TOXICITY OF NEODOL(R) SURFACTANTS

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1.0 INTRODUCTION

This report is the result of Work Assignment #3 of IAG #DW-89930405. Toxicity data from a voluntary submission (FYI-AX-0685-0410 Sequence A) by Shell Chemical Company to EPA's TSCA Existing Chemicals Program and two published reports by Arthur D. Little, Inc. ("Human Safety and Environmental Aspects of Major Surfactants," May, 1977; "Supplement," by Goyer et al. February, 1981) were reviewed to evaluate the toxicity and structure-activity relationships of NEODOL(R) chemicals for which data are available and to identify gaps in the toxicity database. TSCA 8(e) submission 8EHQ-0580-0326 Sequence C was also reviewed for its applicability to NEODOL toxicity.

Today's dishwashing and laundry agents are superior to those of the past because they thoroughly clean man-made fibers, tolerate hard water, form little foam, and are readily biodegraded. These improvements are due largely to the extensive use of three classes of surfactants (NEODOL products) in cleaning formulations. Derived from primary alcohols, these compounds are classified according to the chemical group(s) attached to the alkyl chain: alkyl sulfates, if sulfated; alkyl or alcohol ethoxylates, if ethylene oxides are present; and alkyl or alcohol ethoxysulfates, if ethylene oxides are sulfated.

In addition to the widespread use of NEODOL products as household cleaning agents (primarily the ethoxysulfates), they are extensively used in personal care products such as shampoos, bubble baths, and cosmetics, and also have many industrial applications. NEODOL ethoxylates are also used as analgesics and anesthetics. While recent product/consumption figures have not been provided, a review of the values reported by Arthur D. Little (1977, Goyer et al. 1981) indicates the considerable use of these surface-active agents. Comparing data from 1973 and 1978, annual use of ethoxylates in the United States increased from 188,000 tons to 238,000 tons during the five-year period. Similarly, the use of ethoxysulfates rose from 53,000 tons to 64,000 tons during those same years. As for alkyl sulfates, 90,000 tons were used

worldwide in 1976. Shell Chemical Company is the world's largest producer of linear primary alcohols and alcohol-based surfactants, exceeding 450 million pounds per year in the United States. In England and Japan certain NEODOL products are produced under the name DOBONOL(R).

2.0 DESCRIPTION OF NEODOL(R) PRODUCTS

NEODOL products include:

- o NEODOL alcohols (ROH)
- o NEODOL ethoxylates [$RO(CH_2CH_2O)_xH$]
- o NEODOL sulfates ($ROSO_3^-Na^+$ or NH_4^+)
- o NEODOL ethoxysulfates [$RO(CH_2CH_2O)_xSO_3^-Na^+$ or NH_4^+]

2.1 NEODOL Alcohols

Linear primary alcohols (ROH) included in NEODOL products consist essentially of two groups: chains of C_9 to C_{11} carbon atoms and chains of C_{12} to C_{15} carbon atoms. Nomenclature is based on the length of the alkyl chain. For example, NEODOL 91 indicates that this product is a mixture of mostly C_9 to C_{11} alcohols; NEODOL 25 is a mixture of mostly C_{12} - C_{15} alcohols (Shell b, p. 1).

2.2 NEODOL Ethoxylates

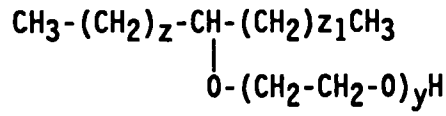
NEODOL ethoxylates are mainly produced from the reaction of ethylene oxide (CH_2CH_2O or EO) with linear primary alcohols, although some branched-chain alcohols are used (Satkowski et al. 1967, as reported in Arthur D. Little, Inc. 1977, p. 240).

Examples:

Primary

$$CH_3\text{-}(CH_2)_x\ CH_2\text{-}O\text{-}(CH_2\text{-}CH_2\text{-}O)_yH$$

Secondary



x - usually C_6 to C_{16}

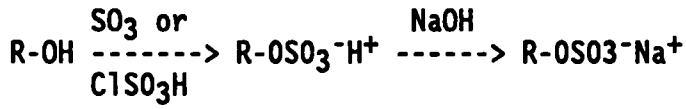
y - usually EO_3 to EO_{20}

$z + z_1$ - usually C_6 to C_{16}

NEODOL 25-3 or C_{12-15} EO_3 indicates that the product is comprised mostly of C_{12} to C_{15} alcohols reacted with an average of 3 molecules of EO to form a 3-unit EO chain (Shell b, p. 2).

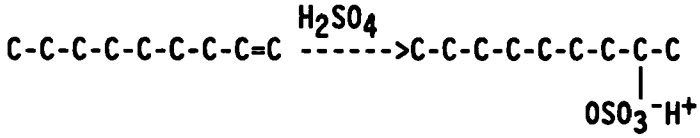
2.3 NEODOL Sulfates

NEODOL sulfates (alcohol or alkyl sulfates or AS) are produced by sulfation of the parent alcohol with either sulfur trioxide or chlorosulfonic acid and subsequent neutralization of the product with an appropriate base as follows:



(R usually averages between 12 and 18 carbons).

