cost of tapwater. Also, MTBE is a man-made chemical. There is no good reason why it should be present in our drinking water.

MTBE is a unique contaminant in water. It spreads into our drinking water aquifers faster than nearly all other constituents in water. It moves faster than regulatory agencies can track it and faster than water utilities can drill new wells to replace the contaminated supplies. Unlike most organic chemicals, MTBE does not biodegrade rapidly in water. Once it has leaked into our groundwater or spilled into our drinking water reservoirs, it persists.

A recent study conducted by the University of California has recommended that use of MTBE be eliminated in California. The Governor of California acting on this recommendation issued Executive Order D-5-99 to phase out the gasoline additive by December 31, 2002. However, this executive order cannot be properly implemented under existing Federal requirements for mandatory use of oxygenates within the Clean Air Act. Changes to Federal law are needed to implement Governor Davis' executive order to phase out MTBE.

Water suppliers in California have already been severely impacted by MTBE. The city of Santa Monica lost 50 percent of its well production capacity and has had to switch to more expensive imported water from Northern California. South Tahoe Public Utilities District has lost one third of its well capacity. Unfortunately, South Tahoe has no imported water to replace its lost supplies and is at risk of water shortages. Many other utilities throughout the State have shut down wells or bypassed water supply reservoirs rather than risk having the fast-moving, persistent MTBE making its way into consumers' taps.

MTBE has also impacted individuals with private wells. Residents of the city of Glenville were drinking water with MTBE levels as high as 20,000 parts per billion, which is 1,000 times greater than the California Public Health Goal of 13 parts per billion. These documented contamination incidents are likely to be a preview of future cases.

The primary source of groundwater contamination by MTBE is leaking underground storage tanks. While ACWA supports increased upgrading, monitoring and enforcement of underground fuel tanks, these actions alone will not solve the MTBE problem. A recent study conducted by the Santa Clara Valley Water District examined 28 underground storage tanks with no reported leak history and all of which met 1998 upgrade standards. The District found that 13 of the 28 tanks had leaked MTBE into the soil or underlying groundwater. This study shows that upgrading underground storage tanks will not stop MTBE from entering the environment and contaminating drinking water sources.

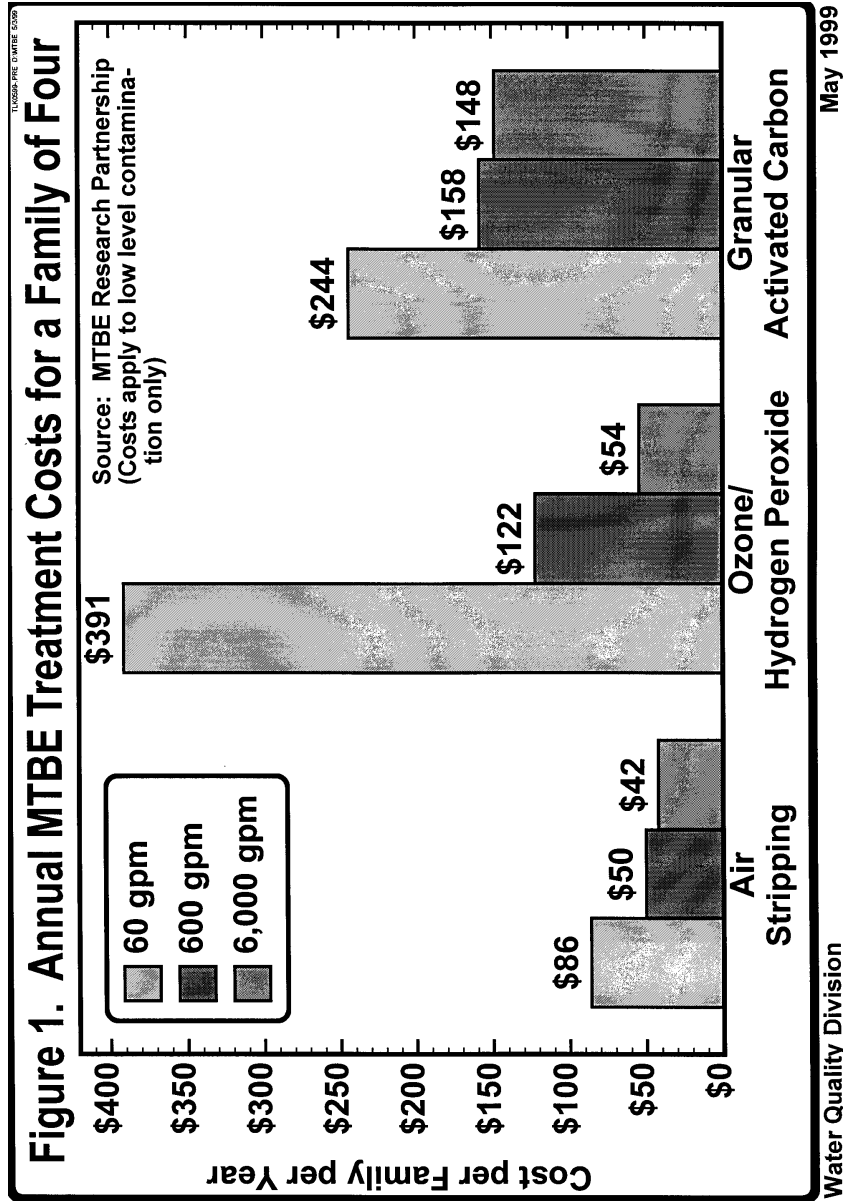
The cost of removing MTBE through treatment is very high. The University of California has estimated these treatment costs to range from \$3450 million to \$1.5 billion in California alone. Other existing treatment processes would be even more expensive (see Figure 1).

To develop new, less expensive treatment technology, an MTBE Research Partnership was created by the Association of California Water Agencies, the Western States Petroleum Association, and the Oxygenated Fuels Association. The Partnership focuses on developing new, cost-effective treatment technology to handle existing contaminated drinking water supplies and developing source protection technology to protect uncontaminated sources. This is a cooperative step in the right direction.

ACWA has also supported all major State legislation on MTBE, including bills by Kuehl/Hayden, Sher, Mountjoy, and Cunneen. However, California MTBE legislation cannot override the Federal mandate for the use of an oxygenate like MTBE.

The oxygenate requirement in Federal law is not necessary to meet clean air standards. California has demonstrated that it can meet all of the health and air requirements of the Clean Air Act without the use of MTBE. With the elimination of MTBE, it will be possible to have both clean air and clean water.

About 50 years ago, the pesticide DDT came into widespread use throughout the world to control mosquitoes. It saved millions of lives by preventing Malaria. Then we found that DDT had unintended consequences on the environment and it had to be phased out. MTBE is similar. It has clearly helped clean up the air; however, there have been unintended consequences to the drinking water. It is time to phase-out MTBE.





[From Oxygenated Fuels Association, Inc., October 5, 1999]

FORMER SENATOR GARN TELLS SENATE PANEL TO SAVE FUEL ADDITIVE MTBE—  
CLEANER FUEL, CLEANER AIR, LOWER COSTS CITED AS REASONS TO STOP EFFORT  
TO BAN

ARLINGTON, VA.—Former Senator Jake Garn (R-UT) today told the Clean Air, Wetlands, Private Property, and Nuclear Safety Subcommittee of the Senate Environment and Public Works Committee that the push to ban or phase-down MTBE will undermine our nation's clean air progress and do nothing to improve clean water standards.

"MTBE has an unparalleled record of cleaning up the air that we all breathe. Banning MTBE will reverse its successful record of cleaning our air—a success which has been accelerating since passage of the 1990 Amendments to the Clean Air Act," Garn testified. He also noted, "Banning MTBE is certainly no substitute for proper enforcement of State and Federal rules which ensure the integrity of underground gasoline storage tanks."

MTBE has been the fuel oxygenate of choice for most refiners because it effectively reduces air pollutants like toxic lead and cancer causing benzene from car emissions. In addition, MTBE has helped keep the cost of cleaner burning gasoline down. Various studies have concluded that banning or restricting MTBE will lead to higher pump prices and gasoline shortages.

The focus of much of today's hearing was leaking underground storage tanks. Trace amounts of MTBE in some sources of drinking water have caused calls for the banning of MTBE in fuel. Mr. Garn testified today that it's the tanks and not MTBE that needs to be the focus of attention in this debate.

In his testimony today, Senator Garn was representing Huntsman Corporation, where he serves as Vice Chairman of the Board of Directors. Huntsman is the largest, privately-owned chemical company in the U.S. and a major supplier of MTBE for America's clean fuels program. Huntsman Corporation is a member of the Oxygenated Fuels Association (OFA).

Speaking on behalf of OFA, Executive Director Terry Wigglesworth said she was confident that the Clean Air, Wetlands, Private Property, and Nuclear Safety Subcommittee of the Senate Environment and Public Works "will consider all of the evidence provided today, and will come to an appropriate conclusion based on science and fact.

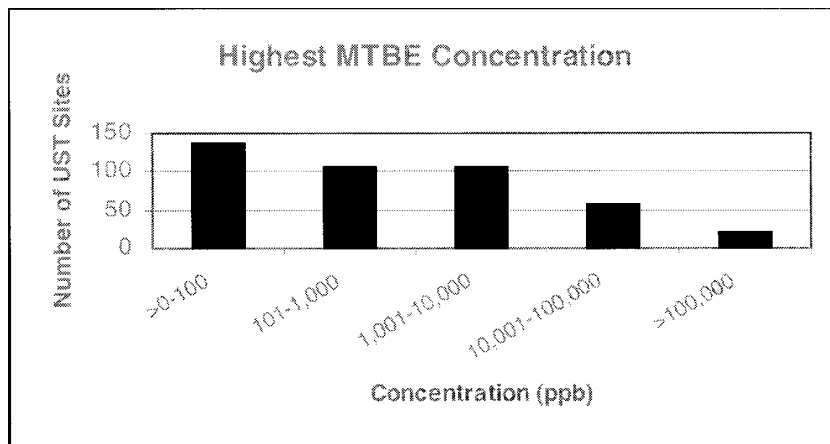
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STATEMENT OF SANTA CLARA VALLEY WATER DISTRICT

The Santa Clara Valley Water District is the water resource management agency serving the wholesale water supply and flood protection needs of the 1.6 million residents in Santa Clara County, California, with its thriving Silicon Valley economy. In fulfilling its water supply mission, SCVWD owns and operates ten reservoirs (total capacity of approximately 163,000 acre feet), three water treatment plants (total capacity 220 million gallons per day), and 393 acres of groundwater recharge ponds. SCVWD is also responsible for protecting water quality of its local groundwater basin that provides approximately 50 percent of the County's water supply needs.

The Santa Clara Valley Water District is implementing a comprehensive program to address Methyl Tertiary Butyl Ether (MTBE) contamination in its water supplies and has been recognized as a leader in the water community on this issue. We have reviewed the findings and recommendations from *The Blue Ribbon Panel on Oxygenates in Gasoline* and generally agree with the recommendations addressing water contamination. In fact, SCVWD is implementing where it can many of the Blue Ribbon Panel's recommendations at the local level, and supporting their implementation at the State level. Because of its chemical properties and widespread use in California, SCVWD has taken the position that MTBE should be completely removed from gasoline.

SCVWD has been monitoring its water sources for MTBE over the past several years and continues to find it. Monitoring of our imported supplies from the Sacramento-San Francisco Bay-Delta periodically shows concentrations of 1–2 parts per billion (ppb). Monitoring has also shown concentrations up to 24 ppb at three of our local surface water reservoirs where we allow motor-powered watercraft recreation.



Our greatest concern, however, continues to be contamination of local groundwater basins from leaking underground storage tanks. The SCVWD operates a Leaking Underground Storage Tank Oversight Program (LUSTOP) to assist State regulators in this area. Most of the underground storage tank sites that are listed as cases in our LUSTOP program have monitored for MTBE and a total of 425 sites have detected MTBE, many at very high levels as shown on the accompanying graph. Almost 60 percent of the sites with detections show MTBE greater than 100 ppb. This phenomenal rate of MTBE contamination is in the shallow groundwater aquifers. Our concern is that this contamination will eventually impact water supply wells deeper in the aquifer. So far, only one water supply well in the County has been impacted; however, the source investigation of this impacted well depicts another problem with MTBE. Because of its high mobility, MTBE plumes are very challenging to define and clean up since they can be long and narrow. A very detailed investigation of the local geology is required to properly assess the impact. The evidence developed to date indicates that a nearby gasoline station with a state of the art, upgraded underground storage tank system, is the source of contamination for this well. Because of MTBE's mobility, we do not believe the current data set fully represents the severity of MTBE contamination from leaking tanks since this data was gathered from current fixed monitoring wells at each site and most sites are not fully investigated.

The SCVWD also initiated a pilot study, which we have included with this testimony, to better determine the ability of upgraded tank systems to adequately contain MTBE. A total of 28 sites with fully upgraded underground storage tank systems were investigated to determine MTBE occurrence originating from these sites. None of these sites have previously shown signs of leakage. Groundwater was encountered at 27 sites and MTBE was detected in groundwater in 13 of these 27 sites at concentrations ranging from 1 ppb to 200,000 ppb. Concentrations over 1,000 ppb were detected at 5 sites. These data indicate that MTBE may be present in groundwater at approximately 50 percent of the underground storage tank facilities that meet 1998 upgrade requirements. This information is consistent with the conclusions of an advisory panel to the Governor of California that concluded there is evidence of MTBE occurrence from new and upgraded underground storage tank systems. The SCVWD study is the first to gather hard data on this issue.

Given the widespread contamination of the shallow groundwater basins from leaking underground storage tanks, the mobility and persistence of MTBE, and stringent California drinking water quality standards for MTBE, we have serious concerns that large portions, or perhaps all of our groundwater basins could become unusable as a water supply source due to MTBE contamination if its use continues indefinitely. Therefore, we feel that MTBE, and other ether oxygenates with similar chemical properties, should be removed from gasoline, and we support the findings and conclusions of *The Blue Ribbon Panel on Oxygenates in Gasoline*.

SOUTH TAHOE PUBLIC UTILITY DISTRICT,  
*South Lake Tahoe, CA, October 4, 1999.*

Hon. BOB GRAHAM,  
*Subcommittee on Clean Air, Wetlands, Private Property and Nuclear Safety,  
Committee on Environment and Public Works,  
U.S. Senate,  
Washington, DC.*

DEAR SENATOR GRAHAM: On behalf of the South Tahoe Public Utility District, I write to request that the enclosed statement be included in the formal record of the proceedings related to the Subcommittee's October 5, 1999 hearing into Federal requirements associated with oxygenates and the Clean Air Act.

Over the past several years, the District has worked diligently, and at great taxpayer expense, to respond to the serious health threats created from the use of the oxygenate MTBE. We believe our experiences with both the tank technology and the chemical and physical attributes of MTBE are especially relevant to the Subcommittee's review of the status of this Federal mandate.

In advance, thank you for including our comments in the record. If you or your staff have any questions, please let me know or contact Eric Sapirstein at (202) 466-3755.

Sincerely yours,

ROBERT BAER,  
*General Manager.*

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MTBE GROUNDWATER IMPACTS IN SOUTH LAKE TAHOE, CA, IVO BERGSOHN, C.HG.,  
HYDROGEOLOGIST, SOUTH TAHOE PUBLIC UTILITY DISTRICT

Methyl Tertiary-Butyl Ether (MTBE) plumes associated with gasoline releases from Service Station operations, in conjunction with water well construction practices and hydro-geologic conditions prevalent in the south shore area of the Tahoe Basin has resulted in the shut-down of 12 (12) of the thirty-four (34) municipal water supply wells owned and operated by the South Tahoe Public Utility District (District). Because of the known environmental behavior and low taste and odor thresholds for MTBE, District wells were put off-line to prevent the migration of MTBE plumes toward District wells and the drawdown of contaminants into deeper portions of the alluvial aquifer. A case example from the South Y Area is presented to show the potential extent of an MTBE plume in a dynamic groundwater environment and its impact on nearby water supply wells.

Groundwater production in the South Y Area is predominantly from a water table aquifer system formed in glacial outwash deposits consisting of fine to medium sands interbedded with silt and clay. A thick section of bedded clay, interpreted as representing Older Lake Bed Deposits, forms a regional aquitard beneath the water table aquifer at depths of approximately 90 to 135 feet across the area. Interbeds of low permeability silt within the outwash deposits form local semi-confined aquifer and perched water table conditions. The water table is relatively shallow and typically occurs at depths less than 30 feet. During seasonal high water table conditions, perched portions of the water table aquifer may rise to within 10 feet of land surface. Because of the historically excellent chemical quality of the water in the shallow system, the majority of the water supply wells completed in the area either wholly or, in part, pump groundwater from the water table system.

Results of contaminant assessment investigations performed in the South Y Area have identified a diving MTBE plume in the water table aquifer system. The delineated plume extends approximately 1,500 feet along its long axis and approximately 650 feet across its short axis. Along its extent, the plume is believed to vary from approximately 25 to 40 feet in thickness. Within the plume and away from the source area, the area of highest MTBE concentration progressively moves downward through the aquifer. Immediately beneath the source area, highest MTBE concentrations are found within the upper 30 feet of the aquifer system. Approximately 600 feet away from the source area, high MTBE concentrations are found in well samples collected from intermediate portions (30-60') of the aquifer system. At distances of approximately 1,000 feet away from the source area, high MTBE concentrations are found in well samples collected from deep portions (>60') of the aquifer system and at depths as great as 85 to 90 feet. Comparison of water level measurements from monitoring well clusters across the area show a strong downward vertical gradient. The observed vertical gradients are believed to be due to a combination of hydrogeologic effects, including seasonal recharge events and the

presence of higher conductivity materials at depth, and pumping effects from nearby District water wells.

The delineated MTBE plume has had a significant impact on District wells in the South Y Area. MTBE has been detected in the Tata No. 4 Well since this well was first sampled for MTBE in June 1996. Operational demands required the continued pumping of this well. In July 1998, MTBE concentrations increased to above the current California Department of Health Service (DHS) action level of 35 ppb and the well was immediately shut-down.

Following this well shut-down, very low levels of MTBE were for the first time identified in four other nearby operating wells. These other wells were subsequently shut-down in August 1998, to prevent the further spread of the plume. The total potential impact from the shut-down of these wells represents a water production loss of approximately 1.25 million gallons per day to the District and the South Lake Tahoe community.

## WELL SHUT-DOWNS

WATER ZONE	WELL	MTBE (ppb)	SHUT - DOWN
ARROWHEAD	Arrwhd 1	3.7	Sep-1997
ARROWHEAD	Arrwhd 2	1.8	Sep-1997
STATELINE	Blkrock 2	ND	Nov-1997
STATELINE	Blkrock 1	ND	Nov-1997
AIRPORT	Helen 2	ND	Mar-1998
AIRPORT	Helen 1	ND	Mar-1998
GARDNER MTN	Tata 4	37	Jul-1998
GARDNER MTN	South Y	0.1 j	Aug-1998
GARDNER MTN	Julie	0.2 j	Aug-1998
AIRPORT	Tata 3	0.2 j	Aug-1998
AIRPORT	Tata 2	0.2 j	Aug-1998
AIRPORT	Tata 1	0.1 j	Aug-1998

### PUBLIC HEALTH GOAL FOR METHYL TERTIARY BUTYL ETHER (MTBE) IN DRINKING WATER

#### SUMMARY

A Public Health Goal (PHG) of 0.013 mg/L (13 µg/L or 13 ppb) is adopted for methyl tertiary butyl ether (MTBE) in drinking water. The PHG is based on carcinogenic effects observed in experimental animals. Carcinogenicity has been observed in both sexes of the rat in a lifetime gavage study (Belpoggi et al. 1995, 1997, 1998), in male rats of a different strain in a 24-month inhalation study (Chun et al. 1992, Bird et al. 1997), and in male and female mice in an 18-month inhalation study (Burleigh-Flayer et al. 1992, Bird et al. 1997). In Sprague-Dawley rats receiving MTBE by gavage, statistically significant increases in Leydig interstitial cell tumors of the testes were observed in males, and statistically significant increases in lymphomas and leukemias (combined) were observed in females. In Fischer 344 rats exposed to MTBE by inhalation, statistically significant increases in the incidences of Leydig interstitial cell tumors of the testes were also observed in males, as well as renal tubular tumors. In CD-1 mice exposed to MTBE by inhalation, statistically significant increases in the incidences of liver tumors were observed in females (hepatocellular adenomas, hepatocellular adenomas and carcinomas combined) and males (hepatocellular carcinomas). The two inhalation studies (Burleigh-Flayer et al. 1992, Chun et al. 1992, Bird et al. 1997) and one gavage study (Belpoggi et al. 1995, 1997, 1998) cited in this document for the development of the PHG provided evidence for the carcinogenicity of MTBE in multiple sites and in both sexes of the rat and mouse. While some reviews have given less weight to the findings of Belpoggi et al. (1995, 1997, 1998) due to the limitations of the studies, Office of En-

vironmental Health Hazard Assessment (OEHHA) scientists found that they contribute to the overall weight of evidence. We reviewed these studies and the reported criticisms carefully, and found the studies are consistent with other MTBE findings, and are of similar quality to studies on many other carcinogens. This conclusion is consistent with the findings in the MTBE report (UC 1998) submitted by the University of California (UC). The results of all available studies indicate that MTBE is an animal carcinogen in two species, both sexes and at multiple sites, and five of the six studies were positive.

For the calculation of the PHG, cancer potency estimates were made, based on the recommended practices of the 1996 United States Environmental Protection Agency (U.S. EPA) proposed guidelines for carcinogenic risk assessment (U.S. EPA 1996f), in which a polynomial [similar to that used in the linearized multistage (LMS) model, but used empirically and without linearization] is fit to the experimental data in order to establish the lower 95 percent confidence bound on the dose associated with a 10 percent increased risk of cancer ( $LED_{10}$ ). It is plausible that the true value of the human cancer potency has a lower bound of zero based on statistical and biological uncertainties. Part of this uncertainty is due to a lack of evidence to support either a genotoxic or nongenotoxic mechanism. However, due to the absence of specific scientific information explaining why the animal tumors are irrelevant to humans at environmental exposure levels, a standard health protective approach was taken to estimate cancer risk. The cancer potency estimate derived from the geometric mean of the cancer slope factors (CSFs) of the combined male rat kidney adenomas and carcinomas, the male rat Leydig cell tumors, and the leukemia and lymphomas in female rats was  $1.8 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>.

The PHG was calculated assuming a *de minimis* theoretical excess individual cancer risk level of  $10^{-6}$  (one in a million) from exposure to MTBE. Based on these considerations, OEHHA adopts a PHG of 0.013 mg/L (13 µg/L or 13 ppb) for MTBE in drinking water using a CSF of  $1.8 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>. This value also incorporates a daily water consumption (DWC) rate of three liters equivalent per day (Leq/day). The range of possible values, based either on different individual tumor sites, or on different multi-route exposure estimates and the average cancer potency of the three sites (male rat kidney adenomas and carcinomas, male rat Leydig interstitial cell tumors, and leukemia and Lymphomas in female rats) was 2.7 to 16 ppb. The adopted PHG is considered to contain an adequate margin of safety for the potential noncarcinogenic effects including adverse effects on the renal and neurological systems.

In addition to the 13 ppb value based on carcinogenicity, a value of 0.047 mg/L (47 ppb) was calculated based on noncancer effects of increased relative kidney weights in the Robinson et al. (1990) 90-day gavage study in rats. The kidney effect is the most sensitive noncarcinogenic effect by the oral route observed in experimental animals with a no observable adverse effect level (NOAEL) of 100 mg/kg/day. This value of 47 ppb incorporates four 10-fold uncertainty factors (UFs) for a less than lifetime study, interspecies and interindividual variation and possible carcinogenicity. This value also incorporates a DWC rate of three Leq/day and a relative source contribution (RSC) default value of 20 percent. The default value for water ingestion is the same as used by U.S. EPA, Office of Water and is also documented in OEHHA's draft technical support document "Exposure Assessment and Stochastic Analysis" (OEHHA 1996). The three Leq/day DWC value represents approximately the 90 percent upper confidence level on tap water consumption and the average total water consumption. The three Leq/day incorporates two liters of direct consumption and one liter for inhalation of MTBE volatilized from drinking water. The use of 20 percent RSC indicates that most of the exposure occurs from ambient air levels. It is used in the noncancer risk assessment, but, consistent with standard practice, is not incorporated into the cancer risk assessment. While the lower value of 13 ppb is adopted as the PHG the difference in the two approaches is less than four-fold.

#### INTRODUCTION

The purpose of this document is to establish a PHG for the gasoline additive MTBE in drinking water. MTBE is a synthetic solvent used primarily as an oxygenate in unleaded gasoline to boost octane and improve combustion efficacy by oxygenation. Reformulated fuel with MTBE has been used in 32 regions in 19 states in the United States (U.S.) to meet the 1990 Federal Clean Air Act Amendments (CAAA) requirements for reducing carbon monoxide (CO) and ozone (O<sub>3</sub>) levels (CAAA of 1990, Title II, Part A, Section 211) because the added oxygenate promotes more complete burning of gasoline. California's cleaner-burning reformulated gasoline (California RFG) has been implemented to meet statewide clean air goals [California Code of Regulations (CCR), Title 13, Sections 2250 to 2297]. While neither

Federal nor State regulations require the use of a specific oxygenate, MTBE is most commonly utilized. MTBE is currently used (11 percent by volume) in California RFG to improve air quality (Demon and Masur 1996). California is the third largest consumer of gasoline in the world. Only the rest of the U.S. and the former Soviet Union surpasses it. Californians use more than 13.7 billion gallons of gasoline a year and another one billion gallons of diesel fuel.

MTBE and other oxygenates such as ethyl tertiary butyl ether (ETBE), tertiary butyl alcohol (TBA) and ethanol are currently being studied to determine the extent of their presence in drinking water and what, if any, potential health implications could result from exposure to them (Freed 1997, Scheible 1997, U.S. EPA 1998a, 1998b). California Senate Office of Research last February released a position paper on MTBE (Wiley 1998). California Energy Commission last October released a mandated report entitled "Supply and Cost of Alternatives to MTBE in Gasoline" (Schremp et al. 1998) evaluating alternative oxygenates and a possible MTBE phaseout. California Bureau of State Audits last December released a report entitled "California's Drinking Water: State and Local Agencies Need to Provide Leadership to Address Contamination of Groundwater by Gasoline Components and Additives" emphasizing the needs for improvements to better protect groundwater from contamination by MTBE (Sjoberg 1998). Maine, New Jersey and Texas are considering alternatives to MTBE in reducing air pollution in their State (Renner 1999).

MTBE was the second most-produced chemical in the U.S. in 1997, whereas previously it was ranked the twelfth in 1995 and eighteenth in 1994 (Cal/EPA 1998, Kirschner 1996, Reisch 1994). In 1994 and 1995, it was estimated that about 70 million Americans were exposed to oxygenated gasoline (oxyfuel) and approximately 57 million were exposed to reformulated gasoline (RFG) (ATSDR 1996, HEI 1996, NRC 1996, NSTC 1996, 1997). About 40 percent of the U.S. population live in areas where MTBE is used in oxyfuel or RFG (USGS 1996) and most people find its distinctive terpene-like odor disagreeable (CDC 1993a, 1993b, 1993c, Kneiss 1995, Medlin 1995, U.S. EPA 1997a). MTBE is now being found in the environment in many areas of the U.S. because of its increased use over the last several years.

Recently MTBE has become a drinking water contaminant due to its high water solubility and persistence. When gasoline with 10 percent MTBE by weight comes in contact with water, about five grams per liter (g/L) can dissolve (Squillace et al. 1996, 1997a). MTBE has been detected in groundwater as a result of leaking underground storage tanks (USTs) or pipelines and in surface water reservoirs via recreational boating activities. MTBE does not appear to adsorb to soil particles or readily degrade in the subsurface environment. It is more expensive to remove MTBE-added gasoline than gasoline without MTBE from contaminated water (Cal/EPA 1998, U.S. EPA 1987a, 1992c, 1996a, 1997a). The discussion of improvements in air quality versus the vulnerability of drinking water surrounding MTBE has raised concerns from the public as well as legislators (Hoffert 1998, McClurg 1998). The controversy and new mandated requirements have made MTBE an important chemical being evaluated by OEHHA.

#### *Background—Prior and Current Evaluations*

MTBE is not regulated currently under the Federal drinking water regulations. The California Department of Health Services (DHS) recently established a secondary maximum contaminant level (MCL) for MTBE as 0.05 mg/L (5 µg/L or 5 ppb) based on taste and odor effective January 7, 1999 (22 CCR Section 64449). An interim non-enforceable Action Level (AL) of 0.035 mg/L (35 µg/L or 35 ppb) in drinking water was established by DHS in 1991 to protect against adverse health effects. OEHHA (1991) at that time recommended this level based on noncarcinogenic effects of MTBE in laboratory animals (Greenough et al. 1980). OEHHA applied large uncertainty factors to provide a substantial margin of safety for drinking water. Since February 13, 1997, DHS (1997) regulations (22 CCR Section 64450) have included MTBE as an unregulated chemical for which monitoring is required. Pursuant to this requirement, data on the occurrence of MTBE in groundwater and surface water sources are being collected from drinking water systems in order to document the extent of MTBE contamination in drinking water supplies.

In California, the Local Drinking Water Protection Act of 1997 [Senate Bill (SB) 1189, Hayden, and Assembly Bill (AB) 592, Kuehl] requires DHS to develop a two-part drinking water standard for MTBE. The first part is a secondary MCL that addresses aesthetic qualities including taste and odor. The second part is a primary MCL that addresses health concerns, to be established by July 1, 1999. DHS is proceeding to establish drinking water standards for MTBE and requested OEHHA to conduct a risk assessment in order to meet the mandated schedule to set this regulation by July 1999. As mentioned above, DHS (1998) also adopts a secondary MCL of five ppb for MTBE to protect the public from exposure to MTBE in drinking

water at levels that can be smelled or tasted, as an amendment to Table 64449-A, Section 64449, Article 16, Chapter 15, Division 4, Title 22 of the CCR.

The 1997 act (SB 1189) also requires the evaluation of MTBE for possible listing under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) as a chemical known to the State to cause cancer or reproductive and developmental toxicity on or before January 1, 1999. This involves consideration of the evidence that MTBE causes these effects by the State's qualified experts for Proposition 65—the Carcinogen Identification Committee (CIC) and the Developmental and Reproductive Toxicant (DART) Identification Committee of OEHHA's Science Advisory Board (OEHHA 1998a, 1998b). These Committees evaluated MTBE in December 1998; MTBE was not recommended for listing under the Proposition 65 by either CIC or DART Committee.

The MTBE Public Health and Environmental Protection Act of 1997 (SB 521, Mountjoy) appropriates funds to the UC for specified studies of the human health and environmental risks and benefits of MTBE. The UC Toxic Substances Research and Teaching Program is managing the following six funded projects: (1) an evaluation of the peer-reviewed research literature on the effects of MTBE on human health, including asthma, and on the environment by UC Los Angeles (UCLA), (2) an integrated assessment of sources, fate and transport, ecological risk and control options for MTBE in surface and ground waters, with particular emphasis on drinking water supplies by UC Davis, (3) evaluation of costs and effectiveness of treatment technologies applicable to remove MTBE and other gasoline oxygenates from contaminated water by UC Santa Barbara (UCSB), (4) drinking water treatment for the removal of MTBE from groundwater and surface water reservoirs by UCLA, (5) evaluation of automotive MTBE combustion byproducts in California RFG by UC Berkeley, and (6) risk-based decisionmaking analysis of the cost and benefits of MTBE and other gasoline oxygenates by UCSB.

Among the SB 521 mandated projects, only the first project regarding human health effects (Froines 1998, Froines et al. 1998) and a part of the second project regarding human exposure to MTBE from drinking water (Johnson 1998) mentioned above are pertinent to the scope of this report. Their report has been submitted to the Governor and posted on their web site ([www.tsrtp.ucdavis.edu/mtbept/](http://www.tsrtp.ucdavis.edu/mtbept/)) on November 12, 1998. In this report, Froines et al. (1998) concluded that MTBE is an animal carcinogen with the potential to cause cancers in humans. Also in this report, Johnson (1998) performed a risk analysis of MTBE in drinking water based on animal carcinogenicity data. The act requires the report be reviewed and two hearings be held (February 19 and 23, 1999) for the purpose of accepting public testimony on the assessment and report. The act also requires the Governor to issue a written certification as to the human health and environmental risks of using MTBE in gasoline in California.

The American Conference of Governmental Industrial Hygienists (ACGIH) lists MTBE as an A3 Animal Carcinogen (ACGIH 1996). That is, MTBE is carcinogenic in experimental animals at relatively high dose(s), by route(s) of administration, at site(s), of histologic type(s), or by mechanism(s) that are not considered relevant to workplace exposure. ACGIH considers that available epidemiological studies do not confirm an increased risk of cancer in exposed humans. Available evidence suggests that the agent is not likely to cause cancer in humans except under uncommon or unlikely routes of exposure or levels of exposure.

In August 1996 the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) released the final report "Toxicological Profile for MTBE" which evaluated the toxic effects of MTBE including carcinogenicity in detail. The cancer effect levels of MTBE through both inhalation and oral exposure routes have been developed based on data of carcinogenicity in animals (ATSDR 1996).

The U.S. National Toxicology Program (NTP) did not find MTBE to be "reasonably anticipated to be a human carcinogen" in December 1998 (NTP 1998a). The National Institute of Environmental Health Sciences (NIEHS) Review Committee for the Report on Carcinogens first recommended (four yes votes to three no votes) that the NTP list MTBE as "reasonably anticipated to be a human carcinogen" in the Ninth Report on Carcinogens in January 1998 (NTP 1998b). The NTP Executive Committee Interagency Working Group for the Report on Carcinogens then voted against a motion to list MTBE (three yes votes to four no votes). Later in December 1998, the NTP Board of Scientific Counselors Report on Carcinogens Subcommittee voted against a motion to list MTBE as "reasonably anticipated to be a human carcinogen. . ." (five yes votes to six no votes with one abstention). The conclusions of these meetings are summarized on the NTP website, however, the supporting documentation on how these conclusions were reached is still under preparation and not available to us for evaluation (NTP 1998a). NTP solicited for final public comments through February 15, 1999 on these actions.

MTBE has been reviewed by the Environmental Epidemiology Section of the North Carolina Department of Environment, Health, and Natural Resources (NCDEHNR) and it was determined that there was limited evidence for carcinogenicity in experimental animals and that the compound should be classified as a Group B2 probable human carcinogen (Rudo 1995). The North Carolina Scientific Advisory Board on Toxic Air Contaminants (TAC) considered MTBE to be eligible as a Group C possible human carcinogen (Lucier et al. 1995). New Jersey (NJDWQI 1994, Post 1994) also classified MTBE as a possible human carcinogen. The State of New York Department of Health is drafting a fact sheet to propose an ambient water quality value for MTBE based on animal carcinogenicity data.

The International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) found "limited", but not "sufficient" evidence of MTBE carcinogenicity in animals. IARC has recently classified MTBE as a Group 3 carcinogen (i.e., not classifiable as to carcinogenicity in humans), based on inadequate evidence in humans and limited evidence in experimental animals. The conclusions of this October 1998 IARC Monographs Working Group Meeting are summarized on the IARC website, however, the supporting documentation on how these conclusions were reached is still under preparation to be published as the IARC Monographs Volume 73 (IARC 1998a).

The International Programme on Chemical Safety (IPCS) of WHO has issued the second draft *Environmental Health Criteria* on MTBE (IPCS 1997) which was scheduled to be finalized in December 1998. IPCS stated that carcinogenic findings in animal bioassays seem to warrant some concern of potential carcinogenic risk to humans, but the document does not contain a risk characterization. However, the final document is not available as of February 1999.

European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC) prepared a technical report (ECETOC 1997) on MTBE health risk characterization mainly on occupational inhalation exposure. ECETOC concluded that MTBE has some potential to increase the occurrence of certain tumors in female mice or male rats after chronic high-dose inhalation exposure.

In February 1996 the Office of Science and Technology Policy (OSTP) through the Committee on Environment and Natural Resources (CENR) of the White House National Science and Technology Council (NSTC) released a draft report titled "Interagency Assessment of Potential Health Risks Associated with Oxygenated Gasoline" (NSTC 1996). This report focused primarily on inhalation exposure to MTBE and its principal metabolite, TBA. In March 1996 NSTC released the draft document "Interagency Oxygenated Fuels Assessment" which addressed issues related to public health, air and water quality, fuel economy, and engine performance associated with MTBE in gasoline relative to conventional gasoline. This document was peer reviewed by the National Academy of Sciences (NAS) under guidance from the National Research Council (NRC) which then published its findings and recommendations in the document "Toxicological and Performance Aspects of Oxygenated Motor Vehicle Fuels" (NRC 1996). The limited review on the potential health effects of MTBE in the NRC report (1996) considered the animal carcinogenicity evidence to be positive. The NRC findings were used to revise the NSTC document and the final report was released in June 1997. The NSTC (1997) concluded: "there is sufficient evidence that MTBE is an animal carcinogen". NSTC (1997) also concluded: ". . . the weight of evidence supports regarding MTBE as having a carcinogenic hazard potential for humans."

