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2 **A Probabilistic Assessment of Household Exposures to MTBE in**
3 **California Drinking Water**
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26

1 ABSTRACT

2 The oxygenate methyl tertiary butyl ether (MTBE) has been added to reformulated
3 gasoline in the United States to meet national ambient air quality standards. Although MTBE
4 has provided significant health benefits in terms of reduced criteria and toxic air pollutants,
5 detections of MTBE in some groundwater and drinking water sources have raised concerns about
6 potential environmental contamination and human exposures. In this paper, we examine the
7 frequency and concentration of MTBE detections in drinking water sources in California from
8 1995 to 1999, and provide a preliminary analysis of the distribution of household exposures to
9 MTBE from water-related activities. Using published data on the toxicity and possible cancer
10 hazard posed by MTBE, we estimate the likely cancer and non-cancer risk for the general
11 population in California from past and potential future MTBE exposures. More highly exposed
12 subgroups were also addressed. Our findings indicate that less than 2% of all sampled drinking
13 water sources in California had detectable levels of MTBE in 1999, with average MTBE
14 drinking water concentrations ranging from 0.09 to 4.9 ppb for this year. Both the detection rate
15 for MTBE and average MTBE concentrations have remained relatively stable since 1995, despite
16 increased sampling of drinking water sources in California. The probabilistic household analysis
17 suggests that drinking water exposures to MTBE are unlikely to pose a significant health risk for
18 the general population or more highly exposed individuals in California.

19

20 **KEY WORDS:** MTBE, drinking water, human exposure, probabilistic analysis, risk assessment

1 INTRODUCTION

2 As the “oxygenate of choice,” methyl tertiary butyl ether (MTBE) has been added to
3 gasoline in significant quantities (11–15% by volume) to meet the requirements of the 1990
4 Clean Air Act Amendments. Specifically, reformulated gasoline containing MTBE has been
5 used since 1992 to reduce carbon monoxide levels in air during winter months, and since 1995 to
6 decrease ground-level ozone formation year-round (HEI, 1996). In California, MTBE has been
7 used year-round since 1996 to meet the state’s Phase II reformulated gasoline or Cleaner Burning
8 Gasoline requirements (Cal-EPA, 1997).

9 Substitution of MTBE-reformulated gasoline for conventional gasoline has notably
10 reduced tailpipe emissions of several toxic and criteria air pollutants in the United States (U.S.),
11 including benzene, 1,3-butadiene, carbon monoxide, and smog-forming volatile organic
12 compounds (USEPA, 1999; NSTC, 1997). In California, the widespread use of reformulated
13 gasoline containing MTBE has been estimated to reduce the cancer risk of conventional gasoline
14 emissions by approximately 40% (Cal-EPA, 1997). Reformulated gasoline use in the Northeast
15 has also been associated with cancer risk reductions relative to the use of conventional gasoline
16 (NESCAUM, 1998). Reductions in non-cancer health effects are expected from the use of
17 MTBE versus conventional gasoline, such as diminished respiratory irritation and asthma
18 attacks, particularly for sensitive individuals in the population (Erdal *et al.*, 1997).

19 The mobility and persistence of MTBE in groundwater, however, coupled with detections
20 of MTBE in some drinking water sources, has raised concerns about potential environmental
21 contamination and public health impacts (USEPA, 1999; UC, 1998; NSTC, 1997). A recent
22 study by the U.S. Geological Survey found that MTBE was one of the most frequently detected
23 volatile organic compounds in untreated ambient groundwater nationwide, with a detection rate

1 of 16.9% in urban areas and 3.4% in rural areas (Squillace *et al.*, 1999). Other studies suggest
2 that the detection frequency of MTBE in groundwater wells is about 5% in 33 states covered by
3 the U.S. Geological Survey, with the majority of detections occurring in shallow groundwater
4 within urban areas (NESCAUM, 1999). In the Northeast, MTBE has been detected in about
5 16% of private wells and public water supplies tested in Maine, 15% of community water
6 supplies sampled in New Jersey, and 6% of the public water supplies tested in Connecticut
7 (NESCAUM, 1999; MDHS, 1998).

8 A study by the Lawrence Livermore National Laboratory also found that 78% of
9 monitored leaking underground fuel tank sites in California had detectable levels of MTBE
10 (Happel *et al.*, 1998). The relatively high detection frequency of MTBE in this study is not
11 surprising, however, because monitoring occurred at tanks that were known or thought to be
12 leaking. It unclear what collective impact storage tank leaks will have on local drinking water
13 supplies, but some research suggests that MTBE leaks may represent a significant threat to
14 drinking water for at least the next ten years (Johnson *et al.*, 2000). Other research suggests that
15 ongoing efforts to upgrade or retrofit leaking underground storage tanks will greatly diminish the
16 potential for MTBE contamination of drinking water supplies (NESCAUM, 1999).

17 The threat of MTBE-contaminated drinking water has resulted in a number of legal
18 claims against MTBE manufacturers and oil companies (BNA, 1999a, 2000a, b, c; Littell, 2000).
19 State and federal agencies have also taken various actions to reduce or eliminate the use of
20 MTBE as a fuel oxygenate. For example, on March 26, 1999, the Governor of California issued
21 an executive order to phase out MTBE use—effective December 31, 2002—due to perceived
22 environmental threats (ARB, 1999). Several states, including Maine and New Hampshire, have
23 sought waivers to opt out of the federal RFG program, and legislation was introduced in

1 Congress to allow all states to stop using MTBE as a fuel oxygenate (BNA, 1999b; NESCAUM,
2 1999). In addition, the Governor of New York has proposed stringent state regulations to tighten
3 the standards for MTBE in surface water, groundwater, and drinking water (BNA, 1999c). More
4 recently, the Environmental Protection Agency (EPA) has taken regulatory action to eliminate
5 MTBE as a fuel additive under the Toxic Substances Control Act (BNA, 2000d; Herbert, 2000;
6 Rogers, 2000; Tansey and Howe, 2000; USEPA, 2000).

