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BENZENE IN FLORIDA GROUNDWATER

AN ASSESSMENT OF THE SIGNIFICANCE TO HUMAN HEALTH

FLORIDA PETROLEUM COUNCIL
A DIVISION OF THE AMERICAN PETROLEUM INSTITUTE

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EXECUTIVE SUMMARY

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Introduction

The purpose of the *Benzene in Florida Groundwater Study* (Study) is to produce, compile, and analyze all available information and thereby comprehensively characterize the probability of potential adverse health effects from human exposure to benzene via groundwater sources. The ultimate goal is to offer the private and public sectors in Florida a meaningful and well informed selection of potential actions to take regarding benzene in groundwater.

Benzene is a known human carcinogen which has produced an increased incidence of leukemia in highly exposed industrial workers. Carcinogenic effects also have been demonstrated to occur in experimental animals after lifetime exposure to high levels of atmospheric benzene or to large doses given daily by the oral route.

On the other hand, benzene is also a natural substance occurring in low concentrations (parts per million) in many plants and as normal metabolite in certain animals. It is present in higher concentrations in crude petroleum and is a component of motor fuels. Benzene is produced by the combustion of most organic materials and is found in the exhaust of both gasoline and diesel powered vehicles. As a pure raw material, benzene is used in the manufacture of detergents, pesticides, and many other chemical products.

At lower concentrations (parts per billion), benzene is ubiquitous in the environment. All humans have a body content, derived primarily from air and food, which exists in equilibrium with the environment. The regulation of benzene in groundwater at such low levels inevitably requires a consideration of its relationship to drinking water and, in turn, the potential impact of that drinking water on the population health risk from benzene from all environmental sources.

The salient findings of the Study are as follows:

Existing Regulations

The Florida "maximum contaminant level" ("MCL") for benzene in both public drinking water systems and the most common class of groundwater (Class G-II) is one $\mu g/l$ (one part per billion). This health based standard, which is five times lower than the proposed U. S. Environmental Protection Agency (EPA) MCL, is based, in part, upon risk analysis considerations and is expressly left subject to alteration in light of further Agency understanding of what potential risks exist. Current Florida regulations allow for at least some case-by-case flexibility in the application of this stringent one $\mu g/l$ standard in groundwater through the use of "exemptions" and "zones of discharge". Invoking these relief mechanisms essentially requires a further assessment of potential health risks and other factors on a site specific basis.

The State of Florida is expected soon to engage in further rulemaking that will determine what concentrations of benzene may be left in groundwater pursuant to the "State Underground Petroleum Environmental Response Act of 1986" ("SUPER Act"). Special care should be taken to insure that the regulations reflect the best scientific information available.

Hazard Assessment

Available literature on benzene toxicity to humans primarily refers to instances involving high dose levels by inhalation. These observational cohort and epidemiological studies demonstrate that acute and chronic toxicological effects can occur as a result of high exposure to benzene in occupational settings. However, there are no reports of significant toxicological effects as a result of benzene ingestion via drinking water or food.

Avenues of human exposure to benzene include food (beef, eggs, etc.) and ambient and indoor air (vehicular sources, cigarette smoke, etc.). Indoor air sources constitute the most significant contributor of human body content of benzene. Drinking water contributes less than 0.2 percent of the average human exposure to benzene.

In summary, available scientific evidence and theory indicates that exposure to benzene through drinking water is unlikely to cause any significant adverse health effect in humans.

Fate and Transport / Containment of Benzene in Groundwater

Gasoline, which typically contains a small amount of benzene, may leak from underground tanks or pipes and thereby release this petroleum product to unsaturated soils above the water table or directly into groundwater. Gasoline is less dense than water, and the primary response to such releases should be to remove as rapidly as possible (by pumping) all of the "free product" that accumulates on top of the water table. This controls further lateral spreading of contamination and greatly reduces the source of gasoline components leaching as a soluble phase into the groundwater.

Mathematical and computer models are available to predict and simulate the transportation and fate of benzene in subsurface systems vulnerable to contamination in Florida. The models developed in this study demonstrate that natural mitigation by volatilization and biodegradation are important factors in the total mitigation process. Field observations have confirmed predictions by the model for the time to reach low concentrations [i.e., 10-100 $\mu g/l$ (ppb)] of benzene. The model indicates continuing reductions in benzene due to volatilization and biodegradation. However, field data indicate that a "leveling off" occurs within the 10-100 $\mu g/l$ range. Concentrations of benzene below this amount are outside the verifiable range of the model due to the geohydrological

irregularities, sampling, analytical, and other uncertainties.