To produce secondary AS, the parent alkene is reacted with sulfuric acid.



A complex mixture of isomers can occur because the sulfate ester group can add at any position along the chain, except at the terminal carbon atoms (Higgins and Burns 1975; Kerfoot and Flammer 1975; Swisher 1970, p. 36; as reported in Arthur D. Little, Inc. 1977, p. 171).

NEODOL 91-S indicates that the alkyl chain is C_9 to C_{11} carbons in length, and that the sulfated alcohol has been neutralized with NaOH to produce a sodium (S) salt of the sulfate. NEODOL 23-A specifies the ammonium (A) salt of the sulfate (Shell b, p. 1).

AS are used in many specialty products such as shampoos, cosmetics, dentifrices, antacids, and depilatories (Gleason et al. 1969 as reported in Arthur D. Little, Inc. 1977, p. 170), and are extensively used in heavy duty laundry products (Kerfoot and Flammer 1975, as reported in Arthur D. Little, Inc. 1977, p. 170).

2.4 NEODOL Ethoxysulfates

Walker et al. (1973, as reported in Arthur D. Little, Inc. 1977, p. 346) described the following procedures for production of NEODOL ethoxysulfates (EOS).

"(1) ethoxylation of a fatty alcohol (prepared from either vegetable oil or petroleum hydrocarbons)

$$R\text{-}OH + \left(\begin{array}{c} CH_2\text{-}CH_2 \\ \diagdown\ \diagup \\ O \end{array}\right)_x \xrightarrow[\text{catalyst}]{\text{KOH}} R\text{-}O\text{-}(CH_2CH_2O)_x\ H \quad (\text{usually } x = 2\text{-}4)$$

(2) sulfation of the product with either sulfur trioxide (SO_3) or chlorosulfonic acid ($ClSO_3H$),

$$R\text{-}O\text{-}(CH_2CH_2O)_x\ H \xrightarrow{SO_3} R\text{-}O\text{-}(CH_2CH_2O)_x\ SO_3\ H$$

(3) and neutralization to form either the sodium or ammonium salt

$$R\text{-}O\text{-}(CH_2CH_2O)_x\ SO_3\ H \xrightarrow{OH^-} R\text{-}O\text{-}(CH_2CH_2O)_x\ SO_3^-Na^+ (\text{or } NH_4^+)"$$

EOS can be designated as, for example, NEODOL 25-3A or $C_{12\text{-}15}EO_3A$, signifying a mixture of 12 to 15 carbon alcohols, an average of three molecules of EO to form a three-unit chain, sulfation of the ethoxylate, and neutralization to form the ammonium salt (Shell b, p. 1).

EOS, high foaming anionic surfactants, are principally used in light-duty dishwashing products and laundry detergent formations. They are also used in shampoos and other household specialty products (Arthur D. Little, Inc. 1977, p. 345).

3.0 EFFECTS ON NON-MAMMALIAN ORGANISMS

3.1 Acute Effects (LC_{50})

Anionic surfactants are less acutely toxic to aquatic organisms than are nonionic surfactants. However, anionics cause more permanent damage to the gill structure of fish than do nonionics (Shell a, p. 4). Aquatic organisms are better able to recover after exposure to nonionic surfactants than after exposure to anionic surfactants. For example, 50% of the barnacle larvae exposed for 30 minutes to the LC_{50} concentration of a nonionic surfactant completely recovered within 20 minutes after removal to clean water. By contrast, barnacle larvae tested under similar conditions recovered no swimming ability within 48 hours after exposure to anionic surfactants ended (Wright 1976, as reported in Shell a, p. 16).

Acute toxicities, expressed as LC_{50}s, of NEODOL surfactants are compared in Appendix A for those aquatic organisms for which sufficient data are available. Chemicals are arranged in order of decreasing toxicity. Results are discussed in the following subsections.

3.1.1 Alcohols

Alcohols are the NEODOL products least toxic to aquatic organisms. Toxicity decreases with increases in the length of the carbon chain (Table 1) because water solubility eventually decreases so that the alcohol floats on the water surface (Shell a, p. 11). This is demonstrated by studies with rainbow trout (Salmo gairdneri) showing an increase in 96 hour LC_{50} values from 6 to 10 mg/L for Dobanol 91 (Shell Internal Report TLGR. 0166.78, as reported in Shell a, p. 11) to a non-toxic response at saturation for Dobanol 45 (Shell Internal Report TLGR. 0162.78, as reported in Shell a, p. 11).