In April 1996 the Health Effects Institute (HEI) released "The Potential Health Effects of Oxygenates Added to Gasoline, A Special Report of the Institute's Oxygenates Evaluation Committee" (HEI 1996). HEI (1996) concluded: "the possibility that ambient levels may pose some risk of carcinogenic effects in human populations cannot be excluded". HEI in summary of studies of long-term health effects of MTBE concluded: "Evidence from animal bioassays demonstrates that long-term, high-level exposures to MTBE by either the oral or inhalation routes of exposure cause cancer in rodents."

The U.S. EPA has not established primary or secondary MCLs or a Maximum Contaminant Level Goal (MCLG) for MTBE but included MTBE on the Drinking Water Contaminant Candidate List (CCL) published in the Federal Register on March 2, 1998 (U.S. EPA 1998c, 1997b, 1997d). An advisory released in December 1997 recommended that MTBE concentration in the range of 20 to 40 ppb or below would assure both consumer acceptance of the water and a large margin of safety from any toxic effects (U.S. EPA 1997a, Du et al. 1998).

On November 30, 1998, the U.S. EPA (1998a) announced the creation of a blue-ribbon panel to review the important issues posed by the use of MTBE and other oxygenates in gasoline so that public health concerns could be better understood. The Panel on Oxygenate Use in Gasoline under the Clean Air Act Advisory Commit-



tee (CAAC), including 12 members and eight Federal representatives serving as consultants to the Panel, is to make recommendations to the U.S. EPA on how to ensure public health protection and continued improvement in both air and water quality after a 6-month study.

In its 1997 advisory, U.S. EPA agreed with the 1997 NSTC conclusions and concluded: "Although MTBE is not mutagenic, a nonlinear mode of action has not been established for MTBE. In the absence of sufficient mode of action information at the present time, it is prudent for EPA to assume a linear dose-response for MtBE. Although there are no studies on the carcinogenicity of MtBE in humans, there are multiple animal studies (by inhalation and gavage routes in two rodent species) showing carcinogenic activity and there is supporting animal carcinogenicity data for the metabolites. The weight of evidence indicates that MtBE is an animal carcinogen, and the chemical poses a carcinogenic potential to humans (NSTC, 1997, page 4-26)." The U.S. EPA (1994a, 1994c) proposed in 1994 to classify MTBE as a Group C possible human carcinogen based upon animal inhalation studies (published in 1992). At that time, U.S. EPA noted that a Group B2 probable human carcinogen designation may be appropriate if oral MTBE exposure studies in animals (published later in 1995) result in treatment-related tumors.

In 1987, MTBE was identified by the U.S. EPA (1987a) under Section Four of the Toxic Substances Control Act (TSCA) for priority testing because of its large production volume, potential widespread exposure, and limited data on long-term health effects (Duffy et al. 1992). The results of the testing have been published in a peer-reviewed journal (Bevan et al. 1997a, 1997b, Bird et al. 1997, Daughtrey et al. 1997, Lington et al. 1997, McKee et al. 1997, Miller et al. 1997, Stern and Kneiss 1997).

California Environmental Protection Agency (Cal/EPA) has reported some background information and ongoing activities on MTBE in California's "cleaner-burning fuel program" in a briefing paper (Cal/EPA 1998). U.S. EPA (1996d, 1996e) published fact sheets on MTBE in water in addition to several advisory documents. While concerns have been raised about its potential health impacts, based on hazard evaluation of the available data, MTBE is substantially less hazardous than benzene (a Group A human carcinogen) and 1,3-butadiene (a Group B2 probable human carcinogen), two carcinogenic chemicals it displaces in California's new gasoline formulations (Spitzer 1997). Potential health benefits from ambient O<sub>3</sub> reduction related to the use of MTBE in RFG were evaluated (Erdal et al. 1997). Whether the addition of MTBE in gasoline represents a net increase in cancer hazard is beyond the scope of this document.

In this document, the available data on the toxicity of MTBE primarily by the oral route based on the reports mentioned above are evaluated, and information available since the previous assessment by NSTC (1997) and U.S. EPA (1997a) is included. As indicated by the summaries provided above, there has been considerable scientific discussion regarding the carcinogenicity of MTBE and the relevance of the animal cancer study results to humans. Also indicated above, especially by some of the reported votes of convened committees, there is a considerable disagreement regarding the quality and relevance of the animal data among scientists. However, some of the disagreement stems from the differences in the level of evidence considered adequate for different degrees of confidence by the scientists considering the evidence. There is a greater level of evidence required to conclude that the data clearly show that humans are at cancer risk from exposure than to conclude that there may be some cancer risk or that it is prudent to assume there is a cancer risk to humans. In order to establish a PHG in drinking water, a nonregulatory guideline based solely on public health considerations, the prudent assumption of potential cancer risk was made. To determine a public health-protective level of MTBE in drinking water, relevant studies were identified, reviewed and evaluated, and sensitive groups and exposure scenarios are considered.

#### CHEMICAL PROFILE

##### *Chemical Identity*

MTBE [(CH<sub>3</sub>)<sub>3</sub>C(OCH<sub>3</sub>), CAS Registry Number 1634-04-4] is a synthetic chemical without known natural sources. The chemical structure, synonyms, and identification numbers are listed in Table I and are adapted from the Merck Index (1989), Hazardous Substances Data Bank (HSDB) of the National Library of Medicine (1997), Integrated Risk Information Systems (IRIS) of U.S. EPA (1997c), TOMES PLUS® (Hall and Rumack 1998) computerized data base, and the ATSDR (1996), Cal/EPA (1998), ECETOC (1997), HEI (1996), NRC (1996), NSTC (1996, 1997), and U.S. EPA (1997a) documents.

TOMES (Toxicology and Occupational Medicine System) PLUS® is a computerized data base which includes the data systems of Hazard Management®,

INFOTEXT®, HAZARDTEXT®, MEDITEXT®, REPROTEXT®, SERATEXT®, HSDB, IRIS, Registry of Toxic Effects of Chemical Substances (RTECS®) of National Institute for Occupational Safety and Health (NIOSH), Chemical Hazard Response Information System (CHRIS) of U.S. Coast Guard, Oil and Hazardous Materials/Technical Assistance Data System (OHM/TADS) of U.S. EPA, Department of Transportation (DOT) Emergency Response Guide, New Jersey Hazardous Substance Fact Sheets (NJHSFS), North American Emergency Response Guidebook Documents (NAERG) of U.S. DOT, Transport Canada and the Secretariat of Communications and Transportation of Mexico, REPROTOX® System of the Georgetown University, Shepard's Catalog of Teratogenic Agents of the Johns Hopkins University, Teratogen Information System (TERIS) of the University of Washington, and NIOSH Pocket Guide<sup>(TM)</sup>. For MTBE, TOMES PLUS® (Hall and Rumack 1998) contains entries in HAZARDTEXT®, MEDITEXT®, REPROTEXT®, REPROTOX®, HSDB, IRIS, RTECS®, NAERG and NJHSFS.

#### *Physical and Chemical Properties*

Important physical and chemical properties of MTBE are given in Table 2 and are adapted from Merck Index (1989), HSDB (1997), TOMES PLUS® (Hall and Rumack 1998), and the ATSDR (1996), Cal/EPA (1998), HEI (1996), NRC (1996), NSTC (1996, 1997), and U.S. EPA (1997a) documents.

MTBE, an aliphatic ether, is a volatile organic compound (VOC) with a characteristic odor. It is a colorless liquid at room temperature. It is highly flammable and combustible when exposed to heat or flame or spark, and is a moderate fire risk. Vapors may form explosive mixtures with air. It is unstable in acid solutions. Fire may produce irritating, corrosive or toxic gases. Runoff from fire control may contain MTBE and its combustion products (HSDB 1997).

MTBE is miscible in gasoline and soluble in water, alcohol, and other ethers. It has a molecular weight of 88.15 daltons, a vapor pressure of about 245 mmHg at 25 °C, an octane number of 110, and solubility in water of about 50 g/L at 25 °C. It disperses evenly in gasoline and water and stays suspended without requiring physical mixing. It does not increase volatility of other gasoline components when it is mixed in the gasoline. MTBE released to the environment via surface spills or subsurface leaks was found to initially partition between water and air (Jeffrey 1997). The log of the octanol-water partition coefficient ( $\log K_{ow}$ ) is reported to range from 0.94 to 1.24 which indicates that there is 10 times more partitioning of MTBE in the lipophilic phase than in the aqueous phase of solvents. The molecular size and  $\log K_{ow}$  of MTBE are characteristic of molecules which are able to penetrate across biological membranes of the skin, lungs and gastrointestinal tracts (Mackay et al. 1993, Nihlen et al. 1995). The octanol-water partition coefficient is reported to be 16 by Nihlen et al. (1997). Fujiwara et al. (1984) reported laboratory-derived octanol-water partition coefficients ranging from 17.2 to 17.5 with a  $\log K_{ow}$  of 1.2. The blood-air, urine-air, saline-air, fat-air and oil-air partition coefficients ( $\lambda$ ) are reported to be 20, 15.6, 15.3, 142 and 138, respectively (Imbriani et al. 1997). One part per million (ppm) of MTBE, volume to volume in air, is approximately 3.6 mg/m<sup>3</sup> of air at 20 °C (ATSDR 1996).

#### ORGANOLEPTIC PROPERTIES

Taste or odor characteristics, often referred to as organoleptic properties, are not used by U.S. EPA or DHS for developing primary drinking water standards, but are used for developing secondary standards. The estimated thresholds for these properties of MTBE reported in the literature are given in Table 3 and are adapted from the ATSDR (1996), Cal/EPA (1998), HEI (1996), HSDB (1997), NSTC (1996, 1997), and U.S. EPA (1997a) documents. Taste and odor may alert consumers to the fact that the water is contaminated with MTBE (Angle 1991) and many people object to the taste and odor of MTBE in drinking water (Killian 1998, Reynolds 1998). However, not all individuals respond equally to taste and odor because of differences in individual sensitivity. It is not possible to identify point threshold values for the taste and odor of MTBE in drinking water, as the concentration will vary for different individuals, for the same individuals at different times, for different populations, and for different water matrices, temperatures, and many other variables.

The odor threshold ranges from about 0.32 to 0.47 mg/m<sup>3</sup> (about 90 to 130 ppb) in air and can be as low as five ppb (about 0.02 mg/m<sup>3</sup>) for some sensitive people. In gasoline containing 97 percent pure MTBE at mixture concentrations of 3 percent, 11 percent and 15 percent MTBE, the threshold for detecting MTBE odor in air was estimated to be 50 ppb (about 0.18 mg/m<sup>3</sup>), 280 ppb (about one mg/m<sup>3</sup>), and 260 ppb (about 0.9 mg/m<sup>3</sup>), respectively (ACGIH 1996). A range of 5 ppb to 53 ppb (about 0.19 mg/m<sup>3</sup>) odor threshold in the air was reported in an American Petroleum Institute (API) document (API 1994).

The individual taste and odor responses reported for MTBE in water are on average in the 15 to 180 ppb ( $\mu\text{g/L}$ ) range for odor and the 24 to 135 ppb range for taste (API 1994, Prah et al. 1994, Young et al. 1996, Dale et al. 1997b, Shen et al. 1997, NSTC 1997). The ranges are indicative of the average variability in individual response. U.S. EPA (1997a) has analyzed these studies in detail and recommended a range of 20 to 40 ppb as an approximate threshold for organoleptic responses. The study (Dale et al. 1997b) by the Metropolitan Water District of Southern California (MWDSC) found people more sensitive to the taste than odor. This result is consistent with API's (1994) findings for MTBE taste and odor thresholds. But in the study by Young et al. (1996), test subjects were more sensitive to odor than taste. The subjects described the taste of MTBE in water as "nasty", "bitter", "nauseating", and "similar to rubbing alcohol" (API 1994).

It is noted that chlorination and temperature of the water would likely affect the taste and odor of MTBE in water. Thresholds for the taste and odor of MTBE in chlorinated water would be higher than thresholds of MTBE in nonchlorinated water. Thresholds for the taste and odor of MTBE in water at higher temperatures (e.g., for showering) would likely be lower than those of MTBE in water at lower temperatures.

There were undoubtedly individuals who could only detect the odor of MTBE at even higher concentrations than 180 ppb (Prah et al. 1994). Odor thresholds as high as 680 ppb have been reported (Gilbert and Calabrese 1992). On the other hand, some subjects in these studies were able to detect the odor of MTBE in water at much lower concentrations, i.e. 2.5 ppb (Shen et al. 1997), five ppb (McKinnon and Dyksen 1984), or 15 ppb (Young et al. 1996). Some sensitive subjects in the taste studies were able to detect MTBE in water at concentrations as low as two ppb (Dale et al. 1997b), 10 ppb (Barker et al. 1990), 21 ppb (Dale et al. 1997b), or 39 ppb (Young et al. 1996). Thus, in a general population, some unknown percentage of people will be likely to detect the taste and odor of MTBE in drinking water at concentrations below the U.S. EPA (1997a) 20 to 40 ppb advisory level. DHS (1997) has recently proposed five ppb as the secondary MCL for MTBE. The lowest olfaction threshold in water is likely to be at or about 2.5 ppb (Shen et al. 1997). The lowest taste threshold in water is likely to be at or about two ppb (Dale et al. 1997b).

Table 1.—Chemical Identity of Methyl Tertiary Butyl Ether (MTBE)

Characteristic	Information	Reference
Chemical Name .....	Methyl tertiary butyl ether .....	Merck 1989
Synonyms .....	Methyl tertiary-butyl ether; .....	Merck 1989
	Methyl tert-butyl ether; tert-butyl methyl ether; tertiary-butyl methyl ether; methyl-1, 1-dimethylethyl ether; 2-methoxy-2-methylpropane; 2-methyl-2-methoxypropane; methyl t-butyl ether; MTBE; MTBE.	
Registered trade names .....	No data .....	
Chemical formula .....	$\text{C}_5\text{H}_{12}\text{O}$ or $(\text{CH}_3)_3\text{C}(\text{OCH}_3)$ .....	Merck 1989
Chemical structure		
Identification numbers:		
Chemical Abstracts Service (CAS) Registry number.	1634-04-4 .....	Merck 1989
National Institute for Occupational Safety and Health (NIOSH) Registry of Toxic Effects of Chemical Substances (RTECS) number.	KN5250000 .....	HSDB 1997
Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code (DOT/UN/NA/IMCO) Shipping number.	UN 2398, IMO 3.2 .....	HSDB 1997
Hazardous Substances Data Bank (HSDB) number.	5847 .....	HSDB 1997
North American Emergency Response Guidebook Documents (NAERG) number.	127 .....	HSDB 1997

Table 1.—Chemical Identity of Methyl Tertiary Butyl Ether (MTBE)—Continued

Characteristic	Information	Reference
National Cancer Institute (NCI) number.	No data .....	
U.S. Environmental Protection Agency (U.S. EPA) Hazardous Waste number.	No data .....	
U.S. EPA Oil and Hazardous Materials/Technical Assistance Data System (OHM/TADS) number.	No data .....	
European EINECS number .....	216.653.1 .....	ECETOC 1997

Table 2.—Chemical Physical Properties of MTBE

Property	Value or information	Reference
Molecular weight .....	88.15 g/mole .....	Merck 1989
Color .....	colorless .....	Merck 1989
Physical state .....	liquid .....	Merck 1989
Melting point .....	-109°C .....	HSDB 1997
Boiling point .....	53.6–55.2°C .....	Mackay et al. 1993
Density at 20°C .....	0.7404–0.7578 g/mL .....	Squillace et al. 1997a
Solubility:		
in water .....	4.8 g/100 g water .....	Merck 1989
in water .....	23.2–54.4 g/L water .....	Garrett et al. 1986, Mackay et al. 1993
in water .....	43–54.3 g/L water .....	Squillace et al. 1997a
in water, 20°C .....	4–5 percent .....	Gilbert and Calabrese 1992
in water, 25°C .....	51 g/L water .....	HSDB 1997
Partition coefficients		
octanol-water .....	16 .....	Nihlen et al. 1997
Log $K_{ow}$ .....	17.2–17.5 .....	Fujiwara et al. 1984
Log $K_{oc}$ .....	0.94–1.16 .....	Mackay et al. 1993
Log $K_{oc}$ .....	1.2 .....	Fujiwara et al. 1984
Log $K_{oc}$ .....	1.24 .....	U.S. EPA 1997a
Log $K_{oc}$ .....	1.05 (estimated) .....	Squillace et al. 1997a
Log $K_{oc}$ .....	2.89 (calculated) .....	U.S. EPA 1995b
Vapor pressure:		
at 25°C .....	245–251 mm Hg .....	Mackay et al. 1993
at 100 °F .....	7.8 psi (Reid Vapor Pressure) .....	ARCO 1995a
Henry's law constant .....	0.00058–0.003 atm-m <sup>3</sup> /mole .....	Mackay et al. 1993
at 25°C .....	$5.87 \times 10^{-4}$ atm-m <sup>3</sup> /mole .....	ATSDR 1996
at 15°C .....	0.011 (dimensionless) .....	Robbins et a. 1993
Ignition temperature .....	224°C .....	Merck 1989
Flash point .....	-28°C .....	Merck 1989
28°C (closed cup) .....		Gilbert and Calabrese 1992
Explosion limits .....	1.65 to 8.4 percent in air .....	Gilbert and Calabrese 1992
Heat of combustion .....	101,000 Btu/gal at 25°C .....	HSDB 1997
Heat of vaporization .....	145 Btu/lb at 55°C .....	HSDB 1997
Stability .....	MTBE is unstable in acidic solution .....	Merck 1989
Conversion factors:		
ppm (v/v) to mg/m <sup>3</sup> in air at 25°C .....	1 ppm = 3.61 mg/m <sup>3</sup> .....	ACGIH 1996
mg/m <sup>3</sup> to ppm (v/v) in air at 25°C .....	1 mg/m <sup>3</sup> = 0.28 ppm .....	ACGIH 1996

Table 3.—Organoleptic Properties of MTBE

Property	Value or information	Reference
Odor Taste .....	terpene-like at 25°C .....	Gilbert and Calabrese 1992
Threshold in air .....	300 ppb .....	Smith and Duffy 1995
99 percent pure MTBE .....	0.32–0.47 mg/m <sup>3</sup> .....	ACGIH 1996
97 percent pure MTBE .....	(-90–130 ppb) .....	
97 percent pure MTBE in gasoline .....	5–53 ppb (detection) .....	API 1994
	8 ppb (recognition) .....	API 1994
	125 ppb (recognition) .....	API 1994

Table 3.—Organoleptic Properties of MTBE—Continued

Property	Value or information	Reference
15 percent MTBE .....	260 ppb .....	ACGIH 1996
11 percent MTBE .....	280 ppb .....	ACGIH 1996
3 percent MTBE .....	50 ppb .....	ACGIH 1996
Threshold in water .....	680 ppb .....	Gilbert and Calabrese 1992
	180 ppb .....	Prah et al. 1994
	95 ppb .....	ARCO 1995a
	55 ppb (recognition) .....	API 1994
	45 ppb (detection) .....	API 1994
	15–95 ppb (mean 34 ppb) .....	Young et al. 1996
	15–180 ppb .....	U.S. EPA 1997a
	13.5–45.4 ppb .....	Shen et al. 1997
	5–15 ppb .....	McKinnon and Dyksen 1984
	2.5 ppb .....	Shen et al. 1997
Taste .....	solvent-like at 25°C .....	U.S. EPA 1997a
	Threshold in water .....	Dale et al. 1997b
Threshold in water .....	21–190 ppb .....	U.S. EPA 1997a
	24–135 ppb .....	Young et al. 1996
	39–134 ppb (mean 48 ppb) .....	API 1994
	39–134 ppb .....	Barker et al. 1990
	10–100 ppb .....	Dale et al. 1997b
	2 ppb (one subject) .....	

#### Production and Uses

MTBE is manufactured from isobutene; also known as isobutylene or 2-methylpropene (Merck 1989), which is a product of petroleum refining. It is made mainly by combining methanol with isobutene, or derived from combining methanol and TBA. It is used primarily as an oxygenate in unleaded gasoline, in the manufacture of isobutene, and as a chromatographic effluent especially in high pressure liquid chromatography (ATSDR 1996, HSDB 1997). MTBE also has had a limited use as a therapeutic drug for dissolving cholesterol gallbladder stones (Leuschner et al. 1994).

MTBE is the primary oxygenate used in gasoline because it is the least expensive and in greatest supply. It is promoted as a gasoline blending component due to its high octane rating, low cost of production, ability to readily mix with other gasoline components, ease in distribution through existing pipelines, distillation temperature depression, and beneficial dilution effect on undesirable components of aromatics, sulfur, olefin and benzene. In addition, the relatively low co-solvent volatility of MTBE does not result in a more volatile gasoline that could be hazardous in terms of flammability and explosivity. The use of MTBE has helped offset the octane specification loss due to the discontinued use of higher toxicity high octane aromatics and has reduced emissions of benzene, a known human carcinogen, and 1,3-butadiene, an animal carcinogen (Cal/EPA 1998, Spitzer 1997).

MTBE has been commercially used in Europe since 1973 as an octane enhancer to replace lead in gasoline and was approved as a blending component in 1979 by U.S. EPA. Since the early 1990's, it has been used in reformulated fuel in 18 states in the U.S. Under Section 211 of the 1990 CAAA, the Federal oxyfuel program began requiring gasoline to contain 2.7 percent oxygen by weight which is equivalent to roughly 15 percent by volume of MTBE be used during the four winter months in regions not meeting CO reduction standards in November 1992. In January 1995, the Federal RFG containing 2 percent oxygen by weight or roughly 11 percent of MTBE by volume was required year-round to reduce O<sub>3</sub> levels. Oxygenates are added to more than 30 percent of the gasoline used in the U.S. and this proportion is expected to rise (Squillace et al. 1997a).

In California, Federal law required the use of Phase I RFG in the worst polluted areas including Los Angeles and San Diego as of January 1, 1995, and in the entire State as of January 1, 1996. By June 1, 1996, State law required that all gasoline sold be California Phase 2 RFG and Federal Phase II RFG will be required by the year 2000 (Cornitius 1996). MTBE promotes more complete burning of gasoline, thereby reducing CO and O<sub>3</sub> levels in localities which do not meet the National Ambient Air Quality Standards (ATSDR 1996, USGS 1996). Almost all of the MTBE produced is used as a gasoline additive; small amounts are used by laboratory scientists (ATSDR 1996). When used as a gasoline additive, MTBE may constitute up to 15 percent volume to volume of the gasoline mixture. Currently, MTBE is added to virtually all of the gasoline consumed in California (Cal/EPA 1998).

The amount of MTBE used in the U.S. has increased from about 0.5 million gallons per day in 1980 to over 10 million gallons per day in early 1997. Of the total amount of MTBE used in the U.S., approximately 70 percent are produced domestically, about 29 percent are imported from other countries, and about 1 percent is existing stocks. Over 4.1 billion gallons of MTBE are consumed in the U.S. annually, including 1.49 billion gallons—more than 36 percent of the national figure—in California (Wiley 1998). California uses about 4.2 million gallons per day of MTBE, about 85 percent of which is imported into the state, primarily by ocean tankers from the Middle East (Cal/EPA 1998). California also imports MTBE from Texas and other major MTBE-producing states in the U.S.

MTBE production in the U.S. began in 1979 and increased rapidly after 1983. It was the second most-produced chemical, in terms of amount, in the U.S. in 1997, whereas previously it was ranked the twelfth in 1995 and eighteenth in 1994 (Cal/EPA 1998, Kirschner 1996, Reisch 1994). The production was 13.61 million pounds in 1994 and 17.62 million pounds in 1995 (Kirschner 1996). MTBE production was estimated at about 2.9 billion gallons in the U.S. and about 181 million gallons in California in 1997 (Wiley 1998). MTBE is manufactured at more than 40 facilities by about 27 producers primarily concentrated along the Houston Ship Channel in Texas and the Louisiana Gulf Coast. Texas supplies about 80 percent of the MTBE produced in the U.S. with about 10 percent produced in Louisiana and about 5 percent in California (Cal/EPA 1998). The major portion of MTBE produced utilizes, as a co-reactant, isobutylene that is a waste product of the refining process (Wiley 1998).

#### ENVIRONMENTAL OCCURRENCE AND HUMAN EXPOSURE

The NSTC (1997) report provides extensive occurrence data for MTBE and other fuel oxygenates, as well as information on applicable treatment technologies. Similar information, specifically based on data in California, can be found in the recent UC (1998) report mandated under SB 521. For additional information concerning MTBE in the environment, the NSTC report can be accessed through the NSTC Home Page via a link from the OSTP. The U.S. Geological Survey (USGS) has been compiling data sets for national assessment of MTBE and other VOCs in ground and surface water as part of the National Water-Quality Assessment (NAWQA) Program (Buxton et al. 1997, Lapham et al. 1997, Squillace et al. 1997a, 1997b, Zogorski et al. 1996, 1997). Information on analytical methods for determining MTBE in environmental media is compiled in the ATSDR (1996) Toxicological Profile document.

The U.S. EPA (1993, 1995a) estimated that about 1.7 million kilograms (kgs) MTBE were released from 141 facilities reporting in the Toxics Release Inventory (TRI) per year, 97.3 percent to air, 2.44 percent to surface water, 0.25 percent to underground injection, and 0.01 percent to land. Cohen (1998) reported that an estimated 27,000 kgs or 30 tons per day were emitted from 9,000 tons of MTBE consumed in California per day. The California Air Resources Board (ARB) estimated that the exhaust and evaporative emission was about 39,000 kgs or 43 tons per day in California in 1996 (Cal/EPA 1998).

A multimedia assessment of refinery emissions in the Yorktown region (Cohen et al. 1991) indicated that the MTBE mass distribution was over 73 percent in water, about 25 percent in air, less than 2 percent in soil, about 0.02 percent in sediment, about  $10^{-6}$  percent in suspended solids, and  $10^{-7}$  percent in biota. A recent laboratory study on liquid-gas partitioning (Rousch and Sommerfeld 1998) suggests that dissolved MTBE concentrations can vary substantially from nominal. The main route of exposure for occupational and non-occupational groups is via inhalation, ingestion is considered as secondary, and dermal contact is also possible.

The persistence half-life of MTBE (Jeffrey 1997) is about 4 weeks to 6 months in soil, about 4 weeks to 6 months in surface water, and about 8 weeks to 12 months in groundwater based on estimated anaerobic biodegradation, and about 20.7 hours to 11 days in air based on measured photooxidation rate constants (Howard et al. 1991, Howard 1993). Church et al. (1997) described an analytical method for detecting MTBE and other major oxygenates and their degradation products in water at sub-ppb concentrations. MTBE appears to be biodegraded under anaerobic conditions (Borden et al. 1997, Daniel 1995, Jensen and Arvin 1990, Mormile et al. 1994, Steffan et al. 1997). Brown et al. (1997) and Davidson and Parsons (1996) reviewed state-of-the-art remediation technologies for treatment of MTBE in water. McKinnon and Dyksen (1984) described the removal of MTBE from groundwater through aeration plus granulated activated charcoal (GAC). Koenigsberg (1997) described a newly developed bioremediation technology for MTBE cleanup in ground-

water. Cullen (1998) reported a one-year field test of a polymer-enhanced carbon technology for MTBE removal at the drinking water supply source.

*Air, Soil, Food, and Other Sources*

The presence of MTBE in ambient air is documented and likely to be the principal source of human exposure. MTBE is released into the atmosphere during the manufacture and distribution of oxyfuel and RFG, in the vehicle refueling process, and from evaporative and tailpipe emissions from motor vehicles. The general public can be exposed to MTBE through inhalation while fueling motor vehicles or igniting fuel under cold startup conditions (Lindstrom and Pleil 1996). The level of inhaled MTBE at the range relevant to human exposures appears to be directly proportional to the MTBE concentrations in air (Big/dynamics, Inc. 1981, 1984c, Niblen et al. 1994). In air, MTBE may represent 5 to 10 percent of the VOCs that are emitted from gasoline-burning vehicles, particularly in areas where MTBE is added to fuels as part of an oxygenated fuel program (ARCO 1995a). MTBE has an atmospheric lifetime of approximately 4 days and its primary byproducts are tert-butyl formate (TBF), formaldehyde (HCHO), acetic acid, acetone, and TBA.

MTBE was found in urban air in the U.S. (Zogorski et al. 1996, 1997) and the median concentrations ranged from 0.13 to 4.6 parts per billion by volume (ppbv). Fairbanks, Alaska reported concentrations ranging from two to six ppbv when the gasoline contained 15 percent MTBE (CDC 1993a). Grosjean et al. (1998) reported ambient concentrations of ethanol and MTBE at a downtown location in Porto Alegre, Brazil where about 74 percent of about 600,000 vehicles use gasoline with 15 percent MTBE, from March 20, 1996 to April 16, 1997. Ambient concentrations of MTBE ranged from 0.2 to 17.1 ppbv with an average of  $6.6 \pm 4.3$  ppbv. This article also cited unpublished data including Cape Cod (four samples, July to August 1995): 39 to 201 parts per trillion by volume (pptv or 1/1,000 ppbv), Shenandoah National Park (14 samples, July to August 1995): less or equal to ( $\leq$ ) seven pptv, Brookhaven (16 samples, July to August 1995): 33 to 416 pptv, Wisconsin (62 samples, August 1994 to December 1996, with all but five samples yielding no detectable MTBE with a detection limit of 12 pptv):  $\leq 177$  pptv, and downtown Los Angeles, California (one sample, collected in 1993 prior to the introduction of California RFG with MTBE): 0.8 ppbv.

Ambient levels of MTBE in California are similar or slightly higher than the limited data suggest for other states. The results of two recent (from 1995 to 1996) monitoring surveys (Poore et al. 1997, Zielinska et al. 1997) indicate that ambient levels of MTBE averaged 0.6 to 7.2 ppbv with sampling for 3 hours at four southern California locations, and 1.3 to 4.8 ppbv with sampling for 24 hours at seven California locations. The Bay Area Air Quality Management District (BAAQMD) has an 18-station network and has been monitoring for MTBE since 1995. The average concentration of MTBE in the San Francisco Bay area is approximately one ppbv (Cal/EPA 1998).

The ARB established a 20-station TAC air-monitoring network in 1985, and began analyzing ambient air for MTBE in 1996 (ARB 1996). Preliminary data suggest a statewide average of approximately two ppbv with higher concentrations in the South Coast of about four ppbv. The limit of detection is 0.2 ppbv. The Desert Research Institute, under contract to ARB as a part of the 1997 Southern California Ozone Study (Fujita et al. 1997), monitored for MTBE in July through September 1995 and 1996 in Southern California, at the Asuza, Burbank, and North Main monitoring sites. The monitoring was designed to determine peak morning rush hour concentrations (6 to 9 a.m.) and was part of a comprehensive study to analyze reactive organics in the South Coast Air Basin. The results showed a mean of approximately four ppbv with a range of one to 11 ppbv. These concentrations are similar to the ARB findings. Although ARB sampled for 24 hours, the highest concentrations are seen in the morning rush hour traffic because MTBE is a tailpipe pollutant.

Industrial hygiene monitoring data for a MTBE operating unit shows an average 8-hour exposure of 1.42 ppm. Average exposure for dockworkers was determined to be 1.23 ppm. Occupational exposure to gasoline containing two to 8 percent MTBE is estimated at one to 1.4 ppm per day (ARCO 1995a, 1995b). In a New Jersey study, MTBE concentrations as high as 2.6 ppm were reported in the breathing zone of individuals using self-service gasoline stations without vapor recovery equipment, and the average MTBE exposure among service station attendants was estimated to be below one ppm when at least 12 percent MTBE was used in fuels (Hartle 1993). The highest Canadian predicted airborne concentration of  $75 \text{ ng/m}^3$  is  $3.9 \times 10^7$  times lower than the lowest reported effect level of  $2,915 \text{ mg/m}^3$  in a subchronic inhalation study in rats (Environmental Canada 1992, 1993, Long et al. 1994).

In a Finnish study based on inhalation exposure (Hakkola and Saarinen 1996), oil company road tanker drivers were exposed to MTBE during loading and delivery at concentrations between 13 and 91 mg/m<sup>3</sup> (about 3.6 to 25 ppm) and the authors suggested some improvement techniques to reduce the occupational exposure. A recent Finnish study, Saarinen et al. (1998) investigated the exposure and uptake of 11 drivers to gasoline vapors during road-tanker loading and unloading. On average the drivers were exposed to vapors for  $21 \pm 14$  minutes, three times during a work shift. The mean concentration of MTBE was  $8.1 \pm 8.4$  mg/m<sup>3</sup> (about 2.3 ppm). Vainiotalo et al. (1999) studied customer breathing zone exposure during refueling for 4 days in summer 1996 at two Finnish self-service gasoline station with "stage 1" vapor recovery systems. The MTBE concentration ranged from less than 0.02 to 51 mg/m<sup>3</sup>. The geometric mean concentration of MTBE in individual samples was 3.9 mg/m<sup>3</sup> at station A and 2.2 mg/m<sup>3</sup> at station B. The average refueling (sampling) time was 63 seconds at station A and 74 seconds at station B. Mean MTBE concentration in ambient air (a stationary point in the middle of the pump island) was 0.16 mg/m<sup>3</sup> for station A and 0.07 mg/m<sup>3</sup> for station B.

Exposure to CO, MTBE, and benzene levels inside vehicles traveling in an urban area in Korea was reported (Jo and Park 1998). The in-vehicle concentrations of MTBE were significantly higher ( $p < 0.0001$ ), on the average 3.5 times higher, in the car with a carbureted engine than in the other three electronic fuel-injected cars. The author considered the in-auto MTBE levels, 48.5 µg/m<sup>3</sup> (about 13 ppb) as a median, as two to three times higher than the measurements in New Jersey and Connecticut. Goldsmith (1998) reported that vapor recovery systems could reduce risks from MTBE.

Unlike most gasoline components that are lipophilic, the small, water-soluble MTBE molecule has low affinity for soil particles and moves quickly to reach groundwater. In estuaries, MTBE is not expected to stay in sediment soil but can accumulate at least on a seasonal basis in sediment interstitial water (ATSDR 1996). There are no reliable data on MTBE levels in food, but food is not suspected as a significant source of exposure to MTBE. There is little information on the presence of MTBE in plants or food chains. The bioconcentration potential for MTBE in fish is rated as insignificant based on the studies with Japanese carp by Fujiwara et al. (1984) generating bioconcentration factors for MTBE ranging from 0.8 to 1.5. Limited data suggest that MTBE will not bioaccumulate in fish or food chains (ATSDR 1996). Based on fugacity modeling and limited information on concentrations in shellfish, it is estimated that the average daily intake of MTBE for the age group of the Canadian population most exposed on a body weight basis, i.e., 5 to 11-year-olds, is 0.67 ng/kg/day (Environmental Canada 1992, 1993, Long et al. 1994).

#### *Water*

MTBE, being a water-soluble molecule, binds poorly to soils and readily enters surface and underground water. MTBE appears to be resistant to chemical and microbial degradation in water (ATSDR 1996). When it does degrade, the primary product is TBA. Two processes, degradation and volatilization, appear to reduce the concentrations of MTBE in water (Baehr et al. 1997, Borden et al. 1997, Schirmer and Baker 1998). The level of ingested MTBE from drinking water at the range relevant to human exposures appears to be directly proportional to the MTBE concentrations in water (Big/dynamics, Inc. 1981, 1984c, Nihlen et al. 1994). The concentrations of MTBE in Canadian surface water predicted under a worst-case scenario is six ppt (or six ng/L), which is  $1.12 \times 10^8$  times lower than the 96-hour LC<sub>50</sub> for fathead minnow of 672 ppm (or 672 mg/L) (Environmental Canada 1992, 1993). The transport, behavior and fate of MTBE in streams have been summarized by the USGS NAWQA Program (Rathbun 1998).

MTBE can be a water contaminant around major production sites, pipelines, large tank batteries, transfer terminals, and active or abandoned waste disposal sites. It tends to be the most frequently detected VOC in shallow groundwater (Bruce and McMahon 1996). The primary release of MTBE into groundwater is from leaking USTs. Gasoline leaks, spills or exhaust, and recharge from stormwater runoff contribute to MTBE in groundwater. In small quantities, MTBE in air dissolves in water such as deposition in rain (Pankow et al. 1997). Recreational gasoline-powered boating and personal watercraft is thought to be the primary source of MTBE in surface water. MTBE has been detected in public drinking water systems based on limited monitoring data (Zogorski et al. 1997). Surveillance of public drinking water systems in Maine, begun in February 1997, has detected MTBE at levels ranging from 1 to 16 ppb in 7 percent of 570 tested systems with a median concentration of three ppb (IPCS 1997, Smith and Kemp 1998). Sampling program conducted during summer of 1998 found trace levels of MTBE in 15 percent of Maine's



drinking water supplies. Concentrations above 38 ppb were found in 1 percent of the wells (Renner 1999).

