

# Exposure of Humans to MTBE from Drinking Water

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## INTRODUCTION

Within a relatively short period of time, methyl tertiary butyl ether (MTBE) has become one of the most highly publicized and widely released contaminants of surface and ground waters. Introduced as a gasoline additive without adequate investigation of its fate, transport, and toxicity, it is now potentially a major threat to human health. Drinking water wells in several locations have been removed from service, and the State of California is faced with the task of identifying additional drinking water supplies that may be contaminated with MTBE. A major question that remains to be addressed is to what extent are members of the population exposed to MTBE from drinking water.

An analysis of exposure of humans to MTBE requires estimating the amount of the MTBE that contacts humans at the lungs, gastrointestinal tract, and skin surface during some specified period of time (U.S. EPA 1987). To perform such an analysis, it is necessary to understand differences among individuals with respect to body size, physiological processes, and behavior. These differences affect how individuals contact the chemical and how they process the chemical at the point of contact with the body. These differences can be explicitly incorporated into an exposure analysis by utilizing a probabilistic framework.

The probabilistic framework allows one to quantify exposure from the three pathways, inhalation, ingestion, and dermal, and to characterize the risk to a randomly selected individual in the population. Typically, this characterization is made by comparison of the estimated exposure to some reference dose that is selected to represent the amount to which an individual can be exposed without experiencing adverse health effects.

While exposure assessments may account for contact from all environmental media, typically they focus on a chemical that is found at a specific source that individuals are assumed to contact with greater frequency. When the source is surface or ground water used as a water supply, individuals may be exposed by all three pathways. Here we estimate the exposure of individuals in a population that draw water from a drinking water supply contaminated with MTBE. We incorporate individual variation by performing a probabilistic analysis to understand the range of possible exposures to MTBE in a water supply. We characterize the potential risk to these individuals by comparison of the McKone and Bogen (1992) framework to the proposed Office of Environmental Health Hazard Assessment (OEHHA) California Public Health

Goal risk analysis. We also evaluate the protectiveness of the PHG dose level by determining the probability that a randomly selected individual in the population will experience exposure to levels of MTBE greater than the PHG *de minimis* exposure/risk level. Finally, we determine the exposure of individuals to MTBE as a result of recreational activities in contaminated surface waters and as a result of consuming contaminated fish.

## EXPOSURE MODEL

The exposure model used in this analysis is based on the framework of McKone and Bogen (1992) who modeled the exposure to tetrachloroethylene from contaminated ground water. For each pathway, a Pathway Exposure Factor (PEF) is calculated which relates the concentration of the contaminant in water to the chronic daily intake during the exposure period. We use a probabilistic approach to incorporate uncertainty and variability in the parameters into the analysis.

### *Ingestion Exposure*

MTBE in tap water can be ingested by drinking and consuming foods prepared with tap water. Cooked foods probably contain little MTBE as it is volatilized during heating. However, many foods and drinks are prepared with water and are not heated.

The exposure due to ingestion is:

$$\begin{aligned} \text{Ingestion} &= C_w(\text{PEF}[\text{water} \rightarrow \text{water ingestion}]) \\ &= C_w(I_w/\text{BW}) \end{aligned}$$

Where  $C_w$  is the concentration of MTBE in tap water in mg/liters, and  $I_w/\text{BW}$  is ingestion rate per unit body weight in liters/kg-day. We used lognormal distribution for ingestion rate per unit body weight (Table 1) taken from McKone and Bogen (1992) which was determined from data compiled by the ICRP (ICRP 1975) and the U.S. EPA (U.S. EPA 1989).