7 Despite the growing controversy over MTBE, few studies have evaluated whether the
8 levels of MTBE detected in most drinking water sources pose a public health risk. Sampling of
9 drinking water and non-drinking water sources nationwide indicates that of those samples with
10 detectable levels of MTBE, median MTBE levels are less than 1 part per billion (ppb) (Squillace
11 *et al.*, 1999). Reported detections of MTBE in drinking water in the Northeastern states are also
12 typically below 2 ppb, and between 0.5 and 1.5% of drinking water supplies tested in the
13 Northeast contain MTBE at concentrations above the most restrictive drinking water standard in
14 the region of 35 ppb (NESCAUM, 1999). Further, the average concentration of MTBE in
15 California drinking water was estimated to be 2.5 ppb (Froines *et al.*, 1998), which is about five
16 times less than the state's Public Health Goal for MTBE in drinking water of 13 ppb (OEHHA,
17 1999). The odor threshold for MTBE in drinking water may be as low as 2 to 5 ppb (Froines *et*
18 *al.*, 1998), but other reports suggest that MTBE is not detectable by taste or odor in the general
19 population until concentrations are well above 100 ppb (NESCAUM, 1999).

20 The purpose of this paper is to provide a preliminary analysis of actual drinking water
21 concentrations of MTBE in California and to assess the likelihood of human health risk based on
22 household drinking water exposures to MTBE. California was chosen as a case study for
23 analysis because extensive drinking water monitoring data on MTBE are available from the

1 California Department of Health Services (CDHS) from 1995–1999. The remainder of this
2 paper consists of the following sections: (1) detections of MTBE and average MTBE
3 concentrations in California’s drinking water sources over time, (2) probabilistic analysis of
4 household exposures to MTBE in drinking water; (3) brief overview of the toxicological
5 literature on MTBE as it relates to the development of a chronic (non-cancer) reference dose and
6 cancer slope factor for MTBE, (4) estimated health risks from past and potential future exposures
7 to MTBE in drinking water, and (5) a discussion of study findings.

8 **MTBE IN CALIFORNIA DRINKING WATER**

9 The current analysis examines available drinking water monitoring data on MTBE in
10 California from April 12, 1995 to November 29, 1999 (CDHS, 2000). In this database, the
11 “XMOD” field indicates whether samples have detectable or non-detectable levels of MTBE,
12 while the “finding” field reports either the concentration of MTBE detected or the analytical
13 detection limit for reporting (DLR) for non-detect samples. Based on discussions with the
14 CHDS database manager, all samples were coded as non-detect if XMOD was reported as “<”
15 and the reported MTBE finding was coded as the DLR for all non-detect samples (Collins,
16 2000). Samples where XMOD was blank or reported as “0” and the reported MTBE finding was
17 also zero were coded as non-detects with an assumed DLR of 5 ppb.¹ Five samples that were
18 coded as “f” in the XMOD field (i.e., false positive result as determined by the analytical
19 laboratory or CHDS) were excluded from further analysis (Collins, 2000).

20 Based on these assumptions, we estimate that a total of 3,937 distinct drinking water
21 sources in California (including active, inactive, standby, and abandoned sites) were sampled and
22 reported during this five-year time period. With the exception of 1999, the number of reported

¹ As of 1997, the mandatory DLR for MTBE in California public drinking water systems was 5 ppb, although many analytical laboratories report lower detection levels. The mandatory DLR is projected to be reduced to 3 ppb on May 17, 2000 (Collins, 2000).

1 drinking water sources tested for MTBE in California has increased steadily over time (see
2 Table 1). Only 117 drinking water sources were tested for MTBE in 1995, with a 14-fold
3 increase in sampled drinking water sources from 1995 to 1996, and a 20% increase in sampled
4 drinking water sources from 1996 to 1997 and 1997 to 1998. Although the number of sources
5 with MTBE detections also increased during this period, due to greater sampling efforts, the
6 detection rate for MTBE remained relatively stable from 1995 to 1999. In 1995, about 3.4% of
7 sampled drinking water sources in California had detectable levels of MTBE, compared to 1.8%
8 in 1999. Evaluations by CDHS (2000), which considers MTBE to be “detected” only if it is
9 present in at least two samples from the same source, suggest that 0.8% of sampled drinking
10 water sources in California had detectable levels of MTBE as of April 7, 2000.

11 *INSERT TABLE 1*

12 To evaluate drinking water levels of MTBE over time, average MTBE concentrations
13 were calculated for each drinking water source and then across all sources for each year. For this
14 analysis, two different methods were used to characterize non-detect samples: MTBE
15 concentrations were either assumed to equal zero or were assumed to equal the specified DLR.
16 This approach estimates the likely range (i.e., lower and upper bound) of MTBE concentrations
17 in California drinking water, because the actual concentration of MTBE for non-detect samples
18 is unknown. This approach may also provide a more appropriate representation of the data than
19 assuming that non-detect values equal a single value or one-half the analytical limit of detection.

20 The average concentration of MTBE for all sampled drinking water sources in California
21 was estimated to range from 2.0 ppb to 6.4 ppb in 1995, depending on whether non-detects were
22 assumed to equal zero or the DLR, respectively (see Figure 1). Average MTBE levels have
23 decreased slightly or remained relatively stable since this time and ranged from 0.09 ppb (non-

1 detects=0) to 4.9 ppb (non-detects=DLR) in 1999. These findings suggest that the increased
2 sampling of drinking water sources in California did not result in higher reported average
3 concentrations of MTBE. Higher MTBE levels observed in 1995 relative to later years may be
4 due, in part, to the initial sampling of drinking water sources that were suspected of MTBE
5 contamination. Note that there is significant variability in reported concentrations of MTBE
6 during the initial sampling period (i.e., 1995—1996), but there is much less variability in later
7 sampling years. Trends in average MTBE concentrations differ slightly between the two
8 approaches due to differences in analytical detection reporting limits over time.

9 *INSERT FIGURE 1*

10 For drinking water sources having at least one detectable level of MTBE (i.e., detects
11 only), average MTBE concentrations were approximately 57.8 ppb in 1995 and 8.8 ppb in 1999
12 (see Figure 2). Although average MTBE levels decreased from 1995 to 1998, there was a slight
13 increase in average MTBE concentrations from 1998 (5.4 ppb) to 1999 (8.8 ppb). Reported
14 concentrations of MTBE for detects only are highly variable during the 1995—1996 sampling
15 period.