MTBE is detected in groundwater following a reformulated fuel spill (Garrett et al. 1986, Shaffer and Uchirin 1997). MTBE in water can be volatilized to air, especially at higher temperature or if the water is subjected to turbulence. However, it is less easily removed from groundwater than other VOCs such as benzene, toluene, ethylbenzene, and xylenes (BTEX) that are commonly associated with gasoline spills. MTBE and BTEX are the most water-soluble fractions in gasoline and therefore the most mobile in an aquifer system. Based on equilibrium fugacity models and especially during warm seasons, the high vapor pressure of MTBE leads to partitioning to air and half-lives in moving water are estimated around 4.1 hours (Davidson 1995, Hubbard et al. 1994). In shallow urban groundwater, MTBE was not found with BTEX. Landmeyer et al. (1998) presented the areal and vertical distribution of MTBE relative to the most soluble gasoline hydrocarbon, benzene, in a shallow gasoline-contaminated aquifer and biodegradation was not a major attenuation process at this site. MTBE may be fairly persistent since it is refractory to most types of biodegradation (Borden et al. 1997, Daniel 1995, Jensen and Arvin 1990). Adsorption is expected to have little effect and dissolved MTBE will move at the same rate as the groundwater. MTBE may be volatilized into air or into soil gas from groundwater and these mechanisms may account for the removal of MTBE from groundwater.

MTBE has been detected in water, mainly by the USGS, in Colorado (Livo 1995, Bruce and McMahon 1996), California (Boughton and Lico 1998), Connecticut (Grady 1997), Georgia, Indiana (Fenelon and Moore 1996), Maine (Smith and Kemp 1998), Maryland (Daly and Lindsey 1996), Massachusetts (Grady 1997), Minnesota, Nevada (Boughton and Lico 1998), New Hampshire (Grady 1997), New Jersey (Terracciano and O'Brien 1997, O'Brien et al. 1998), New Mexico, New York (Stackelberg et al. 1997, Lince et al. 1998, O'Brien et al. 1998), North Carolina (Rudo 1995), Pennsylvania (Daly and Lindsey 1996), South Carolina (Baehr et al. 1997), Texas, Vermont (Grady 1997), Wisconsin and other states. A recent USGS NAWQA survey (Boughton and Lico 1998) reported the detection of MTBE in Lake Tahoe, Nevada and California, from July to September 1997, in concentrations ranging from 0.18 to 4.2 ppb and to depths of 30 meters. Zogorski et al. (1998) summarized the findings and research by the USGS in ground and surface water that MTBE has been detected in 14 percent of urban wells and 2 percent of rural wells sampled from aquifers used for drinking water.

USGS has published the results of the NAWQA Program (Squillace et al. 1995, 1996, 1997a, 1997b, 1998) of monitoring wells, which are not drinking water wells. This program analyzed concentrations of 60 VOCs from 198 shallow wells and 12 springs in eight urban areas (none in California) and 549 shallow wells in 21 agricultural areas (including the San Joaquin Valley). MTBE was detected in 27 percent of the urban wells and springs and 1.3 percent of the agricultural wells. The average MTBE concentration found in shallow groundwater was 0.6 ppb. MTBE was the second most frequently detected VOC (behind chloroform) in shallow groundwater in urban wells with a detection frequency of 27 percent of the 210 wells and springs sampled (Anonymous 1995, Squillace et al. 1996, Zogorski et al. 1998). No MTBE was detected in 100 agricultural wells in the San Joaquin Valley.

A recent evaluation of MTBE impacts to California groundwater resources (Happel et al. 1998), jointly sponsored by the Underground Storage Tank (UST) Program of the California State Water Resources Control Board (SWRCB), the Office of Fossil Fuels of U.S. Department of Energy (DOE), and the Western States Petroleum Association (WSPA), found evidence of MTBE in nearly 80 percent of the 1,858 monitoring wells from 236 leaking underground fuel tank (LUFT) sites in 24 counties examined by the Lawrence Livermore National Laboratory (LLNL). LLNL originally estimated that more than 10,000 LUFT sites out of the recognized 32,409 sites in California are contaminated with MTBE. Recent ongoing monitoring report (UC 1998) confirms that at least 3,000 to 4,500 LUFT sites are contaminated with MTBE. Maximum concentrations found at these sites ranged from several ppb to approximately 100,000 ppb or 100 ppm, indicating a wide range in the magnitude of potential MTBE impacts at gasoline release sites. MTBE plumes are more mobile than BTEX plumes, and the plumes are usually large migrates. Primary attenuation mechanism for MTBE is dispersion. LLNL concluded that MTBE might present a cumulative contamination hazard.

In response to the growing concern over the detection of MTBE in California's groundwater and surface water bodies, the SWRCB was requested to convene an advisory panel to review the refueling facilities and practices at marinas located on surface water bodies serving as drinking water sources to determine if any upgrades should be made to eliminate releases to the water body (Patton et al. 1999a). In ad-

dition, SWRCB's advisory panel was asked to review existing data base of UST contamination sites to determine if there is a leak history and identify appropriate measures to assure the prevention and detection of oxygenate releases from retail marketing facilities (Patton et al. 1999b).

MTBE was detected in municipal stormwater in 7 percent of the 592 samples from 16 U.S. cities during 1991 to 1995 with a range of 0.2 to 8.7 ppb and a median of 1.5 ppb (Delzer et al. 1997). MTBE was found to be the seventh most frequently detected VOCs in municipal stormwater. Among the stormwater samples that had detectable concentrations of MTBE, 87 percent were collected between October 1 and March 31 which is the period of time when oxygenated gasoline is used in CO nonattainment areas (Squillace et al. 1998). Surveys by the U.S. EPA found that 51 public water suppliers in seven responding states had detected MTBE. There are ongoing regional studies of MTBE occurrence in California, New England, Long Island, New Jersey and Pennsylvania (Wiley 1998). MTBE was detected in aquifers (Landmeyer et al. 1997, 1998, Lindsey 1997).

Cal/EPA and other State agencies have taken a proactive approach toward investigating MTBE in water in California. MTBE has recently been detected in shallow groundwater at over 75 percent of about 300 leaking UST sites in the Santa Clara Valley Water District (SCVWD), at 90 out of 131 fuel leak sites under jurisdiction of the San Francisco Regional Water Quality Control Board (SFRWQCB) and at over 200 leaking sites in the Orange County Water District. According to the Santa Ana Regional Water Quality Control Board, MTBE has been found at concentrations higher than 200 ppb at 68 percent of the leaking UST sites in its jurisdiction and at concentrations above 10,000 ppb at 24 percent of the leaking sites. In Solano County, concentrations of MTBE as high as 550,000 ppb have been reported in groundwater at sites with leaking USTs. However, these wells are not sources for drinking water (SCDEM 1997). At sites of gasoline leakage, MTBE concentrations as high as 200,000 ppb have been measured in groundwater (Davidson 1995, Garrett et al. 1986).

In July 1998, the SFRWQCB (1998) has compiled a list of 948 LUFT sites in the nine Bay Area counties in which groundwater has been contaminated with MTBE to a concentration of more than five ppb, which is the detection limit. The MTBE concentrations from the monitoring wells ranged from six ppb to as high as 19,000,000 ppb or 19,000 ppm. The monitoring well with 19,000,000 ppb of MTBE also was reported with benzene contamination in groundwater at 1,900 ppb and a maximum concentration of 6,100 ppb during the past 2 years. The range of MTBE concentrations was from 7 to 390,000 ppb in Alameda County, 6 to 240,000 ppb in Contra Costa County, 6 to 210,000 ppb in Marin County, 12 to 60,000 ppb in Napa County, 6 to 710,000 ppb in San Francisco County, 7 to 2,400,000 ppb in San Mateo County, 6 to 140,000 ppb in Santa Clara County, 9 to 19,000,000 ppb in Solano County, and 7 to 390,000 ppb in Sonoma County.

In 1994, SB 1764 (Thompson, California Health and Safety Code, Section 25299.38) established an independent advisory committee to the SWRCB to review the cleanup of USTs including requesting companies to monitor MTBE (Farr et al. 1996). State and Federal statutes require that all USTs including LUFTs be removed, replaced or upgraded to meet current standards by December 22, 1998. In June 1996, the SWRCB asked local regulatory agencies to require analysis at all leaking UST sites with affected groundwater. MTBE has been detected at a majority of the sites. Concentrations of MTBE in shallow groundwater near the source of the fuel release can exceed 10,000 ppb or 10 ppm (Cal/EPA 1998).

In 1995, ARB requested DHS' Division of Drinking Water and Environmental Management to test for MTBE in the state's drinking water. In February 1996, DHS sent an advisory letter to water suppliers it regulates, requesting voluntary testing for MTBE while a monitoring regulation was being developed. The regulation was adopted on February 13, 1997, and requires monitoring of MTBE as an unregulated chemical by the water suppliers from a drinking water well or a surface water intake at least once every 3 years. DHS routinely updates the reported detection of MTBE in groundwater and surface water sources on its website. DHS uses a detection limit for purposes of reporting (DLR) for MTBE of five ppb based on consideration of the State's commercial laboratories' use of MTBE in other common analyses and the potential for sample contamination and the reporting of false positives. Laboratories are only required to report MTBE analytical results at or above the five ppb DLR, but some laboratories are reporting lower concentrations.

According to the DHS report, from February 13 to June 13, 1997, MTBE had been detected in 14 of the 388 drinking water systems that had been monitored. As of December 22, 1997, 18 of the 516 systems monitored had reported MTBE detection. These are drinking water wells tapping deep aquifers and some aquifers at depths of 200 feet or greater. In addition, approximately 2,500 public drinking water

sources had been sampled and reported. Only 33 sources including 19 groundwater sources and 14 surface water sources, 9 of which are reservoirs, had reported detectable concentrations of MTBE. Three groundwater sources including city of Santa Monica (up to 300 ppb in February 1996), city of Marysville (up to 115 ppb in January 1997), and Presidio of San Francisco (up to 500 ppb in July 1990 from a currently abandoned well) had reported concentrations above the U.S. EPA (1997a) advisory level of 20 to 40 ppb. Otherwise, the range of reported values was less than (<) one to 34.1 ppb in groundwater sources and < one to 15 ppb in surface water sources (DHS 1997).

The city of Santa Monica has shut down two well fields, Charnock and Arcadia, due to MTBE contamination. These well fields used to supply 80 percent of the drinking water to the city residents. Concentrations as high as 610 ppb were observed in the Charnock aquifer and the seven wells in the field have been closed. In the Arcadia well field, two wells have been closed due to MTBE contamination from an UST at a nearby gasoline station (Cal/EPA 1998, Cooney 1997). DHS (1997) reported MTBE concentrations up to 130 ppb in a Charnock well and 300 ppb in another Charnock well in February 1996, and up to 72.4 ppb in an Arcadia well in August 1996. In Santa Clara County, the Great Oaks Water Company has closed a drinking water well in South San Jose due to trace MTBE contamination. The Lake Tahoe Public Utilities District has shut down 6 of their 36 drinking water wells because of MTBE contamination.

MTBE has also been found in many surface water lakes and reservoirs (DHS 1997). The reservoirs allowing gasoline powerboat activities have been detected with MTBE at higher concentrations than those reservoirs prohibiting boating activities. DHS reported MTBE in Lake Tahoe, Lake Shasta, Whiskeytown Lake in the city of Redding, San Pablo Reservoir in East Bay Municipal Utility District (EBMUD) in the San Francisco Bay area, Lobos Creek in Presidio of San Francisco, Del Valle and Patterson Pass of Zone Seven Water Agency serving east Alameda County, Clear Lake in Konocti County Water District, Canyon Lake in the Elsinore Valley Municipal Water District, Lake Perris in the MWDC in the Los Angeles area, and Alvarado, Miramar, and Otay Plant influent in city of San Diego. MTBE concentrations ranged from < 1 to 15 ppb. Donner Lake, Lake Merced, Cherry and New Don Pedro Reservoirs in EBMUD, Anderson and Coyote Reservoirs in the SCVWD, Modesto Reservoir in the Stanislaus Water District, and Castaic Reservoir in MWDC also had detectable levels of MTBE.

The city of Shasta Lake domestic water supply intake raw water was reported with 0.57 ppb MTBE in September 1996 although Lake Shasta had 88 ppb in a surface water sample next to a houseboat at a marina dock. BTEX were found in lower concentrations than MTBE. Water was analyzed for hydrocarbons before and after organized jet ski events held in the summer and fall of 1996 in Orange County and Lake Havasu (Dale et al. 1997a). MTBE was measured in the water at the small holding basin in Orange County at concentrations of up to 40 ppb a few days after the event while there was only negligible BTEX. At the larger Lake Havasu, the MTBE concentrations increased from below the level of detection to 13 ppb. A recent report to the SCVWD described the detection of an average concentration of three ppb MTBE in Anderson, Calero, and Coyote Reservoirs which are drinking water sources where powerboating is allowed. Calero Reservoir banned jet skis in July 1998. The National Park Service is proposing a systemwide ban on similar types of personal watercraft, which are presently allowed in 34 of America's 375 national park units.

The Carson publicly-owned treatment works (POTW) in Carson, California has also reported MTBE in its wastewater. The Carson POTW processes the largest volume of refinery wastewater in the Nation (13 refineries sporadically discharge wastewater to the POTW). Refineries in California perform their own pretreatment prior to discharging to sewers. The refineries' discharges contain average levels from one to 7,000 ppb (seven ppm) with concentrations occasionally as high as 40,000 ppb. California refineries are situated mainly along the coast and discharge directly or indirectly to marine waters. No California refineries discharge their wastewater to sources of drinking water.

#### METABOLISM AND PHARMACOKINETICS

The available information on the metabolism and pharmacokinetics of MTBE is limited to humans and rats with little information from mice. MTBE can be absorbed into the body after inhalation in humans (Johanson et al. 1995, Nihlen et al. 1998a, 1998b, Vainiotalo et al. 1998) and rats (Buckley et al. 1997, Miller et al. 1997, Prah et al. 1994, Savolainen et al. 1985), ingestion or skin contact in rats (Miller et al. 1997). It is metabolized and eliminated from the body within hours.

MTBE caused lipid peroxidation in the liver and induction of hepatic microsomal cytochrome P<sub>450</sub> content in mice (Katoh et al. 1993). The major metabolic pathway of MTBE in both animals and humans is oxidative demethylation leading to the production of TBA (Poet et al. 1997c). In animals, HCHO is also a metabolite (Hutcheon et al. 1996). This reaction is catalyzed by cytochrome P<sub>450</sub> enzymes (Brady et al. 1990, Hong et al. 1997b).

MTBE and TBA have been detected in blood, urine, and breath of humans exposed to MTBE via inhalation for 12 hours. Nihlen et al. (1998b) in a chamber study exposing human subjects for 2 hours suggests that TBA in blood or urine is a more appropriate biological exposure marker for MTBE than the parent ether itself. Bonin et al. (1995) and Lee and Weisel (1998) described analytical methods for detecting MTBE and TBA in human blood and urine at concentrations below one ppb. A recent Finnish study, Saarinen et al. (1998) investigated the uptake of 11 drivers to gasoline vapors during road-tanker loading and unloading. The total MTBE uptake during the shift was calculated to be an average of  $106 \pm 65$  mmole. The mean concentrations of MTBE and TBA detected in the first urine after the work shift were  $113 \pm 76$  and  $461 \pm 337$  nanomole/L, and those found 16 hours later in the next morning were  $18 \pm 12$  and  $322 \pm 213$  nanomole/L, respectively.

#### *Absorption*

There is limited information on the rate and extent that MTBE enters the systemic circulation. MTBE is lipophilic which will facilitate its absorption across the lipid matrix of cell membranes (Nihlen et al. 1997). In its liquid or gaseous state, MTBE is expected to be absorbed into the blood stream (Nihlen et al. 1995). MTBE is absorbed into the circulation of rats following oral, intraperitoneal (i.p.), intravenous (i.v.), or inhalation exposures (BioResearch Laboratories 1990a, 1990b, 1990c, 1990d, Miller et al. 1997, NSTC 1997). Dermal absorption of MTBE is limited, as compared with other routes.

The concentration-time course of MTBE in blood plasma of male rats administered 40 mg/kg/day by oral, dermal, or i.v. routes was followed (Miller et al. 1997). Peak blood concentrations of MTBE ( $C_{max}$ ) were obtained within 5 to 10 minutes. Higher levels of MTBE were seen after oral versus i.v. exposure indicating elimination of the latter via the lungs. Miller et al. (1997) compared the areas under the concentration-time curves (AUC) for MTBE following i.v. and oral administrations and concluded that MTBE was completely absorbed from the gastrointestinal tract. Plasma levels of MTBE following dermal exposure were limited; peak concentrations were achieved 2 to 4 hours after dosing. Absorption ranged from 16 to 34 percent of applied doses of 40 mg/kg/day and 400 mg/kg/day respectively. After inhalation exposure, plasma concentrations of MTBE reached apparent steady state within 2 hours at both low (400 ppm) and high (8,000 ppm) doses. Peak MTBE concentrations were reached at four to 6 hours and were 14 and 493 ppb, respectively.

#### *Distribution*

Once in the blood, MTBE is distributed to all major tissues in the rat. Due to its hydrophilic properties, neither MTBE nor its metabolites would be expected to accumulate in body tissues. TBA appears to remain longer, and chronic exposure could result in accumulation to some steady-state level, but this needs further study. Once absorbed, MTBE is either exhaled as the parent compound or metabolized. Oxidative demethylation by cytochrome P<sub>450</sub>-dependent enzymes is the first step in the metabolism that yields HCHO and TBA. TBA is detected in blood and urine and appears to have a longer half-life in blood than MTBE (Poet et al. 1996, Prah et al. 1994, Prescott-Mathews et al. 1996, Savolainen et al. 1985).

#### *Metabolism*

The metabolism of absorbed MTBE proceeds in a similar fashion regardless of route of exposure. MTBE is metabolized via microsomal enzymes in the cells of organs (Turini et al. 1998). MTBE undergoes oxidative demethylation in the liver via the cytochrome P<sub>450</sub>-dependent enzymes (P<sub>450</sub>IIE1, P<sub>450</sub>IIB1, and P<sub>450</sub>IIA6 are thought to be involved) to give TBA and HCHO (Brady et al. 1990, Hong et al. 1997b). Rat olfactory mucosa displays a high activity in metabolizing MTBE via the cytochrome P<sub>450</sub>-dependent enzymes (Hong et al. 1997a). In vitro studies of MTBE in human (Poet and Borghoff 1998) and rat (Poet and Borghoff 1997b) liver microsomes confirm that MTBE is metabolized by P<sub>450</sub>-dependent enzymes and suggest that the metabolism of MTBE will be highly variable in humans. TBA may be eliminated unchanged in expired air or may undergo secondary metabolism forming 2-methyl-1,2-propanediol and  $\alpha$ -hydroxyisobutyric acid. Both of these latter metabolites are excreted in the urine and account for about 14 percent and 70 percent respectively of urine radioactivity for <sup>14</sup>C-MTBE dosed rats (Miller et al. 1997). Two unidentified minor metabolites are also excreted in urine.

Bernauer et al. (1998) studied biotransformation of  $^{12}\text{C}$ - and  $2\text{-}^{13}\text{C}$ -labeled MTBE and TBA in rats after inhalation or gavage exposure to identify 2-methyl-1,2-propanediol and 2-hydroxyisobutyrate as major metabolites in urine by  $^{13}\text{C}$  nuclear magnetic resonance and gas chromatography/mass spectrometry. In one human individual given five mg  $^{13}\text{C}$ -TBA/kg orally, 2-methyl-1,2-propanediol and 2-hydroxyisobutyrate were major metabolites in urine. The results suggest that TBA formed from MTBE be extensively metabolized by further oxidation reactions. In vitro evidence suggests that TBA may also undergo oxidative demethylation to produce HCHO and acetone (Cederbaum and Cohen 1980). Identification of  $^{14}\text{CO}_2$  in expired air of  $^{14}\text{C}$ -MTBE treated rats suggests some complete oxidation of MTBE or metabolites occurs, probably via HCHO. Studies in humans are more limited but TBA has been observed as a blood metabolite of MTBE. The participation of hepatic cytochrome P<sub>450</sub>-dependent enzymes also indicates a potential role of co-exposure to other environmental chemicals in affecting MTBE metabolism and toxicity (Hong et al. 1997b, NSTC 1997).

#### *Excretion*

Elimination of MTBE and its metabolites by Fischer 344 rats is primarily via the lungs (expired air) and the kidneys (urine). In expired air, MTBE and TBA are the predominant forms. After i.v. administration of  $^{14}\text{C}$ -MTBE to male rats most of the radioactivity was excreted in the exhaled air (60 percent) and urine (34.9 percent) with only 2 percent in the feces and 0.4 percent remaining in the tissues/carcass. Most of the administered dose was eliminated as MTBE during the first 3 hours following administration. About 70 percent of the dose recovered in the urine were eliminated in the first 24 hours and 90 percent in 48 hours. After dermal exposure to MTBE for 6 hours, 70 to 77 percent of the applied radioactivity was unabsorbed while 7.6 to 18.9 percent was excreted in expired air, 6.3 to 16.2 percent in urine, and 0.25 to 0.39 percent in feces at 40 and 400 mg/kg/day respectively. A negligible amount (< 0.2 percent) was found in tissues/carcass. The composition of  $^{14}\text{C}$ -radiolabel in expired air was 96.7 percent MTBE and 3.3 percent TBA at the high dose. After inhalation exposures most of the  $^{14}\text{C}$  was eliminated in the urine with 64.7 percent after single and 71.6 percent after repeated low doses. At the high dose, a larger fraction was eliminated in exhaled air: 53.6 percent compared to 17 percent for single or 21 percent for repeated low doses. Less than 1 percent of the dose was recovered in the feces and < 3.5 percent in the tissues/carcass. The composition of  $^{14}\text{C}$ -radiolabel in exhaled breath in the first 6 hours following administration of MTBE was 66 to 69 percent MTBE and 21 to 34 percent TBA. By 24 hours post-dose 85 to 88 percent of the urine radioactivity was eliminated in rats from all exposure groups (Miller et al. 1997).

Pulmonary elimination of MTBE after intraperitoneal injection in mice (Yoshikawa et al. 1994) at three treated doses (50, 100 and 500 mg/kg) indicated an initial rapid decrease of the elimination ratio followed by a slow decrease at the doses of 100 and 500 mg/kg. The calculated half-lives of the two elimination curves obtained by the least squares method were approximately 45 minutes and 80 minutes. The pulmonary elimination ratios at the three different doses were from 23.2 percent to 69 percent. Most of the excreted MTBE was eliminated within 3 hours.

In a human chamber study (Buckley et al. 1997), two subjects were exposed to 1.39 ppm MTBE, that is comparable to low levels which might be found in the environment for 1 hour, followed by clean air for 7 hours. The results showed that urine accounted for less than 1 percent of the total MTBE elimination. The concentrations of MTBE and TBA in urine were similar to that of the blood ranging from 0.37 to 15  $\mu\text{g/L}$  and two to 15  $\mu\text{g/L}$ , respectively. Human breath samples of end-expiration volume were collected from two individuals during motor vehicle refueling, one person pumping the fuel and a nearby observer, immediately before and for 64 minutes after the vehicle was refueled with premium grade gasoline (Lindstrom and Pleil 1996). Low levels of MTBE were detected in both subjects breaths before refueling and levels were increased by a factor of 35 to 100 after the exposure. Breath elimination indicated that the half-life of MTBE in the first physiological compartment was between 1.3 and 2.9 minutes. The breath elimination of MTBE during the 64-minute monitoring period was about four-fold for the refueling subject comparing to the observer subject.

Johanson et al. (1995) and Nihlen et al. (1998a, 1998b) reported toxicokinetics and acute effects of inhalation exposure of 10 male subjects to MTBE vapor at 5, 25, and 50 ppm for 2 hours during light physical exercise. MTBE and TBA were monitored in exhaled air, blood, and urine. The elimination of MTBE from blood was multi-phasic with no significant differences between exposure levels. The elimination phases had half-lives of 1 minute, 10 minutes, 1.5 hours, and 19 hours respectively. Elimination of MTBE in urine occurred in two phases with average half-

lives of 20 minutes and 3 hours. Excretion of MTBE appeared to be nearly complete within 10 hours. For TBA excretion the average post-exposure half-lives in blood and urine were 10 and 8.2 hours respectively. Some exposure dependence was noted for the urinary half-life with shorter values seen at the highest exposure level (50 ppm  $\times$  2 hour). A low renal clearance for TBA (0.6 to 0.7 mL/hour/kg) may indicate extensive blood protein binding or renal tubular reabsorption of TBA.

#### *Pharmacokinetics*

The plasma elimination half-life ( $t_{1/2}$ ) of MTBE in male rats was about 0.45 to 0.57 hour after i.v., oral (low dose), and inhalation exposures. A significantly longer  $t_{1/2}$  of 0.79 hour was observed with the high oral dose of 400 mg/kg/day. For dermal exposure the initial MTBE elimination  $t_{1/2}$  was 1.8 to 2.3 hours. TBA elimination  $t_{1/2}$  values were 0.92 hour for i.v., 0.95 to 1.6 hours for oral, 1.9 to 2.1 hours for dermal, and 1.8 to 3.4 hours for inhalation exposures. The apparent volume of distribution for MTBE ranged from 0.25 to 0.41 L after i.v., oral, and inhalation dosing and from 1.4 to 3.9 liters (L) after dermal exposures. The total plasma clearance of MTBE, corrected for relative bioavailability, ranged from 358 to 413 mL/hour in i.v., oral, and dermal administrations. Inhalation values ranged from 531 mL/hour for low single dose to 298 mL/hour for high single dose. For oral administration of 40 or 400 mg/kg/day MTBE the AUC values were 17 and 230 ( $\mu\text{g/mL}$ ) hour for MTBE and 39 and 304 ( $\mu\text{g/mL}$ )hour for TBA (Miller et al. 1997).

The disposition and pharmacokinetics observed in these studies are similar to those observed in human volunteers following inhalation and dermal exposures (U.S. EPA 1993). For inhalation exposure to 5 mg/m<sup>3</sup> for 1 hour the  $t_{1/2}$  value for MTBE was 36 minutes. Blood TBA levels rose during exposure and remained steady for up to 7 hours post-exposure suggesting a longer  $t_{1/2}$  for TBA in humans compared to rats. Other more recent data (cited in NSTC 1997) indicate a multi-exponential character to MTBE elimination from human blood with  $t_{1/2}$  values of 2 to 5 minutes, 15 to 60 minutes and greater than 190 minutes. These results possibly indicate a more complex distribution or binding of MTBE in humans than observed in rats. Such differences probably are related to larger fat compartments in humans compared to rats.

Overall, these studies show that following i.v., oral, or inhalation exposures MTBE is absorbed, distributed, and eliminated from the body with a half-life of about 0.5 hour. Dermal absorption is limited. The extent of metabolism to TBA (and HCHO) the major metabolite is somewhat dependent on route and dose. TBA is eliminated from the body with a half-life of 1 to 3 hours or longer in humans. Virtually all MTBE is cleared from the body 48 hours post-exposure.

#### *Physiologically-Based Pharmacokinetic (PBPK) Models*

Computer-based PBPK models have been developed for rats (Borghoff et al. 1996a, Rao and Ginsberg 1997). These models vary in complexity, metabolic parameters, and one chemical specific parameter. The Borghoff et al. (1996a) model uses five compartments for MTBE and either five or two for TBA. While model predictions of MTBE blood concentrations and clearance following inhalation or oral exposures were generally good, the model underpredicted MTBE blood levels at 8,000 ppm by a factor of two. Accurate model predictions of TBA blood levels and clearance were more elusive with the two compartment model giving more accurate predictions at lower oral and inhalation doses than at higher doses or than the five compartment model. The Rao and Ginsberg (1997) model is more complex using eight compartments for MTBE and eight for TBA. While both models assume two Michaelis-Menten processes ( $V_{\text{maxc}}/K_m$ ) from MTBE to TBA namely high capacity to low affinity ( $V_{\text{maxc}_2}/K_{m_2}$ ), and low capacity to high affinity ( $V_{\text{maxc}_1}/K_{m_1}$ ), the Rao and Ginsberg (1997) model uses different parameters than Borghoff et al. (1996a) with a lower  $V_{\text{maxc}_1}/K_{m_1}$ . Rao and Ginsberg (1997) use a lower tissue/blood partition coefficient for TBA in the slowly perfused compartment (e.g., muscle) of 0.4 versus 1. Predictions of blood levels and clearance rates for MTBE and TBA with MTBE inhalation exposures appear to be more accurate with this model. Similar validation is claimed for the oral and i.v. routes for MTBE exposure and for i.p. exposure to TBA although these data have not been seen in detail. Rao and Ginsberg (1997) used their model to evaluate some key uncertainties of acute inhalation exposures to MTBE during bathing and showering and concluded that the acute central nervous system (CNS) toxicity is likely due to MTBE rather than to its TBA metabolite. The simulated brain TBA concentration for CNS effects was in the 500 to 600 mg/L range. In contrast, the simulated brain concentration for MTBE's CNS effects was considerably lower (89 to 146 mg/L). By comparing TBA only versus MTBE exposure studies the authors concluded that under conditions where MTBE

dosing produced acute CNS toxicity, the simulated TBA brain concentrations were too low to be effective.

Despite the lack of human data on tissue/blood partition coefficients and other key parameters, both models have been adjusted to human anatomical and physiological values and estimated metabolic and chemical parameters and compared with limited human blood data. Although the Borghoff et al. (1996a) model was able to predict MTBE levels seen in Cain et al. (1996) during inhalation exposure, it underpredicted MTBE blood concentrations after exposure, resulting in a faster clearance than seen experimentally. The Rao and Ginsberg (1997) model more closely simulated the data (1.7 ppm MTBE for 1 hour) of Cain et al. (1996) but underpredicted the peak and postexposure concentrations at higher inhalation exposures of 5 and 50 ppm MTBE for 2 hours (Johanson et al. 1995). It is clear that while human MTBE PBPK models may be improved considerably, they may prove useful in their present State to assess risks associated with some environmental exposures to MTBE (e.g., exposures when taking a shower).

#### TOXICOLOGY

The toxicology profile of MTBE has been summarized in the U.S. (Von Burg 1992, ATSDR 1996) and in Great Britain (BIBRA 1990). Zhang et al. (1997) used computer modeling to predict metabolism and toxicological profile of gasoline oxygenates including MTBE based on structure activity relationships. Health risk assessment of MTBE has been performed (Gilbert and Calabrese 1992, Hartly and Engle 1992, Hiremath and Parker 1994, Stern and Tardiff 1997, Tardiff and Stern 1997). The general toxicity of MTBE is not considered as "highly hazardous" in a hazard ranking system for organic contaminants in refinery effluents (Siljeholm 1997) and is considered as less hazardous than most chemicals in 10 ranking systems in the Chemical Scorecard of the Environmental Defense Fund (EDF 1998). A substantial amount of health-related research has been conducted or initiated on MTBE in recent years (ATSDR 1996, U.S. EPA 1997a). A recent literature review (Borak et al. 1998) summarizes the exposure to MTBE and acute human health effects including nine epidemiological studies, 10 industrial hygiene studies, and 12 clinical studies. However, most of the studies and reviews focus on the inhalation route of exposure in human health effects and laboratory animal toxicities. No studies were located regarding toxic effects in humans after oral exposure to MTBE alone. Because this document is mainly concerned with the effects of MTBE in drinking water, it focuses on oral toxicity studies in animals. There is limited information on dermal exposure effects in humans and animals. Very little is known about the toxic effects of MTBE in plants and ecosystems.

#### *Toxicological Effects in Animals*

Table 4 summarizes the lowest concentrations resulting in toxicity in laboratory animals via inhalation or oral exposure as reported in the ATSDR (1996) document and the latest U.S. EPA (1997c) advisory. Clary (1997) reviewed the systemic toxicity of MTBE including 12 inhalation and four oral studies. Stelljes (1997) summarized similar information based on only the ATSDR (1996) document. The various noncancer health effects via oral route of exposure in all tested species and the duration of exposure are summarized in Table 5. The highest NOAELs and all the lowest observed adverse effect level (LOAELs) are also included in Table 5. Details of each of the studies listed in Table 5 are described in the following sections on acute, subacute, subchronic and chronic toxicity. The cancer effects observed in animals are discussed in a separate section on carcinogenicity in this chapter. There were no studies located regarding cancer in humans after oral, or any other exposure to MTBE.

In animal studies, oral exposure to MTBE for acute, subacute, subchronic, or chronic duration appears to be without effects on the cardiovascular, musculoskeletal, dermal, ocular, or reproductive systems. In acute and subacute oral exposure studies, limited effects on the respiratory, gastrointestinal, hematological, hepatic, renal, or neurological systems and some minor systemic toxicities have been observed. In subchronic oral exposure, limited effects on gastrointestinal, hematological, hepatic, or renal systems and some minor systemic toxicities have been observed. In chronic oral exposure, the main observation is cancer and preneoplastic effects (ATSDR 1996). In this document, all the potential toxic effects of MTBE have been reviewed with an emphasis on the oral exposure; particularly the potential reproductive, developmental and carcinogenic effects have been extensively reviewed by OEHHA staff.

Some acute, intermediate or chronic duration minimal risk levels (MRLs) have been derived by the ATSDR for inhalation or oral exposure to MTBE (ATSDR 1996). U.S. EPA (1997c) lists in IRIS a Reference Concentration (RfC) for inhalation that

is similar to the ATSDR's inhalation MRL. However, the current IRIS (U.S. EPA 1997c) does not list a Reference Dose (RfD) for ingestion (U.S. EPA 1987b) that is similar to the ATSDR's ingestion MRL. In addition to the key documents from governmental agencies and literature search articles mentioned above, toxicology information in the TOMES PLUS® data base (Hall and Rumack 1998) also has been used in the following summary of toxic effects of MTBE.

Table 4.—Summary of Selected Data on MTBE: Noncancer Toxic Effects in Animals\*

Dose level	Inhalation (mg/m <sup>3</sup> )			Oral (mg/kg/day)	
	Acute	Subacute/ Subchronic	Chronic	Acute	Subacute/ Subchronic
NOAEL .....	1,440	1,440	1,440	40	100
LOAEL .....	3,600	2,880	10,800	90	300
Lethal Dose .....	649,000	NA	NA	3,866	NA

\*Values represent the lowest reported in ATSDR (1996) and U.S. EPA (1997a)



Table 5.—Significant Noncancer Health Effects and Levels of Oral Exposure to MTBE in Animals\*

Species/(Strain)	Exposure/Duration/Frequency (Specific route)	System	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
<b>Acute Exposure</b>					
<i>Death:</i>					
Rat .....	once (gavage) .....	.....	.....	3,866 (LD <sub>50</sub> ) .....	ARCO 1980
Mouse .....	once (gavage) .....	.....	.....	4,000 (LD <sub>50</sub> ) .....	Little et.al. 1979
<i>Systemic Toxicity:</i>					
Rat .....	once (gavage) .....	Respiratory .....	.....	4,080 (labored respiration)	ARCO 1980
		Neurological .....	.....	1,900 (slight to marked CNS depression 2,450 (ataxia).	
Rat (Sprague-Dawley) .....	once (gavage in oil) .....	Gastrointestinal .....	.....	100 (diarrhea) .....	Robinson et al. 1990
		Neurological .....	900 .....	1,200 (profound but transient anesthesia).	
Rat (Fischer 344) .....	once (gavage in water) .....	Neurological .....	40 .....	400 (drowsiness) .....	Bioresearch Labs. 1990b)
Rat (Sprague-Dawley) .....	once (gavage) .....	Neurological .....	.....	90 (salivation) 440 (Male) (hypoactivity, ataxia) 1,750 (Female).	Johnson et al. 1992, Klan et al. 1992
<b>Subacute Exposure</b>					
<i>Systemic Toxicity:</i>					
Rat (Sprague-Dawley) .....	14 days .....	Respiratory .....	1,428 .....	.....	Robinson et al. 1990
	7 days/week .....	Cardiovascular .....	1,428 .....	.....	
	once/day (gavage in oil) .....	Gastrointestinal .....	.....	357 (diarrhea) .....	
		Hematological .....	1,428 (Female) .....	357 (Male) (decreased monocytes).	
		Hepatic .....	714 (Male) .....	1,071 (Male) [increased serum glutamic-oxaloacetic transaminase (SGOT) and lactic dehydrogenase] 1,428 (Female) [decreased blood urea nitrogen (BUN) values].	