Analytical uncertainty in the measurement of benzene below 10 μg/l is high, suggesting that practical demonstration of compliance at low concentrations would be severely complicated by analytical error.

Risk Assessment

The purpose of estimating exposure-related human health risk is to assist in the establishment of regulations that best balance benefits and risks.

In assessing risks associated with the presence of benzene in underground sources of water, several factors weigh in favor of increased flexibility in the Florida benzene groundwater standard because:

- Drinking water exposure to benzene constitutes such a small percentage of total exposure, the relative risk associated with the presence of benzene at concentrations higher than the current Florida MCL is slight;
- Physiological pharmacokinetic considerations show that benzene exposure via water at 20 μg/day (perhaps even higher) would not contribute measurably to the normal body content of benzene; and,
- Quantitative extrapolation of risk is overly conservative.

All previous health risk studies have focused upon the concept of "dose". For an analysis to be truly meaningful, however, it must account for biological processes (pharmacokinetics): i.e., identification of how and at what rates benzene enters the body, is distributed within the body, changes within the body, and is excreted from the body.

The primary factor regulating body content of benzene is average concentration of this constituent in the atmosphere. The body content is usually at an equilibrium with concentrations of benzene in the air due to a relatively quick exchange of benzene across the membranes of the lung. Increments of benzene absorbed from the intestine are largely metabolized in the body with the remainder being excreted by exhalation. Benzene absorbed from the intestine produces only a transitory increase in body content that quickly reverts to a level of equilibrium with the atmosphere.

The variability in an individual's total daily intake of benzene makes it difficult to observe any practical differences caused by changing the benzene intake from drinking water by 20 μg/day (25 μg/l at 0.8 l/day). Even in the statistical analysis of risk associated with benzene at 25 μg/l in drinking water, the upper bound estimate of increased carcinogenic response at that level among individuals *in the exposed population* would be less than 10^5 or, with equal likelihood, it might be essentially zero.

Conclusions

- Once detected on the groundwater, free phase petroleum hydrocarbons should be removed as quickly as possible to minimize the spread of the contaminant plume.
- Plume intercept technology is effective in cleaning up gasoline spill sites to low concentrations of benzene. Once benzene has reached about 100 μg/l in the groundwater, continued use of this cleanup action will not yield a substantial improvement over natural processes. Trace residual hydrocarbons left in the subsurface at these concentrations will degrade in time through natural processes.
- Geohydrological models which predict the relationship between sources of contamination and aquifers are useful tools as planning guides in projecting the extent of contamination and in evaluating cleanup strategies.
- The present Florida benzene water quality standard of one μg/l benzene is unnecessarily restrictive, both from a perspective of feasible site remedial action and from the standpoint of risk to public health and safety. In the 10-100 μg/l concentration range, the risk associated with benzene in groundwater is exceedingly small.
- A drinking water standard which limits intake of benzene from this source to 20 μg/day (25 μg/l at 0.8 l/day) is more than adequate to avoid any detectable risk of leukemia. Such standard should be applied only under circumstances where the groundwater will be used directly for human consumption.
- Should groundwater with traces of benzene be needed as a drinking water source, it may be more economical to treat the water at the well outlet rather than in the aquifer.

NOTE TO READER:

Authors have been free to select data for their work. The American Petroleum Institute has not imposed a requirement to use a given data set. This flexibility does not alter the overall conclusions but should be considered in comparing work by different authors.

Although specific models have been used in this study, other models may be equally valid.

PART TWO: HAZARD ASSESSMENT

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2-1 TOXICOLOGICAL EFFECTS OF BENZENE

2-1.1 Introduction

Several excellent reviews of the toxicological properties of benzene have been published recently: Fishbein (Ref. 2-1.9 [14]), Synder (Ref. 2-1.9 [32]), Aksoy (Ref. 2-1.9 [1]) and Mehlman (Ref. 2-1.9 [22]). This section will therefore address only selected aspects of the extremely large toxicological data base available for this compound in order to provide a perspective from which to evaluate other parts of this document.