16 *INSERT FIGURE 2*

17 If MTBE is detected in a drinking water source at one point in time, this does not mean
18 that MTBE will be detected in the same source at another point in time. Conversely, drinking
19 water sources with undetectable levels of MTBE at one sampling point may contain detectable
20 levels of MTBE at another sampling point. MTBE detections and reported concentrations
21 depend on many factors, including the number of times a source is sampled, the analytical limit
22 of detection and reporting requirements, the method in which non-detect samples are handled,
23 the leakage rate of underground storage tanks, the proximity of MTBE leaks or spills to a

1 drinking water source, and the fate and transport of MTBE in the environment over time. A
2 detailed discussion of these issues is outside the scope of this paper. However, a preliminary
3 analysis of drinking water sources that were sampled for more than one consecutive year reveals
4 that if MTBE is detected over consecutive years, it is usually detected for only one or two years
5 (see Table 2). For example, of the 21 drinking water sources with MTBE detections that were
6 sampled for four consecutive years, ten sources had MTBE detections in only one year, eight
7 sources had MTBE detections in two consecutive years, one source had MTBE detections in
8 three consecutive years, and two sources had MTBE detections in four consecutive years.

9 *INSERT TABLE 2*

10 Table 3 summarizes the location and date of the highest (top 20) reported MTBE
11 concentrations in drinking water sources in California from 1995 to 1999. Drinking water
12 sources in Los Angeles accounted for 17 (85%) of the highest reported levels of MTBE, with a
13 maximum concentration of 610 ppb reported for Charnock Well 13. Note that all of the Los
14 Angeles wells reported in Table 3 are currently on “standby,” which suggests that they are out-
15 of-service but are available as needed. Two drinking water sources in Yuba (Well 03-01) and El
16 Dorado (Tata Well 04) also reported high concentrations of MTBE. Most of the drinking water
17 samples with the highest reported MTBE concentrations were collected in 1996, with two
18 samples collected in 1999.

19 *INSERT TABLE 3*

20 **HOUSEHOLD EXPOSURES TO MTBE FROM DRINKING WATER**

21 To estimate potential human exposures to MTBE in California drinking water, we
22 conducted a probabilistic analysis of household exposures from the ingestion of MTBE in
23 drinking water, dermal contact with MTBE during showering, and the inhalation of MTBE from

1 volatilized water in the home. This latter scenario includes exposure to volatilized MTBE during
 2 showering, in the bathroom, and in the household from multiple sources (e.g., washing dishes,
 3 washing clothes, etc.). Exposure calculations are based on the CalTOX Multimedia Total
 4 Exposure Model by Cal-EPA (1994). Although not particularly important, this model assumes
 5 that everyone showers rather than bathes, which tends to overpredict the actual risk for those
 6 who bathe because of conservative assumptions about inhaled MTBE vapors during showering.

7 To account for the variability and uncertainty in the exposure model, all input parameters
 8 were characterized by distributions rather than point estimates, where appropriate (see Table 4).
 9 The probabilistic analysis was performed using Crystal Ball software and the Latin Hypercube
 10 sampling method for 10,000 iterations. The average daily dose (ADD) and lifetime average
 11 daily dose (LADD) of MTBE were estimated by aggregating doses from each of the three
 12 exposure pathways, which in turn, were calculated using the following equations:

13

14 Ingestion of MTBE in Drinking Water

$$15 \quad LADD \text{ or } ADD = \frac{C * IR * A_{ingest} * EF * ED}{BW * AT}$$

16

17 Inhalation of MTBE from Volatilized Water

$$18 \quad LADD \text{ or } ADD = \frac{[(C_s * ET_s) + (C_b * ET_b) + (C_h * ET_h)] * BR * A_{inhal} * EF * ED}{BW * AT}$$

19

20 Dermal Contact with MTBE during Showering

$$21 \quad LADD \text{ or } ADD = \frac{C * SA * PC * F * ET_s * CF * EF * ED}{BW * AT}$$

1 where:

2 ADD Average daily dose (mg/kg-day);

3 LADD Lifetime average daily dose (mg/kg-day);

4 C Concentration of MTBE in drinking water (mg/L);

5 EF Exposure frequency (days/year);

6 ED Exposure duration (years);

7 BW Body weight (kg);

8 AT Averaging time (days);

9 IR Drinking water ingestion rate (L/day);

10 A_{ingest} Oral absorption of MTBE (unitless);

11 C_s, C_b, C_h MTBE concentrations in shower air, bathroom air, and household air,
12 respectively (mg/m^3);

13 ET_s, ET_b, ET_h Exposure time in the shower, bathroom, and house, respectively (hrs/day);

14 BR Breathing rate (m^3/hr);

15 A_{inhal} Lung absorption of MTBE (unitless);

16 SA Surface area of the skin (cm^2);

17 PC Permeability coefficient (cm/hr);

18 F Fraction of skin in contact with water (unitless); and

19 CF Conversion factor ($0.001 \text{ L}/\text{cm}^3$).

20 Three separate drinking water concentration distributions for MTBE were used in the
21 probabilistic exposure assessment. Each distribution was based on the empirical data for
22 sampled drinking water sources from 1995 through 1999, but different methods were used to
23 estimate non-detect values. As mentioned, two distributions were used in our analysis, to bound

1 MTBE concentrations by assuming that non-detects were either equal to zero or the DLR. The
2 third distribution was based only on samples with detectable levels of MTBE, thereby
3 representing a more highly exposed population group.

4 Because total skin surface area, drinking water ingestion rate, and breathing rate are
5 correlated with body weight, the distributions of the ratios of these parameters with body weight
6 provided by Cal-EPA (1994) were used. These correlations are based on the ratios of body
7 weight to contact rate for household residents of all ages. Default values provided by USEPA
8 (1989) were used for estimates of exposure frequency and averaging time, and distributions
9 provided by Finley *et al.* (1994) were used for other input parameters. Based on review of the
10 available literature, the percent absorption of MTBE via ingestion was assumed to be 100%
11 (ATSDR, 1998; OEHHA, 1999). Research suggests that MTBE absorption via inhalation is less
12 than 50%, and is likely to range from 32 to 42% (Pekari *et al.*, 1996; Johanson *et al.*, 1995). The
13 permeability coefficient was estimated from the log K_{ow} and molecular weight of MTBE.
14 Volatilized concentrations of MTBE in the shower, bathroom, and household air were calculated
15 using the following equation by Finley *et al.* (1993):

$$16 \quad C_i = \frac{W_i * \phi_i * C}{VR_i}$$

17 where:

18 C_i MTBE air concentration in the i th compartment (shower, bathroom, or house)
19 (mg/m^3);

20 W_i Water use rate in the i th compartment (L/hr);

21 ϕ_i Mass transfer efficiency from water to air for the i th compartment (unitless);

22 C MTBE groundwater concentration (mg/L); and

23 VR_i Air exchange rate (m^3/hr).

INSERT TABLE 4

Figure 3 illustrates the distribution of average daily MTBE dose by exposures route for the general population in California for the more conservative scenario (i.e., non-detects = DLR). The estimated ADD of MTBE from all routes of exposure is about 0.17 $\mu\text{g}/\text{kg}/\text{day}$ at the 50th percentile and 0.38 $\mu\text{g}/\text{kg}/\text{day}$ at the 95th percentile. Note that exposures via ingestion account for the greatest contribution to total MTBE daily dose at the 50th percentile, but inhalation exposures (mostly from the shower) account for the greatest contribution to total MTBE dose at the 95th percentile. Dermal contact accounts for less than 5% of the total daily MTBE dose.