Table 5.—Significant Noncancer Health Effects and Levels of Oral Exposure to MTBE in Animals\*—Continued

Species/(Strain)	Exposure/Duration/Frequency (Specific route)	System	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
Mouse (CD-1)	3 weeks, 5 days/week (gavage in oil).	Renal	1,071 (Male) 1,428 (Female)	1,428 (Male) (increased hyaline droplets).	Ward et al. 1994, 1995
		Endocrine	1,428		
		Body weight	714 (Female)	1,071 (Female) (unspecified reduced weight gain).	
		Immunological/Lymphoreticular.		1,428	
		Neurological	1,071	1,428 (profound but transient anesthesia, hypoactivity, ataxia).	
		Reproductive	1,428		
		Other	1,071 (Male) 357 (Female)	1,428 (Male) 714 (Female) (elevated cholesterol).	
		Body weight	1,000		
		Reproductive	1,000		
		Subchronic Exposure			
<i>Death:</i> Rat (Sprague-Dawley)	16 weeks, 4 days/week, once/day, (gavage in oil).			250 (Female) (increased mortality).	Belpoggi et al. 1995
<i>Systemic Toxicity:</i> Rat (Sprague-Dawley)	4 weeks, 5 days/week once/day (gavage)	Respiratory	1,750		Johnson et al. 1992, Klan et al. 1992
		Cardiovascular	1,750		
		Gastrointestinal	440	1,750 (inflammation, submucosal edema, epithelial hyperplasia, stomach ulcers).	
		Hematological	1,750		

.....	Muscle/skeleton .....	1,750 .....	1,750 (increased relative liver weights).
.....	Hepatic .....	440 .....	440 (Male) (increased hyaline droplets in proximal convoluted tubules and increased relative kidney weights).
.....	Renal .....	1,750 (Female)	
.....	Endocrine .....	1,750 .....	
.....	Dermal .....	1,750 .....	
.....	Ocular .....	1,750 .....	
.....	Body weight .....	1,750 .....	
.....	Immunological/ Lymphoreticular .....	1,750 .....	
.....	Neurological .....	.....	440 (hypoactivity, ataxia) ..
.....	Reproductive .....	1,750 .....	
.....	Other .....	440 .....	1,750 (increased serum cholesterol).
.....	Respiratory .....	1,200 .....	
.....	Cardiovascular .....	1,200 .....	
.....	Gastrointestinal .....	.....	all treated doses (diarrhea)
.....	Hematological .....	900 .....	1,200 (increased monocytes, decreased mean corpuscular volume in males, increased red blood cell, hemoglobin, hematocrit and decreased white blood cells in females).
.....	Hepatic .....	.....	BUN values).
.....	Renal .....	900 (Male) .....	1,200 (Male) .....
.....	.....	1,200 (Female)	(hyaline droplets, granular casts).
.....	.....	100 .....	300 (alterations in kidney weights).
.....	Endocrine .....	1,200 .....	
.....	Body weight .....	1,200 .....	
.....	Immunological/ Lymphoreticular .....	.....	
.....	Reproductive .....	1,200 .....	

Rat (Sprague-Dawley) .....

90 days .....

7 days/week .....

once/day (gavage in oil) .....

Table 5.—Significant Noncancer Health Effects and Levels of Oral Exposure to MTBE in Animals\*—Continued

Species/(Strain)	Exposure/Duration/Frequency (Specific route)	System	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
	.....	Other .....	300 (Male) .....	900 (Male) ..... 100 (Female) (elevated cholesterol).	
Chronic Exposure					
<i>Systemic Toxicity:</i> Rat (Sprague-Dawley) .....	104 weeks ..... 4 days/week ..... once/day (gavage in oil) ..... ..... ..... ..... ..... ..... ..... ..... ..... ..... .....	Respiratory ..... Cardiovascular ..... Gastrointestinal ..... Muscle/skeleton ..... Hepatic ..... Renal ..... Endocrine ..... Dermal ..... Body weight ..... Immunological/ Lymphoreticular. ..... Reproductive .....	1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 ..... 1,000 male ..... 1,000 .....	..... ..... ..... ..... ..... ..... ..... ..... ..... ..... 250 (Female) (dysplastic proliferation of lymphoreticular tissues, possibly preneoplastic). .....	Belpoggi et al. 1995

\* adapted from ATSDR (1996) and U.S. EPA (1997c)

## ACUTE TOXICITY

Studies of the systemic effects of MTBE have been conducted in animals, but the majority involves inhalation exposure (Clary 1997). Inhalation or contact with MTBE may irritate or burn skin and eyes. Vapors may cause dizziness or suffocation. Acute toxicity studies in animals demonstrate the extremely low toxicity of MTBE (ARCO 1980, Little et al. 1979, Reese and Kimbrough 1993).

The oral LD<sub>50</sub>s (lethal doses with 50 percent kill) are approximately 3,866 mg/kg or four mL/kg in rats, and approximately 4,000 mg/kg or 5.96 mL/kg in mice. The inhalation 4-hour LC<sub>50</sub>s (lethal concentrations with 50 percent kill) in rats have been calculated to be approximately 39,395 ppm for 96.2 percent MTBE, 33,370 ppm for 99.1 percent MTBE and 23,576 ppm for MTBE. The inhalation 10-minute LC<sub>50</sub> in mice is approximately 180,000 ppm and the inhalation 15-minute LC<sub>50</sub> in mice is approximately 141 g/m<sup>3</sup>. The inhalation LT<sub>50</sub> (time at which death occurs in 50 percent of the exposed animals) in mice exposed to 209,300 ppm MTBE is 5.6 minutes (ATSDR 1996). The dermal LD<sub>50</sub> is estimated to be greater than 10 mL/kg in New Zealand rabbits (HSDB 1997). The i.p. LD<sub>50</sub> is 1.7 mL/kg or approximately 1,100 mg/kg in mice and greater than 148 mg/kg in rats (Arashidani et al. 1993, RTECS 1997).

Zakko et al. (1997) reported cytotoxicity of MTBE to intestinal mucosa of rats via i.p. injection similar to the effects of MTBE treatment for gallstone dissolution in humans. MTBE infused intraduodenally for 3 hours in male New Zealand rabbits caused local intestinal cytotoxic and systemic hepatotoxic effects (Clerici et al. 1997).

At lethal doses, ocular and mucous membrane irritation, ataxia, labored breathing, CNS depression, and general anesthetic effects precede death. An inhalation study also demonstrated inflammation in the nasal mucosa of rats at a dose of 3,000 ppm for 6 hours per day for 9 days (HSDB 1997). Mice that inhaled up to approximately 8,400 ppm MTBE for 1 hour had approximately a 52 percent decrease in breathing frequency (Tepper et al. 1994). The decrease occurred immediately, reached a maximum by 10 minutes and returned to baseline 15 minutes after exposure. High oral doses of greater than 4,080 mg of MTBE/kg caused labored respiration in rats (ARCO 1980). A 4-hour direct exposure to MTBE vapor at concentrations greater than 18,829 ppm in an inhalation study resulted in ocular discharges in rats (ARCO 1980). A 6-hour inhalation study produced signs of reversible CNS depression following exposure to 8,000 ppm and, to a lesser extent, to 4,000 ppm vapor with a NOAEL of 800 ppm (Dodd and Kintigh 1989, Daughtrey et al. 1997). As indicated in Tables 4 and 5, a NOAEL of 40 mg/kg/day and a LOAEL of 90 mg/kg/day are established by these acute oral exposure experiments based on the neurological effects (Bioresearch Laboratories 1990b, Johnson et al. 1992, Klan et al. 1992).

## SUBACUTE TOXICITY

In a consecutive 14-day study, Sprague-Dawley rats (10/sex/dose) were administered MTBE in corn oil by gavage at 0, 357, 714, 1,071 or 1,428 mg/kg/day. MTBE appears to be irritating to the gastrointestinal tract of rats as evidenced by diarrhea and histological lesions at all levels of MTBE by the third day of dosing throughout the 14-day study. Decreased lung weight was observed in female rats at all MTBE doses and at 714 mg/kg/day in male rats. Decreased levels of monocytes in blood were observed in male rats at all MTBE doses. Increased liver enzymes in males at 1,071 mg/kg/day and decreased blood urea nitrogen (BUN) values in females at 1,428 mg/kg/day were observed. At the highest dose, anesthesia was immediate, but recovery was complete within 2 hours. Ataxia and hyperactivity, an increase in the weight of kidneys, adrenal glands, and livers in both genders at 1,428 mg/kg/day, and an increase in hyaline droplet formation in kidneys of male rats at 1,428 mg/kg/day were observed. Increases in relative kidney weights were noted in the males at 1,071 and at 1,428 mg/kg/day and in females at the 1,428 mg/kg/day dose. Although there was a dose-related decrease in body weight gain, it was significant only in females at the highest treatment regimen. At 1,428 mg/kg/day in males and at 714 mg/kg/day in females, elevated cholesterol was observed. There were no gross lesions seen at any treatment level. Based on the increases in relative kidney weight, a NOAEL of 714 mg/kg/day and a LOAEL of 1,071 mg/kg/day are established by these experiments (Robinson et al. 1990). These studies indicate that the male kidney is the primary target of short-term toxicity at relatively high doses. Subchronic toxicity studies of TBA indicated that, in rodents, the urinary tract is a target system and males are more sensitive to TBA toxicity than females (NTP 1995).

## SUBCHRONIC TOXICITY

In a 104-week gavage cancer study, increased mortality was observed in female Sprague-Dawley rats at 250 mg/kg/day beginning at 16 weeks from the start of the study (Belpoggi et al. 1995). Daily oral administration in rats for 4 weeks resulted in increased hyaline droplets and kidney weight in males at 440 mg/kg/day and higher doses, and stomach ulcers, increased liver weights and serum cholesterol at 1,750 mg/kg/day (Johnson et al. 1992, Klan et al. 1992).

Sprague-Dawley rats (10/sex/dose) were treated orally with MTBE in corn oil for 90 days at 0, 100, 300, 900, or 1,200 mg/kg/day. Anesthesia was evident at the highest dose, but as in the 14-day study, full recovery occurred in 2 hours. There was a significant decrease in final body weight of females only at the highest level of treatment. The diarrhea seen in the treated animals was considered to be the consequence of the bolus dosing regime. In female rats, there were significantly increased heart weights at 900 mg/kg/day and increases in relative kidney weights at 300, 900, and 1,200 mg/kg/day. In male rats, increases were noted only at the two highest treatment levels. BUN levels were significantly reduced in both males and females at all MTBE doses. Reductions in serum calcium and creatinine were observed in males and a reduction in cholesterol in females was reported, but there were no clear dose-dependent results. Based on the alterations in kidney weights, a NOAEL and LOAEL of 100 and 300 mg/kg/day, respectively, are identified from this study (Robinson et al. 1990).

The subchronic data from the study by Robinson et al. (1990) were proposed by U.S. EPA (1996a) to develop a draft RfD and a draft Drinking Water Equivalent Level (DWEL) for kidney effects from MTBE. The increase in kidney weights at doses of 300 mg/kg/day and higher was considered to be an adverse effect, since increases in organ weights are a marker for adverse organ effects (Weil 1970). The diarrhea observed was considered to be a gastrointestinal complication of the gavage dosing. Based on the NOAEL of 100 mg/kg/day, a DWEL for kidney effects of 3,500 ppb can be derived for a 70 kg male adult with two liters (L) of daily water consumption (DWC), using an uncertainty factor of 1,000. The uncertainty factor reflects a 10 for the less-than-lifetime duration of the study, a 10 for interspecies variability, and a 10 for intraspecies variability. Using an additional uncertainty factor of 10 for potential carcinogenicity and a 20 percent default relative source contribution (RSC), U.S.EPA (1996a) drafted a lifetime Health Advisory (HA) of 70 ppb or 70 µg/L. Details of the equation and calculation of the HA are described later in the chapter on the calculation of the PHG.

## GENETIC TOXICITY

The results of genetic toxicity studies for MTBE were generally negative; however, positive results have been reported in one in vitro test system in studies that included information on mechanisms of action, and in one in vivo test system. As detailed later in this section, MTBE was not mutagenic in bacteria and tissue culture gene mutation assays, a sister chromatid exchange assay, a *Drosophila* sex-linked recessive lethal test, in vitro and in vivo chromosomal aberration assays, in vivo and in vitro unscheduled DNA synthesis assays, an in vivo DNA repair assay, an in vivo cytotoxicity assay, and in vitro and in vivo micronucleus assays. The only positive in vitro genotoxicity test was for forward mutations in the mouse lymphoma assay with exogenous activation (ARCO 1980, Mackerer et al. 1996) and Mackerer et al. (1996) suggested that HCHO was the metabolite responsible for mutagenic activity in the assay (Gamier et al. 1993). The only positive in vivo genotoxicity test was for DNA strand breaks in the rat lymphocyte comet assay (Lee et al. 1998). ATSDR (1996) indicated that MTBE has little or no genotoxic activity. However, the positive results in the mouse lymphoma and rat lymphocyte assays indicate that the genetic toxicity of MTBE needs to be investigated further.

MTBE was negative in the Ames in vitro assay for reverse mutation in *Salmonella typhimurium* strains TA1535, TA1537, TA1538, TA98, and TA100 in the absence or presence of metabolic activation (ARCO 1980, Cinelli et al. 1992, Life Science Research Roma Toxicology Centre S.P.A. 1989a). Since MTBE is volatile, a closed system was used in a recent microsuspension assay (Kado et al. 1998), and negative results were observed even though some elevated revertant values were seen with TA100 and TA 104. MTBE produced no evidence of a dose-related increase for sister chromatid exchange (ARCO 1980), for gene mutation in Chinese hamster V79 cells (Life Science Research Roma Toxicology Centre S.P.A. 1989b) and for in vitro unscheduled DNA synthesis in primary rat hepatocytes (Life Science Research Roma Toxicology Centre S.P.A. 1989c, Vergnes and Chun 1994). It was negative for micronuclei formation in erythrocytes (Vergnes and Kintigh 1993).

The only *in vitro* test system in which MTBE has tested positive is the activated mouse lymphoma forward mutation assay (ARCO 1980, Mackerer et al. 1996). TBA, one of MTBE's major metabolites, was negative in this assay (McGregor et al. 1988). MTBE was positive for forward mutations in mouse lymphoma L5178Y tk<sup>+</sup>/tk<sup>-</sup> cells in the presence, but not the absence, of metabolic activation (ARCO 1980, Stoneybrook Labs. Inc. 1993). HCHO, another one of MTBE's metabolites, is genotoxic, causing both gene mutations and chromosomal damage in the presence of exogenous metabolic activation systems. HCHO is also a known carcinogen causing nasal tumors in rodents when inhaled at high concentrations, and may also cause nasopharyngeal tumors in humans via inhalation. Work by Mackerer et al. (1996) suggested that HCHO was the MTBE metabolite responsible for mutagenic activity in the activated mouse lymphoma forward mutation assay. Additional studies from this laboratory demonstrated that the HCHO was produced from *in vitro* metabolism of MTBE in this assay system (Gamier et al. 1993).

MTBE was assessed for its *in vivo* mutagenic potential (McKee et al. 1997). It was negative in the sex-linked recessive lethal assay in *Drosophila melanogaster* (Sernau 1989). It was negative for chromosomal aberrations in Fischer 344 rats exposed via inhalation (Vergnes and Morabit 1989), in Sprague-Dawley rats (ARCO 1980) and CD-1 mice (Ward et al. 1994) exposed orally. It was negative for hypoxanthine-guanine phosphoribosyl transferase (hprt) mutant frequency increase in spleen lymphocytes of CD-1 mice exposed orally for 6 weeks (Ward et al. 1994, 1995), for micronuclei formation in bone marrow in mice exposed via inhalation (Vergnes and Kintigh 1993) or via *i.p.* injection (Kado et al. 1998), for *in vivo* DNA repair increase in cultured primary hepatocytes of CD-1 mice exposed via inhalation (Vergnes and Chun 1994) and for an *in vivo* cytotoxicity assay in rats exposed via inhalation (Vergnes and Morabit 1989).

The only *in vivo* test system in which MTBE has tested positive is the rat lymphocyte comet assay, as reported in a recent meeting abstract (Lee et al. 1998). Rats were treated with MTBE by gavage, and lymphocytes assessed for alkaline-labile strand breaks. A significant increase in DNA strand breaks was reported for the highest dose group. An increase in apoptotic comets was also observed in lymphocytes from exposed rats, but this result was not statistically significant for any one dose group.

MTBE is volatile and water-soluble. Given the technical difficulties associated with testing volatile chemicals in bacterial and cultured cell systems, it is possible that careful delivery to genetic materials may have yielded data on reasons for the relative lack of genotoxic activity of MTBE *in vitro* (Mackerer et al. 1996, Kado et al. 1998). Additionally, the *in vivo* test systems used to test MTBE were primarily chromosomal damage assays, with two exceptions being the spleen lymphocyte hprt mutation assay (Ward et al. 1994) and the *in vivo-in vitro* mouse hepatocyte unscheduled DNA synthesis assay (Vergnes and Chun 1994). Only one *in vivo* assay system, the hprt mutation assay, had the potential to detect gene mutations, and it is relatively insensitive in detecting genotoxic chemicals with known false negatives. *In vivo* genotoxicity and metabolism data is not available for a number of the organ systems such as rat kidney, testis, and spleen and bone marrow, which developed tumors in carcinogenicity bioassays.

#### DEVELOPMENTAL AND REPRODUCTIVE TOXICITY

No human studies relevant to MTBE reproductive and developmental toxicity were located. There are a limited number of animal developmental and reproductive toxicity studies, all using the inhalation route of exposure, as listed below:

- one developmental toxicity study in rats exposed to 250 to 2,500 ppm for 6 hours per day on gestation days (gd) 6 to 15 (Conaway et al. 1985, Bio/dynamics, Inc. 1984a),
- two developmental toxicity studies in mice exposed to 250 to 2,500 ppm for 6 hours per day on gestation days 6 to 15 (Conaway et al. 1985, Bio/dynamics, Inc. 1984b), or to 1,000 to 8,000 ppm for 6 hours per day on gestation days 6 to 15 (Bevan et al. 1997b, Tyl and Neeper-Bradley 1989),
- one developmental toxicity study in rabbits exposed to 1,000 to 8,000 ppm for 6 hours per day on gestation days 6 to 18 (Bevan et al. 1997b, Tyl 1989),
- one single generation reproductive toxicity study in rats exposed to 300 to 3,400 ppm (Biles et al. 1987),
- one two-generation reproductive toxicity study in rats exposed to 400 to 8,000 ppm (Bevan et al. 1997a, Neeper-Bradley 1991).

Study designs and results are outlined in Table 6. Some information on reproductive organs can also be obtained from subchronic and chronic toxicity studies (also outlined in Table 6), and there are a few recent studies of possible endocrine effects.

While no effects on fertility endpoints were reported, these studies provide evidence for adverse effects of MTBE on development. Reduced fetal weight and increased frequency of fetal skeletal variations were reported in mice after MTBE exposure during organogenesis, with a NOAEL of 1,000 ppm (Bevan et al. 1997b, Tyl and Neeper-Bradley 1989). Also, in the rat two-generation study, increased post-natal death and decreased postnatal weights were found; the NOAEL was 400 ppm MTBE (Bevan et al. 1997a). A provisional RfC of 173 ppm (48 mg/m<sup>3</sup>) has been derived using U.S. EPA risk assessment methodology (Sonawane 1994) on the basis of developmental toxicity that occurred in the two-generation rat study (Bevan et al. 1997a, Neeper-Bradley 1991). Additionally, a projected no-effect-concentration in drinking water for humans of 2.3 to 9.2 mg/L has been derived by U.S. EPA (1997a) based on a range of NOAELs (250 to 1,000 ppm) in the two developmental toxicity studies in mice. The NSTC (1997) report stated that "MTBE is not expected to pose a reproductive or developmental hazard under the intermittent, low-level exposure experienced by humans".

The developmental and reproductive toxicity studies were of good quality, and generally conformed to U.S. EPA testing guidelines. The highest inhalation concentration used (8,000 ppm) produced hypoactivity, ataxia, and reduced auditory responsiveness in adult males and females during exposure, reflecting the anesthetic properties of MTBE. Prostration, labored respiration, lacrimation, and periocular encrustation were among the clinical signs reported. There was no increase in adult male and female mortality or organ pathology at any inhalation concentration, but lower food intake and weight gain was sometimes seen at the 8,000 ppm concentration. The developmental toxicity study (Conaway et al. 1985) and single generation study (Biles et al. 1987) in rats, and one of the developmental toxicity studies in mice (Conaway et al. 1985) did not include a dose that was minimally toxic to adult males and females. Little developmental or reproductive toxicity was reported in these studies, but it is difficult to interpret this lack of findings because the concentrations were not high enough to induce adult maternal and paternal toxicity.

#### *Developmental Toxicity*

##### *Animal Developmental Toxicity Studies*

Dose-dependent effects on fetal weight and fetal skeletal variations were reported in mice; no fetal effects were reported in the rats and rabbits. Notably, the rat developmental toxicity study (Conaway et al. 1985, Bio/dynamics, Inc. 1984a) was conducted in a lower concentration range. In rabbits, maternal toxicity was reported at the highest concentration (8,000 ppm) as reduced maternal food intake, maternal weight loss, hypoactivity, and ataxia during treatment and increased relative liver weights at term. However, no fetal effects of treatment were reported in rabbits (Tyl 1989).

In mice (Bevan et al. 1997b, Tyl and Neeper-Bradley 1989), an 8,000 ppm concentration produced statistically significant lower pregnancy weight gain (approximately 30 percent lower compared to controls) as well as reduced corrected pregnancy weight gain. Food consumption of dams was lower during the exposure period only. Clinical signs of toxicity, statistically greater in incidence in the 8,000 ppm group on gestation day 6 to 15, were hypoactivity, ataxia, prostration, labored respiration, lacrimation and periocular encrustation. Group observations during daily exposures included hypoactivity, ataxia and forced respiration. Fetal toxicity endpoints at the 8,000 ppm concentration included: increased postimplantation loss, fewer live fetuses per litter, higher percent of litters with external and visceral malformations, increased incidence of cleft palate and partial atelectasis (absence of fetal lung inflation), reduced fetal body weight (21 percent), and increase in the frequency of a number of skeletal variations reflecting delayed ossification.

At the 4,000 ppm exposure, two of these fetal effects (reduced fetal body weight and delayed ossification) were also statistically significant and no maternal toxicity in the form of body weights or clinical signs of toxicity occurred. Group observations at the 4,000 ppm concentrations included hypoactivity and ataxia. The fetal body weight effects and delayed ossification were generally concentration-related at 4,000 and 8,000 ppm, with no indication of treatment related effects at 1,000 ppm, the NOAEL. The mouse developmental toxicity study (Conaway et al. 1985) reported a nonsignificant but apparently concentration-related pattern of increased fetal skeletal malformations in mice exposed to 0, 250, 1,000, or 2,500 ppm (7, 11, 16, and 22 percent affected litters), including fused ribs and sternbrae. Conaway et al. (1985) also evaluated skeletal ossification variations (Big/dynamics, Inc. 1984b), but data were not provided or discussed.



*Animal Reproductive Toxicity Studies*

As noted above, the two rat reproductive toxicity studies used longer exposures than the developmental toxicity studies, beginning prior to mating and continuing through pregnancy and lactation in the dams. Developmental toxicity in the two generation rat study included reduced pup viability and body weights in the postnatal period for both generations (Bevan et al. 1997a, Neeper-Bradley 1991). Viability, as indexed by the number of dead pups on postnatal day four, was lower than controls in the 8,000 ppm group of both the F<sub>1</sub> and F<sub>2</sub> generations; survival indices were not affected. Group difference in pup body weights was not significant on lactation day one; group differences in body weight appeared later in lactation. Pup weights were consistently lower than controls in the 8,000 ppm group after postnatal day 14 in the F<sub>1</sub> generation and after postnatal day 7 in the F<sub>2</sub> generation, and in the 3,000 ppm group after postnatal day 14 in the F<sub>2</sub> generation.

The finding of reduced pup weight gain during lactation in the absence of reduced maternal weight gain is a distinctive finding of the study. Pups were not directly exposed to MTBE during the lactation period but may have been indirectly exposed via dam's milk or MTBE condensation on the dam's fur. The postnatal effects could also have been the result of MTBE effects on maternal behavior or lactation. The findings on postnatal effects are partially supported by the earlier rat single generation study (Biles et al. 1987), which described reduced pup survival and reduced postnatal weights at exposure concentrations of 250 to 2,500 ppm. The statistical significance and dose-related characteristics of these effects varied in the single generation study (see Table 6).

*Reproductive Toxicity**Fertility and general toxicity*

The two rat reproductive toxicity studies used exposures beginning prior to mating and continuing through pregnancy and lactation in the dams. No indication of reduced fertility was reported in either study. No evaluations of ovarian cyclicity or sperm parameters were included in either study.

As mentioned above, a concentration toxic to the adult breeders was not reached in the single generation study (Biles et al. 1987), but was included in the two generation study (Bevan et al. 1997a, Neeper-Bradley 1991). Increased absolute liver weights (8,000 ppm males and females) and increased relative liver weights (3,000 and 8,000 ppm males and 8,000 ppm females) were reported in the F<sub>1</sub> generation. Liver weights of the F<sub>1</sub> generation were the only organ weights reported.

An unexplained effect was greater lactational body weight gain in the 3,000 ppm dams (F<sub>1</sub>) and 8,000 ppm dams (F<sub>0</sub> and F<sub>1</sub>) relative to controls. This was due to less maternal weight loss at the end of the lactation period, postnatal days 14 to 28. Lactational weight gain through postnatal day 14 did not differ from controls. Maternal body weight had not been reduced during gestation or at term. However, pups in the 3,000 and 8,000 ppm groups were smaller than controls at some postnatal ages (see section on developmental toxicity above) and this may have resulted in lower energy requirements for lactation.

*Reproductive organs*

Information on reproductive organs of rats from single and multi-generation studies is varied and incomplete. No effects on reproductive organ weights (testes, epididymides, seminal vesicles, prostate, ovaries) or pathology (testes, epididymides, ovaries) were reported in the rat single generation study (Biles et al. 1987). Reproductive organ weights were not obtained in the rat multi-generation study; no exposure related histopathology of reproductive organs (vagina, uterus, ovaries, epididymides, seminal vesicles, testes, prostate) was reported when 25 rats per sex per generation in the control and 8,000 ppm group were examined (Bevan et al. 1997a, Neeper-Bradley 1991).

Reproductive organ weights and pathology were sometimes reported in subchronic and chronic toxicity and oncogenicity studies in rats. No effects on weight or histopathology of gonads (ovaries and testes) were noted in 14 and 90-day gavage studies in rats (n = 10/sex/group) (Robinson et al. 1990). No effects on histopathology (testes, ovaries, prostate, uterus) were reported in a lifetime (eight weeks to natural death) gavage study in rats (n = 60/sex/group) (Belpoggi et al. 1995). Organ weights were not reported in this oncogenicity study.

*Endocrine effects*

Moser et al. (1996b, 1998) conducted studies in mice of potential antiestrogenic effects of MTBE. Endocrine modulating effects of MTBE were suggested by the rodent tumor profile of endocrine sensitive organs in oncogenicity studies. An additional suggestive finding was reduced incidence of uterine endometrial hyperplasia

in the mouse inhalation cancer bioassays (Burleigh-Flayer et al. 1991), which implies reduced estrogen action on the endometrium throughout the lifetime. Moser et al. (1996b, 1998) demonstrated a number of adverse effects of MTBE on the reproductive system of mice:

- lower relative uterine and ovarian weights compared to controls,
- increase in overall length of estrous cycle, as well as estrus and nonestrus stages,
- lower rate of cell proliferation in the uterine, cervical and vaginal epithelium,
- changes in histology of the uterus, cervix and vagina indicative of decreased estrogen action.

Body weight gain was also lower in MTBE exposed mice than in controls.

In investigating the potential mechanism of MTBE-induced reduction in estrogen action, Moser et al. (1996b) found that estrogen metabolism was increased twofold in hepatocytes isolated from mice exposed to 1,800 mg MTBE/kg/day by gavage for 3 days. This change was associated with greater liver weight and P<sub>450</sub> content. This series of experiments suggested that MTBE might lower circulating estrogen concentrations by increasing estrogen metabolism. However, later studies failed to confirm effects on serum estrogen when female mice were exposed to 8,000 ppm MTBE for 4 or 8 months (Moser et al., 1998). A further series of experiments (Moser et al. 1998) failed to find evidence that MTBE endocrine effects were mediated by the estrogen receptor by studying binding of MTBE and its metabolites to the estrogen receptor, changes in expression of estrogen receptor in MTBE exposed mice, and alterations of estrogen receptor activation and translocation in a transfection assay. The authors suggest that MTBE may exert an antiestrogenic action by a mechanism that does not involve a change in circulating estrogen or estrogen receptor binding.

The consequences of reduced estrogen action induced by MTBE in mice are not known; no fertility studies have been conducted in mice. It is also not clear whether similar effects occur in other species, at other doses, or with other exposure durations, since parallel studies have not been done. The specificity of the effect also needs to be determined. Unleaded gasoline has been found to have some antiestrogenic effects similar to MTBE (MacGregor et al. 1993, Moser et al. 1996b, Standeven et al. 1994). Also, an in vivo study reported recently in abstract form (Okahara et al. 1998) described mild estrogenic and antiestrogenic effects in pubertal mice (21 to 25 days old) gavaged with 600 or 1,500 mg MTBE/kg body weight for 5 days.

#### *Other Relevant Data*

As discussed in the section on metabolism and pharmacokinetics, MTBE is distributed to all major tissues studied in the rat. MTBE is metabolized in the liver to TBA. TBA appears to be widely distributed (Aarstad et al. 1985, Borghoff et al. 1996a, Savolainen et al. 1985). No studies specifically examining distribution of MTBE or TBA to male or female reproductive organs, or the placenta, embryo, or fetus were located in the general published literature. In view of the general widespread distribution, it is plausible that MTBE and TBA distribute to these tissues.

Several studies have examined the developmental toxicity of TBA in mice (oral) and rats (inhalation and oral). No reproductive studies of TBA were located. NTP conducted subchronic and carcinogenesis studies in mice and rats by drinking water that examined some reproductive endpoints. There is also an in vitro study of TBA and mouse sperm.

The specific studies located were:

- one developmental toxicity study in mice, oral (liquid food), 0, 0.5, 0.75, or 1 percent weight to volume, gestation days 6 to 20 (Daniel and Evans 1982),
- one developmental toxicity study in mice, oral (gavage), 0 or 780 mg/kg, twice per day, gestation days 6 to 18 (Faulkner et al. 1989),
- one developmental toxicity study in rats, inhalation, 0, 2,000, 3,500, or 5,000 ppm, 7 hours per day, gestation days 1 to 19 (Nelson et al. 1989a),
- one developmental toxicity study in rats, inhalation, 0, 6,000, 12,000 mg/m<sup>3</sup> (0, 1,660, or 3,330 ppm), 7 hours per day, gestation days 1 to 19 (abstract only) (Nelson et al. 1989b),
- one developmental toxicity study in rats, oral (liquid food), 0, 0.65, 1.3, or 10.9 percent volume to volume, gestation days 8 to 22 (abstract only) (Abel and Bilitzke 1992),
- one developmental toxicity study in rats, gastric cannula, 0, or 0.6 to 2.7 g/kg/day, postnatal day four to seven (Grant and Samson 1982),
- subchronic (13 weeks) and carcinogenesis (2 years) studies in rats and mice (both sexes), oral (water), various concentrations (NTP 1995),
- one in vitro study of mouse sperm fertilization capacity (Anderson et al. 1982).

With the exception of Nelson et al. (1989a), reporting of the data in the developmental studies was incomplete. Developmentally toxic effects were observed in mice and rats orally administered TBA, including prenatal and postnatal death (Abel and Bilitzke 1992, Faulkner et al. 1989, Daniel and Evans 1982) and postnatal developmental retardation (Daniel and Evans 1982). Malformations were not observed (Faulkner et al. 1989). The inhalation study in rats by Nelson et al. (1989a) found developmental retardation, as manifested in lower fetal weights, at concentrations of 2,000, 3,500 and 5,000 ppm TBA, and a higher percent of skeletal variations compared to controls at 3,500 and 5,000 ppm. No increases in resorptions or malformations were observed. Lower maternal weight was reported at 5,000 ppm. Maternal neurobehavioral effects associated with the exposures (narcosis at 5,000 ppm, unsteady gait at 3,500 and 5,000 ppm, unsteady at 2,000 ppm) were also observed in the Nelson et al. (1989a) study.

The NTP subchronic and carcinogenesis studies in mice and rats by drinking water used various concentrations of TBA. In these studies, systemic toxicity was observed at the high concentration, usually including death, reduced weight gain, and altered kidney weight. The studies found little indication of potential reproductive toxicity. Specifically, no effects on testis weight or sperm were observed. Minor and inconsistent effects on testis histopathology and estrous cyclicity were observed at the high concentrations. The *in vitro* study found no effect of TBA on mouse sperm fertilization capacity.

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
Rat (Sprague-Dawley), oral (gavage)	Male: No increase death, reduced body weight gain, or reduced food consumption.	Belpoggi et al. 1995
Male and female, 104 weeks, 4 days/week. 0,250, 1,000	No testicular histopathological effects. Female: No reduced body weight gain, or reduced food consumption. 250, 1,000 mg/kg/day: Increased death (dose-responsive, SS not addressed). No ovarian histopathological effects.	
Mouse (CD-1) inhalation gd 6-15, 6 hours/day.	No maternal death, or altered liver weight.	Bevan et al. 1997b Tyl and Neeper-Bradley 1989
Target concentrations: 0, 1,000, 4,000, 8,000 ppm.	8,000 ppm: Reduced maternal body weight (SS), reduced body weight gain (SS), reduced food consumption during treatment period (SS). Clinical signs (individual observations): maternal, hypoactivity (SS), ataxia (SS) prostration (SS), labored respiration (SS), lacrimation (SS), periocular encrustation (SS).	
Analytical concentrations: 0, 1,035, 4,076, 8,153 ppm.	Clinical signs (group observations during daily exposure periods): maternal hypoactivity, ataxia, labored breathing. 4,000 ppm: Clinical signs (group observations during daily exposure periods): maternal hypoactivity, ataxia. No increased pre-implant loss, early resorptions, or skeletal malformations.	