TABLE 1. General direction of toxicity and rate of biodegradation of linear primary alcohols and derivative surfactants in an aquatic environment as a function of alkyl or ethoxylate (EO) chain length

Class	Chain Length: Alkyl (No. of carbon atoms)	Chain Length: EO (No. of units)	Toxicity	Rate of Biodegradation
Alcohols	↑	NP	↓	→
Alkyl sulfates (AS)	↑	NP	↑	→
Alkyl ethoxylates (EO)	10 ↓ or ↑ 12	—	↓	Reports range from "no effect" to "very slight decrease" as complexity of the molecule increases.
	12 ↑ 10	—	↑	
	—	20 ↑ 2	↓	
Alkyl ethoxy-sulfates (EOS)	↑ 16	—	↓ Less toxic than parent EO	Same as for EO compounds.
	16 ↑	—		
	<16	6 ↑ 2	↓	
	>16	6 ↑ 2	↑	

Key: ↑ = increase; ↓ = decrease; — = no change; NP = not prese; → = moderate or gradual decrease

3.1.2 Alcohol Ethoxylates

The toxicity of alcohol ethoxylates varies according to both the length of the alkyl chain and the number of EO units present. Generally, when the length of the alkyl chain remains the same, increases in the number of EO units decrease the toxicity (Table 1), as shown in Appendix A by the one-hour LC_{50} studies with goldfish (Carassius auratus) (Gloxhuber et al. 1968, as reported in Shell a, p. 14) and the 96-hour LC_{50} studies with rainbow trout (summarized by Shell a, p. 13, Figure 5) and Daphnia (U.S. Food and Drug Administration, as reported in Shell a, p. 15). Shell (a, p. 4) suggests that the toxicity is decreased because the molecule becomes less fat-soluble and, therefore, penetrates the gill membrane less readily. If, however, the number of EO units is unchanged and the length of the alkyl chain is increased, toxicity increases (Shell a, p. 13).

Invertebrates (except Daphnia) are relatively tolerant to alcohol ethoxylates, with most LC_{50} values ranging from 500 to 5,000 mg/L (Shell a, p. 13) compared to bluegill sunfish (Lapomis macrochirus), with LC_{50} values ranging from 1.8 mg/L ($C_{12\text{-}15}EO_3$) to 11.0 mg/L ($C_{12\text{-}15}EO_9$, 98% linear primary), and rainbow trout, with LC_{50} values ranging from 0.8 mg/L ($C_{14\text{-}15}EO_7$) to 8-9 mg/L ($C_{9\text{-}10}EO_5$) (Appendix A). Less active species are perhaps more tolerant to surfactants than the more active species because lower respiratory rates cause less surfactant to pass over their gills (Shell a, p. 4).

3.1.3 Alcohol Sulfates

Alcohol sulfates do not appear to be as acutely toxic to aquatic organisms as are the ethoxylates. According to Kikuchi et al. (as reported in Goyer et al. 1981, p. 100), 24-hour LC_{50} values reported for Japanese killifish (Oryzias latipes) ranged from 0.78 mg/L for $NaC_{16}AS$ to 70 mg/L for $NaC_{12ave}AS$. The variation was attributed to the difference in the length of the alkyl chain (Table 1). However, close examination of the limited LC_{50} data presented in Appendix A does not clarify whether chain length affects toxicity.

3.1.4 Alcohol Ethoxysulfates

Sulfation of ethoxylates appears to reduce their toxicity by a factor of 21 to 23 compared to the parent products (Shell a 1985, p. 16). According to studies using fathead minnows (Pimephales promelas), the most important factor influencing the toxicity of these surfactants is the number of EO units present rather than the number of carbon atoms present (Monsanto Co., unpublished data, as reported in Arthur D. Little, Inc. 1977, p. 363). Increasing the number of EO units when the number of carbon atoms was kept constant and less than 16 decreased toxicity (Table 1); however, when the number of carbon atoms was equal to or more than 16, increasing the number of EO units drastically increased toxicity (see Appendix A). The most toxic surfactant tested was $C_{16}EO_6S$, producing a 24-hour LC_{50} value of 0.8 mg/L. The peak toxicity at C_{16} changed very little with EO units decreasing to EO_2. The least toxic ethoxysulfate tested was $C_{18}EO_2S$ at an LC_{50} of 80 mg/L. Contrary to Monsanto's results with minnows, Gafa (1974, as reported in Arthur D. Little, Inc. 1977, p. 363) found $C_{16}EO_{3.4}S$ to be one of the least toxic surfactants to goldfish. Shell d (unpublished data, as reported in Goyer et al. 1981, p. 199) demonstrated substantial differences in 96-hour LC_{50} values for rainbow trout when the numbers of carbon atoms were changed and the numbers of EO units were only slightly different. The LC_{50} for $C_{12-15}EO_3S$ was 8.9 mg/L compared to an LC_{50} of 400-450 mg/L for $C_{9-10}EO_{2.5}S$. These results indicate that data are insufficient to generalize about the factors influencing the toxicities of various alcohol ethoxysulfates.