2-1.2 Major Sources of Human Benzene Exposure

It must be appreciated that benzene is ubiquitous. According to estimates from the National Research Council (Ref. 2-1.9 [24]), the dietary intake of benzene may be as high as 250 μg daily, perhaps explaining benzene concentrations of 8-20 ppb in the breath of individuals with no known exposure to this compound. The benzene content of specific foods is reported to range from 2 ppb for canned beef to 2100 ppb for a boiled egg (Ref. 2-1.9 [23]). Recent results from the National Toxicology Program (Ref. 2-1.9 [25]) indicate that virtually 100% of benzene administered orally is absorbed into the body.

Benzene concentrations in ambient air (urban and rural) have been estimated to range from 1-100 ppb (Ref. 2-1.9 [17]). Urban air is reported to contain higher levels than rural air, presumably due to the contribution of automotive emissions of benzene (Ref. 2-1.9 [5]). After reviewing these data, the National Research Council (1980) calculated that an individual living in an urban environment containing a mean atmospheric benzene concentration of 16 ppb (50 $\mu g/m^3$) who breathes an average of 24 m^3 of air per day and absorbs approximately 50% of the dose (i.e., equilibrium state) will absorb approximately 600 μg of benzene daily.

[16 ppb = 50 $\mu g/m^3$ x 24 m^3/day x 50% absorption = 600 μg/day]

Benzene is present in cigarette smoke at levels of 47-64 ppm, leading to estimates of the amount of benzene inhaled from a single cigarettes ranging from 10-31 μg. Using the upper estimate, a person smoking two packs (i.e., 40 cigarettes) per day will absorb up to 992 μg of benzene (assumes 80% of dose is absorbed; non-equilibrium state). [Note: This discussion will not consider the contribution of secondary inhalation of cigarette smoke to the overall benzene exposure of a non-smoker.]

[40 cigarettes/day x 31 μg benzene/cigarette x 80% absorption = 992 μg/day]

The solubility of benzene in water is reported to be 1780 mg/l at 25 C (Ref. 2-1.9 [2]). In a report issued by the EPA Office of Drinking Water (Ref. 2-1.9 [11]), it was estimated that 97.7% of the population served by public water systems is receiving water either free of benzene contamination or having levels less than 0.5 μg/l. Estimates of daily human water consumption range from 0.8 to 2 l/day. Assuming that the average adult consumes 2 liters of water daily, it can be calculated that drinking water with a benzene content of 0.5 μg/l would contribute 1 μg of benzene to the average daily dose (100% absorption assumed).

[0.5 μg/l x 2 l/day = 1.0 μg/day]

Summarizing the above calculations for what are considered major sources of human benzene exposure:

	Non-Smoker	Smoker
Food	250 μg/day	250 μg/day
Air	600 μg/day	600 μg/day
Cigarettes	—	992 μg/day
Water	1 μg/day	1 μg/day
TOTAL	851 μg/day	1843 μg/day

2-1.3 The Pharmacokinetics of Benzene

The primary routes of benzene exposure are considered to be oral and inhalation. While there are data that benzene applied as a liquid or in solution to human skin can be absorbed fairly rapidly (Ref. 2-1.9 [4]), this route is generally discounted as being significant because of the rapid evaporation of benzene which effectively reduces the time of skin contact.

Recent data from the National Toxicology Program (Ref. 2-1.9 [26]) indicates that virtually all of an oral dose of benzene dissolved in vegetable oil is absorbed in rats and mice. It is reasonable to assume that dietary fiber content may reduce both the rate and efficiency of benzene absorption somewhat by complexing the hydrocarbon, but experimental evidence for this is unavailable. To be conservative, 100% absorption of benzene from food and water was assumed in the above calculations.

The respiratory absorption of a hydrocarbon vapor such as benzene is a complex process which has only partially been characterized. Initially, when an animal (or man) is placed into an atmosphere containing benzene vapor, virtually all of the hydrocarbon is absorbed into the blood stream and then distributed to the various tissues of the body. Each tissue will absorb some of the benzene from the blood with some tissues (e.g., those having a high