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
	<p>8,000 ppm: Increased post-implant loss (late resorptions and dead fetuses) (SS), reduced live litter size (SS), altered sex ratio (less males) (SS), increased cleft palate (SS) (resulting in increased pooled external malformations, soft tissue malformations, and total malformations (SS)), reduced fetal weight (SS), increased incidence of some skeletal variations (mainly reduced ossification) (SS).</p> <p>4,000 ppm: Reduced fetal weight (SS), increased incidence of some skeletal variations (mainly reduced ossification) (SS).</p>	
Rabbit (New Zealand White) .....	No maternal death, reduced body weight, or clinical signs of toxicity before or after daily exposure periods.	Bevan et al. 1997b, Tyl 1989
Inhalation gd 6–18, 6 hours/day ...	8,000 ppm: Reduced maternal body weight gain (gd 6–12) (SS) (resulting in reduced body weight gain gd 6–18 (SS)), reduced food consumption (gd 6–11, 13–14) (SS) (resulting in reduced food consumption gd 6–18 (SS)), increased relative liver weight (SS). Clinical signs (group observations during daily exposure periods): hypoactivity, ataxia.	
Target concentrations: 0, 1,000, 4,000, 8,000 ppm.	4,000 ppm: Reduced maternal body weight gain (gd 6–9)(SS), reduced food consumption (gd 6–8, 9–10)(SS).	
Analytical concentrations: 0, 1,021, 4,058, 8,021 ppm.	No increased pre- or post-implant loss, reduced litter size, altered sex ratio, reduced fetal weight, increased malformations, or increased skeletal variations..	
Rat (Sprague-Dawley) .....	No adult male or female deaths (F <sub>0</sub> or F <sub>1</sub> ), reduced adult female body weight (F <sub>0</sub> ), reduced adult female body weight gain (F <sub>1</sub> ), or reduced adult female food consumption (F <sub>0</sub> ).	Bevan et al. 1997a, Neeper-Bradley 1991

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
<p>Inhalation 2 generation reproductive.</p> <p>Target concentrations: 0,400, 3,000, 8,000 ppm.</p> <p>Analytical concentrations: 0, 402, 3,019, 8,007 ppm.</p> <p>Male: 6 hours/day, 10 weeks (5 days/week) + mating + gestation.</p> <p>Female: 6 hours/day, 10 weeks (5 days/week) + mating + gestation (gd 1–19) + lactation (pnd 5–28).</p>	<p>8,000 ppm: Reduced adult male body weight (F<sub>0</sub>, F<sub>1</sub>)(SS), reduced adult male body weight gain (F<sub>0</sub>: weeks 0–3, 5–7; F<sub>1</sub>: weeks 0–2, 5–6), reduced adult female body weight (F<sub>1</sub>: weeks 0–8, not gestation or lactation) (SS), reduced adult female body weight gain (F<sub>0</sub>: weeks 0–1, 5–6, not gestation or lactation) (SS), increased female body weight gain during lactation (F<sub>0</sub>, F<sub>1</sub>)(SS), increased adult male and female absolute and relative liver weights (F<sub>1</sub>)(SS), reduced adult female food consumption (F<sub>1</sub>: lactation days 7–14, not pre-breed or gestation) (SS).</p> <p>Clinical signs (individual observations): adult male, perioral wetness (F<sub>0</sub>, F<sub>1</sub>), perioral encrustation and salivation (F<sub>1</sub>); adult female, perioral wetness (F<sub>0</sub>, F<sub>1</sub>), perioral encrustation, salivation and urine stains (F<sub>1</sub>).</p> <p>Clinical signs (group observations during daily exposure periods): adult male and female, ataxia (F<sub>0</sub>, F<sub>1</sub>), hypoactivity (F<sub>0</sub>, F<sub>1</sub>), blepharospasm (F<sub>0</sub>, F<sub>1</sub>), lack of startle reflex (F<sub>0</sub>, F<sub>1</sub>).</p> <p>3,000 ppm: Increased adult male relative liver weights (F<sub>1</sub>) (SS), increased adult female body weight gain (F<sub>1</sub>: lactation) (SS). Clinical signs (group observations during daily exposure periods): adult male and female, hypoactivity (F<sub>0</sub>, F<sub>1</sub>), blepharospasm (F<sub>0</sub>, F<sub>1</sub>), lack of startle reflex (F<sub>0</sub>, F<sub>1</sub>).</p> <p>No ovarian uterine, or vaginal histopathological effects, testicular or other male reproductive organ histopathological effects, reduced mating (F<sub>0</sub>, F<sub>1</sub>), reduced fertility (F<sub>0</sub>, F<sub>1</sub>), reduced live litter size (F<sub>1</sub>, F<sub>2</sub>) reduced postnatal survival after pnd 4 (F<sub>1</sub>, F<sub>2</sub>), reduced live birth, 4-day survival, or lactation indices (F<sub>1</sub>, F<sub>2</sub>), or reduced lactation day one weight (F<sub>1</sub>, F<sub>2</sub>).</p> <p>8,000 ppm: Increased dead pups pnd 4 (F<sub>1</sub>, F<sub>2</sub>)(SS), reduced litter size at end of lactation (F<sub>2</sub>)(SS), reduced postnatal weight (F<sub>1</sub>:pnd 14–28, F<sub>2</sub>:pnd 7–28) (SS), reduced postnatal weight gain (F<sub>1</sub>:pnd 7–21, F<sub>2</sub>:pnd 1–21)(SS).</p>	

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
Exposures for F <sub>0</sub> starting at pnd 42, and F <sub>1</sub> starting on pnd 29–31. Pups not placed in inhalation chambers during lactation.	3,000 ppm: Increased dead pups pnd 4–28 (F <sub>1</sub> )(SS)(NOTR at 8,000 ppm), reduced postnatal weight (F <sub>1</sub> : pnd 4, 14, F <sub>2</sub> : obd 14–28) (SS), reduced postnatal weight gain (F <sub>1</sub> : pnd 1–4, 7–14, F <sub>2</sub> : pnd 7–21) (SS).	Biles et al. 1987, Bio/dynamics 1984c
Rat (Sprague-Dawley) Inhalation	No adult male or female death, or reduced male or female body weight (F <sub>0</sub> ).	
Reproductive: 1 generation, 2 litter.	2,500, 250 ppm: Increased incidence dilated renal pelvis in females (NOT 1,000 ppm).	
Male: 6 hours/day, 12 weeks (5 days/week), + first mating (2 weeks, daily), + 8 weeks (5 days/week), + second mating (2 weeks, daily).	No altered testes or ovary weight (F <sub>0</sub> ), adverse histopathological effects on ovaries or testes (F <sub>0</sub> ), reduced mating, reduced male fertility, reduced female fertility (pregnancy rate), reduced litter size (live or total) (F <sub>1a</sub> , F <sub>1b</sub> ), altered sex ratio (F <sub>1a</sub> , F <sub>1b</sub> ), reduced pup viability at birth (live/total)(F <sub>1a</sub> ), reduced birth weight (F <sub>1a</sub> , F <sub>1b</sub> ), reduced pup survival on pnd 4 (F <sub>1b</sub> ), or reduced pup survival on pnd 21 (F <sub>1a</sub> , F <sub>1b</sub> ).	
Female: 6 hours/day, 3 weeks (5 days/week), + first mating (daily) + first gestation (gd 0–20) + first lactation (pnd 5–21) + 2 weeks (5 days/week) + second mating (daily) + second gestation (gd 0–20) + second lactation (pnd 5–21).	2,500 ppm: Reduced pup viability at birth (live/total) (F <sub>1b</sub> ) (SS) (Note high in controls: control 99 percent, 1,000 and 2,500 ppm 95.5 percent. Authors discount biological significance), reduced postnatal weight on pnd 14, 21 (F <sub>1a</sub> , F <sub>1b</sub> ) (NOT SS).	Burleigh-Flayer et al. 1992
Target concentrations in text: 0, 250, 1,000, 2,500 ppm.	1,000 ppm: Reduced pup viability at birth (live/total) (F <sub>1b</sub> ) (SS) Note high in controls: control 99 percent, 1,000 and 2,500 ppm 95.5 percent. Authors discount biological significance), reduced pup survival from pnd 0–4 (F <sub>1a</sub> ) (NOT 2,500 ppm), reduced postnatal weight on pnd 14, 21 (F <sub>1a</sub> , F <sub>1b</sub> ) (NOT SS).	
Target concentrations in abstract: 0, 300, 1,300, 3,400 ppm.	250 ppm: Reduced pup survival from pnd 0–4 (F <sub>1a</sub> ) (NOT 2,500 ppm) (SS).	
Nominal concentrations, Male/Female: 0/0, 290/300, 1,300/1,300, 3,400/3,400 ppm.		
Analytical concentrations, Male/Female: 0/0, 290/300, 1,180/1,240, 2,860/2,980 ppm.		
Mouse (CD-1) Inhalation, Male and female, 6 hours/day, 5 days/week, 18 months 0,400, 3,000, 8,000 ppm.	Male: 8,000 ppm: Increased death (SS), reduced body weight (SS), increased liver weight (SS), blepharospasm, hypoactivity, ataxia, lack of startle reflex, prostration.	

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
Rat (Fischer 344), Inhalation ..... Male and female 6 hours/day, 5 days/week. Male: 0, 400 ppm, 104 weeks .....	3,000 ppm: Increased liver weight (SS), blepharospasm, hypoactivity, ataxia, lack of startle reflex, stereotypy. 400 ppm: Increased liver weight (SS). No alteration in testes weight, testicular (or other reproductive organ) histopathological effects. Female: No increased death ..... 8,000 ppm: Reduced body weight (SS), increased liver weight (SS), blepharospasm, hypoactivity, ataxia, lack of startle reflex, prostration. 3,000 ppm: Increased liver weight (SS), blepharospasm, hypoactivity, ataxia, lack of startle reflex, stereotypy. No ovarian (or other reproductive organ) histopathological effects. Male: No altered liver weight to 400 ppm (see note).	Chun et al. 1992
Male: 3,000 ppm, 97 weeks .....	8,000 ppm: Increased death (SS), reduced body weight (SS), (increased) nephropathy, ataxia, hypoactivity, blepharospasm, lack of startle reflex.	
Male: 8,000 ppm, 82 weeks .....	3,000 ppm: Increased death (SS), nephropathy, ataxia, hypoactivity, blepharospasm, lack of startle reflex.	
Female: 0, 400, 3,000, 8,000 ppm, 104 weeks.	400 ppm: Increased death (SS), nephropathy No altered testes weight to 400 ppm (see note).	
	8,000, 3,000, 400 ppm: Increased testicular mineralization (see note). Note: Remaining males in 8,000 and 3,000 ppm groups were sacrificed early due to high group mortality. Authors attribute mortality and mineralization of "numerous tissues" to nephropathy. No statistical evaluation of testes or other organ weight, or, apparently, histopathological changes, was performed by the authors for the 8,000 or 3,000 ppm groups.	
	Female: No increased death ..... 8,000 ppm: Reduced body weight (SS), increased liver weight (SS), ataxia, hypoactivity, blepharospasm, lack of startle reflex, nephropathy.	

Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
Rat (Sprague-Dawley), Inhalation gd 6–15, 6 hours/day.  Target concentrations: 0, 250, 1,000, 2,500 ppm.  Analytical concentrations: 0, 250, 1,000, 2,430 ppm.  Nominal concentrations: 0, 260, 1,100, 3,300 ppm.	3,000 ppm: Increased liver weight (SS), ataxia, hypoactivity, blepharospasm, lack of startle reflex, nephropathy. No ovarian (or other reproductive organ) histopathological effects. No maternal death, reduced ma- ternal body weight, altered water consumption, or altered liver weight. 2,500, 1,000, 250 ppm: Reduced maternal food consumption on gd 9–12 (SS). No increased pre- or post-implant loss, reduced live litter size, re- duced fetal weight, reduced crown-rump distance, altered sex ratio, increased malforma- tions, or increased ossification variations.	Conaway et al. 1985, Bio/dynamics, Inc. 1984a
Mouse (CD-1) Inhalation, gd 6– 15, 6 hours/day.  Target concentrations: 0, 250, 1,000, 2,500 ppm.	No maternal death, reduced ma- ternal body weight, altered food or water consumption, altered liver weight. No increased pre- or post-implant losses, reduced live litter size, reduced fetal weight, reduced crown-rump distance, altered sex ratio, increased malforma- tions.	Conaway et al 1985, Bio/dynamics, Inc. 1984b
Analytical concentrations: 0, 280, 1,110, 2,710 ppm.  Nominal concentrations: 0, 280, 1,200, 3,500 ppm.	[Fetuses with skeletal malforma- tions: control, 1.6 percent; 250 ppm, 1.7 percent; 1,000 ppm, 2.4 percent; 2,500 ppm, 3.1 per- cent (NOT SS). Litters with skeletal malformations: control, 7.4 percent; 250 ppm, 11.5 per- cent; 1,000 ppm, 16 percent; 2,500 ppm, 22.2 percent (NOT SS).].	
Rat (Sprague-Dawley), oral (ga- vage). Male and female, 14 days, 0, 357, 714, 1,071, 1,428 mg/kg/day.	Male: No increased death ..... 1,428 mg/kg/day: Reduced body weight gain (SS), anesthesia, loose stools. 1,071, 714 mg/kg/day: Reduced body weight gain (SS), loose stools. 357 mg/kg/day: Loose stools ..... No altered absolute testes weight, or testicular histopathological effects. 1,071, 714 mg/kg/day: Increased relative testes weight (NOT at 1,428 mg/kg/day) (SS). Female: No increased death .....	Robinson et al. 1990



Table 6.—MTBE: Developmental and Reproductive Toxic Effects (studies in alphabetical order by author)—Continued

Study design <sup>(1)</sup>	Reported effects <sup>(2)</sup>	Reference
Rat (Sprague-Dawley), oral (gavage). Male and female, 90 days 0, 100, 300, 900, 1,200 mg/kg/day.	1,428 mg/kg/day: Reduced body weight gain (SS), anesthesia, loose stools. 1,071 mg/kg/day: Reduced body weight gain (SS), loose stools. 714, 357 mg/kg/day: Loose stools No altered ovary weight, or ovarian histopathological effects. Male: No increased death ..... 1,200 mg/kg/day: Reduced body weight (NOT SS), increased relative liver weight (SS), increased absolute and relative kidney weight (SS), anesthesia, diarrhea. 900 mg/kg/day: Increased relative liver weight (SS), increased absolute and relative kidney weight (SS), diarrhea. 300, 100 mg/kg/day: Diarrhea ..... No altered testes weight, or testicular histopathological effects. Female: No increased death ..... 1,200 mg/kg/day: Reduced body weight (SS), anesthesia, diarrhea. 900, 300 mg/kg/day: Reduced body weight (NOT SS), diarrhea. 100 mg/kg/day: Diarrhea ..... No altered ovary weight, or ovarian histopathological effects.	Robinson et al. 1990

(1) Abbreviations: gd = gestation day, pnd = postnatal day.

(2) Effects reported by authors to be statistically significant (SS) or biologically noteworthy.

#### IMMUNOTOXICITY

Oral administration of 1,428 mg MTBE/kg/day for 14 days reduced absolute spleen weights and absolute and relative thymus weights in female rats but not in males and did not produce histopathological lesions in the spleen or thymus. Similar results were observed following 90 days treatment with an oral dose of 100 to 1,200 mg MTBE/kg/day (Robinson et al. 1990). An increased incidence of dysplastic proliferation of lymphoreticular tissues was observed in female rats gavaged with 250 or 1,000 mg MTBE/kg/day, 4 days per week for 104 weeks (Belpoggi et al. 1995). The authors discussed the possibility that these lesions had the potential to develop into the lymphomas and leukemias also observed in this study.

Administration of MTBE to Sprague-Dawley male rats by daily gavage for 28 days with 40, 400, or 800 mg MTBE/kg/day produced an overall increased percentage of apoptotic-type comets in peripheral blood lymphocytes but no dose produced a statistical increase over vehicle controls. DNA strand breakage was significantly increased in the 800 mg/kg/day group and depressed body weight gain and high corticosterone levels were observed at 28 days (Lee et al. 1998).

#### NEUROTOXICITY

Acute oral exposure in rats caused marked CNS depression at doses greater than 1,900 mg/kg, ataxia at doses greater than 2,450 mg/kg, loss of righting reflex at doses greater than 3,160 mg/kg, and tremors and labored breathing at doses greater than 4,080 mg/kg. A no observed effect level (NOEL) of 40 mg/kg for adverse but reversible neurological effects for acute oral exposure was identified (BioResearch Laboratories 1990b) and an acute oral MRL of 0.4 mg/kg/day was calculated by ATSDR (1996).

Scholl et al. (1996) measured the duration of ataxia and hypnosis in male Fischer 344 rats pretreated with P<sub>450</sub> inducers following a single sub-hypnotic (0.5 mg/kg)

and hypnotic (1.2 mg/kg) i.p. dose of MTBE. Pretreatment with phenobarbital, and to a lesser extent clofibrate but not beta-naphthoflavone, prolonged the duration of ataxia or narcosis from MTBE compared with the vehicle control. The data suggested that the biotransformation status is a major potential determinant of sensitivity to the CNS depression effects of MTBE.

Two inhalation studies indicated that MTBE might be a weak neurotoxicant in adult rats with primary effects of acute impairment. A 6-hour inhalation study and a 13-week repeated vapor inhalation study produced signs of reversible CNS depression following exposure to 8,000 ppm and, to a lesser extent, to 4,000 ppm vapor with a NOAEL of 800 ppm (Dodd and Kintigh 1989, Daughtrey et al. 1997). MTBE induced some mild and reversible CNS toxicity but did not appear to be a neurotoxicant under the conditions of these studies (Fueta et al. 1994).

#### CHRONIC TOXICITY

Sprague-Dawley rats (60 animals per sex, per dose group) were given 0, 250 or 1,000 mg MTBE/kg/day in olive oil via gavage, 4 days per week, for 104 weeks. This dosing regimen gives a 7-day time-weighted average daily dose of 0, 143, and 571 mg/kg/day. Survival appeared to be decreased in female rats after 16 weeks, but no statistical treatments on data were reported. There was no reporting of hematological, clinical chemistry or urinalysis parameters, or any indication as to whether or not these endpoints were evaluated. The authors did not observe any differences in food consumption or final body weights in the various groups. In addition, they did not report any noncancer histopathological changes (Belpoggi et al. 1995, 1997, 1998). Due to the limited scope, intermittent treatment schedule and scant data reporting on noncancer endpoints in this study, it is not possible to identify an adequate NOAEL or LOAEL.

Kidney toxicity was observed in both males and females in the 2-year inhalation study in Fischer 344 rats by Chun et al. (1992) discussed in the next section on carcinogenicity. U.S. EPA derived a RFC of three mg/m<sup>3</sup> based on the kidney and liver effects of MTBE (U.S. EPA 1993, 1997c). These data support the conclusion that, after MTBE exposure, kidney toxicity is of toxicological concern. However, the use of the Robinson et al. (1990) study for evaluation of kidney effects, as detailed in the previous section on subchronic toxicity, has two significant uncertainties. One is that the study was for 90 days and not for a lifetime, and the second is the extrapolation of dose from a single daily bolus dose in corn oil to the continuous small doses from drinking water exposure. In general, it would be anticipated that a 90-day exposure period would tend to underestimate the toxicity, while the bolus dose (a NOAEL of 100 mg/kg/day) would be more likely to overestimate the toxic response. However, the relative effects of these two factors are uncertain.

Animal studies conducted at very high levels of exposure to MTBE, i.e., at greater than 1,000 ppm, through inhalation caused increased liver, kidney, spleen, and adrenal weights; decreased brain weight, body weight, and body weight gain; swollen periocular tissue; and ataxia in rodents. Increased prostration (lying flat) or exhaustion was reported in female rodents only.

#### CARCINOGENICITY

No data on long-term effects of human exposure to MTBE relevant to cancer risk were found in recent literature searches performed by OEHHHA.

The carcinogenic activity of MTBE has been investigated in male and female Sprague-Dawley rats administered MTBE by gavage (Belpoggi et al. 1995, 1997, 1998) and in male and female Fischer 344 rats (Chun et al. 1992, Bird et al. 1997) and CD-1 mice (Burleigh-Flayer et al. 1992, Bird et al. 1997) exposed to MTBE by inhalation. In rats receiving MTBE by gavage for 24 months, statistically significant increases in Leydig interstitial cell tumors of the testes were observed in males, and statistically significant increases in lymphomas and leukemias (combined) were observed in females. An increase in the incidence of uterine sarcomas was also observed in MTBE-exposed female rats, but was not statistically significant at the  $p < 0.05$  level. In rats exposed to MTBE by inhalation for up to 24 months, statistically significant increases in the incidences of renal tubular tumors and Leydig interstitial cell tumors of the testes were observed in males. In mice exposed to MTBE by inhalation for up to 18 months, statistically significant increases in the incidences of liver tumors were observed in females (hepatocellular adenomas; hepatocellular adenomas and carcinomas combined) and males (hepatocellular carcinomas). These studies are described in more detail below.

#### *Oral Exposure Studies*

Rat gavage exposure studies: Belpoggi et al. (1995, 1997, 1998)

Groups of 60 male and 60 female 8-week old Sprague-Dawley rats were administered MTBE in olive oil by gavage at doses of 0 (oil only), 250 or 1,000 mg/kg body weight/day, 4 days per week for 104 weeks. Animals were maintained until natural death; the last animal died at 174 weeks of age. No difference in water or food consumption, or in mean body weights was observed between treated and control animals of either sex. A dose-related decrease in survival was observed in females. At 56 weeks of age, survival was approximately 98 percent, 85 percent, and 78 percent in controls, low- and high-dose females, respectively; at 88 weeks of age, survival in those same groups was approximately 76 percent, 60 percent, and 43 percent. In males, there was no difference in survival between the controls and the low-dose animals. However, after 88 weeks, survival in high-dose males exceeded that of low-dose and control males. At 104 weeks of age, survival was approximately 30 percent in low-dose and control males and 43 percent in high-dose males; at 120 weeks of age, survival in those same groups was approximately 11 percent and 32 percent.

A dose-related increase in the combined incidence of lymphomas and leukemia was observed in female rats (Table 7). The authors reported that the increase was highly significant ( $p < 0.01$ ) in the high-dose group and marginally significant in the low-dose group, when analyzed using a log-ranked test as described by Mantel (1966) and Cox (1972). When analyzed using the Fisher Exact test, the combined incidence of lymphomas and leukemia in high-dose females was significantly different from controls at the  $p = 0.001$  level. Historical control incidence rates in this laboratory for lymphomas and leukemias (combined) was  $< 10$  percent in female Sprague-Dawley rats (Belpoggi et al. 1995). The authors also noted an increase in uterine sarcomas in the low-dose females ( $5/60$  versus  $1/60$  in controls), however, this increase did not reach statistical significance ( $p = 0.1$  by Fisher's Exact test). In males, a statistically significant increased incidence of Leydig cell tumors of the testes was observed in the high-dose group (Table 7). The authors reported that this increase was significant at the  $p = 0.05$  level using a prevalence analysis for non-lethal tumors (Hoer and Walburg 1972).

Subsequent to the initial report of this study, a pathology review was undertaken (Belpoggi et al. 1998) in which slides from the original study were re-examined, and diagnostic criteria reviewed. This was undertaken by an independent panel of the Cancer Research Centre (where the study authors are based), assisted by an outside pathologist. Tumor incidences according to the review are also presented in Table 7. Both observed types of tumor were re-examined:

1. *Testicular tumors.*—Diagnosis was carried out according to criteria developed by NTP, and adenomas and hyperplasia were reported separately. In addition, adenomas were further characterized as single or multiple histiotype, and the number of multifocal adenomas in each dose group was reported. The results confirmed the diagnosis of the Leydig cell tumors as adenomas, as reported in the initial papers. According to the NTP diagnostic criteria, the incidence of Leydig cell adenomas was 3, 5, and 11 in the control, low- and high-dose groups, respectively. Hyperplasia was found in four, eight, and nine animals of the three dose groups. This compares with the originally reported incidences of 2, 2, and 11 in control, low- and high-dose animals. The latest report indicated that all four multifocal adenomas observed occurred in the high-dose group. No dose related increase of atrophy or degeneration of testicular tissue was observed, although these pathologies were reported. Thus, the tumors were not considered likely to be secondary to cell death.

2. *Lymphoid tumors.*—The cell type of origin and tumor sites were reported. All neoplasms were of lymphoid origin. Corrected incidences were 2, 7, and 12 in the control, low- and high-dose groups, respectively. For comparison, the previously reported incidence data were 2, 6, and 12 in the same groups. Cancers were classified as lymphoblastic lymphomas, lymphoblastic leukemias and lymphoimmunoblastic lymphomas. The latter category was the most prevalent, accounting for one, six, and eight of the tumors observed in the respective dose groups. The data on distribution by site indicated that most animals with lymphoid cancers were affected at multiple sites. The tissues involved in treated animals were lung, liver, spleen and lymph node, and "other", with the lung being the most commonly affected site in treated animals.

Table 7.—Tumors in Sprague-Dawley Rats Receiving MTBE by Gavage, 0, 250 or 1,000 mg/kg/day, 4 days/week for 104 Weeks (Belpoggi et al. 1995, 1997, 1998)

Tumor site and type	Dose <sup>a</sup> (mg/kg/day)		
	0	250	1,000
<i>Females:</i>			
Hemolymphoreticular tissue (including mesenteric lymph nodes)			
Lymphomas and leukemias (Belpoggi et al. 1995) .....	2/58 <sup>b</sup> (3.4%)	6/51 <sup>b</sup> (11.8%)	12/47 <sup>b,c,d,e</sup> (25.5%)
Lymphomas and leukemias of lymphoid origin (Belpoggi et al. 1998) .....	2/58 <sup>b</sup> (3.4%)	7/51 <sup>b</sup> (13.7%)	12/47 <sup>b,d,e</sup> (25.5%)
<i>Males:</i>			
<i>Testes</i>			
Leydig interstitial cell tumors (Belpoggi et al. 1995) .....	2/26 <sup>f</sup> (7.7%)	2/25 <sup>f</sup> (8.0%)	11/32 <sup>f,g,h</sup> (34.4%)
Leydig interstitial cell adenomas (Belpoggi et al. 1998) .....	3/26 <sup>f</sup> (11.5%)	5/25 <sup>f</sup> (20.0%)	11/32 <sup>f,h</sup> (34.4%)

<sup>a</sup> Administered in olive oil, 4 days per week, for 104 weeks.

<sup>b</sup> Number of lesion-bearing animals/total alive at 56 weeks of age, when the first leukemia was observed.

<sup>c</sup> Incidence relative to control group was significant ( $p < 0.01$ ) using a log-ranked test (Mantel 1966, Cox 1972), as reported by Belpoggi et al. (1995).

<sup>d</sup> Incidence relative to control group was significant by the Fisher Exact test ( $p = 0.001$ ).

<sup>e</sup> Dose-related trend was significant by the Cochran-Armitage trend test ( $p < 0.01$ ).

<sup>f</sup> Number of lesion-bearing animals/total alive at 96 weeks of age, when the first Leydig cell tumor was observed.

<sup>g</sup> Incidence relative to control group was significant at the  $p = 0.05$  level using prevalence analysis for non-lethal tumors (Hoer and Walburg 1972), as reported by Belpoggi et al. (1995).

<sup>h</sup> Incidence relative to control group was significant by the Fisher Exact test ( $p < 0.05$ ).

#### *Inhalation Exposure Studies*

Groups of 50 male and 50 female 8-week old Fischer 344 rats were exposed to 0, 400, 3,000, or 8,000 ppm MTBE vapor by inhalation (corresponding to analytical mean concentrations of 403, 3,023, or 7,977 ppm, or 1,453, 10,899, 28,760 mg/m<sup>3</sup>). The animals were exposed for 6 hours per day, 5 days per week for 24 months, except for the mid- and high-dose males, which were terminated at 97 and 82 weeks, respectively, due to a dose-dependent increased mortality rate from chronic progressive nephropathy. Low-dose males also experienced an increase in nephropathy that was associated with a slight increase in mortality and a decrease in survival. Survival times for females were not significantly different between exposed and control rats. However, there were slightly more deaths due to chronic progressive nephropathy in the mid- and high-dose females than in the low-dose and control females. Body weight gain and absolute body weight were decreased in both sexes of the high-dose group. Exposure-related increases in kidney and liver weights were reported in mid- and high-dose females, but not in males. Chun et al. (1992) concluded that the maximum tolerated dose (MTD) was exceeded in both sexes at high- and mid-dose levels, based on increased mortality. Other observed effects of MTBE exposure included anesthetic effects in rats of both sexes in the mid- and high-dose groups.

A detailed histopathology examination was performed on all animals in the control and high-dose groups, and on all animals that died or were sacrificed moribund. Only a limited histopathology examination was performed on non-moribund animals from the low- and mid-dose groups that survived to terminal sacrifice; for males, only the liver, kidneys, testes and gross lesions were evaluated, while for females, only the liver and gross lesions were examined microscopically (Bird et al. 1997). At the request of the MTBE Task Force, Experimental Pathology Laboratories, Inc. (1993) re-evaluated the histopathologic slides of kidneys from all male and female rats used in the Chun et al. (1992) study, and confirmed the study pathologist's conclusion that MTBE increased the severity of chronic progressive nephropathy in rats of both sexes. No histopathologic re-evaluation of the kidney tumors was performed.

In males, a statistically significant increase in renal tubular adenoma and carcinoma (combined) was observed in the mid-dose group (Table 8). In high-dose males renal tubular adenomas were increased, however, this increase did not reach statistical significance (Table 8). The sensitivity of the bioassay to detect a dose-related

increase in renal tumors in the high-dose group is likely to have been reduced by the high rate of early mortality, and the early termination of this treatment group at week 82. Despite the reduced sensitivity of the bioassay, a statistically significant increase in Leydig interstitial cell testicular tumors was observed in mid- and high-dose males, with a clear dose-response evident (Table 8). Historical laboratory control values for Leydig testicular tumors in Fischer rats ranged from 64 to 98 percent (Bird et al. 1997).

In female Fischer 344 rats exposed to MTBE vapor, a single rare renal tubular cell adenoma was observed in one mid-dose animal; no treatment-related increases in tumor incidence were observed (Chun et al. 1992, Bird et al. 1997). MTBE treatment of females was associated with several nonneoplastic kidney lesions, however. Both female and male rats exposed to MTBE experienced a dose-related increase in mortality from chronic progressive nephropathy. Increases in microscopic kidney changes indicative of chronic nephropathy were seen in all treated males and in mid- and high-dose females. All treated males had increases in the severity of mineralization and interstitial fibrosis of the kidney, while increases in mild to moderate glomerulosclerosis, interstitial fibrosis, and tubular proteinosis were observed in females.

Table 8.—Tumors in Male Fischer 344 Rats Receiving MTBE by Inhalation, 0, 400, 3,000, or 8,000 ppm, for up to 24 Months<sup>a</sup> (Chun et al. 1992, Bird et al. 1997)

Tumor site and type	Concentration <sup>b</sup> (ppm)			
	0	400	3,000	8,000
<i>Kidney:</i>				
renal tubular adenoma .....	1/35 <sup>c</sup>	0/32 <sup>c</sup>	5/31 <sup>c</sup>	3/20 <sup>c</sup>
renal tubular carcinoma .....	0/35 <sup>c</sup>	0/32 <sup>c</sup>	3/31 <sup>c</sup>	0/20 <sup>c</sup>
renal tubular adenoma and carcinoma (combined) .....	1/35 <sup>c</sup> (3%)	0/32 <sup>c</sup> (0%)	8/31 <sup>c,d</sup> (26%)	3/20 <sup>c</sup> (15%)
<i>Testes:</i>				
Leydig interstitial cell tumors .....	32/50 (64%)	35/50 (70%)	41/50 <sup>e</sup> (82%)	47/50 <sup>f</sup> (94%)

<sup>a</sup> Mid- and high-dose animals were terminated at 97 and 82 weeks, respectively, due to a dose-dependent increased mortality rate from chronic progressive nephropathy.

<sup>b</sup> Administered as MTBE vapor 6 hours per day, 5 days per week.

<sup>c</sup> Survival-adjusted tumor incidence rates were used to attempt to control for excess early mortality in the mid- and high-dose groups (U.S. EPA, 1995c).

<sup>d,e,f</sup> Incidence relative to control group was significant by the Fisher Exact test (<sup>d</sup>p < 0.01, <sup>e</sup>p < 0.05, <sup>f</sup>p < 0.001).

Groups of 50 male and 50 female 8-week old CD-1 mice were exposed to 0, 400, 3,000, or 8,000 ppm MTBE vapor by inhalation (corresponding to analytical mean concentrations of 402, 3,014, or 7,973 ppm or 1,442, 10,816, or 28,843 mg/m<sup>3</sup>). The animals were exposed for 6 hours per day, 5 days per week, for 18 months. Increased mortality and decreased mean survival time were observed only for male mice in the high-dose group. A slightly increased frequency of obstructive uropathy, a condition that occurs spontaneously in this mouse strain, was observed in high-dose males, however, deaths due to the condition were within the range noted for historical controls. Body weight gain and absolute body weights were decreased in high-dose males and females. Dose-dependent increases in liver weights were observed in both sexes. Kidney weights were increased in high-dose females and in low- and mid-dose males. Burleigh-Flayer et al. (1992) concluded that the MTD was exceeded in both sexes at the high-dose level. Other observed effects of MTBE exposure included anesthetic effects in mice of both sexes in the mid- and high-dose groups.

A detailed histopathology examination was performed on all animals in the control and high-dose groups, and on all animals that died or were sacrificed moribund. Only a limited histopathology examination was performed on non-moribund animals from the low- and mid-dose groups that survived to terminal sacrifice; for males, only the liver, spleen and submandibular lymph nodes were evaluated, while for females, only the liver, uterus and stomach were examined microscopically (Bird et al. 1997).

In females, a statistically significant increased incidence of hepatocellular adenomas was observed in the high-dose group (Table 9). The incidence of hepatocellular adenomas and carcinomas (combined) was also increased in high-dose females, how-

ever, only two hepatocellular carcinomas were reported, one each in the low- and high-dose groups. In males, a statistically significant increase in hepatocellular carcinomas was observed in the high-dose group (Table 9). Bird et al. (1997) noted that the combined incidence of adenomas and carcinomas in high-dose males was similar to the historical incidence for male CD-1 mice of 33 percent. However, after correcting for the number of animals alive at 49 weeks, when the first hepatocellular adenoma was observed in males, the incidence in the high-dose group was 43 percent (16/37, see Table 9), representing a clear increase above the cited historical incidence in male CD-1 mice. Burleigh-Flayer et al. (1992) concluded that the increased incidence of liver tumors in the high-dose groups (adenomas in females and carcinomas in males) could be attributed to MTBE exposure. The ability of this study to detect increases in tumor incidence was likely decreased by the shortened study length (18 versus 24 months).

Table 9.—Tumors in CD-1 Mice Receiving MTBE by Inhalation, 0, 400, 3,000 or 8,000 ppm, for up to 18 Months<sup>a</sup> (Burleigh-Flayer et al. 1992, Bird et al. 1997)

Tumor site and type	Dose <sup>b</sup> (ppm)			
	0	400	3,000	8,000
<i>Females (Liver):</i>				
hepatocellular adenoma .....	2/50	1/50	2/50	10/50 <sup>c</sup>
hepatocellular carcinoma ....	0/50	1/50	0/50	1/50
hepatocellular adenoma and carcinoma (combined) .....	2/50	2/50	2/50	11/50 <sup>d</sup>
<i>Males (Liver):</i>				
hepatocellular adenoma.				
hepatocellular carcinoma ....	11/47 <sup>e</sup>	11/47 <sup>e</sup>	9/46 <sup>e</sup>	12/37 <sup>e</sup>
hepatocellular carcinoma ....	2/42 <sup>f</sup>	4/45 <sup>f</sup>	3/41 <sup>f</sup>	8/34 <sup>e,f</sup>
hepatocellular adenoma and carcinoma (combined) .....	12/47 <sup>e</sup>	12/47 <sup>e</sup>	12/46 <sup>e</sup>	16/37 <sup>e</sup>

<sup>a</sup> Male mice in the high-dose group experienced early mortality.

<sup>b</sup> Administered as MTBE vapor 6 hours per day, 5 days per week.

<sup>c,d</sup> Incidence relative to control group was significant by the Fisher Exact test ( $p < 0.05$ ,  $p < 0.01$ ).

<sup>e</sup> Number of lesion-bearing animals per total alive at 49 weeks, when the first hepatocellular adenoma was observed.

<sup>f</sup> Number of lesion-bearing animals per total alive at 63 weeks, when the first hepatocellular carcinoma was observed.

#### Other Relevant Data

##### Structure-Activity Comparisons

MTBE and similar ethers generally undergo metabolism at the ethereal bond to form the corresponding alcohol and an aldehyde (Savolainen et al. 1985). Other structurally similar ethers include ETBE and tertiary-amyl methyl ether (TAME). No studies have been reported to date on the carcinogenicity of ETBE or TAME. Published data on the genotoxic potential of ETBE and TAME are few in number; ETBE and TAME tested negative in the Salmonella reverse mutation assay, and TAME did not induce micronuclei in mouse bone marrow cells following exposure in vivo (NSTC 1997). In a recent review of gasoline toxicity, Caprino and Togna (1998) briefly refer to an unpublished report in which TAME induced "chromosomal effects" in Chinese hamster ovary cells. MTBE is made by isobutene and methanol, or TBA and methanol. NTP has documented some evidence of carcinogenic activity for isobutene in male rats (NTP 1997), and for TBA in male rats and female mice (NTP 1995).

##### Pathology

The tumors observed by Belpoggi et al. (1995, 1997, 1998) in hemolymphoreticular tissues in the female Sprague-Dawley rat were diagnosed as lymphomas and leukemias. The re-analysis of the pathology data (Belpoggi et al. 1998) confirmed that these neoplasms were all of lymphoid origin, and further identified them as lymphoblastic lymphomas, lymphoblastic leukemias, and lymphoimmunoblastic lymphomas. IARC (IARC, 1993) classifies all three of these tumor types as malignant lymphomas. The aggregation of these tumor types for carcinogen identification and risk assessment purposes is therefore appropriate.

The testicular tumors observed in both the Sprague-Dawley (Belpoggi et al. 1995, 1997, 1998) and Fischer 344 (Chun et al. 1992, Bird et al. 1997) rat strains were diagnosed as Leydig interstitial cell tumors. The spontaneous incidence of these tumors is typically much lower in the Sprague-Dawley rat, as compared to the Fischer

344 rat (approximately 5 percent and 88 percent, respectively at 24 months) (Clegg et al. 1997). The control incidence of these tumors reported by Belpoggi et al. (1995) (i.e., 7.7 percent) is consistent with levels typically observed in the Sprague-Dawley strain. The control incidence observed by Chun et al. (1992), (i.e., 64 percent) was reported in the published study (Bird et al. 1997) as being lower than that typically observed in the Fischer 344 strain. However, this control incidence was similar to that (i.e., 64.9 percent) reported for male Fischer 344 rats in another oncogenicity study from the same laboratory (Burleigh-Flayer et al., 1997), the same as the historical control rate for male Fischer 344 rats in NTP inhalation studies (Nyska et al. 1998), and within the range (64 to 98 percent) reported for aged male rats of this strain (Bird et al. 1997, Haseman and Arnold 1990). The lower spontaneous Leydig cell tumor incidence observed in the Chun et al. (1992) study is likely to have facilitated the detection of the dose-dependent increase in Leydig cell tumors in MTBE-treated males, despite the early termination of the mid- and high-dose groups.

The tumors observed in male Fischer 344 rat kidney tissues (Chun et al. 1992, Bird et al. 1997) were diagnosed as renal tubular adenomas and carcinomas. These two tumor phenotypes are generally considered to be related in origin, with the possibility that adenomas may progress to carcinomas (Borghoff et al. 1996b). Therefore, they are normally aggregated for carcinogen identification and risk assessment purposes (U.S. EPA 1991). The possibility that the male rat-specific  $\alpha_{2u}$ -globulin nephropathy plays a significant role in the pathogenesis of MTBE rat kidney tumors has been investigated, and reported to be unlikely (NSTC 1997, U.S. EPA 1997a). The data indicate that MTBE induces only mild accumulation of  $\alpha_{2u}$ -globulin and mild or partial expression of  $\alpha_{2u}$ -globulin associated nephropathy in male rats, while clearly exacerbating the expression of non- $\alpha_{2u}$ -globulin rat nephropathy in both males and females (NSTC 1997). Support for this conclusion includes the observation that a dose-dependent increase in mortality from chronic progressive nephropathy was observed in male rats at all dose levels, and in females at the mid- and high-dose levels in the rat inhalation bioassay (Bird et al. 1997). Observed microscopic kidney changes included increases in the severity of mineralization and interstitial fibrosis in all treated males, and increases in mild to moderate glomerulosclerosis, interstitial fibrosis, and tubular proteinosis in mid- and high-dose females (Chun et al. 1992). In addition, a rare renal tubular tumor was observed in one MTBE-treated female rat (Chun et al. 1992). In a separate analysis of a 13-week inhalation exposure study of male rats conducted at the Bushy Run Research Center laboratory, Swenberg and Dietrich (1991) measured the levels of  $\alpha_{2u}$ -globulin associated with hyaline droplets in MTBE-treated and control kidney sections by immunohistochemical staining techniques. Although a slight increase in renal cortex staining for  $\alpha_{2u}$ -globulin was observed in MTBE-treated animals, as compared with controls, there was no relationship between the level of  $\alpha_{2u}$ -globulin staining and the dose of MTBE received (U.S. EPA 1997c, Swenberg and Dietrich 1991). In a study by Lington et al. (1997), inhalation of 4,000 and 8,000 ppm MTBE for 13 weeks resulted in a moderate increase in the size of hyaline droplets in male rat kidney, but no MTBE-associated increase in the area or intensity of  $\alpha_{2u}$ -globulin immunostaining was observed, as reported by Bird et al. (1997). In a 4-week inhalation study, exposure to 3,000 and 8,000 ppm MTBE increased the levels of protein accumulated in male rat kidney tubule epithelial cells, but not the levels of  $\alpha_{2u}$ -globulin, as compared with controls (Bird et al. 1997).

