

c. The systemic toxic effects of acetone cyanohydrin are caused by free cyanide ions, and

d. Hydrogen cyanide has a far higher vapor pressure than acetone cyanohydrin.

From these facts it was concluded that with every exposure to acetone cyanohydrin a concomitant exposure to hydrogen cyanide will occur. It therefore seemed reasonable to apply the AEGL-3 values (on a ppm basis) derived for hydrogen cyanide to acetone

cyanohydrin. This procedure is supported by a close similarity of acetone cyanohydrin and hydrogen cyanide regarding lethal effects in rats exposed for 6 hours.

The proposed AEGL values are listed in Table 2 of this unit.

TABLE 2.—SUMMARY TABLE OF PROPOSED AEGL VALUES FOR ACETONE CYANOHYDRIN^A

Classification	10-minutes	30-minutes	1-hour	4-hours	8-hours	Endpoint (Reference)
AEGL-1 (Nondisabling)	1.1 ppm (3.9 mg/m ³)	1.1 ppm (3.9 mg/m ³)	0.84 ppm (2.9 mg/m ³)	0.53 ppm (1.9 mg/m ³)	0.35 ppm (1.2 mg/m ³)	No red nasal discharge in rats (Monsanto Co., 1986a)
AEGL-2 (Disabling)	6.8 ppm (24 mg/m ³)	6.8 ppm (24 mg/m ³)	5.4 ppm (19 mg/m ³)	3.4 ppm (12 mg/m ³)	2.2 ppm (7.7 mg/m ³)	No respiratory distress in rats (Monsanto Co., 1986a)
AEGL-3 (Lethal)	27 ppm (95 mg/m ³)	21 ppm (74 mg/m ³)	15 ppm (53 mg/m ³)	8.6 ppm (30 mg/m ³)	6.6 ppm (23 mg/m ³)	Application of AEGL-3 values for hydrogen cyanide

^a Cutaneous absorption may occur; direct skin contact with the liquid should be avoided; fatal intoxications have been reported upon skin contact.

ii. *References.* a. El Ghawabi A.; M. Gaafar; A. El Saharta; S.H. Ahmed; K.K. Malash; and R. Fares. 1975. Chronic cyanide exposure: a clinical radioisotope and laboratory study. *British Journal of Industrial Medicine*. 32:215–219.

b. Monsanto Co. 1982b. Male fertility study of Sprague-Dawley rats exposed by inhalation route to acetone cyanohydrin. Monsanto Co. Report No. ML-82-144. Monsanto Co., St. Louis, MO, USA.

c. Monsanto Co. 1982c. Female fertility study of Sprague-Dawley rats exposed by inhalation route to acetone cyanohydrin. Monsanto Co. Report No. ML-82-125. Monsanto Co., St. Louis, MO, USA.

d. Monsanto Co. 1986a. One-month inhalation toxicity of acetone cyanohydrin in male and female Sprague-Dawley rats with cover letter dated 04-25-86. Report No. BN-81-178. Monsanto Co., St. Louis, MO, USA.

e. Monsanto Co. 1986b. Three-month inhalation toxicity of acetone cyanohydrin in male and female Sprague-Dawley rats with cover letter dated 04-25-86. Report No. ML-82-143. Monsanto Co., St. Louis, MO, USA.

2. Ammonia—i. Description.

Ammonia is a colorless, corrosive, alkaline gas that has a very pungent odor. The odor detection level ranges from 5–53 ppm. Ammonia is used as a compressed gas and in aqueous solutions. It also is used as a household cleaning product, in fertilizers, and as a refrigerant. Exposures to ammonia occur as a result of accidents during highway and railway transportation, by releases

at manufacturing facilities, and from farming accidents.

Ammonia is very soluble in water. Because of its exothermic properties, ammonia forms ammonium hydroxide and produces heat when it contacts moist surfaces, such as mucous membranes. The corrosive and exothermic properties of ammonia can result in immediate damage (severe irritation and burns) to eyes, skin, and mucous membranes of the oral cavity and respiratory tract. In addition, ammonia is effectively scrubbed in the nasopharyngeal region of the respiratory tract because of its high solubility in water.

The data for deriving AEGL values were obtained primarily from case studies of accident victims, experimental studies in humans, and experimental studies on lethality and irritation in animals. The case studies were of limited use for quantitative evaluation, but the experimental studies in humans and animals contained quantitative data that would be used for deriving AEGL values.

No reliable quantitative lethality data were available for humans dying as a result of exposure to ammonia. One case study reported the death of an individual exposed to a high but unknown concentrations of ammonia. Other case studies also contained no exposure estimates, but showed that high concentrations of ammonia cause severe damage to the respiratory tract, particularly in the tracheobronchial and pulmonary regions. Death, however, is most likely to occur when damage causes pulmonary edema. Non-lethal, irreversible, or long-term effects occur

when damage progresses to the tracheobronchial region, manifested by reduced performance on pulmonary function tests, bronchitis, bronchiolitis, emphysema, and bronchiectasis. Nondisabling, reversible effects are manifested by irritation to the eyes, throat, and nasopharyngeal region of the respiratory tract. The odor of ammonia is detected by humans at concentrations >5 ppm; the odor is highly penetrating at 50 ppm (10 minutes). Experimental studies on human volunteers, showed that slight irritation may occur at 30 ppm (10 minutes), moderate irritation to the eyes, nose, throat, and chest occurs at 50 ppm (10 minutes to 2 hours), moderate to highly intense irritation occurs at 80 ppm (30 minutes to 2 hours), highly intense irritation occurs at 110 ppm (30 minutes to 2 hours), unbearable irritation occurs at 140 ppm (30 minutes to 2 hours), and excessive lacrimation and irritation at 500 ppm. In addition, some subjects were able to breathe 140 ppm for up to 2 hours or 500 ppm for 30 minutes without suffering long-lasting effects. Reflex glottis closure, a response to irritant vapors, occurred at 570 ppm for 21- to 30-year-old subjects, 1,000 ppm for 60-year-old subjects, and 1,790 ppm for 86- to 90-year-old subjects.

Acute lethality studies in animals showed that the LC₅₀ values for rats ranged from 40,300 ppm for a 10-minute exposure to 7,338 and 16,600 ppm for 60-minute exposures. For the mouse, LC₅₀ values were 21,430 ppm for a 30-minute exposure (almost all animals died in less than 13 minutes), 10,096 ppm for a 10-minute exposure, and 4,230 and 4,837 ppm for 60-minute