### *Inhalation Exposure*

Inhalation of MTBE originating in tap water is the result of the volatilization during a variety of activities including showering/bathing, cooking, and laundering. Given the high volatility of MTBE, it is possible that a large portion of the exposure can be the result of inhalation. McKone's (1987) review found that inhalation of volatile chemicals in tap water could be as large or larger a component of the total exposure, than exposure due to ingestion. Concentrations of MTBE in indoor air are a function of the volatilization of MTBE out of the water into the indoor air. Volatilization is a function of the chemical mass transfer of MTBE from water to air and is governed by the diffusion of the contaminant in air and water, and the Henry's law constant for MTBE. The transfer efficiency is calculated relative to radon McKone (1987), and is discounted by the ventilation rate in the shower, bathroom, or house. Consequently, inhalation exposure would be increased with greater transfer

## Exposure of Humans to MTBE from Drinking Water

efficiency, and lower with an increase in ventilation rate in the house.

The exposure due to inhalation is:

$$\begin{aligned} \text{Inhalation exposure} &= C_w(\text{PEF}[\text{water} \rightarrow \text{inhalation}]) \\ &= C_w\{[\text{BR}/\text{BW}] * ([\text{Et}_s * W_s * \text{TE}_s/\text{VR}_s] + [\text{Et}_b * W_s * \text{TE}_s/\text{VR}_b] + \\ &\quad [\text{Et}_h * W_h * \text{TE}_h/\text{VR}_h]) / (24 \text{ hrs} / \text{d})\} \end{aligned}$$

where  $C_w$  is the concentration of MTBE in the tap water in mg/liter, BR/BW is breathing rate per unit body weight,  $\text{Et}_s$  is the exposure time in the shower in hours per day,  $W_s$  is the shower water use rate per person in liters per hour,  $\text{Et}_b$  is the exposure time in the bathroom in hours per day,  $W_h$  is the water use rate in the house in liters/hour,  $\text{Et}_h$  is the exposure time in the house in hours per day,  $\text{VR}_s$  is the ventilation rate in the shower in  $\text{m}^3$  per hour,  $\text{VR}_b$  is the ventilation rate in the bathroom in  $\text{m}^3$  per hour, and  $\text{VR}_h$  is the ventilation rate in the house in  $\text{m}^3$  per hour,  $\text{TE}_s$  is the transfer efficiency of MTBE from shower water to shower air, and  $\text{TE}_h$  is the transfer efficiency of MTBE from shower water to household air. Transfer efficiencies are calculated according to McKone and Bogen (1992) as:

$$\text{TE} = \text{TE}_{\text{radon}} * \{2.0 \times 10^6 (\text{m}^2/\text{sec})^{-2/3} / (2.5/D_w^{2/3} + [R * T * D_a^{2/3}/H])\}$$

where  $\text{TE}_{\text{radon}}$  is the transfer efficiency for radon,  $D_w$  is the diffusion coefficient of MTBE in water,  $D_a$  is the diffusion coefficient of MTBE in air,  $R$  is the Universal gas constant in Pa-liters/mol-K,  $T$  is the temperature in degrees Kelvin, and  $H$  is the Henry's law constant for MTBE in Pa-liters/mol.  $\text{TE}_{\text{radon}}$  is 0.70 for showers and 0.54 for all household water uses including showering (McKone and Bogen 1992).

### Dermal Exposure

Dermal contact with MTBE occurs as a result of showering, bathing, and general washing. The contact rate is the rate at which MTBE passes through the stratum corneum layer of the skin per unit body weight per hour. The model of McKone and Bogen (1992) is based on an earlier model by Brown et al. (1984). The assumptions of the model include: (1) dermal uptake is the result of passive diffusion through the stratum corneum, (2) once through the stratum corneum, resistance to diffusion through other layers is negligible, and (3) diffusive flux is proportional to the difference in the concentration of MTBE in tap water and internal body water. Similar to McKone and Bogen (1992), our model also assumes that the exposure time of children and adults during showering and bathing is the same, and that the exposure time of adults during showering and bathing is the same as exposure time for showering alone that we use in the simulations.