INSERT FIGURE 3

The distribution of MTBE average daily dose by exposure route for more highly exposed households is presented in Figure 4. In this scenario, the estimated ADD for MTBE drinking water exposures is about 0.1 $\mu\text{g}/\text{kg}/\text{day}$ and 1.4 $\mu\text{g}/\text{kg}/\text{day}$ at the 50th and 95th percentiles, respectively. Exposures via ingestion account for the greatest contribution to total MTBE daily dose at the 50th and 95th percentiles.

*INSERT FIGURE 4***MTBE TOXICITY AND CARCINOGENIC POTENTIAL**

The toxicology of MTBE has been reviewed extensively by others and is discussed here only briefly (HEI, 1996; Clary, 1997; Mennear, 1997; UC, 1998; USEPA, 1997; NSTC, 1997; ATSDR, 1998; OEHHA, 1999). Animal studies suggest that oral exposures to MTBE do not affect the cardiovascular, musculoskeletal, dermal, ocular, or reproductive systems, and limited effects have been reported for the gastrointestinal, hematological, respiratory, hepatic, renal, and neurological systems in acute and subchronic exposure studies (ATSDR, 1998). Two subchronic

1 (Johnson *et al.*, 1992; Klan *et al.*, 1992; Robinson *et al.*, 1990) and one chronic (Belpoggi *et al.*,
2 1995) oral exposure studies have evaluated the systemic toxicity of MTBE. Based on these
3 studies, the no-observed-adverse-effect level (NOAEL) for systemic effects in rodents associated
4 with subchronic and chronic oral exposures to MTBE ranges from 90 to 1,750 mg/kg/day. In
5 general, the renal and hepatic systems appear to be the most sensitive to MTBE exposures.
6 Johnson *et al.* (1992) and Robinson *et al.* (1990) reported NOAELs of 90 and 100 mg/kg/day,
7 respectively, for increased kidney weight associated with oral MTBE exposures. Similar
8 systemic effects were observed in inhalation exposure studies (ATSDR, 1998), and the NOAEL
9 for renal effects associated with chronic exposures of MTBE via inhalation is 400 ppm (Chun *et*
10 *al.*, 1992).

11 No epidemiological data on long-term systemic effects or carcinogenic effects of human
12 exposure to MTBE have been reported in the literature (OEHHA, 1999; ATSDR, 1998). A
13 limited number of studies, however, have examined potential acute human health effects from
14 exposure to MTBE in gasoline vapors. These studies were recently reviewed by Borak *et al.*
15 (1998). Self-reported symptoms from MTBE inhalation exposures include headache, eye
16 irritation, burning of the nose and throat, cough, nausea, and dizziness (Mohr *et al.* 1994;
17 Anderson *et al.* 1995). Although there is anecdotal evidence that inhaled MTBE may cause
18 short-term health effects, the available studies have not shown a significant correlation between
19 the reported acute health effects and the MTBE exposures experienced by the general public
20 from the use of MTBE in gasoline (ATSDR, 1998; Balter, 1997; NSTC, 1996, 1997; USEPA,
21 1997). Mehlman (1998) reports that exposures to water contaminated with MTBE-containing
22 gasoline may produce neurotoxic and respiratory effects in some individuals. No studies were
23 identified on the acute effects of ingested MTBE or dermal contact with MTBE. However, pure

1 MTBE has been used in medicine since the mid-1980s to dissolve gallstones, resulting in human
2 intake of up to 500 mL of MTBE (Hellstern *et al.*, 1998; Saraya *et al.*, 1990). Aside from short-
3 term effects such as nausea, vomiting, and mild drowsiness, no lasting side effects have been
4 associated with the clinical use of MTBE (ECETOC, 1997).

5 The EPA has not yet derived a chronic reference dose (RfD) for the ingestion of MTBE.
6 Extrapolation from the chronic reference concentration (RfC) of 3 mg/m³ for inhaled MTBE
7 (IRIS, 2000) yields an equivalent oral dose of approximately 1 mg/kg/day (Dourson and Felter,
8 1997). ATSDR (1998) has established a Minimal Risk Level (MRL) of 0.3 mg/kg/day for
9 intermediate-duration oral exposures to MTBE based on the lowest-observable-adverse-effect
10 level for hepatic effects from Robinson *et al.* (1990). The U.S. EPA (1997) has also proposed a
11 Drinking Water Advisory Consumer Acceptability Advice of 20–40 ppb MTBE based on
12 organoleptic effects.

13 The primary health effect of interest in chronic inhalation and oral MTBE exposure
14 studies is cancer. No data are available on the carcinogenicity of MTBE to humans (OEHHA,
15 1999; ATSDR, 1998). The carcinogenicity of MTBE has been investigated in male and female
16 Sprague-Dawley rats administered MTBE by gavage (Belpoggi *et al.*, 1995, 1997, 1998) and in
17 male and female Fisher 344 rats (Bird *et al.*, 1997; Chun *et al.*, 1992) and CD-1 mice (Bird *et al.*,
18 1997; Burleigh-Flayer *et al.*, 1992) exposed to MTBE by inhalation. In rats exposed by gavage,
19 a statistically significant increase in Leydig cell tumors of the testes was reported, as well as a
20 statistically significant increase in combined lymphomas and leukemias in females. In rats
21 exposed to MTBE by inhalation, a statistically significant increase in renal tubular tumors and
22 Leydig cell tumors of the testes were observed in males. A statistically significant increase in
23 the incidence of liver tumors was also observed in female and male mice exposed to MTBE by

1 inhalation. Although these findings suggest that MTBE is carcinogenic to animals, the available
2 bioassay data have several important limitations. First, significant increases in MTBE-related
3 tumors have occurred primarily at the highest administered doses, which are generally toxic to
4 test animals (Mennear, 1997). Second, some of the observed tumor sites by other measurement
5 endpoints (e.g., Leydig cell of the testes) are those that have historically had a high background
6 rate of tumor occurrence in rodents, making it difficult to assess the statistical and biological
7 significance of such findings (Mennear, 1997). Third, limited pathology reporting makes it
8 difficult to interpret the increased incidence of combined lymphomas and leukemias reported by
9 Belpoggi *et al.* (1995).

