

APPENDIX G

**MINNESOTA DEPARTMENT OF HEALTH EXPOSURE EVALUATION - VAPOR
EXPOSURE TO VINYL CHLORIDE**

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Exposure to volatile organic compounds (VOCs) from use of contaminated water through pathways other than ingestion

Some older studies suggested that exposure to VOCs in drinking water through inhalation or skin contact during activities such as showering, bathing, or washing dishes could be significant in certain situations. The ratio of inhalation uptake versus direct ingestion of contaminated water was estimated to be as high as six to one (McKone 1989) or as low as less than one to one (Lindstrom and Pleil 1996). A more recent, accurate study (Kerger et al 2000) using water and air measurements taken in actual home bathrooms estimated that the exposure through inhalation of volatile organics (such as vinyl chloride, or VC) from showering and bathing in contaminated water is less than the ingestion exposure by a factor of three to four. Previous studies typically used laboratory or simulated shower facilities, which tend to be smaller than standard home showers and less well ventilated, resulting in higher estimates of exposure through inhalation.

A large number of variables are involved in assessing inhalation exposure, making accurate estimates very difficult. These variables include such things as water temperature, size of the shower enclosure, the type of shower head used, length of time spent in the shower, and the ventilation rate. One study (Lee et al 2002) identified the contaminant level and the time spent in the shower as the key variables that determine the level of exposure. Several studies have demonstrated that simply ventilating the shower stall can greatly reduce the estimated exposure to VOCs in shower air (McKone and Knezovich 1991; Aggarwal 1994).

Estimates of additional exposure through skin contact with contaminated water are generally thought to be less than for inhalation exposure, and have been estimated to be in the range of one to one or less (McKone 1989). One study (Lee et al 2002) estimated that intake through dermal absorption would account for only about 2% of the total intake through inhalation and dermal contact while showering. This is especially true for VC due to its extreme volatility.

The route of exposure, however affects the rate at which VOCs are absorbed and metabolized by the body; even if the same dose is received via different routes (i.e., ingestion, inhalation, or dermal contact) the resulting toxicity may be different (Weisel and Jo 1996). A compilation of studies conducted by ATSDR and summarized in their toxicological profile for VC suggests that absorption of VC through the gastro-intestinal tract as a result of oral exposure is "rapid and virtually complete" based on studies in rats. While absorption of inhaled VC in human lungs is "rapid," on average only 42% of inhaled VC is retained in the body, at least at higher concentrations (ATSDR 1997). Some pharmacokinetic models developed by EPA also suggest that the levels of VOC metabolites formed by the body may be higher as a result of oral exposure than inhalation exposure. For instance, small amounts of VOCs that are ingested are often quickly metabolized by the liver, while small amounts of VOCs that are inhaled or absorbed through the skin are typically distributed throughout the body prior to metabolism by the liver, and are therefore metabolized more slowly. The toxic effects of exposure to VOCs are mainly due to the action of their metabolites within the body. This implies that for

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equal (low) doses the ingestion of VOCs in water may be of greater consequence within the body than inhalation or dermal absorption because ingestion produces higher concentrations of toxic metabolites in a shorter amount of time.

References

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