The tumors observed by Burleigh-Flayer et al. (1992) and Bird et al. (1997) in mouse liver were diagnosed as hepatocellular adenomas and carcinomas. These two tumor phenotypes are generally considered to be related in origin, with the possibility that adenomas may progress to carcinomas. They are normally therefore aggregated for carcinogen identification and risk assessment purposes. The sensitivity of the study to detect treatment-related tumors, especially in the low- and mid-dose groups, may have been compromised by the less-than-lifetime length of the study (18 months).

#### *Mechanism*

The mechanism(s) by which MTBE induces tumors at multiple sites in rats and mice is unknown at this time. It is unclear whether MTBE itself plays a direct role in the observed tumorigenesis, or whether metabolism to one or more active metabolites is required. The two major metabolites of MTBE, HCHO (Kerns et al. 1983, Sellakumar et al. 1985, Til et al. 1989, Woutersen et al. 1989) and TBA (NTP 1995), have both been shown to possess tumorigenic activity in animal studies. Interestingly, there is a commonality of tumor sites observed for MTBE, HCHO, and TBA. Leukemias were observed in male and female Sprague-Dawley rats administered HCHO in drinking water (Soffritti et al. 1989), and renal tubular cell adeno-

mas and carcinomas were observed in male Fischer 344 rats administered TBA in drinking water (NTP 1995, Cirvello et al. 1995). IARC (1995) concluded that the evidence on the carcinogenicity of HCHO was sufficient in animals and limited in humans, and classified the agent in Group 2A probably carcinogenic to humans. NTP (1995) in reviewing the results of 2-year drinking water studies with TBA concluded that "there was 'some' evidence of carcinogenic activity of TBA in male Fischer 344/N rats based on increased incidences of renal tubule adenoma or carcinoma (combined)".

It is presently unknown whether the nature or degree of MTBE metabolism is tissue- or sex-specific, or whether there is any relationship between the site of metabolism and target tumor sites. Comparison of the target tumor sites in rats administered MTBE by two different routes of administration is inherently limited by the use of different rat strains in these studies; however, these findings suggest that route-specific distribution and metabolism of MTBE may be of importance in the development of some (e.g., leukemias and lymphomas, renal tumors), but not all treatment-associated tumors (e.g., testicular tumors). It has also been suggested that sex-specific differences in metabolism may underlie the development of leukemias and lymphomas in female, but not male rats (Belpoggi et al. 1995, 1997, 1998). This hypothesis remains untested, however.

MTBE was negative in a number of genotoxicity assays as noted in the section on genetic toxicity in this document and by ATSDR (1996), testing positive only in the activated mouse lymphoma forward mutation assay (ARCO 1980, Mackerer et al. 1996) and the rat lymphocyte comet assay (Lee et al. 1998). The MTBE metabolite TBA was not mutagenic in either the Salmonella assay (Zeiger et al. 1987) or the mouse lymphoma assay (McGregor et al. 1988). HCHO is genotoxic, testing positive in numerous assay systems (IARC 1995). Data on HCHO-related genotoxicity in MTBE tumorigenesis are too limited to draw any conclusions at this time. Studies conducted in freshly isolated mouse hepatocytes from female CD-1 mice (Casanova and Heck 1997) did not find any dose-related increase in HCHO-associated DNA-protein cross-links or RNA-HCHO adducts following MTBE-treatment. Similar results were obtained with freshly isolated hepatocytes from male B6C3FI mice and male Fischer 344 rats (Casanova and Heck 1997). These data suggest that HCHO is not the active species responsible for MTBE liver tumorigenesis in the mouse. In studies using the mouse lymphoma assay, however, HCHO has been implicated as the active species responsible for MTBE's mutagenic activity (Gamier et al. 1993, Mackerer et al. 1996). DNA-protein cross-link data and RNA-HCHO adduct data are not available for the other tumor sites noted after MTBE exposure in laboratory animals.

Several hypotheses have been put forward suggesting that MTBE may act via a variety of nongenotoxic mechanisms, such as the involvement of endocrine modulation in mouse liver and rat testicular tumorigenesis (Bird et al. 1997, Moser et al. 1996b) and  $\alpha_{2u}$ -globulin nephropathy in male rat kidney tumorigenesis (Bird et al. 1997, Poet and Borghoff 1997a, 1997b, Prescott-Mathews et al. 1997a). While MTBE exposure of the mouse is associated with various endocrine-related tissue and cellular responses (see the section on developmental and reproductive toxicity in this document), the available data are insufficient to support an endocrine-mediated mode of action for MTBE-associated liver (Moser et al. 1996a, 1996b, Moser et al. 1998, Okahara et al. 1998) or testicular tumors (Day et al. 1998) at this time.

Data which suggest that  $\alpha_{2u}$ -globulin nephropathy may be involved in MTBE kidney tumorigenesis include the following:

- A mild to moderate increase in the number and size of hyaline droplets in the renal proximal tubule cells of MTBE-treated male rats has been observed.
- In a 10-day inhalation study, MTBE increased the number of protein droplets within the renal proximal tubules of male rats with a statistically significant concentration-related positive trend (Prescott-Mathews et al. 1997a).
- In a 14-day gavage study, MTBE increased the formation of hyaline droplets in male rat kidney proximal tubular epithelial cells at the highest dose tested (Robinson et al. 1990).
- In a 28-day inhalation study, MTBE slightly increased protein accumulation in male rat kidney tubular epithelial cells (Bird et al. 1997).
- In a 13-week inhalation study, MTBE slightly increased hyaline droplet formation in male rat kidney (Swenberg and Dietrich 1991).
- In another 13-week inhalation study, MTBE slightly increased the size of hyaline droplets in male rat kidney (Bird et al. 1997 reporting on findings of Lington et al. 1997).
- In a 90-day gavage study, MTBE slightly increased the number of hyaline droplets in male rat kidney proximal tubular epithelial cells (Robinson et al. 1990).



- Protein in the renal proximal tubule cells of MTBE-treated male rats stains weakly for  $\alpha_{2u}$ -globulin.
- In a 13-week inhalation study, MTBE slightly increased hyaline droplet formation and staining for  $\alpha_{2u}$ -globulin in male rat kidney but these increases were not dose-dependent (Swenberg and Dietrich 1991).
- In a 10-day inhalation study, no dose-related increase in  $\alpha_{2u}$ -globulin staining could be detected in MTBE-treated male rat kidney by immunohistochemical staining (Prescott-Mathews et al. 1997a).
- Using an ELISA-based method, a mild dose-dependent increase in  $\alpha_{2u}$ -globulin-immunoreactivity (approximately 150  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in controls versus 200  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in the high-dose animals) has been observed in rat kidney cytosol of male rats exposed to MTBE by inhalation for 10 days (Prescott-Mathews et al. 1997a).
  - MTBE binds weakly to  $\alpha_{2u}$ -globulin in vitro. Using a kidney homogenate system, only a very weak interaction between MTBE and male rat renal proteins was detected (Poet and Borghoff 1997a). This interaction did not survive dialysis or anion exchange chromatography (Poet and Borghoff 1997a).
  - A dose-dependent increase in cell proliferation has been observed in the renal cortex of male rats exposed to MTBE by inhalation for 10 days (Prescott-Mathews et al. 1997a).
  - Agents which are thought to induce renal tubular tumors via an  $\alpha_{2u}$ -globulin-mediated mechanism are nongenotoxic. MTBE has demonstrated little or no genotoxicity in vitro or in vivo.
- Data which argue against a significant role for  $\alpha_{2u}$ -globulin nephropathy in MTBE kidney tumorigenesis include the following:
  - Male rat specificity for nephropathy and renal tumorigenicity has not been observed.
    - In a 2-year inhalation study, MTBE exacerbated chronic progressive nephropathy and increased mortality associated with chronic progressive nephropathy in a dose-dependent manner in both in female and male rats (Chun et al. 1992, Bird et al. 1997).
    - A rare kidney tumor was observed in one MTBE-treated female rat in the 2-year inhalation study (Chun et al. 1992, Bird et al. 1997).
    - A clear exposure-related increase in staining for  $\alpha_{2u}$ -globulin, an effect typical of classical  $\alpha_{2u}$ -globulin nephropathy-inducing agents, has not been observed in male rats treated with MTBE.
      - In a 13-week inhalation study, MTBE slightly increased hyaline droplet formation and staining for  $\alpha_{2u}$ -globulin in male rat kidney but these increases were not dose-dependent (Swenberg and Dietrich 1991).
      - In another 13-week inhalation study, MTBE slightly increased the size of hyaline droplets in male rat kidney, but no increase in the area or intensity of  $\alpha_{2u}$ -globulin staining was observed (Bird et al. 1997 reporting on findings of Lington et al. 1997).
      - In a 28-day inhalation study, MTBE slightly increased protein accumulation in male rat kidney, but did not increase  $\alpha_{2u}$ -globulin immunohistochemical staining (Bird et al. 1997).
      - In a 10-day inhalation study, no dose-related increase in  $\alpha_{2u}$ -globulin staining could be detected in MTBE-treated male rat kidney by immunohistochemical staining, but using a more sensitive ELISA-based assay a mild increase in the concentration of  $\alpha_{2u}$ -globulin (approximately 150  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in controls versus 200  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in the high-dose animals) was observed (Prescott-Mathews et al. 1997a). This small increase is in contrast to the marked increase seen with classical  $\alpha_{2u}$ -globulin nephropathy-inducing agents, such as 2,2,4-trimethylpentane (approximately 200  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in controls versus 550  $\mu\text{g}\alpha_{2u}$ -globulin/mg total protein in treated animals) (Prescott-Mathews et al. 1997a).
      - $\alpha_{2u}$ -Globulin-positive proteinaceous casts, another effect typical of classical  $\alpha_{2u}$ -globulin nephropathy-inducing agents, were not seen at the junction of the proximal tubules and the thin loop of Henle in several short-term studies, including a 10-day inhalation study (Prescott-Mathews et al. 1997a), a 28-day inhalation study (Bird et al. 1997), or a 13-week inhalation study (Swenberg and Dietrich 1991, U.S. EPA 1997c). However, in a 90-day oral study a small number of granular casts were observed (Robinson et al. 1990).
        - Linear mineralization of papillary tubules, another effect typical of classical  $\alpha_{2u}$ -globulin nephropathy-inducing agents, has not been reported in rats exposed to MTBE to date.
        - To date, published reports have not detected the binding of MTBE to  $\alpha_{2u}$ -globulin or male rat renal proteins in vivo (Prescott-Mathews et al. 1997b), although

Borghoff and colleagues report indirect evidence for an in vivo association between MTBE and male rat renal proteins (Borghoff, personal communication). Only a very weak interaction between MTBE and male rat renal proteins has been detected in vitro, using a kidney homogenate system (Poet and Borghoff 1997a). This interaction did not survive dialysis or anion exchange chromatography (Poet and Borghoff 1997a), in contrast to observations with classical  $\alpha_{2u}$ -globulin nephropathy-inducing agents, where typically 20 to 40 percent of bound ligand is retained after dialysis (NSTC 1997).

The available data on renal tumorigenesis indicate that MTBE induces only mild accumulation of  $\alpha_{2u}$ -globulin and mild or partial expression of  $\alpha_{2u}$ -globulin associated nephropathy in male rats, while clearly exacerbating the expression of non- $\alpha_{2u}$ -globulin rat nephropathy in both males and females (NSTC 1997). The U.S. EPA (1991) established three criteria for causation of an  $\alpha_{2u}$ -globulin effect:

- (1) increased number and size of hyaline droplets in renal proximal tubule cells of treated male rats;
- (2) accumulating protein in the hyaline droplets is  $\alpha_{2u}$ -globulin; and
- (3) additional aspects of the pathological sequence of lesions associated with  $\alpha_{2u}$ -globulin nephropathy are present.

If the response is mild all of the typical lesions may not be observed, however, some elements consistent with the pathological sequence must be demonstrated to be present.

Evaluation of the available data indicates that the first U.S. EPA criterion has been satisfied, but not the second or third (NSTC 1997, U.S. EPA 1997a).

In late 1997, IARC held a workshop to examine, among other issues, the scientific basis for possible species differences in mechanisms by which renal tubular cell tumors may be produced in male rats (IARC 1998b). The final draft of the consensus report from this workshop outlines seven criteria which all must be met by agents causing kidney tumors through an  $\alpha_{2u}$ -globulin-associated response in male rats. These criteria are the following:

- (1) Lack of genotoxic activity (agent and/or metabolite) based on an overall evaluation of in vitro and in vivo data
- (2) Male rat specificity for nephropathy and renal tumorigenicity
- (3) Induction of the characteristic sequence of histopathological changes in short-term studies, of which protein droplet accumulation is obligatory
- (4) Identification of the protein accumulating in tubular cells as  $\alpha_{2u}$ -globulin
- (5) Reversible binding of the chemical or metabolite to  $\alpha_{2u}$ -globulin
- (6) Induction of sustained increased cell proliferation in the renal cortex
- (7) Similarities in dose-response relationship of the tumor outcome with the histopathological end-points (protein droplets,  $\alpha_{2u}$ -globulin accumulation, cell proliferation)

The data summarized above indicates that the second, fourth and seventh IARC (1998b) criteria have not been satisfied. With regard to the third criterion, the classical  $\alpha_{2u}$ -globulin-associated accumulation of granular casts has not been observed in several shorter-term studies. Similarly, linear mineralization of papillary tubules, which is also part of the characteristic sequence of histopathological changes, has not been observed. With regard to the fifth criterion, MTBE appears to reversibly bind to  $\alpha_{2u}$ -globulin only very weakly. As to the sixth criterion, there are no data available to evaluate whether MTBE induces a sustained increase in cell proliferation in the renal cortex.

Thus, based on both the U.S. EPA and IARC criteria,  $\alpha_{2u}$ -globulin nephropathy does not appear to play a significant role in MTBE kidney tumorigenesis.

#### *Summary of the Evidence*

Epidemiological studies of the carcinogenic effects of MTBE are not available. Carcinogenicity of MTBE has been observed in both sexes of the rat in a lifetime gavage study (Belpoggi et al. 1995, 1997, 1998), in male rats of a different strain in a 24-month inhalation study (Chun et al. 1992, Bird et al. 1997), and in male and female mice in an 18-month inhalation study (Burleigh-Flayer et al. 1992, Bird et al. 1997). Statistically significant increases in Leydig interstitial cell tumors of the testes were observed in two different strains of rats by two separate routes of administration. Other statistically significant increases in the rat were leukemias and lymphomas (combined) in females and renal tubular tumors in males. Statistically significant increases in hepatocellular carcinomas were observed in male mice and statistically significant increases in adenomas and combined adenomas and carcinomas were observed in female mice. MTBE has demonstrated little or no genotoxicity in vitro or in vivo. The mechanism by which MTBE induces tumors at multiple sites in animals remains unknown (NSTC 1997, Mennear 1995, 1997a, 1997b). Additional supporting evidence is provided by the carcinogenic activity of

HCHO and TBA, two primary metabolites of MTBE, which share target tumor sites in common with MTBE. Both TBA and MTBE cause renal tumors in one strain of rat, and both orally administered HCHO and MTBE were associated with lymphohematopoietic cancers in a different strain.

#### *Conclusion*

Based on the information reviewed in the preparation of this document, there is evidence for the carcinogenicity of MTBE at multiple sites in both sexes of the rat and the mouse in five of the six available studies; MTBE is a two-species, multi-strain, two-sex, two-route, and multi-site carcinogen. Positive animal carcinogenicity data for HCHO and TBA, metabolites of MTBE, provide support for this conclusion.

#### ECOTOXICITY

Concern has been raised about the effects of MTBE in water on plants, animals and ecosystems (UC 1998). Rowe et al. (1997) summarized aquatic toxicity information and water quality criteria for VOCs including MTBE being monitored in the NAWQA Program by the USGS. The species tested so far for toxic effects of MTBE have high thresholds in the ppm or mg/L range indicating that MTBE has limited acute and chronic toxicity for aquatic species (Mancini 1997, Stubblefield et al. 1997). Acute studies generated MTBE LC<sub>50</sub> values with the freshwater green algae of 184 ppm, the freshwater Ceriodaphnia fleas of 348 ppm, the freshwater Daphnia water fleas of 542 and 681 ppm, the freshwater fathead minnows of 672, 706, 929 and 979 ppm, the freshwater rainbow trout of 887 and 1237 ppm, the freshwater tadpoles of 2,500 ppm, the marine mysid shrimps of 44 and 136 ppm, the marine inland silverside of 574 ppm, the marine bleak of > 1,000 ppm, the marine copepod of > 1,000 ppm, and the marine sheepshead minnows of > 2,500 ppm.

Toxicity of MTBE to *Daphnia magna* and *Photobacterium phosphoreum* was reported (Gupta and Lin 1995). A recent laboratory toxicity study with three unicellular algae suggests that the dissolved MTBE may alter algal community composition in the natural environment (Rousch and Sommerfeld 1998). Research by the API and others on ecological hazards of MTBE exposure is continuing. Because of the large amount of MTBE usage in California, high water and lipid solubility of MTBE, and lack of information on toxic effects of long-term exposure to low doses of MTBE (e.g., reproductive impairment in plants or animals), Cal/EPA (1998) has a continuing interest in reviewing current and proposed research to fill in these data gaps.

#### TOXICOLOGICAL EFFECTS IN HUMANS

No studies were located regarding toxic effects of MTBE in humans following ingestion or skin contact. No studies were located regarding toxic effects of ingested or inhaled or skin-contacted MTBE in drinking water in humans. No studies were located regarding acute effects, subchronic effects, chronic effects, death, systemic effects including respiratory, gastrointestinal, cardiovascular, hematological, musculoskeletal, hepatic, renal, endocrine, dermal, ocular, or body weight effects, immunological or lymphoreticular effects, neurological effects, developmental or reproductive effects, genotoxic effects, or cancer in humans after oral exposure to MTBE alone (ATSDR 1996).

No epidemiological study data on long-term effects and the carcinogenic effects of human exposure to MTBE were found in an earlier search by ATSDR (1996) or more recently by OEHHA. The U.S. EPA has not classified MTBE with respect to potential human carcinogenicity based on animal studies. The NSTC (1997) report concluded that "there is sufficient evidence to indicate that MTBE is an animal carcinogen and to regard MTBE as having a human hazard potential." Nevertheless, health complaints from the public have raised the concern of Federal and State health agencies (Begley 1994, Begley and Rotman 1993, CDC 1993a, 1993b, 1993c, Drew 1995, Joseph 1995, Mehlman 1995, 1996, 1998a, 1998b, 1998c, 1998d).

No studies were located regarding death, cardiovascular effects, hematological effects, musculoskeletal effects, hepatic effects, renal effects, endocrine effects, body weight effects, developmental and reproductive effects, genotoxic effects, or cancer in humans after inhalation exposure to MTBE. No studies were located regarding death, respiratory effects, gastrointestinal effects, cardiovascular effects, hematological effects, musculoskeletal effects, hepatic effects, renal effects, endocrine effects, body weight effects, immunological or lymphoreticular effects, neurological effects, developmental and reproductive effects, genotoxic effects, or cancer in humans after dermal exposure to MTBE (ATSDR 1996).

#### *Acute Toxicity*

A recent literature review (Borak et al. 1998) summarizes the exposure to MTBE and acute human health effects including nine epidemiological studies, ten indus-

trial hygiene studies, and 12 clinical studies. No studies were located regarding acute toxic effects of ingested or skin-contacted MTBE in humans. There are very limited data on the acute toxicity of MTBE in humans through inhalation exposure. Several studies undertaken over the past four to 5 years were unable to find any correlation between reported acute health effects and MTBE exposures experienced by the general public, mainly through inhalation, from the use of MTBE in gasoline (ATSDR 1996, Balter 1997, McCoy et al. 1995, NSTC 1996, 1997, U.S. EPA 1997a). The acute effects of combustion products and atmospheric chemistry of gasoline, and of gasoline formulated with MTBE, deserve further study within the context of sensitive populations (McConnell and Taber 1998).

Ingestion of gasoline-MTBE mixtures may result in aspiration and pneumonitis. Two recent reviews by Mehlman (1998a, 1998d) reported neurotoxic, allergic, and respiratory effects in humans from water and air contaminated by MTBE in gasoline. Symptoms reported by 82 participants ingesting water containing MTBE from a spill in North Carolina for approximately 5 years include headache, anxiety, inability to concentrate, lightheadedness, ear, nose and throat irritation, skin rashes, sneezing and breathing problems, shortness of breath and bronchitis. Similar acute illnesses in petroleum workers were reported. Acute symptoms in Alaska and New Jersey were summarized and allergic symptoms from one Alaska resident were detailed.

Complaints of acute effects from exposure to oxygenates such as MTBE in gasoline, mainly via inhalation, have been received by health authorities (Fiedler et al. 1994, McCoy et al. 1995, Raabe 1993). However, the limited epidemiological studies that have been conducted to date have not demonstrated a causal association between acute effects and inhalation exposure in a relatively small population (ATSDR 1996). Three human volunteer inhalation studies did not show increased symptoms among healthy adults (Cain et al. 1996, Johanson et al. 1995, Prah et al. 1994).

In 1993, the J.B. Pierce Laboratory of Yale University (Cain et al. 1996) and U.S. EPA (Prah et al. 1994), in two separate studies, exposed individuals to clean air and air mixed with MTBE. In cases where 37 or 43 human volunteers were exposed to low levels of MTBE in air (1.39 or 1.7 ppm) for 1 hour, there was no significant increase in symptoms of eye, nasal, or pulmonary irritation when the results for periods of exposure to MTBE were compared to results from exposure to ambient air. There were also no significant effects on mood or in the results from several performance-based neurobehavioral tests. In both studies, the females ranked the general quality of the air containing MTBE lower than the control atmosphere. However, in the study by Cain et al. (1996), where the subjects were also exposed to an atmosphere containing a total of 7.1 ppm mixture of 17 VOCs that are frequent air contaminants in areas around gasoline stations, the air quality of the MTBE-containing atmosphere ranked higher than that with the VOC mixture. No increase in acute symptoms was observed in individuals exposed to MTBE at concentrations that would be encountered while refueling a car.

The studies by Hakkola (1994), Hakkola et al. (1996, 1997) and White et al. (1995) compared the effects in two groups exposed to different concentrations of MTBE from treated gasoline because of their lifestyles. The moderately exposed individuals either drove a gasoline delivery truck, worked in a gasoline station, or worked on car repairs. The minimally exposed individuals merely used a gasoline-powered vehicle to go to and from work or as part of their job. In the study by White et al. (1995), the odds ratio was 8.9 (95 percent confidence interval = 1.2 to 75.6) for the reporting of one or more symptoms when 11 individuals with blood MTBE levels of  $> 2.4 \mu\text{g/L}$  were compared with 33 individuals with lower levels. The odds ratio increased to 21 (95 percent confidence interval = 1.8 to 539) when commuters were excluded from the population studied and eight workers with blood levels of  $> 3.8 \mu\text{g/L}$  were compared to 22 individuals with lower blood MTBE levels. All individuals lived and worked in the area around Stamford, Connecticut.

In a series of studies conducted in Finland where the gasoline contains 10 percent MTBE, Hakkola (1994) first evaluated neuropsychological symptoms among 61 male tanker drivers with exposure to organic solvents at work. The differences between the exposed group and the two control groups (56 males with occasional exposure at work and 31 male with no exposure) were found not to be statistically significant. Hakkola et al. (1996) again found that there were no statistically significant differences between the signs and symptoms reported by 101 drivers of tanker trucks and 100 milk truck drivers. Blood concentrations of MTBE or its metabolites were not monitored. However, the latest Hakkola et al. (1997) study comparing symptoms and moods among 101 road tanker drivers with 100 milk delivery drivers found results different from the previous studies. The tanker drivers with long exposure to gasoline during the work week reported significantly higher changes in fatigue

scores than drivers with short exposure, and 20 percent of tanker drivers reported acute symptoms connected to MTBE exposure.

In the winter of 1992, the State of Alaska began using 15 percent MTBE in wintertime oxygenated gasoline as part of the Federal requirements to reduce emissions of CO in Fairbanks and Anchorage. There were reports of headaches, dizziness, nausea, and spaciness after refueling and/or working around oxygenated gasoline (Smith and Duffy 1995). The Centers for Disease Control (CDC), U.S. EPA, and the State of Alaska investigated these complaints but were unable to associate them with MTBE exposure. Instead, it was suggested that the increase in price of the new Federal RFG, the odor of MTBE, and the harsh climate of Alaska resulted in some of the public associating changes in fuel with the reported symptoms. The State is now using ethanol in its gasoline during the winter (Belier et al. 1992, Chandler and Middaugh 1992, CDC 1993a). Gordian et al. (1995) reported no increase in claims for respiratory illness in Anchorage or Fairbanks after introduction of MTBE in Alaska.

A study in Alaska (Moolenaar et al. 1994) compared effects and blood levels of MTBE from a time period when oxygenated fuels were in use (Phase I) to those after the oxygenated fuels use had stopped (Phase II). The subjects were volunteers who were occupationally exposed to motor vehicle exhaust or gasoline fumes. Eighteen workers participated in Phase I and 22 in Phase II. Twelve of those that participated in Phase I of the study also participated in Phase II. A questionnaire was used to gather information on signs and symptoms and blood samples were collected for measurement of MTBE at the beginning and end of a typical workday. In Phase I, the median post-shift MTBE level was higher than the pre-shift value (1.80 versus 1.15 ppb). During Phase II, the values were more comparable (0.25 versus 0.21 ppb). Median post-shift blood measurements of TBA were higher during Phase I than in Phase II (5.6 versus 3.9 ppb).

Signs and symptoms that could be associated with MTBE exposure were reported more frequently during Phase I than Phase II (Moolenaar et al. 1994). During Phase I, 50 percent or more of the participants reported headaches, eye irritations, and nose and throat irritations. Reporting of these symptoms occurred in less than 10 percent of the participants during Phase II. However, it is difficult to evaluate if psychosomatic factors and individual sensitivity had influenced these results. The volunteers may have chosen to participate because of their sensitivity to contaminants in the atmosphere. A follow-up survey of workers exposed to oxygenated fuel in Fairbanks, Alaska (Moolenaar et al. 1997) detected higher blood benzene concentrations in mechanics than drivers and other garage workers.

Milwaukee, Wisconsin began to use MTBE in its gasoline as part of the Federal RFG program in November 1994. Similar health complaints, as voiced in Alaska (Beller et al. 1992), were registered in Wisconsin. U.S. EPA, the Wisconsin Department of Health, CDC, and the University of Wisconsin investigated complaints from approximately 1,500 people. They wrote two reports (May and September 1995) and concluded that they could find no relationship between reported health effects and MTBE exposure. It was suggested that the odor of MTBE, increase in price of wintertime gasoline, and negative media coverage were responsible for the reports of health problems associated with exposure to gasoline (Anderson et al. 1995).

National Institute for Working Life in Sweden (Nihlen et al. 1998a, 1998b) assessed acute effects up to the Swedish occupational exposure limit value with both objective measurements and questionnaires. The healthy male volunteers were exposed to MTBE vapor for 2 hours at 5, 25, and 50 ppm during light physical work. In the questionnaire, only the ratings of solvent smell increased up to 50 percent of the scale as the volunteers entered the chamber and declined slowly with time. No ocular effects were observed. Nasal airway resistance blockage index increased but was not related to exposure levels. Decreased nasal volume was seen but with no dose-effect relationship. The authors concluded no or minimal acute effects of MTBE vapor upon short-term exposure at these relatively high levels.

An interview questionnaire study (Fiedler et al. 1994) was conducted, first to assess exposure and the symptomatic responses of individuals with multiple chemical sensitivities (MCS) while using gasoline products with MTBE, second to compare their responses to individuals with chronic fatigue syndrome (CFS) which can not contribute to exposure to chemicals, and third to compare with normal controls. Fourteen MCS, five CFS, and six normal control subjects of comparable age, education, gender, and ethnicity completed several structured interview and assessment sessions. It was concluded that while the sample was limited, MTBE symptoms were not uniquely associated with chemical sensitivity or with situations where MTBE was more prevalent.

Several additional major literature reviews on the acute health effects of MTBE have been conducted. Reviews from studies in Connecticut (CDC 1993b, White et

al. 1995), Montana (MCCHD 1993), New Jersey (Mohr et al. 1994), New York (CDC 1993c), Illinois and Wisconsin (Anderson et al. 1995) and the HEI (1996) could find no evidence linking acute health effects with exposure to MTBE from gasoline use. In 1993, the Environmental and Occupational Health Sciences Institute (EOHSI) surveyed New Jersey garage workers and service station attendants, some of whom were exposed to MTBE, and some of whom were not. No significant differences in the frequency of reported symptoms were observed between the two groups (Hartle 1993, Mohr et al. 1994). EOHSI is conducting a study on individuals who have reported sensitivity to MTBE and were recruited from the "Oxybuster" group in New Jersey. The Oxybuster group is a citizens' group which claims their members experience acute health effects from breathing MTBE (Joseph 1995). Those individuals will be exposed to gasoline with and without MTBE. Results are expected later in 1998.

In response to the negative publicity associated with the use of Federal winter-time oxygenated fuel, the White House OSTP through the NSTC in September 1995 directed Federal agencies to review fuel economy and engine performance issues, water quality, air quality benefits, and health effects of oxygenates in fuel with a final report issued in June 1997. NSTC (1997) concluded that with the information collected to date there was no evidence that MTBE is causing increases in acute symptoms or illnesses at concentrations experienced by the general population, but anecdotal reports of acute health symptoms among some individuals cannot yet be explained or dismissed. NSTC also recommended that greater attention should be given to the potential for increased symptom reporting among workers exposed to high concentrations of oxygenated gasoline containing MTBE. Regarding the issue of acute sensitivity to MTBE, NRC which peer-reviewed an earlier draft of the NSTC report, concluded that there was no reason to believe that some people have extreme sensitivity to MTBE. The final NSTC report concluded "an examination of possible predisposing factors might be useful to better understand the occurrence of various symptoms in the general public following exposure to MTBE-containing gasoline."

MTBE has had a limited use as a therapeutic drug for dissolving cholesterol gallbladder stones (ATSDR 1996, HSDB 1997). Perfusion of MTBE through the bile duct and gallbladder by a percutaneous transhepatic catheter under local anesthesia was once used as a medical treatment to dissolve gallstones as an alternative to surgery (Diaz et al. 1992, Edison et al. 1993, Lin et al. 1994). Leuschner et al. (1994) reported identical side effects of manual and automatic gallstone dissolution with MTBE in 228 patients. Hellstern et al. (1998) surveyed 268 European patients from one hospital comparing with 535 patients from 20 other centers and reported that method-related lethality amounted to 0 percent and 30-day-lethality to 0.4 percent. Another solvent, ethyl propionate, has been suggested to be preferable to MTBE in this investigational procedure due to intestinal mucosa damages (Hofmann et al. 1997).

Acute exposure of humans to MTBE has occurred via injection through the catheter into the gallbladder. During this procedure, some of the MTBE enters the blood stream and is distributed systemically. Side effects reported in patients treated by this procedure included nausea, vomiting, coughing, bronchitis, sleepiness, sedation, perspiration, bradycardia (slow heart beat), elevation of liver enzymes, apnea, CNS depression, and respiratory depression (Allen et al. 1985, Juliani et al. 1985, Wyngaarden 1986). A case of acute renal failure was also reported (Ponchon et al. 1988). These signs cannot be attributed totally to MTBE because of the confounding effects of anesthesia and the infusion process itself. Borak et al. (1998) reviewed 12 dissolution studies and reported that the peak MTBE blood levels averaged 40,000 g/L in one study and ranged up to 10,000 g/L in another study.

#### *Immunotoxicity*

There are very limited human studies available on the immunotoxicity of MTBE-added fuels through inhalation or MTBE-contaminated water. Duffy (1994) concluded that single day exposures to oxyfuel and its combustion products did not show an immediate effect on the immune system as measured by serum plasma interleukin six (IL-6) levels. In this study, blood samples from 22 individuals exposed to auto emissions derived from oxyfuel were analyzed for effects on the immune system by monitoring IL-6 levels at the beginning and at the end of the 8-hour workday during a 4-week period in late November and early December 1992 (Duffy 1994).

Vojdani et al. (1997b) reported the detection of MTBE antibodies in seven out of 24 gasoline station attendants (six females and 18 males ranging in age from 21 to 58 years) who were employed for more than 2 years in service stations, and none out of the 12 healthy control subjects (four females and eight males 24 to 60 years

of age). The results indicated that these IgG and IgM antibodies were produced against the methyl or tert-butyl group of MTBE. They also indicated that the immune reactions to MTBE occurred through hapten carrier reactions that could be related to airborne exposures to TBF. However, the antibody response did not correlate with claimed symptoms.

The same group (Mordechai et al. 1997, Vojdani et al. 1997a) also reported reversible but statistically significant increased rates of abnormal apoptosis (programmed cell death) and cell cycle progression in peripheral blood lymphocytes in 20 Southern California residents exposed to MTBE and benzene contaminated water as compared to ten healthy human controls. Similar observations on 80 patients were reported again by the same group (Vojdani and Brautbar, 1998). Apoptosis is an organism's way of maintaining healthy cell populations, the process can lead to the development of disease if it is unduly suppressed or stimulated (Thompson 1995). For example, cancer may be the result of a failure in the apoptotic process, in which mutant cells are allowed to proliferate freely rather than being recognized as damaged and destroyed.

#### *Neurotoxicity*

Burbacher (1993) reviewed gasoline and its constituents as neuroactive substances and recommended future studies to focus on examining the dose-response relationship between chronic low-level exposure and subtle toxic effects in CNS functions. The results from human studies of neurological effects, e.g. headache, dizziness, disorientation, fatigue, emotional distress, gastrointestinal problems, e.g. nausea or diarrhea, and symptoms of respiratory irritation in individuals exposed to MTBE vapors through MTBE-containing fuels are inconclusive (Hakkola et al. 1996, Hakkola and Saarinen 1996, Moolenaar et al. 1994, White et al. 1995). The three studies cited were different in their design and utilized slightly different parameters for monitoring effects. All studies evaluated exposure to an MTBE-gasoline mixture and not MTBE alone.

However, in the most recent study by Hakkola et al. (1997) comparing neuropsychological symptoms and moods among 101 road tanker drivers from three Finnish oil companies with 100 milk delivery drivers from two milk companies, the tanker drivers with long exposure to gasoline during the work week reported significantly higher changes in fatigue scores than drivers with short exposure, and 20 percent of tanker drivers reported acute symptoms of headache, dizziness, nausea, dyspnoea, and irritation of saliva excretion. These symptoms have been connected to MTBE exposure. The authors suggested that exposure to MTBE during the work-week could be reason for acute symptoms among the tanker drivers in this study.

### DOSE-RESPONSE ASSESSMENT

#### *Internal Dose Estimation*

Due to the lack of a clear mode of action of TBA or other MTBE metabolites in MTBE-induced carcinogenesis in experimental animals, OEHHA has necessarily had to treat the parent compound MTBE as the cause of the observed effects in animal studies for the purpose of determining dose metrics. In order to estimate internal doses of MTBE, in addition to simple continuous applied doses, a simplified PBPK model was employed. This model is based on both the Borghoff et al. (1996a) model, in that it has five compartments for MTBE and five compartments for TBA, and the Rao and Ginsberg (1997) model with its MTBE metabolic parameters and slowly perfused compartment/blood partition coefficient for TBA. The PBPK model employs compartments loosely representing "Fat, Liver, Kidneys, Muscle, and rapidly perfused tissues termed as Vessel Rich Group (VRG)". The model's fundamental structure is based on that developed by Hattis et al. (1986) for perchloroethylene and was formulated in Stellar® software (ithink® v. 3.0.6b for the Power Macintosh, High Performance Systems Inc., Hanover, New Hampshire 03755). The model units for the whole animal are moles, L, moles/L, hour, moles/hour, L/hour, and ppm in alveolar air. Simulations of up to 32 hours were run at approximately 1,000 steps per simulated hour, using the Runge-Kutta four computation method on a Power Macintosh 7100/80. The model parameters were obtained from Borghoff et al. (1996a) or Rao and Ginsberg (1997) and are listed in Table 10. In addition to simulations of the pharmacokinetic data of Miller et al. (1997) with a model 0.22 kg rat, simulations of cancer bioassay doses were conducted assuming 0.35 kg for female and 0.5 kg for male lifetime average body weights. Physiological and metabolic parameters were scaled to these body weights as described in Borghoff et al. (1996a).