The few data available for invertebrates suggest that they may be slightly less susceptible to EOS than are fish. LC_{50} values (24-hour) ranged from 5 mg/L ($C_{12}EO_3S$) to 37 mg/L ($C_{12}EO_3S$, Ziegler or natural fatty alcohol-derived) in Daphnia (Lundahl et al. 1972, as reported in Arthur D. Little, Inc. 1977, p. 364).

3.2 Sublethal Effects

3.2.1 Aquatic Animals

Surfactants have been shown to cause a variety of sublethal effects in aquatic organisms, such as changes in ventilation rates, inhibition of larval development, and immobilization. Alcohol ethoxylates and ethoxysulfates affect the ventilation rates of bluegill sunfish. For example, forty-eight hour tests by Maki (1979a, as reported in Goyer et al. 1981, p. 158) demonstrated that concentrations ranging from 0.26 mg/L to 1.2 mg/L of $C_{12.5}EO$ suppress ventilation rates in bluegills by 30 to 50% compared to controls. To a lesser extent, similar effects were also caused by $C_{14.5}EO$. However, 48 hours of exposure to 0.39 mg/L $C_{16}EO_3S$ significantly increased the ventilation rate of bluegills (Maki 1979a, as reported in Goyer et al. 1981, p. 198). Larval development was inhibited in the Eastern oyster (Crassostrea virginica) after 48 hours exposure to a 0.11 mg/L concentration of $C_{14.5}EO$ (Maki 1979b, as reported in Goyer et al. 1981, p. 159), in the Pacific oyster (Crassostrea gigas) after 48 hours exposure to a 0.84 mg/L (average) concentration of $NaC_{12}AS$ (Cardwell et al. 1977, as reported in Goyer et al. 1981, p. 104), and in the horse clam (Tresus capax) after 48 hours exposure to a 0.4 mg/L concentration of $NaC_{12}AS$ (Cardwell et al. 1978, as reported in Goyer et al. 1981, p. 104). Immobilization of barnacle nauplii occurred after 30 minutes exposure to 580 mg/L of a concentration of $C_{10}EO_{20}$ (Wright 1976, as reported in Goyer et al. 1981, p. 159). Daphnia were immobilized by concentrations of sulfates ranging from 42 mg/L for $C_{13}AS$ to 8200 mg/L for C_5AS indicating a trend of increasing AS toxicity with increasing numbers of carbon atoms (Lundahl and Cabridenc 1978, as reported in Goyer et al. 1981, p. 97). Similarly, Wright (1976, as reported in Goyer et al. 1981, p. 97) found $C_{10}AS$ to be approximately ten times as toxic as C_8AS in barnacle larvae (Elininius modestus). However, Bode et al. (1978, as reported in Goyer et al. 1981, p. 101) found toxicity decreased with increasing chain length when budding Hydra attenuata were exposed to C_{10}, C_{12}, C_{14}, and $C_{16}AS$. The decrease was attributed to reduced water solubility at the assay temperature of 20°C.

Most data indicate that increasing the length of the alkyl chain of alcohol sulfates tends to increase toxicity (Table 1). Insufficient data are available on sublethal effects to make such generalizations about ethoxylates.

Another type of effect was detected in whitefish (Caregonus clupeaformis) by Hara and Thompson (1978, as reported in Goyer et al. 1981, p. 103). The olfactory bulbar electric response was suppressed with 0.1 mg/L $C_{12ave}AS$, the lowest concentration at which sublethal effects were observed. The authors considered this an adverse effect because feeding and migrating behavior could be impaired by reduced olfactory sensitivity.

Feather oils of ducks were dissolved after 30 minutes exposure to a solution of 19 mg/L $C_{12}AS$ in distilled water (Choules et al. 1978, as reported in Goyer et al. 1981, p. 110). Such an effect could obviously place waterfowl at increased risk of hypothermia in waters polluted with detergents.

3.2.2 Plants

Surfactants are toxic to aquatic plants. Alcohol ethoxylates have been shown to inhibit the growth of algae. $C_{12-14}AE_6$ was algistatic to populations of the diatom (Navicula seminulum) at concentrations of 5-10 mg/L and to the green algae (Selenastrum capricornutum) at concentrations of 50 mg/L. The same surfactant was algicidal to the diatom at 100 mg/L and to the green algae at 1000 mg/L (Payne and Hall 1979, as reported in Goyer et al. 1981, p. 156).

The growth of 12 species of marine phytoplankton (chlorophyceae) was completely inhibited by $MgC_{12ave}AS$ at concentrations of 100 and 1000 mg/L. Nannochloris sp. and Stichococcus sp. were completely inhibited by this surfactant at 10 mg/L (Ukeles 1965, as reported in Arthur D. Little, Inc. 1977, p. 194). Rockstroh (1967, as reported in Arthur D. Little, Inc. 1977, p. 196) demonstrated the toxicity of $Na\text{-}C_{12ave}AS$ to ciliates (Cyrtolophosis). Exposures of 4 and 15 minutes to concentrations of 0.1 and 0.2 mg/ml caused autolysis of the cytoplasm, fissures in the mitochondrial membrane, and formation of a diffuse mitochondrial edema.