10 To date, no national or international regulatory agency has classified MTBE as a known
11 human carcinogen, and the available genotoxicity data suggest that MTBE is not highly
12 mutagenic (ECETOC, 1997). However, the U.S. EPA (1997) has determined that MTBE is an
13 animal carcinogen and poses a carcinogenic potential to humans. The California Office of
14 Environmental Health Hazard Assessment also considers MTBE to be an animal carcinogen and
15 a possible human carcinogen (OEHHA, 1999). Based on the presumption that MTBE is
16 carcinogenic to humans, OEHHA (1999) recently derived an upper-bound cancer slope factor
17 (CSF) for MTBE in drinking water of $1.8 \times 10^{-3} \text{ (mg/kg/day)}^{-1}$. This estimate is based on the
18 geometric mean of three potency estimates obtained from Chun *et al.* (1992) and Belpoggi *et al.*
19 (1995) for which tumors were observed at multiple target sites and under inhalation and gavage
20 MTBE dosing regimes. A modified physiologically based pharmacokinetic (PBPK) model was
21 also used to estimate the absorbed dose of MTBE in animals (OEHHA, 1999).

22

1 ESTIMATED MTBE LIFETIME EXPOSURES AND HEALTH RISKS

2 The estimated lifetime cancer risk from drinking water exposures to MTBE, assuming
3 that MTBE is carcinogenic to humans, is calculated by the following equation:

$$4 \text{ Risk} = \text{LADD} \times \text{CSF}$$

5 where:

6 Risk Lifetime cancer risk from MTBE exposures;

7 LADD Sum of lifetime average daily dose for all three exposure pathways
8 (mg/kg/day); and

9 CSF Theoretical upper-bound cancer potency of MTBE (mg/kg/day)⁻¹

10 The LADD was calculated for three different exposure durations. First, a five-year
11 exposure duration was used to estimate potential cancer risks based on MTBE drinking water
12 exposures in California from 1995 through 1999. Second, an 8-year exposure duration was used
13 to estimate potential cancer risks based on past and projected exposures to MTBE from 1995
14 through 2002—i.e., the phase-out date for MTBE. Because MTBE drinking water exposures
15 may not cease after MTBE use is discontinued, due to a lag time in the fate and transport of
16 released MTBE in groundwater, a third exposure duration of 13 years was evaluated (i.e., 1995–
17 2007). This latter exposure duration was selected after modeling the maximum time it would
18 take for MTBE to be observed at a drinking water well 100 meters downgradient of a leaking
19 underground storage tank. Assuming an infinite source and sandy-loam soil conditions,
20 preliminary modeling efforts suggest that MTBE would reach steady state in about 5 years.

21 The OEHHA (1999) cancer slope factor was used to estimate potential carcinogenic risks
22 to Californians from exposures to MTBE in drinking water. We rely on the OEHHA (1999)
23 estimate for illustrative purposes only, and do not make any claims about whether MTBE is

1 likely to be carcinogenic to humans, or about the reliability of the estimated cancer slope factor.
2 Table 4 provides the estimated LADDs and upper-bound cancer risk associated with household
3 exposures to MTBE in drinking water for the general population and more highly exposed
4 individuals. The estimated lifetime MTBE cancer risk for the general population in California at
5 the 50th percentile (assuming non-detects = DLR) is 2×10^{-8} for a 5-year exposure period, 3×10^{-8}
6 for an 8-year exposure period, and 6×10^{-8} for a 13-year exposure period. At the 95th percentile,
7 estimated MTBE cancer risks over a lifetime are 5×10^{-8} , 8×10^{-8} , and 1×10^{-7} at 5, 8, and 13 years,
8 respectively. Exposures to MTBE in drinking water are also estimated to pose a negligible
9 lifetime cancer risk (i.e., less than 1 per million) for more highly exposed individuals. For
10 example, the estimated lifetime cancer risk for more highly exposed persons in California at the
11 95th percentile is 5×10^{-7} for a 13-year exposure period.

12 *INSERT TABLE 5*

13 To assess potential non-cancer health effects from MTBE exposures, estimated MTBE
14 concentrations from all routes were compared to the chronic RfC reported in IRIS (2000) of
15 3 mg/m^3 for inhalation exposures, and the intermediate MRL reported in ATSDR (1998) of
16 0.3 mg/kg/day for ingestion and dermal exposures. Specifically, the following equation was
17 used to establish a Hazard Index (HI) for MTBE in drinking water:

18
$$HI = HQ_{inhalation} + HQ_{ingestion} + HQ_{dermal}$$

19 where:

20 $HQ_{inhalation}$ Hazard quotient for inhalation estimated as the concentration of MTBE
21 inhaled \div RfC;

22 $HQ_{ingestion}$ Hazard quotient for ingestion estimated as the ADD from oral MTBE
23 exposures \div oral MRL; and

1 HQ_{dermal} Hazard quotient for dermal uptake estimated as the absorbed dermal
2 MTBE dose \div oral MRL.

3 A hazard index less than one ($HI < 1$) means that estimated exposures are not expected to
4 pose an adverse health hazard. In Table 6, the HI for MTBE is significantly less than one at the
5 95th percentile for the general population and more highly exposed individuals, suggesting that
6 household exposures to MTBE in drinking water are not expected to pose significant non-cancer
7 health effects in California.

8 *INSERT TABLE 6*

9 **DISCUSSION**

10 When Congress first decided that the use of oxygenates in gasoline could improve air
11 quality in the U.S., several choices of oxygenating additives were available (NFGH, 1999). For
12 a number of reasons, MTBE was selected by refiners as the most appropriate option and soon
13 became the “oxygenate of choice” in the U.S. Although it is not clear from the historical record
14 which factors were most influential in the selection of MTBE in favor of existing alternatives,
15 this chemical appears to have had many of the characteristics that would be sought in a fuel
16 additive. The available data suggested that MTBE had moderately low acute toxicity (certainly
17 no greater than conventional gasoline), was an effective fuel oxygenate, and could be produced
18 in large quantities within a reasonable period of time. MTBE was also not considered to be
19 genotoxic, and therefore, was not suspected to pose a cancer hazard. The excellent warning
20 properties (odor) of MTBE, which could help prevent exposure to contaminated drinking water
21 by humans, may also have been a consideration in the decision-making process.