Exposure due to dermal contact is:

$$\text{Dermal exposure} = C_w(\text{PEF}[\text{water} \rightarrow \text{dermal uptake}])$$

## *Exposure of Humans to MTBE from Drinking Water*

$$= C_w([SA/BW] * F_{sa} * P * ET_s * CF)$$

where  $C_w$  is the concentration of MTBE in the water,  $[SA/BW]$  is the total skin surface area per unit body weight averaged over an individual's lifetime in  $m^2/kg$ ,  $F_{sa}$  is the fraction of the skin surface exposed during showering/bathing (unitless),  $P$  is the permeability constant for MTBE across the stratum corneum in  $m$  per hour,  $ET_s$  is the exposure time in the shower, and  $CF$  is a conversion factor to convert liters to  $m^3$  (liters/ $m^3$ ).

### *Distributions and Assumptions*

Distributions used in the analyses are taken from McKone and Bogen (Table 1). Total exposure is calculated as the sum of all three exposure pathways. The simulations use random values for the parameters that are selected from the distributions in Table 1. However, for any individual exposed to MTBE within his/her home, the concentration of MTBE in the water is the same for all exposure pathways, and the exposure time in the shower is the same for the inhalation and dermal exposure pathways. Therefore, each simulation was run with the same value for those parameters in all calculations, i.e., the correlation between these parameters was 1.0. No additional correlations were factored into the analysis.

The exposure calculations do not estimate the dose of MTBE that reaches the target organ(s). Experimental evidence indicates that 100% of MTBE entering the body via ingestion is moved to the target organs, while only 50% of the MTBE inhaled is retained in the body (Nihlen et al. 1998). Previous modeling studies indicated that ingestion and inhalation exposure are approximately equal, but that only 50% of inhaled MTBE retained (OEHHA draft report). Our simulations indicated that exposure from inhalation is approximately 2.4X greater than exposure due to ingestion (see below). Consequently, we performed two sets of simulations. Model I used the ingestion and dermal exposures calculated as above, and simply added 50% of the ingestion exposure value as the contribution from inhalation.

This exposure model is:

$$\text{Total Dose} = \text{Ingestion Exposure} + \text{Dermal Exposure} + (50\% \text{ of Ingestion Exposure})$$

Model II used the exposures calculated from the full exposure model above and reduced the inhalation exposure by one-half to agree with the experimental data. This exposure model is:

$$\text{Total Dose} = \text{Ingestion Exposure} + \text{Dermal Exposure} + (50\% \text{ of Inhalation Exposure})$$

### *Uncertainty Analyses*

Uncertainty analysis is defined (McKone and Bogen 1992) as "the determination of the variation or imprecision in an output function based on the collective

variation of model inputs.” Variation of model inputs is the result of normal random variability in the parameters and data used in the models, and the imprecision in the analysts understanding of the models, parameters, and/or their predictions (McKone and Bogen 1992). Three approaches are used in the assessment of uncertainty and sensitivity, (1) differential analysis, (2) response-surface analysis, and (3) Monte-Carlo methods. We used a variation of the Monte-Carlo methodology, Latin hypercube sampling. Latin hypercube sampling involves stratifying the cumulative probability distribution of an input parameter into intervals of equal probability, and sampling randomly from within each interval. Latin hypercube sampling is designed to accurately recreate the input distribution through sampling with fewer iterations as compared to simple Monte Carlo methods. Uncertainty in the response variable is represented by a distribution of output values that characterize the range of output responses.

#### *Sensitivity Analysis*

Sensitivity analysis is defined as “the determination of the changes in model response as a result of changes in individual model parameters” (McKone and Bogen 1992). Sensitivities were determined for the general exposure model by examining covariation in model inputs and response. Sensitivities can be used to evaluate which inputs are most critical to the model response and are particularly informative within a risk management framework.

#### *Probabilistic Framework*

All simulations were performed using @RISK (Palisade Corporation, NY), a software package that is an add-in to Microsoft® Excel. All simulations were performed using a Latin hypercube sampling with 10,000 iterations per simulation. It was determined that approximately 10,000 iterations were required for the simulations to reach convergence, even with the more efficient Latin hypercube sampling scheme. Convergence of an output distribution was defined as a change of less than 1.5% in the mean and variance of the distribution where the moments were calculated every 100 iterations. The number of stratifications in the Latin hypercube sampling was based on the number of iterations, i.e., 10,000 intervals were established in the input distributions. Sampling was conducted without replacement.

