DICHLOROETHENE (VDC)

COMMENTS - Chemicals and Metals (S. Hearn)

Sect 1.1 (p.1)
DCE is also used as a chemical intermediate, not only as an ingredient in plastic products.

Sect 1.2 (p.1)
In this section it is stated that "air concentrations within manufacturing facilities have been measured from 6 ppm to 1900 ppm which exceeds the levels observed to affect health in animals". In section 5.5 (p.90), other exposure data are presented which indicate that typical DCE exposures are far lower (<5 ppm-ppb). The 1900 ppm level is obviously an extreme. In these cases, the median values must be presented to make a meaningful judgement. It also is not stated whether this exposure level was over an 8-hour work day or was a short term excursion. Lastly, OSHA has recently established a Permissible Exposure Limit of 1 ppm (8-hr. TWA). This new PEL should significantly reduce worker exposure potential in the future.

In the last paragraph in this section, potential exposure to DCE from plastic packaging films is discussed. In addition to the data presented, it should also be mentioned that these packaging uses are regulated by the FDA and that small DCE residuals are considered by the FDA to present no hazard to the consumer.

Sect 1.3 (p.2)
In this section, it is stated that "DCE can probably also enter the body through the skin". No data to support this statement ~~is~~ are presented in sect 2.2.3 and, therefore, this statement is inappropriate for section 1 (Public Health Statement) and should be removed.

Sect 1.4 (p.2)
This section starts out by stating "the health effects of DCE in humans are unknown. Yet there are data summarized in sect. 2.2.1.2 which describe some of the acute effects of overexposure (neurotoxicity). Why are these acute effects overlooked in sect. 1.

There have been 18 chronic studies of DCE exposure in animals. Only one study has demonstrated a possible increased risk of cancer. In addition, there are metabolic and mechanism of action data which ~~may~~ suggest that the increased risk of cancer observed in this one species may not be relevant to humans. The sentence "an increased risk of cancer has been demonstrated in animals exposed to DCE" does not reflect the preponderance of data available from animal tests.

If birth def cts hav been noted along with "sickness of the mothers", it should be pointed out that such effects may be secondary to the maternal toxicity. This is important since non-maternally toxic exposures would not be expected to result in birth defects.

Sect. 1.5 (p.3)

What is the level of detection (analytical sensitivity) for medical tests? Is it likely that low level environmental exposures such as those encountered at waste sites or from plant emissions can be detected in exhaled air or body fluids?

Sect. 1.6 (p.4)

In the second paragraph on this page, effects observed in oral studies are summarized. What is the relevance to humans of the data from animals receiving 200 mg/kg/day (4000 pm) placed in their stomachs experimentally? If this section is supposed to draw some conclusions about potential human health effects, data from the most relevant routes of exposure should be used to draw these conclusions. At the very least, some statement regarding the relevancy of the route is needed in this section.

Table 1-1 (p.5)

In this table, it should be noted that these levels are extrapolated from animal data. If they are, the use of the term "short-term exposure" for exposures less than 14 days becomes meaningless for health effects information especially when the document cites exposure levels of 500 and 4000 ppm. Clearly, these exposure levels are well above the permissible exposure limit (1 ppm) and ACGIH TLV (5 ppm) and it is unrealistic to imply that these levels should or could be endured for up to 14 days.

Sect. 2.2 (p.12)

It appears a zero is missing in the following: "risk of one in 10,000 to one in 10,00,000 (10^{-4} to 10^{-7})".

Sect. 2.2.1.2 (p.23)

The reference to neurotoxicity after short-term exposure should describe whether the effect is transient or permanent. Levels of exposure should be provided if available. This is also true for "upper airway irritation" in humans noted on p.24.

Sect. 2.2.1.4 (p.29)

If the two cases of persistent cranial nerve disorders were more likely due to dichloroacetylene, it is inappropriate to mention them in this DCE profile. This reference should be removed.

Sect. 2.2.1.8 (p.52)

It is not appropriate to characterize the retrosp ctive cohort mortality studies as having severe design flaws. As epidemiology is an observational science and these studies, in particular, evaluated post occurrences, the investigators have only marginal control over the size of the cohort and no control over the number of deaths due to specific causes.

Sect. 2.3 (p.48)

In the second sentence, possible human exposure situations are listed. In discussing the potential for human exposure, it should be pointed out that the levels of exposur will vary greatly from situation to situation and even within each situation for different individuals.

In the next paragraph, the effects of metabolism are briefly reviewed. However, one observation discussed in sect. 2.6.3 (p.65) is omitted here. That is the observation that though the pathways are similar in the rat and mouse, the rate of metabolism was greater in the mouse (resulting in a greater concentration of toxic metabolite).

Sect. 4.4 (p.82)

In the second paragraph it is stated that DCE has been found at 16% of all hazardous waste sites. At what level was DCE found? Range? Median? This data should be provided.