An unusual relationship between toxicity of a coconut-alcohol-derived ethoxysulfate and a red tide dinoflagellate (Gymnodinium breve) was reported by Kutt and Martin (1974, as reported in Goyer et al. 1981, p. 201). Mortality decreased with increasing concentrations of the surfactant (87% with 2.5 ug/L, 63% with 12.5 mg/L, and 44% with 50 ug/L). No explanation was given for these abnormal results.

Surfactants also affect the growth and developmnt of higher plants. Aquatic duckweed (Lemna minor) was adversely affected by exposures to $C_{14.5}AE$. On the basis of frond count, the 7-day EC_{50} was 21 mg/L and on the basis of root length, it was 1.9 mg/L (Bishop and Perry 1979, as reported in Goyer et al. 1981, p. 166). Of ten AE surfactants tested on rye and barley grasses by Valores and Letez (1978, as reported in Goyer et al. 1981, p. 166), n-pri-$C_{12-15}AE_3$ and n-pri-$C_{12-15}AE_3$ were the most toxic to both grasses. Barley growth was reduced 25% and 20%, respectively, and rye growth was reduced 50% and 80%, respectively. All surfactants tested inhibited growth in both grasses at concentrations of 100 mg/L. The least phytotoxic compounds were n-pri $C_{12-15}AE_{20}$, n-pri $C_{9-11}AE_6$, and n-pri-$C_{9-11}AE_8$.

Grain yield was reduced in paddy rice plants watered with 50 mg/L AS. Water absorption by the roots was markedly inhibited, photosynthesis was inhibited, and considerable yellowing of the leaf blade also occurred (Taniyama and Nomura 1978, as reported in Goyer et al. 1981, p. 109). However, a stimulatory effect was demonstrated with corn seeds watered with 0.01, 0.1, or 1 g/L $C_{12ave}AS$ (Nadasy et al. 1972, as reported in Arthur D. Little, Inc. 1977, p. 197). Seeds weighed 97%, 130%, and 136% of controls, respectively. Similar increases also occurred in length and dry weight of the corn plants. Treatment of barley seeds (Hordeum vulgare L.) with 100% active $NaC_{12ave}AS$ ($10^{-3}M$) for 24 hours before germination resulted in significant growth inhibition as determined by shoot length (Antonielli and Lupatteli 1977, as reported in Goyer et al. 1981).

3.3 Chronic Effects

Few data are available on the chronic effects of NEODOL products. Much of the data that are available are "no observed effect concentrations" (NOEC) for ethoxylates derived by Maki from studies with fathead minnows and Daphnia. For example, a chronic toxicity test emphasizing egg production and spawning rate in minnows resulted in a NOEC of 0.32 mg/L, the highest concentration tested for $C_{12.5}EO$ (Maki 1979c, as reported in Goyer et al. 1981, p. 160). For Daphnia, a similar NOEC, 0.27 mg/L, was obtained with a chronic exposure to $C_{13.67}EO_{2.25}S$ (Maki 1979d, as reported in Goyer et al. 1981, p. 200). Growth was inhibited, however, in the fathead minnow after a one-year exposure to 0.22 mg/L concentration of $C_{13.7}EO_{2.25}S$ (Maki, 1979d, as reported in Arthur D. Little, Inc. p. 200). Maki (1979a, as reported in Shell a, p. 18) demonstrated that chronic exposure to low levels of alcohol ethoxylates decrease respiratory rates in fathead minnows, whereas the rates are increased by exposures to ethoxysulfates. The mode of action is unknown.

Other studies have shown that egg fertilization can be inhibited in crustaceans by exposure to surfactants. Grammo and Jorgensen (1975, as reported in Goyer et al. 1981, p. 160 and Shell a, p. 19) caused almost complete inhibition of egg fertilization by exposing mussels (Mytilus edulis) to 2 mg/L TAE_{10} (ethoxylated tallow alcohol) for five months. Some inhibition occurred at concentrations as low as 0.1 mg/L. Arthur D. Little, Inc. (1977) reviewed the results of a chronic toxicity test on clam (Mercenaria mercenaria) and oyster (Crassostrea virginica) larvae. At 1 mg/L AS, fertilized egg development was significantly retarded compared to controls, while complete inhibition of development occurred at 2.5 mg/L. After a 10-day exposure to 5 mg/L, clam mortality was 68%; oyster mortality was 82% after 12 days exposure.

More studies are needed before conclusions can be reached about the long-term toxicity of surfactants to aquatic systems. The limited data available indicate that, generally, concentrations exceeding 0.2 mg/L can cause adverse effects in aquatic organisms when exposures last for several months.