content of fat) absorbing relatively greater proportions of benzene compared to other tissues. The benzene absorbed by each tissue may be sequestered, released back to the blood unchanged, or processed by the specific metabolic enzymes which characterize the cells of that tissue. In time, a state of dynamic equilibrium (often called "steady state") is established between the air, blood, and other body compartments. As more benzene is absorbed from the air, some of the previously absorbed benzene is exhaled unchanged from the body, some is metabolized to specific derivatives which may be further metabolized even in other distant tissues, and some is removed by the kidneys and excreted in the urine. As the animal approaches steady state, the apparent (i.e., net) absorption of the compound into the body can be seen to decrease substantially. As noted in Section 2-1.2 above, conservative estimates of 50-80% absorption have been assumed in calculating the contribution of ambient air and cigarette smoking to the total burden of benzene. Recent data from the NTP (1986) indicate that from a 6 hour exposure to 11 ppm of benzene vapor, the apparent absorption of benzene in mice is 50%, whereas at 1000 ppm, the apparent absorption is 9.7%. Values for similarly exposed rats are 33% absorption at 13 ppm and 15% absorption at 870 ppm.

Significant differences in the respiratory characteristics of commonly used laboratory animals exist and greatly influence the amount and rate at which a hydrocarbon vapor is absorbed into the body. Small animals, with their high metabolic rate, must have a relatively large supply of oxygen to the body (Ref. 2-1.9 [34]). While this could have been accomplished evolutionarily by development of larger lungs relative to their body size, small animals such as mice and rats instead breathe more rapidly. It might be expected therefore that rodents should reach steady state more quickly than a man exposed to the same vapor concentration, but definitive experimental data in this regard have not been identified.

The Lovelace Inhalation Toxicology Research Institute (ITRI), under contract with NTP, have characterized the respiratory parameters of the B6C3F1 mouse and the F344 rat, (unpublished information from the butadiene research program). According to ITRI data, the average B6C3F1 mouse weighed 27.5 g and breathed 35 ml of air per minute (i.e., minute volume); the average F344 rat weighed 392 g and had a minute volume of 289.7 ml. Expressed on a kilogram body weight basis, the minute volume of the mouse is 1274 ml/kg and of the rat is 737 ml/kg. From this perspective, it is understandable why Sabourin *et al.* (1986) concluded that at similar exposure levels of benzene vapor, mice received 150-200% of the dose received by rats per kilogram body weight. Such differences in respiratory characteristics between species are especially important to exposures where the steady state condition has either not been reached or has been perturbed. Consideration of species differences must be incorporated in the quantitative modeling of animal inhalation data for estimation of human health risks. In particular, if one assumes a 70-kg man who breathes 7500 ml of air per minute, it can be calculated that the human minute volume/kg body weight is about one-seventh that of the rat and about one-twelfth that of the mouse.

A similar picture of steady-state equilibrium can be developed for benzene absorbed from the gastrointestinal tract. As noted above, recent data from NTP (Sabourin *et al.*, 1986) suggest that essentially all of a dose of benzene (dissolved in corn oil) administered by gavage is absorbed by rats and mice. Two points should be emphasized with regard to oral exposures. First, the concentration of benzene in the blood as a result of the administration of an oral bolus is seen to be a transient peak or spike. Dissolving benzene in a vehicle such as corn oil essentially results in a retardation in the absorption of benzene into the blood stream, such that all of the benzene is eventually absorbed, but it requires a longer period of time.

Benzene is metabolized via a number of possible pathways, summarized in Figure 2-1.8 [1]. Available information indicates that benzene must be metabolized in order to exert its toxic effects. However, it is not clear at the present time which of the various metabolic pathways activates benzene to more toxic derivatives and which pathways result in detoxification. Various investigators have suggested that toxicity may be the result of covalent binding of benzene metabolites to cellular macromolecules such as DNA and protein, and a reactive benzene epoxide was postulated to be formed during the metabolism of benzene by the cytochrome P-450 enzyme system (Ref. 2-1.9 [19]). Recent data, however, call into question whether benzene oxide is in fact formed to a significant degree during the metabolism of benzene (Ref. 2-1.9 [20]). Investigators at the Chemical Industry Institute of Toxicology (Ref. 2-1.9 [16], [18]) suggest that polyphenol derivatives (e.g., hydroquinone and 1,2,4 -benzenetriol) may be responsible for the toxic effects of benzene, perhaps via the autooxidative formation of highly reactive radicals which could bind to macromolecular targets. Goldstein *et al.* (Ref. 2-1.9 [15]) suggest that the benzene ring is cleaved metabolically forming muconaldehyde which is also capable of reacting with essential macromolecules. The role of various isomers of diphenol formed by cellular peroxidases, such as myeloperoxidase in the bone marrow in benzene toxicity remains to be evaluated (Ref. 2-1.9 [28]). See Figure 2-1.8 [1].