Table 10.—Parameters Used in the PBPK Model Simulations for MTBE and TBA

Parameter	Female rat	Male rat	Source
Body weight (kg) .....	0.35	0.5	Estimated from Belpoggi et al. 1995
Compartment volumes (L):			
Liver .....	0.014	0.020	Borghoff et al. 1996a
Kidney .....	0.00245	0.0035	Borghoff et al. 1996a
Muscle .....	0.2625	0.375	Borghoff et al. 1996a
Fat .....	0.0245	0.035	Borghoff et al. 1996a
Vessel Rich Group (VRG) ...	0.01505	0.0215	Borghoff et al. 1996a
Flows (L/hour):			
Alveolar ventilation .....	6.4	8.32	Borghoff et al. 1996a
Cardiac output .....	6.4	8.32	Borghoff et al. 1996a
Liver .....	1.6	2.88	Borghoff et al. 1996a
Kidney .....	1.6	2.88	Borghoff et al. 1996a
Muscle .....	0.96	1.248	Borghoff et al. 1996a
Fat .....	0.576	0.7488	Borghoff et al. 1996a
VRG .....	1.664	2.1632	Borghoff et al. 1996a
Partition coefficients (MTBE):			
Blood/Air .....	11.5	11.5	Borghoff et al. 1996a
Liver/Blood .....	1.1826	1.1826	Borghoff et al. 1996a
Kidney/Blood .....	3.113	3.113	Borghoff et al. 1996a
Muscle/Blood .....	0.565	0.565	Borghoff et al. 1996a
Fat/Blood .....	10.05	10.05	Borghoff et al. 1996a
VRG/Blood .....	3.113	3.113	Borghoff et al. 1996a
Partition coefficients (TBA):			
Blood/Air .....	481–75	481–75	Borghoff et al. 1996a*
Liver/Blood .....	0.8316	0.8316	Borghoff et al. 1996a
Kidney/Blood .....	1.1289	1.1289	Borghoff et al. 1996a
Muscle/Blood .....	0.4	0.4	Rao & Ginsberg 1997
Fat/Blood .....	0.3971	0.3971	Borghoff et al. 1996a
VRG/Blood .....	1.1289	1.1289	Borghoff et al. 1996a
Metabolism (MTBE):			
Vmax <sub>1</sub> (mole/hour) .....	2.05 × 10 <sup>-6</sup>	2.66 × 10 <sup>-6</sup>	Rao & Ginsberg 1997
Vmax <sub>2</sub> (mole/hour) .....	2.27 × 10 <sup>-4</sup>	2.94 × 10 <sup>-4</sup>	Rao & Ginsberg 1997
Km <sub>1</sub> (M) .....	2.27 × 10 <sup>-6</sup>	2.27 × 10 <sup>-6</sup>	Rao & Ginsberg 1997
Km <sub>2</sub> (M) .....	1.25 × 10 <sup>-3</sup>	1.25 × 10 <sup>-3</sup>	Rao & Ginsberg 1997
Metabolism (TBA):			
Vmax (mole/hour) .....	2.46 × 10 <sup>-5</sup>	3.21 × 10 <sup>-5</sup>	Rao & Ginsberg 1997
Km (M) .....	3.79 × 10 <sup>-4</sup>	3.79 × 10 <sup>-4</sup>	Rao & Ginsberg 1997
GI absorption (hour <sup>-1</sup> ) .....	0.8	0.8	Model assumption

\*Note: see text

The PBPK model simulation results for oral exposures to MTBE are summarized in Table 11. The italic boldface values are observed experimental data from Miller et al. (1997). The simulated or predicted values for 0.215 kg, 0.35 kg female, and 0.5 kg male rats are shown in normal type. In general, better predictions were obtained for MTBE than for TBA both for maximum blood concentration and the area under the blood concentration x time curve, or AUC.

Adequate simulation of TBA blood kinetics became increasingly difficult with increased body size and lower TBA blood-air partition coefficients of 150 and 75 had to be employed to achieve stable simulations. In all cases MTBE doses were cleared within 24 hours and there was no need for multi-day simulations to estimate an average daily MTBE AUC for the bioassays. In all cases MTBE AUC was linear with applied dose for a particular body size.

Table 11.—Comparison of PBPK Predictions with Experimental Data from Oral MTBE Administrations\*

Oral dose/Body weight	MTBE mM C <sub>max</sub>	TBA mM C <sub>max</sub>	MTBE AUC mM hour	TBA AUC mM hour	Blood:Air MTBE/TBA
40 mg/kg, 0.215 kg rat ....	0.068	0.176	0.150	0.863	11.5/481
<b>Observed:</b>					
<b><i>Frat</i></b> .....	<b><i>0.127</i></b>	<b><i>0.12</i></b>	<b><i>0.142</i></b>	<b><i>0.495</i></b>	
<b><i>Mrat</i></b> .....	<b><i>0.195</i></b>	<b><i>0.135</i></b>	<b><i>0.193</i></b>	<b><i>0.526</i></b>	



Table 11.—Comparison of PBPK Predictions with Experimental Data from Oral MTBE Administrations\*—Continued

Oral dose/Body weight	MTBE mM C <sub>max</sub>	TBA mM C <sub>max</sub>	MTBE AUC mM hour	TBA AUC mM hour	Blood:Air MTBE/TBA
250 mg/kg:					
0.35 kg Frat .....	0.527	0.974	1.03	6.3	11.5/75
0.5 kg Mrat .....	0.813	1.42	2.32	10.7	
400 mg/kg:					
0.215 kg rat .....	0.801	2.26	1.88	30.7	11.5/150
<b>Observed:</b>					
<b>Frat</b> .....	<b>1.30</b>	<b>0.66</b>	<b>2.19</b>	<b>3.90</b>	
<b>Mrat</b> .....	<b>1.41</b>	<b>0.68</b>	<b>2.61</b>	<b>4.10</b>	
1,000 mg/kg:					
0.35 kg Frat .....	2.36	3.03	6.08	30.9	11.5/75
0.5 kg Mrat .....	3.81	3.26	11.9	30.6	

\*Note: Mrat = male rat; Frat = female rat, in both cases values are for assumed lifetime average body weights. Simulation values are single day results and not averaged over a week.

Table 12 gives the average daily doses based on the blood MTBE AUC values for male and female rat simulations and the linear relations for each with applied oral dose.

Table 12.—MTBE AUC-Based PBPK Doses

Nominal dose mg/kg/day	Average applied mg/kg/day	MTBE AUC females mg/kg/day	MTBE AUC males mg/kg/day
0 .....	0	0	0
250 .....	143	116.1	124.2
1,000 .....	571	576.0	575.1

Males: mg/kg/day = 26.28 + 82.36 (mM hour), r = 0.998;  
Females: mg/kg/day = 38.95 + 159.37 (mM hour), r = 0.996.

Table 13 presents similar simulation results for inhalation exposures with the observed experimental values in italic boldface. The results are similar to the oral exposures with predictions of MTBE blood concentrations and AUCs being closer to observed values than TBA predictions. On the basis of comparison of MTBE AUC values, a 3,000 ppm × 6-hour exposure appeared to be equivalent to a 1,000 mg/kg oral Savage dose to a 0.5 kg rat. As seen in the oral exposures, the MTBE AUC in mM hour varied linearly with applied dose [ppm × 6-hour/day = 145.84 + 255.17 (mM hour), r = 0.999]. Also given in the lower part of Table 13 are dose conversions from MTBE AUC to oral mg/kg/day averaged for lifetime daily intake. This conversion assumes that the same relation exists between AUC and mg/kg/day as seen above in the oral simulations. If this assumption holds, the oral equivalent male doses from the inhalation bioassay would be 0, 82.9, 618.8, and 1,848.3 mg/kg/day. The male oral doses from the Savage bioassay study would be 0, 124.2, and 575.1 mg/kg/day.

Table 13.—Comparison of MTBE PBPK Predictions with Experimental Data:  
Rat Inhalation

Inhalation dose/Body weight	MTBE mM C <sub>max</sub>	TBA mM C <sub>max</sub>	MTBE AUC mM hour	TBA AUC mM hour	Blood:Air MTBE/TBA
400 ppm × 6 hours:					
0.215 kg rat .....	0.219	1.34	1.31	15.8	11.5/350
<b>Observed 400 ppm:</b>					
<b>Mrat</b> .....	<b>0.169</b>	<b>0.535</b>	<b>0.956</b>	<b>5.45</b>	
<b>Frat</b> .....	<b>0.171</b>	<b>0.531</b>	<b>0.884</b>	<b>5.05</b>	
400 ppm × 6 hours, 0.5 kg Mrat .....	0.182	0.914	1.09	12.2	11.5/350
3,000 ppm × 6 hours, 0.5 kg Mrat .....	1.7	5.4	10.2	125est	11.5/150
8,000 ppm × 6 hours, 0.215 kg rat .....	5.65	9.83	33.9	22.6	11.5/150
<b>Observed 8,000 ppm:</b>					
<b>Mrat</b> .....	<b>6.3</b>	<b>7.2</b>	<b>33.6</b>	<b>81.0</b>	

Table 13.—Comparison of MTBE PBPK Predictions with Experimental Data:—Continued  
Rat Inhalation

Inhalation dose/Body weight	MTBE mM C <sub>max</sub>	TBA mM C <sub>max</sub>	MTBE AUC mM hour	TBA AUC mM hour	Blood:Air MTBE/TBA
<b>Frat</b> .....	<b>6.4</b>	<b>3.3</b>	<b>32.6</b>	<b>34.4</b>	
8,000 ppm × 6 hours, 0.5 kg Mrat .....	5.2	9.6	31.1	487est	11.5/150
Male rats	Nominal dose ppm × 6 hours	MTBE AUC mM hour	Dose from MTBE AUC ppm	Dose from MTBE AUC* mg/kg/day	
	400	1.09	424	82.9	
	3,000	10.2	2,749	618.8	.....
	8,000	31.1	8,082	1,848.3	

\*Note: This conversion assumes the same relation between AUC and mg/kg/day as seen in oral studies or what single oral dose would give the predicted MTBE AUC seen during the 6-hour inhalation exposures. See also Dourson and Felter (1997) for alternative route-to-route extrapolation.

Overall, the PBPK pharmacokinetic correction for delivered dose when based on MTBE blood AUC is relatively modest compared to the simple applied dose. It is presently uncertain whether other dose metrics would be superior to MTBE AUC and will probably remain so until a more definitive mode(s) of action of MTBE carcinogenesis develops.

#### NONCARCINOGENIC EFFECTS

The most sensitive noncarcinogenic effect by oral route is in the kidney based on the Robinson et al. (1990) 90-day gavage study with a NOAEL of 100 mg/kg/day. As noted above this value was used by U.S. EPA (1996a) to derive a proposed lifetime HA of 70 ppb (or 0.07 mg/L) in drinking water for MTBE. In its more recent document (U.S. EPA 1997a), U.S. EPA employed this toxicity endpoint along with two other noncancer endpoints, neurological and reproductive and developmental, as well as three cancer endpoints in a margin of exposure (MOE) analysis to develop longer-term HAs. Other states also used this toxicity endpoint to develop regulatory guidelines for MTBE as described later in this document.

#### CARCINOGENIC EFFECTS

##### *Possible Modes of Action*

There are limited data available on the mechanism of action of MTBE. It remains unknown whether biotransformation is required for expression of MTBE's carcinogenic activity. The data from several in vitro and in vivo tests indicate that MTBE lacks significant genotoxic activity and suggest that a genotoxic mode of action is unlikely. It has been proposed that MTBE's induction of renal tubular cell tumors in the male rat is the result of  $\alpha_{2u}$ -globulin nephropathy. Although some characteristic features of  $\alpha_{2u}$ -globulin nephropathy have been associated with MTBE, the absence of others leads to the overall conclusion that  $\alpha_{2u}$ -globulin nephropathy is not likely to account for the induction of kidney tumors by MTBE. Although endocrine-mediated modes of action have been suggested for MTBE's induction of testicular tumors in rats and liver tumors in mice, there are insufficient data to support these hypotheses. In summary, the data available at this time do not provide sufficient evidence in support of a specific mode of action of MTBE carcinogenicity.

##### *Estimation of Carcinogenic Potency*

According to the proposed guidelines for carcinogen risk assessment (U.S. EPA 1996f) the type of extrapolation employed for a given chemical depends on the existence of data supporting linearity or nonlinearity or a biologically based or case-specific model. When insufficient data are available supporting either approach the default is to use a linear extrapolation. MTBE seems to fit this category, since no mode of action is known (U.S. EPA 1994a, 1994c). Although the lack of genotoxicity and the nonlinearity of the carcinogenic response in some studies might be argued as supportive of a mechanism other than direct genotoxicity via covalent modification of DNA, attempts to identify positively an alternative mechanism have not so far succeeded. Dourson and Felter (1997) attempted to perform an extrapolation of the cancer potency of MTBE from inhalation route (Chun et al. 1992) to oral route.

Cancer potency or cancer potency factor (CPF) is a slope derived from a mathematical function used to extrapolate the probability of incidence of cancer from a bioassay in animals using high doses to that expected to be observed at the low doses which are likely to be found in chronic human exposure. The mathematical model, such as the LMS model, is commonly used in quantitative carcinogenic risk

assessments in which the chemical agent is assumed to be a complete carcinogen and the risk is assumed to be proportional to the dose at very low doses.  $q_1^*$  is the upper 95 percent confidence limit on the cancer potency slope calculated by the LMS model. Or another cancer slope factor (CSF) is a potency value derived from the lower 95 percent confidence limit on the 10 percent tumor dose ( $LED_{10}$ ).  $LED_{10}$  is the 95 percent lower bound on the dose that is predicted to give a 10 percent tumor incidence. The CSF equals to 10 percent dividing by  $LED_{10}$ .

Earlier guidelines for cancer risk assessment, including those formerly used by OEHHA (DHS 1985) have required the use of the LMS model to estimate an upper bound on the low-dose potency ( $q_1^*$ ). However, more recent OEHHA methodologies, and the draft proposed U.S. EPA (1996f) guidelines for carcinogen risk assessment, recommend a linear extrapolation approach based on the  $LED_{10}$ . A multistage polynomial is used to fit data in the observable range, unless some other dose-response curve is specifically indicated by the available data. Because adequate data do not exist for MTBE, the default curve-fitting approach is appropriate. Interspecies scaling for oral doses (and internal doses calculated from a single-species pharmacokinetic model) is based on  $(\text{body weight})^{3/4}$  as proposed by U.S. EPA (1996f, 1992b) instead of the  $(\text{body weight})^{2/3}$  used previously. For inhalation exposures U.S. EPA has in the past used an assumption of equivalence between different species of exposures to a given atmospheric concentration. This provides roughly similar scaling in effect, due to the way that breathing rate and related parameters affecting uptake scale with body weight. More recently PBPK modeling has been seen as a preferable approach to both dose estimation and interspecies scaling of inhalation exposures, where data are available to support this. Since pharmacokinetic data are available for MTBE in the rat, the modeling approach was feasible in this case for that species only.

Table 14 summarizes the cancer potency values derived by both the  $LED_{10}$  method and the LMS model (for comparison with earlier results) from the available statistically significant rodent cancer bioassay data sets for MTBE described earlier in the section on carcinogenicity. In all cases the Tox—Risk v.3.5 (Crump et al. 1993) program was used to fit the multistage model to the quantal data sets. The  $q_1$  cancer potencies or the 95 percent upper bound on the LMS linear slope at low dose were calculated directly by the program. CSF's are based on the  $LED_{10}$ . The CSF is  $0.1/LED_{10}$ , in units of  $(\text{mg/kg-day})^{-1}$ . For the curve fitting to estimate the  $LED_{10}$ , we have employed a  $p \geq 0.05$  criterion for the Chi-squared goodness of fit statistic of the optimized polynomial. In order to obtain an adequate fit it was necessary to exclude the data for kidney tumors in the high dose (8,000 ppm) males rats in the study by Chun et al. (1992). As can be seen from Table 14, the potency estimates for all tumors are similar whether based on the  $q_1$  or the CSF. Results in the inhalation studies (shun et al. 1992, Burleigh-Flayer et al. 1992) are effectively the same (within a factor of two) for the different sites in rats and mice, except that the potency for testicular interstitial cell tumors in male rats is about five times higher. Comparison between different routes and experiments for the rat is easiest by examining the data calculated using the pharmacokinetic model to convert the inhalation exposures to equivalent oral doses. In this case it is apparent that all the results are comparable, with the testicular interstitial cell tumors in the Chun et al. (1992) males again showing a slightly higher value than those found at other sites or in the testis in the Belpoggi et al. (1995, 1997, 1998) oral study.

Table 14.—Dose Response Parameters for MTBE Carcinogenicity Studies

Species	Sex	Tumor site and type	$q_1^*$ (ppm <sup>-1</sup> )	$LED_{10}$ (ppm)	CSF (ppm <sup>-1</sup> )
(a) Inhalation studies—ppm in air as dose metric					
Mouse .....	Female	hepatocellular adenoma + carcinoma.	$3.2 \times 10^{-4}$	320	$3.2 \times 10^{-4}$
	Male .....	hepatocellular adenoma + carcinoma.	$7.3 \times 10^{-4}$	140	$7.0 \times 10^{-4}$
Rat .....	Male .....	renal tubular cell adenoma + carcinoma testicular interstitial cell tumors.	$4.4 \times 10^{-4}$	240	$4.2 \times 10^{-4}$
			$2.3 \times 10^{-3}$	46	$2.2 \times 10^{-3}$

Assumed:

Data reassessment by U.S. EPA (1994c, 1995c).

Duration correction based on  $(t_e/t_1)^2$ :  $t_1 = 104$  weeks for both rats and mice.

Interspecies correction: ppm equivalency.

Study	Sex	Tumor site and type	$q_1^*$ (mg/kg-day) <sup>-1</sup>	LED <sub>10</sub> mg/kg/day	CSF (mg/kg-day) <sup>-1</sup>
(b) Rat oral study—Administered dose as dose metric					
Belpoggi, et al., 1995, 1998	Male .....	Leydig cell tumors:			
		Original 1995 report	$1.38 \times 10^{-3}$	76	$1.38 \times 10^{-3}$
	Female .....	Leukemia/lymphoma:			
		Original 1995 report	$2.13 \times 10^{-3}$	49	$2.03 \times 10^{-3}$
		Revised 1998 data ...	$1.63 \times 10^{-3}$	64	$1.55 \times 10^{-3}$
		Revised 1998 data ...	$2.20 \times 10^{-3}$	48	$2.09 \times 10^{-3}$
Assumed: No duration correction: $t_c = t_1$ . Interspecies correction: $BW^{3/4}$ .					

Route	Sex	Tumor site and type	$q_1^*$ (mM.hour/day) <sup>-1</sup>	LED <sub>10</sub> mM.hour/day	CSF (mM.hour/day) <sup>-1</sup>
(c) Rat oral and inhalation studies—AUC as dose metric					
Inhalation (Chun et al. 1992).	Male .....	renal tubular cell adenoma + carcinoma.	0.037	2.9	0.035
	Male .....	testicular interstitial cell tumors.	0.16	0.66	0.15
Gavage (Belpoggi et al 1995, 1998)	Male .....	Leydig cell tumors:			
		Original 1995 report	0.044	2.4	0.041
	Female .....	Leukemia/lymphoma:			
		Original 1995 report	0.051	2.1	0.048
		Revised 1998 data ..	0.044	2.4	0.041
		Revised 1998 data ..	0.051	2.1	0.048
Assumed: Data reassessment by U.S. EPA (1994c, 1995c) for Chun et al. (1992) study. Duration correction based on $(t_c/t_1)^3$ : $T_1 = 104$ weeks for rats. Interspecies correction: AUC equivalency.					

Route	Sex	Tumor site and type	$q_1^*$ (mg/kg-day) <sup>-1</sup>	LED <sub>10</sub> mg/kg/day	CSF (mg/kg-day) <sup>-1</sup>
(d) Rat oral study—Equivalent oral dose as dose metric					
Inhalation (Chun et al. 1992).	Male .....	renal tubular cell adenoma + carcinoma.	$1.9 \times 10^{-3}$	55	$1.8 \times 10^{-3}$
	Male .....	testicular interstitial cell tumors.	$9.2 \times 10^{-3}$	11	$8.7 \times 10^{-3}$
Gavage (Belpoggi et al ..... 1995, 1998)	Male .....	Leydig cell tumors:			
		Original 1995 report	$1.38 \times 10^{-3}$	76	$1.38 \times 10^{-3}$
	Female .....	Leukemia/lymphoma:			
		Original 1995 report	$2.13 \times 10^{-3}$	49	$2.03 \times 10^{-3}$
		Revised 1998 data ...	$1.63 \times 10^{-3}$	64	$1.55 \times 10^{-3}$
		Revised 1998 data ...	$2.20 \times 10^{-3}$	48	$2.09 \times 10^{-3}$
Assumed: Data reassessment by U.S. EPA (1994c, 1995c) for Chun et al. (1992) study. Duration correction based on $(t_c/t_1)^3$ : $T_1 = 104$ weeks for rats. Interspecies correction: $BW^{3/4}$ .					

Species	Route	Sex	Body weight	Study duration	Lifetime assumed	Dosing schedule	Concentrations	Study
(e) Oral and inhalation studies—Study design								
Rat .....	Inhalation	Male .....	500g ...	97 weeks	104 weeks	6 hour/day, 5 day/week.	0, 400, 3,000, 8,000* ppm.	Chun et al. 1992
Mouse ...	Inhalation	Male .....	35g .....	68 weeks	104 weeks	6 hour/day, 5 day/week.	0, 400, 3,000, 8,000* ppm.	Chun et al. 1992
		Female ..	30g .....	68 weeks	104 weeks	6 hour/day, 5 day/week.	0, 400, 3,000, 8,000* ppm.	
Rat .....	Gavage ..	Male .....	500g ...	lifetime ..	104 weeks	4 day/week .....	0, 250, 1,000 mg/kg, day.	Belpoggi et al. 1995
		Female ..	350g ...	lifetime ..	104 weeks	4 day/week .....	0, 250, 1,000 mg/kg/day.	

\*8,000 ppm dose group not used in analysis of male rat renal tubule tumors due to inability of multistage polynomial to achieve adequate

Carcinogen risk assessment guidelines used by OEHHA normally recommend selection of human cancer potency estimates based on the most sensitive site and species, unless there is evidence to indicate that the most sensitive site(s) are not relevant to human cancer induction, or represent data sets with unusually wide error bounds. As an alternative, where several equally plausible results are available and are sufficiently close to be regarded as concordant, the geometric mean of all such estimates may be used.

The pharmacokinetic model, that allows comparison of different routes and corrects for nonlinearities in the relationship between applied and internal dose, is not available for the mouse. Therefore, the potency estimates obtained in the rat are preferred for risk assessment purposes. Because the results in rats and mice are comparable, the use of the rat data is consistent with the policy of selecting appropriately sensitive species as the basis for the estimate of potency in humans.

In terms of the relevance to human cancer and the mechanism of the observed effects, the results of the studies by Chun et al. (1992) and Burleigh-Flayer et al. (1992) are limited by the relatively severe mortality seen in the highest dose groups, and the less-than lifetime exposure given the mice and the male rats. These experimental flaws are not so severe as to exclude the use of the data in risk assessment, nor more prohibitive than the experimental flaws associated with many studies on other compounds that have been successfully used for this purpose. There are, however, additional problems in the case of the testicular interstitial cell tumors observed in male rats by Chun et al. (1992). The study authors stated that the control incidence of these tumors was lower than the historical incidence observed in animals from the colony from which these experimental animals were obtained. In view of this, the slightly divergent value for the potency estimate obtained with this data set is regarded with lower confidence than the other values obtained in this analysis.

An attempt was made to allow for the severe impact of mortality on the male rat kidney adenoma and carcinoma incidence in the study by Chun et al. (1992) by applying the time-dependent version of the LMS model to the individual time-to-tumor incidence data in this study. A suitable model available in the Tox—Risk program (multistage in dose, Weibull in time) was used, and an adequate fit was obtained. The program provided an estimate of  $q_1^* = 7.6 \times 10^{-2}$ , (mg/kg-day)<sup>-1</sup>, which is substantially higher than the value estimated from the quantal data. The calculated end-of-life LED<sub>10</sub> indicated a CSF of  $7.2 \times 10^{-2}$  (mg/kg-day)<sup>1</sup>. However, the fit obtained involved a large Weibull exponent ( $z = 8.7$ , whereas more usual values are in the range of three to six), implying a very late appearance of this tumor. This observation may be of interest in addressing the unsolved question of the mechanism of induction of this tumor by MTBE. However it implies a marked reduction in the confidence which can be placed in the potency estimate using this model. Few tumor data were obtained during the final third of the expected lifetime of the exposed rats (due to the early death of all the rats dosed with 8,000 ppm, and most of the rats dosed with 3,000 ppm by this time). The potency estimate therefore involves a substantial extrapolation outside the range of the observed data, even using the LED<sub>10</sub>/CSF methodology that is designed to avoid such problems. The extreme time dependency, deficiency in genotoxicity data, and other uncertainties described previously also raise the question of how appropriate it is to use this particular model to fit these data. Its use for extrapolation outside the range of observed data

(as opposed to merely as a curve-fitting device within the range of observed data) implies an acceptance of the classic Armitage-Doll theory of action for genotoxic carcinogens, which may not be warranted in the case of MTBE. Because the mechanistic information and the technical resources which would be required to undertake a more appropriate analysis of these time-to-tumor data are lacking, it was decided not to include the results of the time-dependent analysis in the final risk estimate.

In view of the closeness of the other values obtained in the rat, and their similar confidence levels, the preferred value for the cancer potency is therefore the geometric mean of the potency estimates obtained for the male rat kidney adenomas and carcinomas combined ( $1.8 \times 10^{-3}$ ) (Chun et al. 1992), and the male rat Leydig interstitial cell tumors ( $1.55 \times 10^{-3}$ ) and the leukemia and lymphomas in female rats ( $2.09 \times 10^{-3}$ ) (Belpoggi et al. 1995, 1998). The combined use of these data yields an estimated CSF of  $1.8 \times 10^{-3}$  (mg/kg-day). While it is theoretically possible that the true human CSF could exceed this value, that is considered unlikely. On the other hand it is plausible that the lower bound on the human CSF includes zero. This is a result of statistical uncertainty with a zero lower bound estimate on  $q_1$ , by the LMS method with some MTBE data sets and biological uncertainties due to interspecies extrapolation and mode of action.

A unit risk value is similarly derived from the geometric mean of the respective LED<sub>10</sub> values for the blood MTBE AUC (Table 14c) as follows:

(a) the geometric mean of  $2.1 \text{ mM} \times \text{hour}$  is converted to external concentration (in ppm) using the regression expression derived above i.e.,  $145.84 + 225.17(2.1) = 618.7 = 619 \text{ ppm}$ ;

(b) this value is converted to  $\text{mg}/\text{m}^3$  using the  $3.6 \text{ mg}/\text{m}^3/\text{ppm}$  conversion factor, or  $619 \text{ ppm} \times 3.6 \text{ mg}/\text{m}^3/\text{ppm} = 2,230 \text{ mg}/\text{m}^3$ ,

(c) the unit risk is calculated as  $0.1/2230 \text{ mg}/\text{m}^3$  or  $4.5 \times 10^{-5} (\text{mg}/\text{m}^3)^{-1}$  or  $4.5 \times 10^{-8} (\mu\text{g}/\text{m}^3)^{-1}$ .

Since the LED values were in human equivalent doses no additional interspecies scaling is required. This unit risk would indicate negligible theoretical lifetime cancer risk at ambient MTBE air concentrations below about 6.2 ppbv (ppb by volume).

#### CALCULATION OF PHG

Calculations of public health-protective concentrations of chemical contaminants in drinking water associated with negligible risks for carcinogens or noncarcinogens must take into account the toxicity of the chemical itself, as well as the potential exposure of individuals using the water. Tap water is used directly as drinking water, for preparing foods and beverages. It is also used for bathing or showering, and in washing, flushing toilets, and other household uses resulting in potential dermal and inhalation exposures.

#### *Noncarcinogenic Effects*

Calculation of a public health-protective concentration (C, in mg/L) for MTBE in drinking water for noncarcinogenic endpoints uses the following general equation adopted by U.S. EPA (1990, 1992a, 1996c):  $C = \text{NOAEL}/\text{LOAEL} \times \text{BW} \times \text{RSC}/\text{UF} \times \text{DWC}$

where:

NOAEL/LOAEL = no observable adverse effect level or lowest observed adverse effect level.

BW = body weight (a default of 70 kg for a male or 60 kg for a female adult).

RSC = relative source contribution (a default of 20 percent to 80 percent as explained below).

UF = Uncertainty factors (UFs) are included to account for gaps in our knowledge (uncertainty) about the toxicity of chemicals and for recognized variability in human responses to toxic chemicals.

In determining UFs for chronic effects it is conventional to apply an UF where data are only available from short- or medium-term exposures of animals, rather than full lifetime exposures. In the case of MTBE noncarcinogenic effects, there is no adequate chronic study in experimental animals of the critical effect (increase in kidney weight in rats): the key study is of 90 days duration or about 10 percent the life span of a rat. Because of this, we consider that a 10-fold UF is justified.

For interspecies extrapolation of toxic effects seen in experimental animals to what might occur in exposed humans an UF of up to 10-fold is generally recommended. This is usually considered as consisting of two parts: one that accounts for metabolic or pharmacokinetic differences between the species; and another that addresses pharmacodynamic differences, i.e. differences between

the response of human and animal tissues to the chemical exposure. Based on the limited metabolic studies of MTBE in humans that indicate possible differences from metabolism in rodents, and unresolved questions of its toxic potential for neurological, immunological and endocrine effects we believe a 10-fold UF for interspecies differences is appropriate.

Exposed humans are known to vary considerably in their response to toxic chemical and drug exposures due to age, disease states, and genetic makeup, particularly in genetic polymorphisms for enzymes (isozymes) for detoxifying chemicals. While little is known about individual variation of MTBE metabolism and toxicity the use of a 10-fold UF seems prudent considering the widespread use of tap water in the population.

Finally an additional 10-fold UF is used to account for possible carcinogenicity. This follows an U.S. EPA policy applied to their Group C contaminants. OEHHA has previously employed this additional UF for other PHGs in situations where either a nonlinear dose response was applied to a carcinogen or where both linear and nonlinear approaches were used.

DWC = daily water consumption rate (a default of two L/day for an adult has been used by the U.S. EPA (1996b), or L equivalent/day (Leq/day) to account for additional inhalation and dermal exposures from household use of drinking water as explained below).

Based on the NOAEL of 100 mg/kg/day of the most sensitive noncarcinogenic effect in the kidney from the 90-day gavage (Robinson et al. 1990) study, the following calculation can be made:

$$C = 100 \text{ mg/kg/day} \times 70 \text{ kg} \times 0.2 = 0.0467 \text{ mg/L} = 47 \text{ ppb (rounded)} / 10 \times 1,000 \times 3 \text{ Leq/day}$$

In this calculation an additional UF of 10 is employed to account for potential carcinogenicity and a DWC value of three Leq/day is used to account for inhalation exposures via typical household use as well as ingestion of tap water. The RSC addresses other non-drinking-water sources, principally airborne MTBE from vehicular exhaust. Support for these values is presented below in a discussion of exposure factors.

#### *Exposure Factors*

The U.S. EPA (1994b) estimated scenarios of potential human exposure to MTBE related to RFG. In terms of the equation for calculating the public health-protective concentrations of chemical contaminants in drinking water as shown above, the first exposure factor to be considered is the RSC (OEHHA 1996, U.S. EPA 1994b). The RSC is a factor that is based on an estimate of the contribution of drinking water exposure relative to other sources such as food, air, etc. While food is often a significant source of chronic chemical exposure, in the case of MTBE, airborne exposures are likely to be most significant, if highly variable. U.S. EPA typically uses 20 percent as the default RSC. Maine Department of Human Services used 10 percent RSC for their proposed MCL for MTBE of 35 ppb (Smith and Kemp 1998) based on the same renal toxicity (Robinson et al. 1990) NOAEL in the 90-day oral study.

Estimates for combined population's airborne exposures and occupational sub-populations' exposures vary by three orders of magnitude or more and include few California data sets. Some of these estimates are collected in Table 15 where RSC values are calculated for a range of drinking water concentrations. The analyses of Brown (1997) include a combined population grand average of 0.00185 mg/kg/day for various activity associated airborne exposures and an average ambient water concentration of 0.36 ppb. The NSTC (1997) report gives MTBE concentrations in groundwater and surface water ranging from 0.2 to 8.7 ppb with a median value of 1.5 ppb, presumably resulting from nonpoint sources. Although the air exposure analysis of Brown (1997) is the most comprehensive it may underestimate MTBE exposures to the general public in local areas in California (e.g., the Los Angeles basin), possibly by a factor of two. Also due to the year-round and universal use of MTBE in California gasoline, commuters, other drivers, gasoline station customers and neighbors, and the general public are likely to receive greater exposures than elsewhere in the U.S. For this reason a health-protective value of 0.2 (or 20 percent), equal to the default value used by U.S. EPA (1994a, 1994b, 1996a), is used here for the RSC.

The other exposure factor in the equation to calculate the public health-protective concentrations of chemical contaminants in drinking water as shown above is DWC, the daily water intake in Leq/day. DWC represents the amount of tap water consumed as drinking water as well as that mixed with beverages and used in cooking. The default for an adult is two L/day. For children a default value of one Leq/day is used. For VOCs, additional exposures occur via the inhalation and dermal

routes (i.e., multi-route) during and after showering, bathing, flushing of toilets, washing clothes and dishes, and other domestic uses (OEHHA 1996, U.S. EPA 1994b).

Estimates of inhalation and dermal exposure of MTBE relative to ingestion exposure vary from 15 percent at 0.36 ppb in water (Brown 1997) to 45 percent to 110 percent at 70 ppb in water based on predictions of the CalTox™ Model (DTSC 1994) assuming only 50 percent of inhaled MTBE is absorbed. Nihlen et al. (1998a) observed a respiratory uptake of 42 percent to 49 percent in human subjects exposed to MTBE for 2 hours at 5, 25, and 50 ppm. A value of 50 percent inhalation absorption seems supported by actual human data. Based on this assumption and a range of values for Henry's Law constant, the estimated total MTBE intake ranges from 2.5 Leq/day to 4 Leq/day as shown in Table 16. For this analysis, OEHHA scientists concluded that one liter of additional exposure would incorporate the expected exposure to MTBE volatilized from water and inhaled. Therefore, three Leq/day for total MTBE exposure would appear to be a reasonable estimate for the purpose of calculating the PHG. The Henry's Law constant for MTBE is about  $6 \times 10^{-4}$  atm-m<sup>3</sup>/mole at 25 °C which is approximately one quarter (1/4) that of benzene and one fourteenth (1/14) that of perchloroethylene, the two common VOCs that have been studied previously (Robbins et al. 1993). MTBE is less volatile and its solubility in water is significantly higher than these VOCs. Accordingly, the correction for showering and other activities for assumed daily water consumption for MTBE is smaller than these other common VOCs. This is consistent with the conclusions of Johnson (1998) as documented in the UC (1998) MTBE report.

Table 15.—Relative Source Contribution (RSC) Estimates (Percent) for Different Combinations of Air and Drinking Water Exposures to MTBE\*

Air exposure estimate (mg/kg/day)	Air exposure scenario	RSC (In Percent)				Reference
		0.36 ppb*	2 ppb*	12 ppb*	70 ppb*	
0.00185	Combined U.S. population grand average.	0.6	3	16	52	Brown 1997
0.01	One million exposed U.S. nationwide.	0.1	0.6	3.3	17	Brown 1997
0.002	Los Angeles basin at 4 ppbv ambient.	0.5	2.8	15	50	ARB 1996
0.0093	Scenario I annual	0.1	0.6	3.6	18	NSTC 1996
0.0182	Scenario II annual	0.06	0.3	1.8	10	NSTC 1996
$6.7 \times 10^{-5}$	Milwaukee, Wisconsin Air	13	46	84	97	HEI 1996
0.37	MTBE distribution of fuel mixture Time-Weighted-Average (TWA) for workers.	0.003	0.02	0.09	27	HEI 1996
$1.3 \times 10^{-4}$	Albany, New York air	7	30	72	94	NSTC 1997
Geometric mean		0.28	1.5	6.4	34	
Arithmetic mean		2.6	10.4	24.5	45.6	

Note:  
 $RSC = (I_{water} \times 100) / (I_{water} + I_{air})$ . Food and soil sources are considered negligible for MTBE.  
 $I_{water}$  = uptake by ingestion of tap water containing MTBE at the concentrations noted assuming two L/day and 100 percent intestinal absorption.  
 $I_{air}$  = uptake by inhalation of airborne MTBE assuming 20 m<sup>3</sup> air inhaled/day and 50 percent absorption.  
 Both  $I_{water}$  and  $I_{air}$  are assumed for a 70 kg human.  
 \*The concentrations of MTBE in drinking water were taken from the reports noted rather than using arbitrary values: 0.36 ppb (Brown 1997); two ppb (NSTC 1997 rounded); 12 ppb (rounded 10<sup>-6</sup> risk estimate, U.S. EPA 1996a); and 70 ppb (proposed Longer-Term and Lifetime HA, U.S. EPA 1996a). However, any plausible range could have been used, e.g., 5, 10, 20, 40, etc.