22 However, MTBE also had certain characteristics that could be problematic from an
23 environmental or public health perspective. The degradation or environmental half-life of

1 MTBE in surface soil or surface water was acceptable, but little was known about the
2 degradation of MTBE in groundwater, where volatilization would be negligible. The chemical
3 properties of MTBE also suggested that this compound would be very mobile in groundwater,
4 which could pose a problem if MTBE was released in underground aquifers due to leaking
5 underground storage tanks. On the other hand, if MTBE traveled faster than other gasoline
6 constituents in a groundwater plume, then MTBE would pose a public health advantage because
7 its odor would prevent persons from drinking water contaminated with gasoline's more toxic
8 constituents such as benzene. The use of additives to prevent human overexposure to toxic
9 agents is not uncommon, such as the addition of hydrogen sulfide to natural gas. It is true,
10 however, that in some environments benzene and other gasoline constituents might never reach a
11 drinking water well where MTBE was detected due to the relatively rapid biodegradation of
12 these constituents. Regardless, the EPA announced a 10-year program to upgrade, replace, or
13 close leaking underground storage tanks in 1988, which suggested that the frequency of gasoline
14 leaks should have been minimal in subsequent years (NFGH, 1999).

15 As evidenced by the widespread publicity and controversy surrounding MTBE in recent
16 years, it is clear that much of the forethought that went into the initial selection of MTBE as a
17 fuel oxygenate has been largely forgotten. In particular, the threat of MTBE contamination of
18 drinking water supplies can arguably be attributed to continuing problems with underground fuel
19 storage tanks, including poor management practices (e.g., tanks are overfilled), the continued
20 presence of old leaking tanks in the ground, and a lack of regulatory enforcement for leaking
21 underground tanks (BNA, 2000e; Davidson and Creek, 1999; White, 1999). To reach an
22 informed decision about the impact of leaking underground storage tanks on public health,
23 decisionmakers will need an analysis of all of the relevant information. The State of California

1 has historically relied extensively on the use of MTBE to meet air quality standards, yet it was
2 one of the first states to make policy decisions about eliminating MTBE from reformulated
3 gasoline. A careful review of the drinking water data in California therefore should be useful in
4 helping other states and regulatory bodies reach their own decisions about the future use of
5 MTBE as a fuel oxygenate.

6 Preliminary review of the available monitoring data suggests that the percentage of
7 sampled drinking water sources with detectable levels of MTBE in California is quite low (i.e.,
8 less than 2%). Detections of MTBE in drinking water, as well as average MTBE concentrations,
9 have remained relatively stable from 1995 through 1999. This finding is in contrast to many
10 media reports that suggest MTBE contamination of public drinking water supplies is widespread
11 and growing. A major limitation of this evaluation, however, is the inability to evaluate potential
12 MTBE contamination of drinking water sources over a longer time horizon. Although some
13 research suggests that MTBE leaks from underground storage tanks may represent a significant
14 threat to drinking water over the next decade (Johnson *et al.*, 2000), such predictions are not
15 readily apparent based on existing monitoring data.

16 Our probabilistic analysis of household exposures to MTBE in drinking water suggests
17 that past and predicted future MTBE exposures from contaminated water supplies are unlikely to
18 pose a significant health risk for the general population or more highly exposed individuals in
19 California. However, there are some uncertainties in this assessment due to incomplete data on
20 MTBE. For example, even though there is no clear evidence to indicate that this is likely, certain
21 segments of the population, such as young children and the elderly, could potentially be more
22 susceptible to the toxic effects of MTBE than the general population (ATSDR, 1998). The
23 current analysis is also based on available data for public drinking water supplies in California

1 and does not consider potential MTBE contamination of private drinking water wells. Estimated
2 exposures to MTBE in the future are based on the assumption that the incidence of MTBE
3 detections and the levels of MTBE in drinking water will not change dramatically in California
4 from current estimates. In reality, household drinking water exposures to MTBE may increase or
5 decrease over time depending on many factors, including future releases of MTBE in the
6 environment, the transport and degradation rate of MTBE in groundwater, and ongoing efforts to
7 upgrade or retrofit leaking underground storage tanks.

8 Despite these limitations, our analysis should provide important information to decision-
9 makers both within and outside the state of California. The data clearly indicate that few
10 drinking water sources in California are contaminated with MTBE. It also appears that few, if
11 any, persons in California have been exposed to concentrations of MTBE in drinking water that
12 might pose a significant health risk. Further, it is possible that in some instances the presence of
13 MTBE in contaminated drinking water has been beneficial from a public health standpoint,
14 because exposure to potentially hazardous concentrations of gasoline in groundwater would be
15 avoided. On the other hand, if regulatory bodies are unable to implement an effective plan for
16 dealing with leaking underground storage tanks, the future use of MTBE as an oxygenate will
17 create a number of challenges. For example, it has become apparent that MTBE moves quickly
18 in groundwater and is slow to degrade in groundwater. Remediation of MTBE-contaminated
19 water may also be more difficult compared to remediation of conventional gasoline plumes.
20 Finally, the toxicological database on MTBE is not complete or necessarily robust, and there is
21 some possibility that human exposures to relatively low concentrations of MTBE might pose a
22 chronic health risk.

1 Due to its relatively low taste and odor threshold (i.e., as low as 2 ppb), MTBE in
2 drinking water above certain concentrations clearly poses an aesthetic concern. However,
3 drinking water concentrations of aesthetic concern, or those used to establish secondary drinking
4 water standards, do not necessarily pose a public health hazard. Therefore, decisions about
5 MTBE need to rely on accurate assessments of MTBE exposure and the possible health risks, as
6 well as a comprehensive assessment of the risks and benefits of MTBE relative to other gasoline
7 formulations.

8

9

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1 Table 1. Detections of MTBE in sampled drinking water sources in California from 1995–1999.

Year	Number of Sampled Drinking Water Sources	Number of Sources with MTBE Detections	Percentage of Sources with MTBE Detections
1995	117	4	3.4%
1996	1,677	22	1.3%
1997	2,027	30	1.5%
1998	2,435	44	1.8%
1999	1,951	35	1.8%

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4

- 1 Table 2. MTBE detections in California drinking water for sources sampled for more than one
 2 consecutive year.

Number of Consecutive Years Sampled	Number of Sources with MTBE Detections in Consecutive Years				
	One	Two	Three	Four	Five
Two	17	11	--	--	--
Three	6	8	4	--	--
Four	10	8	1	2	--
Five	3	0	1	1	0

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- 1 Table 3. Twenty highest reported MTBE concentrations in California drinking water sources
 2 from 1995–1999.