## **RESULTS**

#### *General Exposure Model - Deterministic Analysis*

A preliminary simulation of the exposure model was performed using the mean value of all the distributions as input parameters. This deterministic simulation was used to assess the general contributions of each exposure route to the total exposure. The total daily exposure is  $6.8 \times 10^{-4}$  mg/kg-d and the results indicate that the primary exposure pathway is inhalation followed by ingestion and dermal exposure. Dermal exposure is several orders of magnitude smaller than either ingestion or inhalation and made little impact on the results of the simulation. Inhalation exposure is approximately 2.4X greater than ingestion exposure.

## *Exposure of Humans to MTBE from Drinking Water*

Compared to a previous exposure analysis (OEHHA 1998), our relative exposures of inhalation and ingestion are somewhat different. Using the CalTox™ model, the OEHHA analysis found that inhalation exposure was between 45% and 110% of ingestion exposure, depending on the level of water intake. At higher levels of water intake, ingestion of MTBE was approximately twice the inhalation exposure (OEHHA 1998, Table 16). The OEHHA analysis further used experimental evidence to make the assumption that only one-half of the inhaled MTBE is retained, and the other half of the MTBE is exhaled (Nihlen et al. 1998). Therefore, the OEHHA analysis makes the assumption that exposure due to inhalation is about half of the exposure due to ingestion.

### *Exposure and the OEHHA Draft Public Health Goal*

The OEHHA draft document proposes 14 ppb MTBE in drinking water as the Public Health Goal (PHG). This level is the concentration of MTBE at which the increased risk of developing cancer is  $1 \times 10^{-6}$ . Using Model I and the 14 ppb PHG value as our target, we calculated the total exposure as  $6.8 \times 10^{-4}$  mg/kg-d. This is the average daily exposure at which the increased risk of cancer is  $1 \times 10^{-6}$  and is considered the *de minimis* exposure/risk level. We then determined the concentration of MTBE in drinking water that would be necessary to generate the *de minimis* exposure/risk level using the assumptions of Model II. This concentration is 10.1 ppb which reflects the increased exposure of MTBE from inhalation of indoor air.

Sensitivity analysis indicates that four factors possess relatively high positive sensitivities, i.e., changes in the values of those parameters result in large changes in the average exposure. Breathing rate per unit body weight had the highest sensitivity (.741) followed by shower duration (.219), water use rate in the shower (.176), and exposure time in the bathroom (.152). Negative sensitivities were associated with the ventilation rates in the home, i.e., greater ventilation rates in the home result in lower average exposure

### *Model I - Probabilistic Analysis*

Using the McKone and Bogen framework and the OEHHA PHG document assumptions (Model I), we performed a probabilistic risk assessment using 14 ppb as a point estimate for the concentration of MTBE in water, and the range of model parameter values and the distributions obtained from McKone and Bogen (1992). The mean average daily exposure is  $7.5 \times 10^{-4}$  mg/kg-d ( $\pm 5.6 \times 10^{-4}$  st. dev.), slightly above the *de minimis* exposure of  $6.8 \times 10^{-4}$  mg/kg-d. The range is very broad with a minimum average daily exposure of  $1.2 \times 10^{-4}$  mg/kg-d and a maximum exposure of  $130 \times 10^{-4}$  mg/kg-d. Examining the cumulative frequency distribution reveals that approximately 40% of the population is exposed to concentrations of MTBE that are above the *de minimis* exposure/risk level of  $6.8 \times 10^{-4}$  mg/kg-d. Therefore, variability in parameter values such as breathing rate per unit body weight, ingestion rate per unit body weight, shower use rate, and time spent indoors results in a substantial proportion of the population experiencing levels of exposure greater than the *de minimis* risk level.