Table 16.—CalTox™ Predictions of Inhalation (I), Oral (O) and Dermal (D) Exposures (mg/kg/day) from 70 ppb MTBE Contaminated Tap Water: Effects of Varying Henry's Law Constant and Drinking Water Intake Level

Henry's Law constant (PA m <sup>3</sup> /mole)		Water intake (mL/kg/day)		
		19.4	33.3	43.9
66.5	I = $1.16 \times 10^{-3}$	$1.16 \times 10^{-3}$	$1.16 \times 10^{-3}$	$1.16 \times 10^{-3}$
	O = $1.11 \times 10^{-3}$	$1.91 \times 10^{-3}$	$1.91 \times 10^{-3}$	$2.52 \times 10^{-3}$
	D = $4.41 \times 10^{-6}$	$4.41 \times 10^{-6}$	$4.41 \times 10^{-6}$	$4.41 \times 10^{-6}$
	$2.28 \times 10^{-3}$	$3.08 \times 10^{-3}$	$3.08 \times 10^{-3}$	$3.69 \times 10^{-3}$
	All 2.46 Leq/day	3.30 Leq/day	3.30 Leq/day	3.97 Leq/day



Table 16.—CalTox™ Predictions of Inhalation (I), Oral (O) and Dermal (D) Exposures (mg/kg/day) from 70 ppb MTBE Contaminated Tap Water: Effects of Varying Henry's Law Constant and Drinking Water Intake Level—Continued

Henry's Law constant (PA m <sup>3</sup> /mole)	Water intake (mL/kg/day)		
	19.4	33.3	43.9
142 .....	I = $1.17 \times 10^{-3}$ .....	ND .....	ND
	O = $1.09 \times 10^{-3}$ .....		
	D = $4.43 \times 10^{-6}$ .....		
	= $2.26 \times 10^{-3}$ .....		
	All 2.48 Leq/day .....		
228 .....	I = $1.18 \times 10^{-3}$ .....	$1.18 \times 10^{-3}$ .....	$1.18 \times 10^{-3}$
	O = $1.09 \times 10^{-3}$ .....	$1.88 \times 10^{-3}$ .....	$2.47 \times 10^{-3}$
	D = $4.34 \times 10^{-6}$ .....	$4.3 \times 10^{-6}$ .....	$4.34 \times 10^{-6}$
	$2.27 \times 10^{-3}$ .....	$3.06 \times 10^{-3}$ .....	$3.65 \times 10^{-3}$
	All 2.51 Leq/day .....	3.33 Leq/day .....	4.03 Leq/day

Note: The CalTox™ model vadose and root zone compartments were loaded to predict 70 ppb MTBE in the groundwater used for residential drinking water. Various values for Henry's Law constant and water intake in mL/kg/day for a 62 kg female were used. MTBE parameters for molecular weight, octanol-water partition coefficient, melting point, vapor pressure, and water solubility were entered. Water intake values (mL/kg/day) correspond to median tap water for 20- to 64-year-old females (19.4), median total water intake for 20- to 64-year-old females (33.3), and average total water intake for all females (43.9) based on the Western Regional data (Ershov and Cantor 1989). Inhalation (I) value assumes 50 percent of inhaled MTBE is absorbed. Oral (O) and dermal (D) values assume 100 percent absorption. Total intakes by all routes are also expressed as L equivalents (Leq) per day.

#### Carcinogenic Effects

For carcinogens, the following general equation can be used to calculate the public health-protective concentration (C) for a chemical in drinking water (in mg/L):

$$C = BW \times R/q_1^* \text{ or } CSF \times DWC = \text{mg/L}$$

where:

BW = adult body weight (a default of 70 kg).

R = *de minimis* level for lifetime excess individual cancer risk (a default of  $10^{-6}$ ).

$q_1^*$  or CSF = cancer slope factor. The  $q_1^*$  is the upper 95 percent confidence limit on the cancer potency slope calculated by the LMS model, and CSF is a potency derived from the lower 95 percent confidence limit on the 10 percent (0.1) tumor dose (LED<sub>10</sub>). CSF =  $0.1/\text{LED}_{10}$ . Both potency estimates are converted to human equivalent [in (mg/kg-day)<sup>-1</sup>] using BW<sup>3/4</sup> scaling.

DWC = daily volume of water consumed by an adult (a default of two L/day or other volume in Leq/day to account for additional inhalation and dermal exposures from household use of drinking water as explained above).

Two cancer potency estimates,  $q_1^*$  or CSF, were calculated because our current experience with the LMS model is extensive whereas the new methodology proposed by U.S. EPA (1996f) in its draft guidelines for carcinogen risk assessment is based on the LED<sub>10</sub> for which little is known about the problems and outcome of using this procedure. The LMS model focuses on the linear low-dose extrapolation and analysts (e.g., U.S. EPA) have often accepted relatively poor fits to the observed tumor incidence data. The new method places a higher premium on fitting the observed data to estimate the ED<sub>10</sub> and the 95 percent lower bound LED<sub>10</sub>, the point from which the low dose extrapolation is made (U.S. EPA 1996a). In the case of the estimates obtained for carcinogenic potency of MTBE, the values calculated using the LMS model are not significantly different from that obtained using the preferred LED<sub>10</sub> approach.

The calculated public health-protective concentration accounting for carcinogenic effects of MTBE is based on a carcinogenic potency of  $1.8 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>. This estimate is the geometric mean of the potency estimates (CSFs) obtained for the combined male rat kidney adenomas and carcinomas in the inhalation study by Chun et al. (1992), the male rat Leydig cell tumors in the oral study by Belpoggi et al. (1995, 1998), and the leukemia and lymphomas in female rats, also in the study by Belpoggi et al. (1995, 1998). It is consistent with potencies obtained at other sites in another species (mice). The estimate for the inhalation route was converted to an oral intake using the pharmacokinetic model described earlier. The public health-protective concentration was therefore calculated using the following values:

BW = 70 kg (the default male adult human body weight).

R =  $10^{-6}$  (default *de minimis* lifetime excess individual cancer risk).

$q_1^*$  or CSF =  $1.8 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup> (CSF estimated as above).

DWC = 3 Leq/day (daily water consumption. As described previously in the section on RSCs, there are various probable routes of exposure in addition to ingestion that would result from contamination of water supplies. To allow for these additional exposures as shown in calculations in Table 16, the assumed daily volume of water consumed by an adult is increased from the default of two L/day to three Leq/day). Thus,

$$C = 70 \times 10^{-6} / 1.8 \times 10^{-3} \times 3 = 13 \times 10^{-3} \text{ mg/L} = 13 \text{ } \mu\text{g/L} = 13 \text{ ppb}$$

Since the calculated public health-protective concentration based on noncancer toxicity of 47 ppb is less protective of public health than the above cancer based value of 13 ppb, the recommended PHG level for MTBE is therefore 13 ppb (0.013 mg/L or 13  $\mu$ g/L). The adopted PHG is considered to contain an adequate margin of safety for the potential noncarcinogenic adverse effects including adverse effects on the renal, neurological and reproductive systems.

#### RISK CHARACTERIZATION

MTBE is used as an additive in cleaner burning automotive fuel in California. This results in opportunities for airborne exposures as well as drinking water exposures through leaking USTs and to a lesser extent from certain powered watercraft and air deposition. The public health risks of exposure to MTBE can be characterized as follows:

##### *Acute Health Effects*

Acute health effects are not expected to result from typical exposure to MTBE in drinking water. This includes household airborne exposures from showering, flushing toilets, etc. Reports of health complaints of various nonspecific symptoms (e.g., headache, nausea, cough) associated with exposure to gasoline containing MTBE have not been confirmed in controlled studies and remain to be fully evaluated.

##### *Carcinogenic Effects*

Inhalation exposure to MTBE produced increased incidences of kidney and testicular tumors in male rats and liver tumors in mice. Oral administration of MTBE produced leukemia and lymphoma in female rats and testicular tumors in male rats. A summary of our evaluation is listed below.

- As a result of this assessment OEHHA considers MTBE to be an animal carcinogen and a possible human carcinogen.
- Three cancer bioassays have shown MTBE induced tumors at several sites, in two species, in both sexes, by oral and inhalation routes of exposure; five of six studies were positive.
- Cancer study results exhibit consistency. For example, testicular tumors were induced in rats by both routes of MTBE administration.
- The oral rat study by Belpoggi et al. (1995, 1997, 1998) was found to be adequate for risk assessment purposes despite early mortality in the females.
- The inhalation studies in rats and mice were also considered adequate for risk assessment despite early mortality in both studies.
- In general the quality of the three studies was as good or better than those typically available for chemical risk assessment.
- While there are varying degrees of uncertainty as to the relevance to human cancer causation for each of the tumor types induced by MTBE in rodents (i.e., hepatocellular adenoma and carcinoma, renal tubular adenoma and carcinoma, Leydig interstitial cell tumors of the testes, leukemias and lymphomas), the occurrence of tumors at all of these sites adds considerably to the weight of evidence supporting the conclusion that MTBE should be considered a possible human carcinogen.
- MTBE genotoxicity data is weak, and there is no clear evidence that genotoxicity of its metabolites is involved in the carcinogenicity observed.
- There is no evidence to support a specific nongenotoxic mode of action (e.g., hormone receptor binding) and no evidence that metabolism of MTBE is required for carcinogenicity. In the absence of sufficient evidence, dose metrics based on the parent compound, MTBE, were necessarily chosen for the dose-response assessment.
- In the absence of specific scientific information explaining why the animal tumors are irrelevant to humans at environmental exposure levels, a standard health protective approach was taken to estimate cancer risk.
- Cancer potency estimates derived from different studies, sites, and routes of administration are similar.
- Cancer potency estimates are low compared to other known carcinogens despite the health conservative default assumptions employed.

- The adopted PHG of 13 ppb is based on an average of three quantitatively similar CSFs for three sites (kidney tumors, testicular tumors, leukemia and lymphoma). If the PHG value was based on individual tumor sites instead of an average, the values would range from 2.7 to 15 ppb.

- The CSFs are upper-bound estimates defined by the 95 percent confidence limit on the ED<sub>10</sub>. It is theoretically possible that the true value of the cancer potency of MTBE in humans could exceed these values, but that is considered unlikely. It is plausible that the true value of the human cancer potency for MTBE has a lower bound of zero based on statistical and biological uncertainties including interspecies extrapolation and mode of action.

- The estimate of multi-route exposure employed in the PHG calculation was three Leq/day. The range of exposure estimates based on different Henry's Law constants and water ingestion rates was 2.5 to four Leq/day. The range of possible PHGs based on this range and the average CSF of 0.0018 (mg/kg-day)<sup>-1</sup> is 10 to 16 ppb.

- Additional peer review of all the cancer bioassays would be useful, as would be a separate bioassay of MTBE in drinking water. However, these supplemental data should be seen in the context of the data already available, which are substantial and of better quality than is available for some other compounds for which risk assessments have been undertaken.

- Lack of knowledge of the mode(s) of action of MTBE or its metabolites is a major limitation of this risk assessment.

- Lack of evidence of cancer causation in humans is also a significant limitation, although widespread use and potential exposure is relatively recent in California and the rest of the U.S.

- Additional pharmacokinetic data in humans and improved PBPK models in animals and humans are desirable.

- Lack of information on the role that interindividual variability (i.e., stemming from metabolic polymorphisms, age-related differences, and concurrent disease conditions) may play in determining susceptibility to the carcinogenicity of MTBE severely hinders identification of sensitive subgroups in the California population.

The cancer potency estimate derived from the geometric mean of the CSFs of the combined male rat kidney adenomas and carcinomas, the male rat Leydig cell tumors, and the leukemia and lymphomas in female rats was  $1.8 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>. Individual tumor endpoint CSFs ranged from  $1.55 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup> to  $8.7 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>, or a range of about six-fold. Potencies based on the LMS model were similar ranging from  $1.63 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup> to  $9.2 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup>, also a range of six-fold. A time-to-tumor analysis gave much higher values of 0.076 (mg/kg-day)<sup>-1</sup> and 0.072 (mg/kg-day)<sup>-1</sup> for the LMS and LED<sub>10</sub> approaches, respectively. However this latter estimate has a low degree of confidence.

The findings of the oral gavage studies conducted by Belpoggi and colleagues have been given less weight by some reviewers, based on criticisms of various aspects of the study design, study reporting, and data analysis employed. The NAS (NRC 1996) review of the studies of Belpoggi et al. (1995) noted the following as study deficiencies: (1) the dosage schedule of Monday, Tuesday, Thursday, and Friday, rather than five consecutive days; (2) use of doses in apparent excess of the Maximum Tolerated Dose (MTD), based on a dose-related decrease in survival among treated females; (3) the combining of leukemia and lymphoma incidence; (4) incomplete description of tumor pathology and diagnostic criteria; and (5) lack of mortality adjusted analysis to account for differences in survival times. As noted above, OEHHA has considered these criticisms and considers that, although these experiments, like the others available for MTBE, do have certain limitations or difficulties of interpretation, they contribute considerably to the overall evidence available for MTBE risk assessment. Further, our conclusion is that the study is valid, not critically flawed, and is consistent with other reported results.

In criticizing the dosing schedule, NAS (NRC 1996) is correct in pointing out that 5 days per week is more usual. However, there is no evidence from the pharmacokinetic analyses that the proportionately higher peak dose and longer recovery periods would make any difference relative to the same time-averaged dose given over 5 days. The criticism that the MTD was exceeded appears misguided, in that a substantial proportion of the animals in all groups survived for a major part of the standard lifetime. The authors specifically noted no dose-related differences between control and exposed animals in food and water consumption or mean body weights (important indicators of non-specific toxicity). In any event, such a flaw, if real, would reduce rather than enhance the power of the studies to detect a positive response. The questions as to the advisability of combining leukemias and lymphomas, and the desire for clarification of the diagnostic criteria for these and the Leydig cell tumors, have been addressed by pathology review undertaken by

Belpoggi et al. (1998), and reviewed elsewhere in this document. OEHHA shares the NAS preference for availability of full mortality data whenever possible, but notes that extensive quantal statistical analyses were undertaken by Belpoggi et al. (1998), as well as by OEHHA for this report, and considers that the data as presented provide an adequate basis for use in this risk assessment.

In its critique of the Belpoggi et al. studies, the NAS (NRC 1996) also stated that "an in-depth review of the data, especially the pathology (microscopic slides) of the critical lesions, is warranted (as was done with the inhalation studies) before the data are used for risk assessment." As mentioned above, Belpoggi and colleagues have recently published the results of a pathology review in which slides from the original study were re-examined, and diagnostic criteria reviewed by an independent panel of pathologists from the Cancer Research Centre, with the participation of an outside pathologist (Belpoggi et al. 1998). This review confirmed the authors' previous findings, and addressed the concerns expressed in the NAS report. As was correctly pointed out in the NSTC report (1997), the pathological findings of the MTBE inhalation studies (Burleigh-Flayer et al. 1992, Chun et al. 1992) have not undergone peer review, moreover, "independent peer review of pathological findings are not routinely performed in carcinogenesis studies used by the risk assessing community and (U.S.) EPA."

The water concentration associated with a  $10^{-6}$  negligible theoretical extra lifetime cancer risk calculated from this analysis is 13 ppb. This includes an estimate of inhalation exposure from showering in MTBE contaminated water, flushing toilets, and other household activities involving tap water. The estimate of one Leq/day of additional exposure via the inhalation route is lower than the default value of two Leq/day of additional exposure suggested by U.S. EPA (1996b) based on average estimated showering exposures of a number of typical VOCs. This reflects the fact that MTBE is less volatile and more water-soluble than other VOCs commonly found in drinking water. The adopted PHG value of 13 ppb also compares favorably with the Provisional Health and Consumer Acceptability Advisory range of 20 to 40 ppb established by U.S. EPA (1997a) using a MOE approach. Since the adopted value of 13 ppb was calculated for a  $1 \times 10^{-6}$  theoretical lifetime extra risk from a linear extrapolation, the values of 130 ppb and 1,300 ppb (1.3 ppm or 1.3 mg/L) would be associated with the higher risk estimates of  $1 \times 10^{-5}$  to  $1 \times 10^{-4}$ , respectively.

For PHGs, our use of the RSC has, with a few exceptions, followed U.S. EPA drinking water risk assessment methodology. U.S. EPA has treated carcinogens differently from noncarcinogens with respect to the use of RSCs. For noncarcinogens, RfDs (in mg/kg/day), DWELs (in mg/L) and MCLGs (in mg/L) are calculated using UFs, body weights and DWC (in Leq/day) and RSC, respectively. The typical RSC range is 20 percent to 80 percent (0.2 to 0.8), depending on the scientific evidence.

U.S. EPA follows a general procedure in promulgating MCLGs:

- if Group A and B carcinogens (i.e., strong evidence of carcinogenicity) MCLGs are set to zero;
- if Group C (i.e., limited evidence of carcinogenicity), either an RfD approach is used (as with a noncarcinogen) but an additional UF of 1 to 10 (usually 10) is applied to account for the limited evidence of carcinogenicity, or a quantitative method (potency and low-dose extrapolation) is used and the MCLG is set in the  $10^{-5}$  to  $10^{-6}$  cancer risk range;
- if Group D (i.e., inadequate or no animal evidence) a RfI approach is used to promulgate the MCLG.

For approaches that use low-dose extrapolation based on quantitative risk assessment, U.S. EPA does not factor in a RSC. The use of low-dose extrapolation is considered by U.S. EPA to be adequately health-protective without the additional source contributions. In developing PHGs, we have used the assumption that RSCs should not be factored in for carcinogens grouped in U.S. EPA categories A and B, and for C carcinogens for which we have calculated a cancer potency value based on low-dose extrapolation. This is an area of uncertainty and scientific debate and it is not clear how this assumption impacts the overall health risk assessment.

#### OTHER REGULATORY STANDARDS

The IPCS of WHO is issuing the final version of an environmental health criteria document on MTBE (IPCS 1997). The Dutch Expert Committee on Occupational Standards (Wibowo 1994) recommended a health-based 8 hour-Time-Weighted Average (TWA) exposure limit for MTBE of 180 mg/m<sup>3</sup> or 50 ppm to be averaged over an 8-hour working day, and a short-term 15-minute-TWA limit of 360 mg/m<sup>3</sup> or 100 ppm in the Netherlands. Czechoslovakia has an Occupational Exposure Limit (OEL) TWA of 100 mg/m<sup>3</sup> and a Short-Term OEL (STEL) of 200 mg/m<sup>3</sup> since January 1993. Russia has a STEL of 100 mg/m<sup>3</sup> since January 1993 (RTECS 1997). Sweden

established a TWA of 50 ppm and a 15-minute STEL of 75 ppm in 1988 (ACGIH 1996). The British Industrial Biological Research Association (BIBRA) compiled a toxicological profile on MTBE in 1990. The Danish Environmental Protection Administration is considering setting a 30 ppb limit of MTBE in groundwater. More recently, ECETOC (1997) recommended an occupational exposure limit of 90 mg/m<sup>3</sup> or 25 ppm to be 8 hour-TWA and a short-term peak 15-minute-TWA limit of 270 mg/m<sup>3</sup> or 75 ppm.

In the U.S., the OSHA and NIOSH established the TLV-TWA as 40 ppm in air (144 mg/m<sup>3</sup>) in 1994 as proposed by ACGIH in 1993. ACGIH (1996) also lists MTBE as an A3 animal carcinogen in 1995 as proposed in 1994. MTBE is on the Emergency Preparedness and Community Right-to-Know Section of the Superfund Amendments and Reauthorization Act of 1986 (SARA Title III) Extremely Hazardous Substances (EMS) list and in the TSCA Test Submission (TSCATS) Data base. It is one of the TRI chemicals to be routinely inventoried. MTBE is on the Hazardous Air Pollutant (HAP) list with 189 other chemicals to be regulated under the Air Toxics Program of the 1990 CAAA. Article 211 (b) of Title III of the CAAA requires that oil companies conduct gasoline inhalation studies and U.S. EPA sent the testing requirement notification on August 20, 1997. Negotiations with industry on the extent of these studies are ongoing. Animal research will focus on short and long-term inhalation effects of conventional gasoline and gasoline with MTBE. The Article 211 studies will also include human exposure research. The research will be completed at varying intervals over the next 5 years. HEI is funding three new studies designed to answer key questions on the metabolism of MTBE and other ethers in animals and humans.

MTBE is listed as a California TAC mandated under AB 1807 by virtue of its status as a HAP. It is one of the California Air Toxics "Hot Spots" chemicals mandated under AB 2588. ARB is proposing to place MTBE into subcategory b as substances nominated for review for development of health values. A chronic Reference Exposure Level, which is the same as the three mg/m<sup>3</sup> RfC for inhalation of MTBE in air as listed in the U.S. EPA (1997c) IRIS data base, is being developed in the draft Hot Spots document by OEHHA mandated under SB 1731. Texas established a half-hour limit in ambient air of 0.6 mg/m<sup>3</sup> and an annual limit of 0.288 mg/m<sup>3</sup> in 1992 (Sittig 1994).

MTBE is not a priority pollutant under the Clean Water Act and is not a target analyte in routine water quality monitoring and assessment programs. MTBE is included in the draft and final Drinking Water Contaminant Candidate List (CCL) required by the Safe Drinking Water Act (U.S. EPA 1997b, 1997d, 1998b). The final list is published on March 2, 1998 with descriptions on how to make decisions on whether to establish a standard on the contaminants. CCL is divided into categories representing next steps and data needs for each contaminant. U.S. EPA will choose at least five contaminants from the Regulatory Determination Priorities category and determine by August 2001 whether or not to regulate them based on occurrence, exposure and risk. If regulations are deemed necessary they must be proposed by August 2003 and promulgated by February 2005. MTBE is proposed for inclusion on the Federal "National Drinking Water Contaminant Occurrence Data Base".

In the interim, the Office of Water has initiated a data base based on voluntary reporting from some states, USGS data, and other available sources. MTBE is on the U.S. EPA Drinking Water Priority List for future regulation. The U.S. EPA's Office of Research and Development is working to identify MTBE research needs, including monitoring, exposure, health effects, and remediation. A workshop was held on October 7, 1997 to present an initial assessment of research needs to industry and academic groups. A draft report (U.S. EPA 1998b) has been issued for public comment ending by August 28, 1998. Other U.S. EPA activities include development of a protocol to collect data on potential CO reductions using Federal oxygenated gasoline. USGS is conducting urban land use studies this year to characterize VOCs, including MTBE contamination as a part of the larger national NAWQA program.

Since the early 1990's, U.S. EPA has evaluated MTBE to quantify its toxic effects (Farland 1990, Hiremath and Parker 1994, Klan and Carpenter 1994, Gomez-Taylor et al. 1997). U.S. EPA (1996a) proposed a 70 ppb HA for MTBE in its December 1996 draft report based on noncarcinogenic kidney and liver effects in laboratory animals with large uncertainty factors (U.S. EPA 1996f). U.S. EPA also included an extra uncertainty factor in its draft report to account for the possible carcinogenicity of the substance. The laboratory animal cancer bioassays of MTBE by the inhalation route were performed by Bushy Run Research Center (Burleigh-Flayer et al. 1992, Chun et al. 1992) and the ones by the oral route were performed by Cancer Research Centre of the European Foundation for Oncology and Environmental

Sciences "B. Ramazzini" in Italy (Belpoggi et al. 1995, 1997, 1998). U.S. EPA has not had an opportunity to audit the studies even though reviews of pathological findings are not routinely performed (NSTC 1997). Nevertheless, in the 1996 draft, U.S. EPA indicated that the animal studies would suggest that 12.5 ppb would equate to a theoretical risk level of one excess fatal case of cancer per million people per 70-year lifetime (a  $10^{-6}$  risk), a level usually viewed as *de minimis*, for MTBE as a Group B2 probable human carcinogen. The 12.5 ppb was calculated based on an oral cancer potency estimate ( $q_1^*$ ) of  $3 \times 10^{-3}$  (mg/kg-day) $^{-1}$  derived from the default LMS method and a scaling factor of body weight raised to  $3/4$  power using the combined lymphoma and leukemia in the female rats in the gavage study.

The U.S. EPA (1997c) IRIS data base lists the RfC for inhalation of MTBE in air as three mg/m $^3$  as last revised on September 1, 1993. The RfC is based on increased liver and kidney weights, increased prostration in females, and swollen periocular tissues in male and female rats. The RfD for oral exposure to MTBE is under review by U.S. EPA (1997c). In 1992, U.S. EPA derived a draft long-term HA range for MTBE in drinking water of 20 to 200 ppb (or 0.02 to 0.2 mg/L) based on a RfD of 0.1 mg/kg/day from a 90-day rat drinking water study with dose-related increases in relative kidney weights in both sexes (Robinson et al. 1990). The range is due to the uncertainty for the carcinogen classification. The guideline would be either 20 ppb if MTBE were classified as a Group B2 or C carcinogen, or 200 ppb if MTBE is a Group D carcinogen. In 1994, U.S. EPA drafted a proposal in reviewing data from animal studies for the possibility of listing MTBE as a Group B2 probable human carcinogen, and derived an oral cancer potency estimate ( $q_1^*$ ) of  $8.6 \times 10^{-3}$  (mg/kg-day) $^{-1}$  and a HA of four ppb for a  $10^{-6}$  risk.

The States of Vermont and Florida established drinking water standards for MTBE of 40 ppb and 50 ppb, respectively. The New York State Department of Public Water promulgated a MCL of 50 ppb in 1988. The New York State Department of Health is drafting an ambient water quality value for protection of human health and sources of potable water for MTBE based on the evaluation of animal oncogenicity data. The New Jersey Department of Environmental Protection (NJDEP) proposed in 1994 and established in 1996 a health-based MCL for MTBE in drinking water of 70 ppb, reducing from 700 ppb. This is in agreement with the 1993 evaluation of the U.S. EPA except for an uncertainty factor of 10,000 used by NJDEP instead of the 3,000 applied by the U.S. EPA (NJDWQI 1994, Post 1994). The Illinois Environmental Protection Agency listed a human threshold toxicant advisory concentration of 230 ppb in 1994 and has proposed a health-based MCL for MTBE in drinking water ranging from 70 to 2,000 ppb. The Massachusetts Department of Environmental Protection in 1995 proposed to decrease the guideline for MTBE in drinking water from 700 ppb to 70 ppb (MORS 1995). The Maine Department of Human Services listed a drinking water threshold of 50 ppb in 1995 and is considering to adopt 35 ppb based on noncancer health effects with a RSC of 10 percent (Smith and Kemp 1998). NCDEHNR has proposed a primary MCL of 70 ppb. The Wisconsin Department of Natural Resources in 1995 established a groundwater enforcement standard for MTBE of 60 ppb (WDOH 1995). The guideline for MTBE in drinking water is 35 ppb in Arizona, 40 ppb in Michigan, 50 ppb in Rhode Island, and 100 ppb in Connecticut and New Hampshire (ATSDR 1996, HSDB 1997, Sittig 1994).

The UC report mandated under SB521 concluded that MTBE is an animal carcinogen with the potential to cause cancers in humans (Froines et al. 1998). Using several models for exposure analysis, Johnson (1998) calculated a *de minimis* theoretical excess individual cancer risk level of  $10^{-6}$  from exposure to MTBE of 10 ppb which, the author concluded, is comparable to the level recommended in this report.

DHS has added MTBE to a list of unregulated chemicals that require monitoring by drinking water suppliers in California in compliance with the California Safe Drinking Water Act, Sections 116300 to 116750. An interim Action Level of 35 ppb or 0.035 mg/L for drinking water was adopted by the DHS in 1991. The level was recommended by OEHHA (1991) using the oral RfD of 0.005 mg/kg/day then reported on the U.S. EPA IRIS data base for an anesthetic effect in rats in a 13-week inhalation study performed in Europe (Greenough et al. 1980). DHS is proceeding with establishing drinking water standards for MTBE in California.

The initial standard to be developed for MTBE is a secondary MCL. The secondary MCL of five ppb is adopted by DHS as a regulation effective January 7, 1999. Secondary MCLs address aesthetic qualities of drinking water supplies. In the case of MTBE, the focus is on its organoleptic qualities, that is, its odor and taste. The purpose of the secondary MCL is to protect the public from exposure to MTBE in drinking water at levels that can be smelled or tasted. Secondary MCLs in California are enforceable standards, which means that drinking water should not be served by public water systems if it contains MTBE higher than the secondary

standard. Enforceable secondary standards are unique to California. The proposed secondary MCL for MTBE is based on data from experiments that have been performed by researchers, using panels of subjects who were exposed to varying concentrations of MTBE in water to determine levels at which it could be smelled or tasted. As part of the process by which regulations are adopted under California's Administrative Procedures Act, the proposed regulation (R-44-97) was available for public comment since July 3, 1998, and September 8, 1998 was the close of the written comment period (DHS 1998).

The next standard to be developed is a primary MCL that protects the public from MTBE at levels that can affect public health. A primary MCL for MTBE will include consideration of the health risk assessment, the technical feasibility of meeting the MCL (in terms of monitoring and water treatment requirements for MTBE) and costs associated with compliance. DHS has requested the OEHHA to provide a risk assessment for MTBE that is required for the development of the primary standard. DHS requested that the risk assessment be completed in order to meet the scheduled adoption of this regulation by July 1999. The proposed primary MCL is anticipated to be available for public comment in early 1999.

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#### LIST OF ABBREVIATIONS

AB—Assembly Bill

AL—Action Level

ACGIH—American Conference of Governmental Industrial Hygienists

API—American Petroleum Institute

ARB—California Air Resources Board  
 ATSDR—Agency for Toxic Substances and Disease Registry, USDHHS  
 AUC—area under the concentration-time curve  
 BAAQMD—Bay Area Air Quality Management District, San Francisco, California  
 BIBRA—British Industrial Biological Research Association  
 BTEX—benzene, toluene, ethylbenzene, and xylenes  
 BUN—blood urea nitrogen  
 BW—body weight  
 CAAA—1990 U.S. Clean Air Act Amendments  
 Cal/EPA—California Environmental Protection Agency  
 CAS—Chemical Abstracts Service  
 CCL—Drinking Water Contaminant Candidate List, U.S. EPA  
 CCR—California Code of Regulations  
 CDC—Centers for Disease Control and Prevention, USDHHS  
 CFS—chronic fatigue syndrome  
 CENR—Committee on Environment and Natural Resources, White House OSTP  
 CHRIS—Chemical Hazard Response Information System, U.S. Coast Guard  
 CNS—central nervous system  
 CO—carbon monoxide  
 CSF—cancer slope factor, a cancer potency value derived from the lower 95 percent confidence bound on the dose associated with a 10 percent (0.1) increased risk of cancer (LED<sub>10</sub>) calculated by the LMS model.  $CSF = 0.1/LED_{10}$ .  
 CPF—cancer potency factor, cancer potency, carcinogenic potency, or carcinogenic potency factor  
 DHS—California Department of Health Services  
 DOE—U.S. Department of Energy  
 DOT—U.S. Department of Transportation  
 DOT/UN/NA/IMCO—U.S. Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code  
 DLR—detection limit for purposes of reporting  
 DWC—daily water consumption  
 DWEL—Drinking Water Equivalent Level  
 EBMUD—East Bay Municipal Utility District, California  
 ECETOC—European Centre for Ecotoxicology and Toxicology of Chemicals  
 EHS—Extremely Hazardous Substances, SARA Title III  
 EOHSI—Environmental and Occupational Health Sciences Institute, New Jersey  
 ETBE—ethyl tertiary butyl ether  
 GAC—granulated activated charcoal  
 gd—gestation day  
 g/L—grams per liter  
 HA—Health Advisory  
 HAP—Hazardous Air Pollutant  
 HCHO—formaldehyde  
 HEI—Health Effects Institute, Boston, Massachusetts  
 HSDB—Hazardous Substances Data Bank, U.S. NLM  
 IARC—International Agency for Research on Cancer, WHO  
 i.p.—intraperitoneal  
 IPCS—International Programme on Chemical Safety, WHO  
 IRIS—Integrated Risk Information Systems, U.S. EPA  
 i.v.—intravenous  
 kg—kilograms  
 L—liter  
 LC<sub>50</sub>—lethal concentrations with 50 percent kill  
 LED<sub>50</sub>—lethal doses with 50 percent kill  
 LED<sub>10</sub>—lower 95 percent confidence bound on the dose associated with a 10 percent increased risk of cancer  
 Leq/day—liter equivalent per day  
 LLNL—Lawrence Livermore National Laboratory, California  
 LMS—linearized multistage  
 LOAEL—lowest observed adverse effect level  
 LUFT—leaking underground fuel tank  
 MCCHD—Missoula City—County Health Department, Montana  
 MCL—Maximum Contaminant Level  
 MCLG—Maximum Contaminant Level Goal  
 mg/L—milligrams per liter  
 µg/L—micrograms per liter  
 MCS—multiple chemical sensitivities  
 mL—milliliter

MOE—margin of exposure  
 MORS—Office of Research and Standards, Department of Environmental Protection, the Commonwealth of Massachusetts  
 MRL—minimal risk levels  
 MTBE—methyl tertiary butyl ether  
 MTD—maximum tolerated dose  
 MWDSC—Metropolitan Water District of Southern California  
 NAERG—North American Emergency Response Guidebook Documents, U.S., Canada and Mexico  
 NAS—U.S. National Academy of Sciences  
 NAWQA—National Water-Quality Assessment, USGS  
 NCDEHNR—North Carolina Department of Environment, Health, and Natural Resources  
 NCEH—National Center for Environmental Health, U.S. EPA  
 NCI—U.S. National Cancer Institute  
 ng—nanograms  
 NIEHS—U.S. National Institute of Environmental Health Sciences  
 NIOSH—U.S. National Institute for Occupational Safety and Health  
 NJDEP—New Jersey Department of Environmental Protection  
 NJHSFS—New Jersey Hazardous Substance Fact Sheets  
 NJDWQI—New Jersey Drinking Water Quality Institute  
 NLM—National Library of Medicine  
 NOAEL—no observable adverse effect levels  
 NOEL—no observable effect levels  
 NRC—National Research Council, U.S. NAS  
 NSTC—U.S. National Science and Technology Council  
 NTP—U.S. National Toxicology Program  
 OEHHA—Office of Environmental Health Hazard Assessment, Cal/EPA  
 OEL—Occupational Exposure Limit  
 OHM/TADS—Oil and Hazardous Materials/Technical Assistance Data System, U.S. EPA  
 OSTP—White House Office of Science and Technology Policy  
 O<sub>3</sub>—ozone  
 oxyfuel—oxygenated gasoline  
 PBPK—physiologically-based pharmacokinetic  
 PHG—Public Health Goal  
 PHS—Public Health Service, USDHHS  
 pnd—postnatal day  
 POTW—publicly-owned treatment works  
 ppb—parts per billion  
 ppbv—ppb by volume  
 ppm—parts per million  
 ppt—parts per trillion  
 pptv—ppt by volume  
 Proposition 65—California Safe Drinking Water and Toxic Enforcement Act of 1986  
 q<sub>1</sub>\*—a cancer potency value that is the upper 95 percent confidence limit of the low dose extrapolation on cancer potency slope calculated by the LMS model  
 RfC—Reference Concentration  
 RfD—Reference Dose  
 RFG—reformulated gasoline  
 RSC—relative source contribution  
 RTECS—Registry of Toxic Effects of Chemical Substances, U.S. NIOSH  
 SARA—U.S. Superfund (CERCLA) Amendments and Reauthorization Act of 1986  
 SB—Senate Bill  
 SCVWD—Santa Clara Valley Water District, California  
 SFRWQCB—San Francisco Regional Water Quality Control Board  
 SGOT—serum glutamic-oxaloacetic transaminase  
 SS—statistically significant  
 STEL—Short-Term Occupational Exposure Limit  
 Superfund—U.S. Comprehensive Environmental Response, Compensation and Liability Act of 1980, a.k.a. CERCLA  
 SWRCB—California State Water Resources Control Board  
 TAC—toxic air contaminant  
 TAME—tertiary amyl methyl ether  
 TBA—tertiary butyl alcohol  
 TBF—tertiary butyl formate  
 TERIS—Teratogen Information System, University of Washington  
 TOMES—Toxicology and Occupational Medicine System, Micromedex, Inc.

TRI—Toxics Release Inventory, U.S. EPA  
TSCA—U.S. Toxic Substances Control Act  
TWA—Time-Weighted Average  
 $t_e$ —experimental duration  
 $t^1$ —lifetime of the animal used in the experiment  
 $t_{1/2}$ —plasma elimination half-life  
UC—University of California  
UCLA—UC Los Angeles  
UCSB—UC Santa Barbara  
UF—uncertainty factors  
U.S.—United States  
USCG—U.S. Coast Guard  
USDHHS—U.S. Department of Health and Human Services  
U.S. EPA—U.S. Environmental Protection Agency  
USGS—U.S. Geological Survey  
UST—underground storage tanks  
VOC—volatile organic compound  
VRG—vessel rich group  
WDOH—Wisconsin Division of Health, Department of Natural Resources  
WHO—World Health Organization  
WSPA—Western States Petroleum Association