Source Name	City	Sampling Date	MTBE Level (ppb)
CHARNOCK WELL 19 - STANDBY	LOS ANGELES	03/25/96	610
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	03/12/96	490
CHARNOCK WELL 19 - STANDBY	LOS ANGELES	03/12/96	490
CHARNOCK WELL 19 - STANDBY	LOS ANGELES	02/26/96	400
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	03/19/96	340
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	03/22/96	340
CHARNOCK WELL 19 - STANDBY	LOS ANGELES	02/26/96	300
CHARNOCK WELL 19 - STANDBY	LOS ANGELES	02/07/96	290
CHARNOCK WELL 13 – STANDBY	LOS ANGELES	10/17/95	250
WELL 03-01	YUBA	08/02/99	234
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	02/26/96	136
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	02/07/96	130
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	02/26/96	130
WELL 03-01	YUBA	01/15/97	115
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	08/28/95	95
ARCADIA WELL 05 - STANDBY	LOS ANGELES	09/04/96	87
CHARNOCK WELL 13 - STANDBY	LOS ANGELES	05/14/96	81
CHARNOCK WELL 15 - STANDBY	LOS ANGELES	06/13/96	73
ARCADIA WELL 05 - STANDBY	LOS ANGELES	08/26/96	72
TATA WELL 04 (ANGORA WELL 10)	EL DORADO	07/27/99	68

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1 Table 4. Description of input parameters used in household probabilistic exposure model.

Input Parameter	Symbol	Distribution Type	Distribution Parameters
MTBE concentration in drinking water: upper bound (mg/L)	C	Empirical	Based on assumption that non-detects equal DLR. Mean = 0.005; std. dev. = 0.006
MTBE concentration in drinking water: lower bound (mg/L)	C	Empirical	Based on assumption that non-detects equal zero. Mean = 0.0002; std. dev. = 0.0057
MTBE concentration in drinking water: detects only (mg/L)	C	Empirical	Based on dataset containing only detected concentrations. Mean = 0.012; std. dev. = 0.039
Drinking water ingestion rate per body weight (L/kg-day)	IR/BW	Lognormal	Mean = 0.022; std. dev. = 0.0044
Breathing rate per body weight (m ³ /kg-hr)	BR/BW	Lognormal	Mean = 0.0127; std. dev. = 0.00413
Total skin surface area per body weight (cm ² /kg)	SA/BW	Lognormal	Mean = 0.026; std. dev. = 0.00182
Absorption via ingestion	A _{ingest}	--	1
Absorption via inhalation	A _{inhal}	Uniform	Minimum=0.32; maximum=0.42
Averaging time (days)	AT	--	25,550 days
Exposure duration (years)	ED	--	5, 8, or 13 years
Exposure frequency (days/year)	EF	--	350 days/year
Exposure time – shower (hrs/day)	ET _s	Lognormal	Mean = 0.13; std. dev. = 0.085

Input Parameter	Symbol	Distribution Type	Distribution Parameters
Exposure time – bathroom (hrs/day)	ET_b	Lognormal	Mean = 0.33; std. dev. = 0.22
Exposure time – house (hrs/day)	ET_h	Uniform	Min. = 8; max. = 20
Water use rate – shower (L/hr)	W_s	Lognormal	Mean = 480; std. dev. = 160
Water use rate – bathroom (L/hr)	W_b	Lognormal	Mean = 40; std. dev. = 15
Water use rate – house (L/hr)	W_h	Lognormal	Mean = 40; std. dev. = 15
Mass transfer efficiency – shower (unitless)	ϕ_s	Triangular	Mode = 0.57; min. = 0.1; max. = 0.9
Mass transfer efficiency – bathroom (unitless)	ϕ_b	Triangular	Mode = 0.44; min. = 0.1; max. = 0.9
Mass transfer efficiency – house (unitless)	ϕ_h	Triangular	Mode = 0.44; min. = 0.1; max. = 0.9
Air exchange rate – shower (m^3/hr)	VR_s	Uniform	Min. = 4; max. = 20
Air exchange rate – bathroom (m^3/hr)	VR_b	Uniform	Min. = 10; max. = 100
Air exchange rate – house (m^3/hr)	VR_h	Uniform	Min. = 300; max. = 1200
Permeability coefficient (cm/hr)	PC	--	0.054 cm/hr
Fraction of skin in contact with water (unitless)	F	Uniform	Min. = 0.4; max. = 0.9

- 1 Table 5. Estimated lifetime average daily doses (LADDs) and cancer risk for Californians
 2 exposed to MTBE in drinking water.

Exposure Duration	LADD (mg/kg/day)		Cancer Risk	
	50 th Percentile	95 th Percentile	50 th Percentile	95 th Percentile
<i>General Population^a</i>				
1995–1998 (5 years)	1.2x10 ⁻⁵	2.7x10 ⁻⁵	2x10 ⁻⁸	5x10 ⁻⁸
1995–2002 (8 years)	1.9x10 ⁻⁵	4.3x10 ⁻⁵	3x10 ⁻⁸	8x10 ⁻⁸
1995–2008 (13 years)	3.1x10 ⁻⁵	7.0x10 ⁻⁵	6x10 ⁻⁸	1x10 ⁻⁷
<i>Highly Exposed Population^b</i>				
1995–1998 (5 years)	7.3x10 ⁻⁶	9.9x10 ⁻⁵	1x10 ⁻⁸	2x10 ⁻⁷
1995–2002 (8 years)	1.2x10 ⁻⁵	1.6x10 ⁻⁴	2x10 ⁻⁸	3x10 ⁻⁷
1995–2008 (13 years)	1.9x10 ⁻⁵	2.6x10 ⁻⁴	3x10 ⁻⁸	5x10 ⁻⁷

3 ^aAssumes that non-detect drinking water samples are equal to the reporting detection limit.

4 ^bBased on drinking water samples with detectable MTBE levels.

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1 Table 6. Estimated hazard quotients (HQs) and hazard index (HI) for Californians exposed to
 2 MTBE in drinking water.