We extended the analysis to determine the proportion of a population that would exceed the *de minimis* exposure/risk level when exposed to concentrations

## *Exposure of Humans to MTBE from Drinking Water*

of MTBE that range from 1 ppb to 30 ppb in their water supply. We used each concentration as a point estimate and the same parameter values and distributions as above. Results indicate that at least a portion of the population would be above the *de minimis* exposure/risk level at all concentrations of MTBE at or above 6 ppb (Table 2). If the PHG were set at 10 ppb, only 15-20% of the population would experience exposures greater than the  $10^{-6}$  *de minimis* level. At approximately 15 ppb, half of the population would be exposed above the *de minimis* exposure/risk level.

### *Model II – Probabilistic Analysis*

Using the McKone and Bogen framework and the assumption of a 50% dose from inhalation (Model II), we performed a probabilistic risk assessment using 14 ppb as a point estimate for the concentration of MTBE in water. The range of model parameter values and the distributions were the same as for the Model I simulations. The mean average daily exposure is  $11 \times 10^{-4}$  mg/kg-d ( $\pm 14 \times 10^{-4}$  st. dev.), well above the *de minimis* exposure/risk level of  $6.8 \times 10^{-4}$  mg/kg-d. The range is again very broad with a minimum average daily exposure of  $1.6 \times 10^{-4}$  mg/kg-d and a maximum exposure of  $610 \times 10^{-4}$  mg/kg-d. In this scenario, approximately 60% of the population would experience exposure above the *de minimis* exposure/risk level.

### *MTBE in drinking water supplies*

Drinking water can be obtained from either surface or groundwater, or a combination of the two. We therefore made additional estimates of exposure based on the modeled concentration of MTBE in surface and groundwater. A distribution of MTBE concentrations was obtained from a one-dimensional lake model developed to predict the concentration of MTBE in surface waters that support recreational boating and the use of personal watercraft. Model II was run as before using the model-generated distribution of MTBE concentrations with a mean of 9 ppb.

Average daily exposure was  $1.09 \times 10^{-3}$  mg/kg-d ( $\pm 1.5 \times 10^{-3}$ ) with inhalation exposure again being the greatest contributor to the total exposure. The maximum exposure was  $2.7 \times 10^{-2}$  mg/kg-d. The sensitivity analysis indicated that breathing rate had the highest positive sensitivity (.674) followed by concentration of MTBE in the water (.274), shower duration (.204), and water use rate in the shower (.172) exposure time in the bathroom (.126). Again, ventilation rate in the shower and the bathroom had negative sensitivities (-.149 and -.129 respectively).

### *Additional Exposure Scenarios*

Two additional exposure scenarios were explored, exposure due to swimming and exposure from consumption of contaminated fish. Exposure during swimming is due to dermal contact and incidental ingestion of water while swimming. We used a uniform distribution to represent time in the water assuming a minimum of 30 minutes per day and a maximum of 2 hours per day. The ingestion rate of water while swimming was the U.S. EPA default value of  $7 \times 10^{-4}$  liters per kilogram of body weight per day. It was assumed that during the entire time spent swimming, 65% of the body would be exposed to MTBE in the

water. Concentration of MTBE in the water was taken from an empirical distribution with a mean of 9 ppb. With these assumptions, the exposure during swimming is an order of magnitude lower than that for exposure to indoor water.

For the exposure analysis due to consumption of contaminated fish, we followed the Oregon Department of Environmental Quality guidelines and made the following assumptions (1) all fish consumed originated in a lake contaminated with MTBE, (2) the fish spent all of their time in the upper portions of the lake where the concentrations of MTBE are the highest, (3) the fish bioaccumulate MTBE at a rate of 1.5X the concentration of MTBE in the water, (4) fish are consumed daily, (5) between 20% to 60% of the fish is edible, and (6) all MTBE from the fish is absorbed in the gastrointestinal tract. Assumption four was made to account for a potential subsistence fishery scenario and is an extremely conservative assumption. Because of the differences in intake rate per unit body weight, we broke the analysis into three age groupings, child, juvenile, and adult. We used the same concentration of MTBE in the water as in the analysis for exposure while swimming. Even with the restrictive assumption of daily consumption of fish, average daily exposures are between 50% and 75% of the *de minimis* level generated by the 14 ppb PHG level. Using a less restrictive assumption of consumption of fish three times per week, the exposure drops to an order of magnitude lower than the *de minimis* exposure/ risk level.