While it is not yet possible to identify which metabolite(s) of benzene is responsible for the profound cellular toxicity associated with this compound, quantitative risk modeling must recognize that significant qualitative and quantitative differences in the specific metabolic pathways for benzene are seen between species. The phenolic metabolites in the urine of benzene-treated mice, for example, have been found to comprise 50-65% *glucuronide* conjugates, 26-38% sulfate conjugates, and approximately 5% unconjugated phenol (Ref. 2-1.9 [33]). In contrast, Cornish and Ryan (Ref. 2-1.9 [6]) observed that approximately 70% of the phenolic metabolites found in the urine of benzene-treated rats were *sulfate* conjugates. In man, virtually all of the phenolic compounds in the urine following benzene exposure exist as *sulfate* conjugates until the concentration of phenol in the urine reaches approximately 400 mg/l when the sulfate pathway has apparently been saturated and products of glucuronide conjugation begin to appear (Ref. 2-1.9 [30]). Such data

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2-4 NONDRINKING-WATER EXPOSURE

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2-4.1 Occurrence

2-4.1(1) Food

Data on the occurrence of benzene in food are limited. Mara and Lee (Ref. 2-4.4 [11]) reported that benzene occurs naturally in fruits, fish, vegetables, nuts, dairy products, beverages, and eggs. These authors report concentrations ranging from 2 micrograms per kilogram (μg/kg) for canned beef to 2,100 μg/kg for eggs. Cooked meats are reported to have higher benzene levels than raw meats, and it is postulated that the increased benzene levels observed after cooking meats is due to the breakdown of aromatic amino acids such and tyrosine (Ref. 2-4.4 [5]).

Low levels (<10 μg/kg) of benzene in food could be due to a partitioning from the ambient levels of atmospheric benzene. The high levels observed in eggs indicate an intrinsic mechanism for the biochemical formation of benzene. Table 2-4.3 [1] summarizes the reported occurrence of benzene in foods.

2-4.1(2) Air

The materials balance for benzene indicates that 95 percent of environmental releases of benzene are to the atmosphere, and three-quarters of this release is associated with fuel combustion (Ref. 2-4.4 [5]). As a result of these emissions, it is not surprising that ambient air levels of benzene have been correlated with traffic volumes (Ref. 2-4.4 [2]).

Atmospheric benzene is ubiquitous; remote regions have measured concentrations usually ranging from 1 to 3.5 micrograms per cubic meter (μg/m^3). Higher levels are observed in urban and industrial environments. Table 2-4.3 [2] summarizes benzene concentration ranges and averages for various atmospheric environments.

Indoor benzene levels have been studied in industrial settings. Inside chemical plants, reported concentrations range from 2,000 to 10,000 μg/m^3. The current Occupational Safety and Health Administration (OSHA) regulation on workplace exposure is 32,000 μg/m^3 (10 parts per million, ppm) for the time-weighted average (TWA) concentration for an 8 hour exposure with a peak maximum concentration of 160,000 μg/m^3 (50 ppm) for any 15 minute period during an 8 hour day (Ref.2-4.4 [1]).

Indoor benzene levels in residences have been reported by Sample and Gilbert (Ref. 2-4.4 [14]) to have a median value of 15.0 μg/m^3 and an arithmetic mean of 25.8 μg/m^3 for 353 nighttime observations of benzene. There is a minimal correlation between indoor concentrations and outside ambient levels. The impact of smoking on indoor benzene concentrations appears to be important in households containing one or more smokers. These households exhibit at least 50 percent greater concentrations than households of nonsmokers. One cigarette can generate approximately 90 micrograms (μg) of benzene (Ref. 2-4.4 [5]). The portion of the benzene in the mainstream smoke is predominantly absorbed by the smoker and not exhaled. Mainstream smoke is that which the smoker inhales; however, the sidestream smoke which is released to the room often contains twice the quantity of some chemicals as the mainstream smoke (Ref.2-4.4 [6]). Therefore, of the 90 μg of benzene released from each cigarette, possibly 60 μg is released to the smoker's environment through the sidestream smoke.