4.0 ENVIRONMENTAL FACTORS INFLUENCING AQUATIC TOXICITY

4.1 Water Hardness

Water hardness appears to play a role in the toxicity of at least the alcohol sulfates. With an increase in water hardness, the toxicity (and uptake) of AS increases (Arthur D. Little, Inc. 1977, p. 165; Goyer et al. 1981, p. 85 and 106). The effect of water hardness on ethoxylate and ethoxysulfate toxicity is less certain, however. Studies by Maki and Bishop (1979) and Maki et al. (1979) using Daphnia and $C_{14.5}EO_7$ suggest a slight decrease in EO toxicity with increased water hardness (as reported in Goyer et al. 1981, p. 161). However, no such trends were apparent in similar studies by Procter and Gamble Company (unpublished data, as reported in Goyer et al. 1981). No intra-species, water hardness data were available for EOS.

4.2 Biodegradability

It is generally agreed in all reports reviewed (Shell a and b; Arthur D. Little, Inc. 1977, Goyer et al. 1981) that the linear primary alcohol-based surfactants do not persist in laboratory or field tests. Even slightly branched or secondary structures are easily degraded, albeit at a somewhat slower rate (Table 1). Concentrations of EO as high as 1000 mg/L in shake flask tests simulating spills, were 70 to 80% degraded in three days (Kravetz et al. 1979, as reported in Shell a, p. 5). Goyer et al. (1981, p. 143) suggests, however, that a study using an atmosphere containing 70% oxygen to enable "complete surfactant oxidation to CO_2" may not be indicative of degradation rates occurring in the same time period under natural conditions. Ethoxysulfates added to activated sludge were completely metabolized to carbon dioxide and water within five to ten days (Miura et al. 1979 and Itoh et al. 1979, as reported in Goyer et al. 1981, p. 195). Numerous other studies using sludge or river, estuarine or ocean waters have demonstrated the rapid breakdown of these compounds. Degradation of ethoxylates has been shown to be generally faster in saltwater than in freshwater, and faster in freshwater at high temperatures than at lower temperatures (Schoberl and Mann, 1976, as reported in Goyer et al. 1981, p. 142). The degradation rate of ethoxylates

is also influenced by the length of the EO chain; increased length of the chain caused degradation to be slower, especially in freshwater at low temperature. Ethoxylates having 100 EO units/mole of alcohol biodegrade considerably slower (20% ultimate biodegradation in 21 days) than those having up to 30 EO units/mole of alcohol (90% or more in 21 days) (Kravetz et al. 1979, as reported in Shell p. 5; Goyer et al. 1981, p. 144). Shell claims to market only products having a maximum of 13 EO units/mole (NEODOL 45-13). Biodegradation was essentially complete 10 days after DOBONOL 45-7, simulating a spill condition, was added to an activated sludge medium (Cook 1979, as reported in Shell a, p. 5).

Degradation results in a rapid loss of toxicity of surfactants to aquatic organisms. Products resulting from EO biodegradation were much less toxic to rainbow trout and goldfish than were the parent compounds (Reiff 1976 and Kurata et al. 1977, as reported in Goyer et al. 1981, p. 153). Maki et al. (1979, as reported in Goyer et al. 1981, pp. 151 and 152) concluded that initial concentrations of 3 mg/L or less of $C_{14.5}$ EO_7 in stream water effluent was non-toxic to fathead minnows within 24 hours. At 10 mg/L, toxicity was observed for five days in stream water and for two to three days in secondary effluent. NEODOL-type surfactants are readily utilized as a carbon (energy) source by bacteria present in activated sludge and natural waters (Shell a, p. 5).

4.3 Exposure

By comparing an organism's sensitivity to a chemical with the concentration of that chemical likely to be present in the environment, one can often predict with reasonable accuracy the potential threat posed by the chemical to the organism. Modeling techniques have been used to estimate surfactant concentrations in 20 estuarine locations (Maki 1979b, as reported in Goyer et al. 1981, pp. 163 and 164). Estimated maximum EO concentrations ranged from 0.2 ug/L in Penobscot Bay, Maine to 19.8 ug/L in the Hudson River; the geometric mean for all estuaries was 3.2 ug/L. It should be noted that these values are probably high because the model was provided with an elevated

estimate of inflow from sewage treatment plants. Further, degradation was not factored in.

Comparing these estimated concentrations with the acute sensitivities of test species, it appears unlikely that, short of a direct spill, concentrations of surfactants acutely dangerous to aquatic organisms will be attained in the environment.

5.0 MAMMALIAN TOXICITY

5.1 Acute Effects

All LD_{50} data reviewed indicate that, at their worst, NEODOL surfactants are moderately toxic (0.5 to 5 g/kg) when rated according to Gosselin et al. (1976, as reported in Shell b, p. 10). LD_{50}s for all types of surfactants generally exceed 1.0 g/kg. Alcohols appear to be the least toxic (oral LD_{50}s >5 g/kg), becoming more so with either sulfation or ethoxylation. Sulfation, however, decreases the toxicity of alcohol ethoxylates (Shell b, pp. 9-11).