Exposure Route	HQ or HI	
	50 th Percentile	95 th Percentile
<i>General Population^a</i>		
Inhalation	6.5x10 ⁻⁵	3.0x10 ⁻⁴
Ingestion	3.4x10 ⁻⁴	4.8x10 ⁻⁴
Dermal	1.6x10 ⁻⁵	4.6x10 ⁻⁵
Total (all routes)	4.4x10 ⁻⁴	7.1x10 ⁻⁴
<i>Highly Exposed Population^b</i>		
Inhalation	4.0x10 ⁻⁵	7.7x10 ⁻⁴
Ingestion	2.0x10 ⁻⁴	2.4x10 ⁻³
Dermal	8.7x10 ⁻⁵	1.6x10 ⁻⁴
Total (all routes)	2.6x10 ⁻⁴	3.2x10 ⁻³

3 ^aAssumes that non-detect drinking water samples are equal to the reporting detection limit.

4 ^bBased on drinking water samples with detectable MTBE levels.

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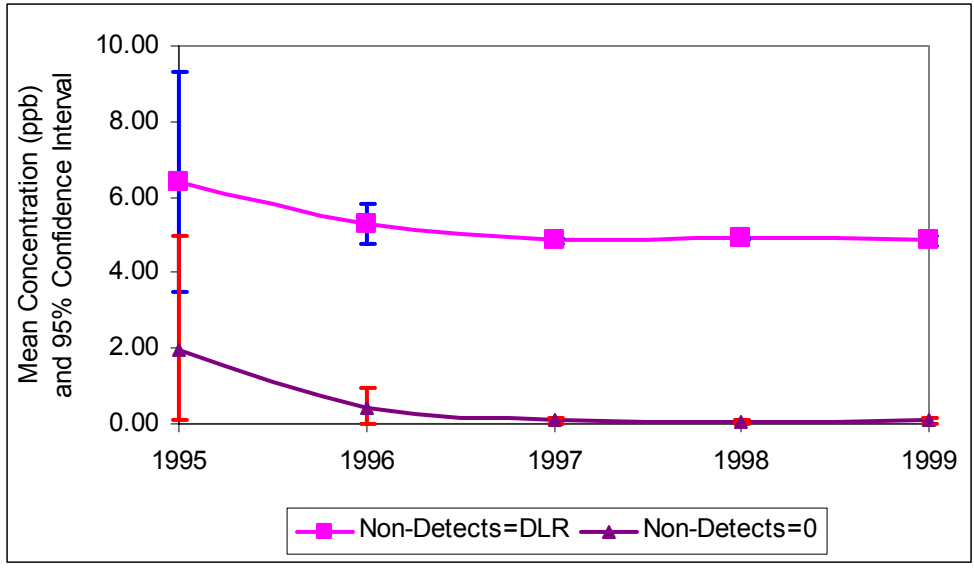
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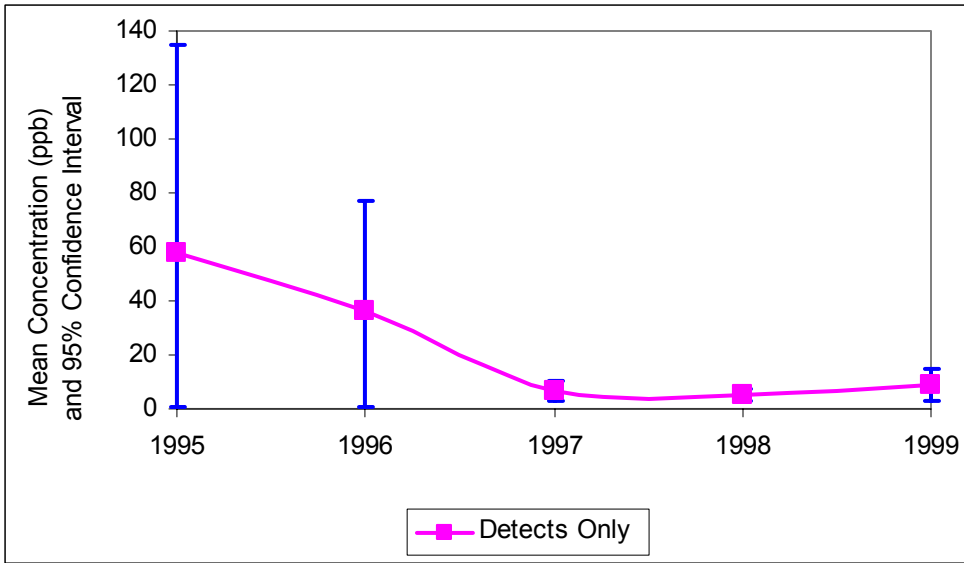
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1 Figure 1. Average MTBE concentration in California drinking water sources from 1995 to 1999
2 for all sampled sources.



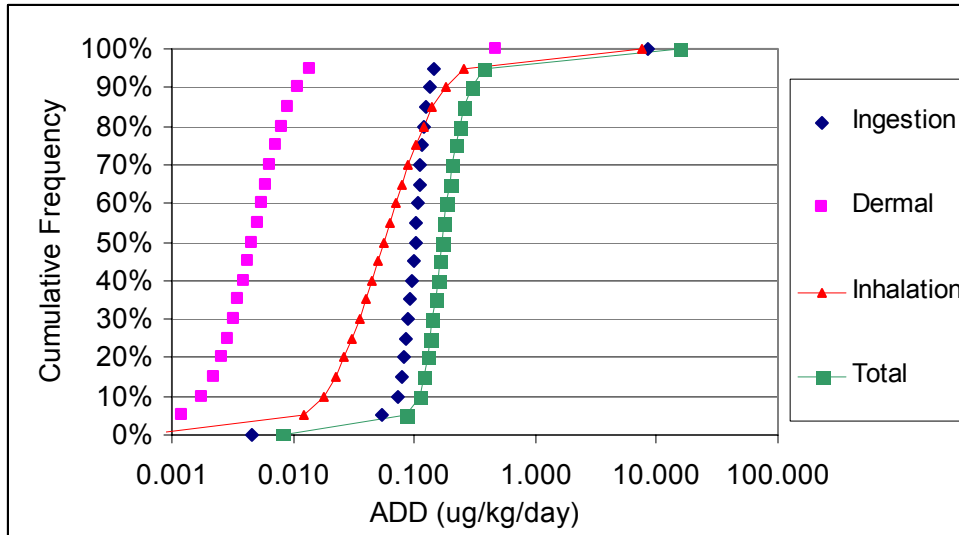
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1 Figure 2. Average MTBE concentration in California drinking water sources from 1995 to 1999
2 for sources with detectable levels of MTBE (detects only).



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1 Figure 3. Distribution of estimated average daily MTBE dose by exposure route for the general
2 population in California.



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1 Figure 4. Distribution of estimated average daily MTBE dose by exposure route for more highly
 2 exposed individuals in California.

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