2-4.2 Exposure

Reported benzene concentrations in foods do not involve all food groups. It is not known how representative these concentrations are of the concentrations in foods in general. Dietary intake of benzene has been estimated to be as high as 250 micrograms per day (μg/day) from beef, eggs, and rum alone (Ref. 2-4.4 [13]). Assuming that the average adult male weighs 70 kg, an intake of 250 μg/day would be equivalent to 3.6 micrograms per kilogram per day (μg/kg/day). In the absence of further data, Letkiewicz *et al.* (Ref. 2-4.4 [8]) assumed the dietary intake of benzene was at that level. Gilbert *et al.* (Ref. 2-4.4 [5]) estimated the ingestion due to only those foods with reported benzene concentrations (i.e., butter, cooked beef, eggs, and haddock), resulting in a daily ingestion intake of 31 to 108 μg/day.

Exposure to benzene in the atmosphere is highly variable; reported levels range between low parts-per-billion values in outside air to low parts-per-million in certain industrial settings. Median air concentrations of benzene have been calculated by Brodzinsky and Singh (Ref. 2-4.4 [3]) for rural/remote areas (4.5 μg/m^3), urban/suburban areas (8.9 μg/m^3), and source-dominated areas (9.6 μg/m^3). Thus, in urban/ suburban areas, people inhale approximately 180 μg of benzene each day (at 20 cubic meters, m^3, of air inhaled each day). As a comparison, a one-pack-per-day smoker inhales approximately 600 μg/day of benzene from mainstream smoke. Exposure by these routes is substantial, but highly variable in the general population.

Average annual atmospheric benzene concentrations and the size of exposed populations have been calculated by Mara and Lee (Ref. 2-2.4 [11]) based on air dispersion models. Approximately half of the population of the United States was estimated to be exposed to average atmospheric benzene concentrations between 3.5 and 13 μg/m^3.

A newborn, formula-fed infant's respiratory intake of benzene can be expected to range from 1.0 to 2.2 μg/kg/day, whereas the intake of a nonsmoking, 70-kilogram (kg) adult male may vary between 1.5 to 360 μg/kg/day, depending on ambient benzene concentrations (Ref. 2-4.4 [8]).

TABLE 2-4.3 [1] Foods Reported to Contain Benzene

Fruits[a]	Vegetables[a]	Meat, Fish, and Poultry
Apple	Bean	Cooked beef (2 to 19 μg/kg)[c]
Citrus Fruit	Leek	Chicken (<10 μg/kg)[d]
Cranberry and Bilberry	Mushroom	Egg, hard boiled (500 to 1,900 μg/kg)[e]
Currants	Onion, *roasted*	Egg, uncooked (2100 μg/kg)[h]
Guava	Parsley	Haddock (100 to 200 μg/kg)[f]
Pineapple	Potato	Lamb, heated (<10 μg/kg)[d]
Strawberry	Soya Bean	Mutton, heated (<10 μg/kg)[d]
Tomato	Trassi, *cooked*	Veal, heated (<10 μg/kg)[d]
Nuts[a]	**Dairy Products**	**Beverages**
Filbert, *roasted*	**Butter (0.5 μg/kg)[b]**	**Cocoa[a]**
Peanut, *roasted*	Blue Cheese[a]	Coffee[a]
Macademia Nut	Cheddar Cheese[a]	Jamaican Rum (120 μg/kg)[g]
	Other Cheese[a]	Tea[a]
		Whiskey[a]

[a] Ref. 2-4.4 [16]
[b] Ref. 2-4.4 [15]
[c] Ref. 2-4.4 [13]
[d] Ref. 2-4.4 [12]
[e] Refs. 2-4.4 [9], [10]
[f] Irradiated and non-irradiated haddock, respectively.
[g] Ref. 2-4.4 [7]
[h] Ref. 2-4.4 [11]

Source: Ref. 2-4.4 [5]

TABLE 2-4.3 [2] Summary of Benzene Occurrence in Air

Environment	Benzene Concentration (μg/m³)
Remote (Range)	1 to 3.5
Urban (Range)	4 to 160
Residential — Remote from Traffic (Average)	4.5
Near Chemical Plant (Average)	14
Near Refineries (Average)	9
Gas Stations (Range)	<1 to 32

Sources: Refs. 2-4.4 [5], [8]

2-4.4 REFERENCES: NONDRINKING-WATER EXPOSURE

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