Acute oral toxicity studies with rats indicate that the degree of ethoxylation has some influence on toxicity. For example, NEODOL 91-2.5 produced LD_{50}s ranging from 2.7 to 10 g/kg (Shell Internal Reports HSE-78-0156, TLGR.124.79, and TLGR. 088.80, as reported in Shell b, p. 11), whereas NEODOL 91-8 was more toxic, exhibiting LD_{50}s of 1.0 or 2.7 g/kg (Shell Internal Reports TLGR.088.80 and TLGR.0024.76, as reported in Shell b, p. 4). The length of the alkyl chain does not appear to influence the acute toxicity of alcohol ethoxylates (Shell b, Table V, p. 12).

Skin and eye irritation tests with rabbits have demonstrated that NEODOL surfactants, except the alcohols, are generally severe irritants at high or undiluted concentrations (Shell b, pp. 17 and 20). Shell b (p. 21), however, reports that when directions are followed, actual use concentrations for NEODOL products are $\leq$0.04%. At 0.1% dilutions, NEODOL products tested ranged from non-irritating for alcohols and alcohol sulfates to non-irritating to mildly irritating for alcohol ethoxylates (Shell b, p. 21).

Most NEODOL products produced negative results in skin sensitization tests (Shell b, p. 25, Table XIV). Most exceptions showed some, weak or moderate, sensitivity in one type of test but none in other tests. By contrast, NEODOL 25-3 was found to be a very weak sensitizer in the Maximization Test (Shell b, p. 25). Results of repeated-insult patch tests of NEODOL products in human volunteers agree with most observations in animal studies that NEODOL products are not skin sensitizers (Shell b, pp. 25 and 27).

5.2 Subchronic Effects

Available data indicate that effects of subchronic exposures to surfactants mainly involve changes in organ and body weights. Studies exposing rats for 16 weeks to a diet containing C_{12} sulfate (4% of the total diet) resulted in reduced body weights (Arthur D. Little, Inc. 1977 and 1981 and Fitzhugh and Nelson 1948, as reported in Shell b, p. 29). Compared to controls, ethoxylates ($C_{13}EO_6$ and $C_{14}EO_7$) produced elevated liver weights in rats exposed to concentrations equivalent to 1% of the total diet (Brown and Benke 1977, Arthur D. Little, Inc. 1977, and Goyer et al. 1981, as reported in Shell b, p. 29). Lower body weight was also observed with exposure to $C_{13}EO_6$. Other subchronic feeding studies exposing rats to sulfates and ethoxysulfates produced no major biological effects at concentrations up to 0.1% of the diet for 13 weeks (Shell Internal Report R(T)-12-66 and Walker et al. 1967, as reported in Shell b, p. 29). Increases in serum urea or protein concentrations and increases in some organ weights occurred at concentrations of 0.5% of the diet. Because histopathology was normal, these effects were considered minor.

Another type of effect observed was slight inhibition of the progression of cholesterol-induced atherosclerosis in rabbits (Kivak et al. 1975 as reported in Goyer et al. 1981). The mechanism of action is not known, although a reduction in accumulation of cholesterol esters in aortic tissue was suggested as a possibility (Morin et al. 1974, as reported in Goyer et al. 1981).

5.3 Chronic Effects

The few data available on the chronic effects of surfactants do not demonstrate any alarming effects. One or two year studies using 1.0% alcohol sulfate (Goyer et al. 1981, p. 118) or 0.5% alcohol ethoxysulfate (Arthur D. Little, Inc. 1977, p. 377), respectively, in the diets of rats have produced no adverse effects. However, 1% alcohol ethoxylate, C_{12-13} $EO_{6.5}$, added to the diets of rats for two years resulted in reduced body weight, elevated organ to body weight ratios for liver, kidney, brain, and heart in females and for liver in males and increased incidence of focal myocarditis, a common spontaneous lesion found in aging populations of rats (Procter and Gamble, unpublished data, as reported in Goyer et al. 1981, p. 174). Food consumption was also reduced in groups having reduced body weights, and was attributed to poor palatability of the diet. Reduced body weights and increased organ to body weight ratios also occurred in females at the 0.5% treatment level. A second feeding study using as much as 1.0% C_{14-15} EO_7 in the diets of rats for two years resulted in reduced body weight gains for females and males, and in decreased absolute organ weights for liver, kidney, heart, and thyroid/ parathyroid glands in females and for brain and adrenals in males in the 1% treatment groups. Gross incidences of focal myocarditis increased with increasing treatment levels for all groups of rats at 12 months, but severity of lesions was not treatment-related (Proctor and Gamble, unpublished data, as reported in Goyer et al. 1981, p. 175). Eighteen months exposure to repeated dermal applications of up to 5.0% $C_{12-13}EO_{6.5}$ produced no notable results in ICR Swiss mice (Procter and Gamble Company, unpublished data, as reported in Goyer et al. 1981, p. 176).