## DISCUSSION

The difference between our analysis and the OEHHA analysis is approximately 30% (14 ppb vs 10 ppb). However, the OEHHA draft document states that due to differences in the Henry's law coefficient for MTBE and differences among individuals in water intake rate, the possible range of PHGs is 10 ppb to 18 ppb. Thus, our cancer risk level of 10 ppb is comparable to the low end of the OEHHA range.

Both of these estimates are based on a deterministic approach with single parameter values used in the analyses. Using a probabilistic approach and the assumptions of Model I, we determined that the potential range of exposures resulting from a 14 ppb concentration of MTBE in drinking water is very large ( $1.2 \times 10^{-4}$  to  $130 \times 10^{-4}$  mg/kg-d). Those individuals experiencing the largest exposures are at approximately double the *de minimis* risk of developing cancer. If we use the assumptions of Model II, the range is even greater ( $1.6 \times 10^{-4}$  to  $610 \times 10^{-4}$  mg/kg-d). Those individuals experiencing the greatest exposure are at an order of magnitude greater risk than the  $10^{-6}$  *de minimis* exposure/ risk level.

Due to variation in the size of individuals, the rate at which they ingest water, the rate at which they breathe, and their behavior, e.g., shower duration, approximately 35-40% of the population could experience exposures above the *de minimis* exposure/ risk level. The parameter with the highest sensitivity is breathing rate per unit body weight, a factor not generally responsive to risk management options. Other factors with high sensitivity are also not amenable

## *Exposure of Humans to MTBE from Drinking Water*

to risk management as these are at the discretion of individuals, e.g., ventilation rates in the home, the amount of water used in showering and bathing, and the duration of showers.

The sensitivity analyses also provide an indication of the portion of the population that may be at greater risk from exposure to MTBE. The parameter with the highest sensitivity is breathing rate per unit body weight. The positive sensitivity indicates that an increased breathing rate results in greater exposure. The highest breathing rate per unit body weight generally occurs in children and infants indicating that these groups may be at an increased risk of developing cancer due to exposure to MTBE. These groups may also spend a greater proportion of their time in the home, increasing their potential exposure even more.

The probabilistic analysis also suggests that to be completely protective of all individuals in the population with respect to cancer risk, the concentration of MTBE in drinking water should be 5 ppb or lower. This level coincides with the proposed secondary standard for taste and odor. Also, at concentrations of MTBE at 25-26 ppb, 95% of the population would experience exposure greater than the *de minimis* exposure/risk level. At concentrations above 30 ppb, essentially all of the population would experience exposures greater than the *de minimis* level.

Depending on the source, individuals may experience either a relatively constant concentration of MTBE in their drinking water or a variable concentration of MTBE. Even though average concentration over a period of time may be below the PHG concentration, individuals could experience high concentrations of MTBE for short periods of time. For example, during some holiday periods in the summer, boat traffic on surface waters increases greatly. Also, pipeline leaks may allow large quantities of MTBE to enter surface waters over a short time period. Private wells could become contaminated due to local spills and leaks of gasoline. For a few days, the concentration of MTBE could be much higher than the 10 ppb to 14 ppb range. Peaks of exposure are known to be correlated with physiological impairment in plants, but it is unknown what the effects of these short-term peaks in MTBE exposure to humans might be.

## CONCLUSIONS AND RECOMMENDATIONS

### *Conclusions*

- Using an exposure model developed for this analysis, a 10 ppb Public Health Goal concentration of MTBE in drinking water was calculated. This concentration is 30% lower than the draft PHG developed by the Office of Environmental Health Hazard Assessment using a different model. However, it is within the 10 to 18 ppb PHG range identified by OEHHA as possible depending on the values used for the Henry's Law coefficient and water intake.
- The  $10^{-6}$  *de minimis* exposure/risk level corresponding to the 14 ppb concentration of MTBE in drinking water is  $6.8 \times 10^{-4}$  mg/kg-d.
- With a 14 ppb concentration of MTBE in drinking water, a probabilistic assessment indicates that approximately 40% of the population would experience exposures greater than the *de minimis* exposure/risk level due to variation in parameters such as breathing rate per unit body weight, shower duration, and time spent indoors. With a 10 ppb PHG, 20% of the population would experience exposures greater than the *de minimis* exposure/risk level.
- Individuals potentially at the greatest risk of developing cancer are those with higher breathing rate per unit body weight, which typically are infants and children.
- To be completely protective of all members of the population with respect to cancer risk, the concentration of MTBE in drinking water should not exceed 5 ppb.
- If a population is exposed to concentrations of MTBE in drinking water at 30ppb or greater, essentially all members of the population would experience exposures greater than the  $10^{-6}$  *de minimis* exposure/risk level.

### *Recommendations*

- In utero exposure and effects of MTBE should be evaluated.
- Dose reconstruction and epidemiological studies should be developed to assess exposure and health effects from drinking water contaminated with MTBE.
- Analyses should be conducted to assess the potential additional exposure from atmospheric MTBE in urban areas.

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**Table 1. Constants, Distributions and Moments Used in the Simulations**

All distributions are from McKone and Bogen (1992).

Permeability of the skin to MTBE is from Brown (1997), and the transfer efficiencies are calculated using the formulas in McKone and Bogen (1992).

Parameter description	Symbol	Distribution type	Arithmetic mean	Arithmetic standard deviation
Fluid intake per unit body weight (liters/kg-d)	IR/BW	Lognormal	3.0 x 10 <sup>-2</sup>	1.2 x 10 <sup>-2</sup>
Breathing rate per unit body weight (m <sup>3</sup> /kg-d)	BR/BW	Lognormal	0.4	0.5
Shower duration (hrs/d)	ETs	Lognormal	0.13	0.085
Shower water use rate per person (liters/hr)	Ws	Lognormal	480	160
Total water use in the house (liters/hr)	Wh	Lognormal	42	15
Exposure time in the bathroom (hr/d)	ETb	Lognormal	0.33	0.22
Surface area per unit body weight (m <sup>2</sup> /kg)	SA/BW	Lognormal	0.027	0.0025
Exposure time in the house (hr/d)	ETh	Uniform	8	20
Ventilation rate in the shower (m <sup>3</sup> /hr)	VRs	Uniform	4	20
Ventilation rate in the bathroom	VRb	Uniform	10	100
Ventilation rate in the house (m <sup>3</sup> /hr)	VRh	Uniform	300	1200
Permeability of the skin (m/hr)	P	Constant	0.006	
Fraction of skin exposed during showering and bathing	FS	Uniform	0.4	0.9
Transfer efficiency from water to shower air	TEs	Constant	0.52	
Transfer efficiency from water to household air	TEh	Constant	0.4	

*Exposure of Humans to MTBE from Drinking Water*

**Table 2. Model I simulations of exposure to water supply with concentrations of MTBE from 1 to 30 ppb**

*Values in the percentage column are the percent of the population that would be exposed to levels above the de minimis exposure level of  $6.8 \times 10^{-4}$  mg*

Concentration (in ppb)	percentage	Concentration (in ppb)	percentage	Concentration (in ppb)	percentage
1	0	11	25	21	75
2	0	12	30	22	80
3	0	13	35	23	82
4	0	14	40	24	90
5	0	15	47	25	95
6	6	16	55	26	>95
7	8	17	60	27	>95
8	10	18	65	28	>95
9	15	19	67	29	>95
10	20	20	72	30	>95