5.4 Carcinogenicity

No evidence for carcinogenic potential of NEODOL products has emerged from the limited data available from long-term oral or dermal studies exposing rats or mice to C_{12-13} $EO_{6.5}$ or C_{14-15} EO_7 (Proctor and Gamble Company, unpublished data, as reported in Goyer et al. 1981, pp. 174-176); $C_{12}S$ (Goyer et al. 1981, p. 119); or $C_{12}EO_3S$ (Tusing et al. 1962, as reported in Arthur D. Little, Inc. 1977, p. 378).

5.5 Mutagenicity

Mutagenicity has not been demonstrated for any NEODOL product tested with *in vitro* or *in vivo* mammalian systems or in bacterial or yeast systems. The following NEODOL products have been tested for mutagenicity: C_{12ave} AS (Hope 1977, as reported in Goyer et al. 1981, p. 120); n-pri-$C_{12-13}EO_3$ (Shell Toxicology Laboratory unpublished data, as reported in Goyer et al. 1981, p. 177); $C_{12-15}EOS$ (Hope 1977, as reported in Goyer et al. 1981, p. 209); $C_{12-13}EO_{2.5}S$ (53:43) (Inoue et al. 1980, as reported in Goyer et al. 1981, p. 210); and n-pri-$C_{12-15}EO_3S$ (Shell Research Limited, unpublished data, as reported in Goyer et al. 1981, p. 210).

5.6 Teratogenicity/Reproduction

Few teratogenesis/reproduction studies have been performed with NEODOL products; no teratogenesis studies have been performed with AES administered alone. However, formulations containing AES administered orally to mice, rats, or rabbits have produced no teratogenic effects (Iimori et al. 1973, Iseki 1972, Nolan et al. 1975, and Palmer et al. 1975 as reported in Arthur D. Little, Inc. 1977, p. 379). Results from testing the following chemicals have shown no cause for concern: C_{14-15} EO_7 and $C_{12}EO_6$ (Proctor and Gamble unpublished data, as reported in Arthur D. Little, Inc. 1977, p. 322) and $C_{12}EO_3S$ (Tusing et al. 1962, as reported in Arthur D. Little, Inc. 1977, p. 378).

An alcohol sulfate whose chain length was not identified has also been tested (Nomura et al. 1980, as reported in Goyer et al. 1981, p. 119). Dermal applications of 10 to 20% concentrations of the alcohol sulfate to pregnant mice on days 1 to 10 of gestation interfered with embryonic development at the cleavage stage. Applications of 2% on days 1 through 17 also reduced the number of pregnancies, but the number of animals compared was too small to be statistically significant. Dermal applications of 10% alcohol sulfate twice a day prior to implantation (days 0 to 3) resulted in an elevated incidence of deformed embryos, compared to controls (29.1% vs 4.9 of 0 in controls) (Nomura

et al. 1980, as reported in Goyer et al. 1981, p. 120). Dermal application of the alcohol sulfate during late pregnancy did not interfere with gestation.

More tests, especially with AES products, are necessary before conclusions can be reached about the teratogenic potential or reproductive effects of surfactants.

5.7 Studies in Humans

Studies using human volunteers have demonstrated the skin irritation properties of NEODOL products (Shell b, p. 27). In most cases, 1% dilutions caused very slight to mild irritation with repeated exposures. Alcohol ethoxylates appear to be the least irritating, with only non-to-mild irritations caused by repeated exposures to dilutions up to 25%. Use of certain alcohol ethoxylates as analgesics and anesthetics have caused no adverse reactions in humans (Goyer et al. 1981, p. 130).

5.8 Metabolism

Alcohol sulfates, short-chain ethoxylates, and ethoxysulfates (Goyer et al. 1981, pp. 121, 178, and 211, respectively) are readily absorbed when administered orally to rats, and are primarily excreted in urine. Increasing the alkyl chain length of an ethoxylate decreases its excretion in urine and feces, and increases the amount in expired air (Goyer et al. 1981, p. 178). Increasing the length of the EO unit of an ethoxysulfate causes it to be poorly absorbed and excreted primarily unchanged in the feces (Arthur D. Little, Inc. 1977, p. 381).

Cutaneous absorption of alcohol ethoxylates (about 50%) is somewhat slower than absorption after oral administration (>75%) (Drotmann 1977 and 1980, as reported in Shell b, p. 33). Dermal absorption of similar alcohol ethoxylates is greater than dermal absorption of alcohol sulfates or ethoxysulfates (Black and Howes 1979, as reported in Shell b, p. 33). Maximum absorption of alcohol sulfates on human callus occurred with a chain length of 12 carbons (Dominguez et al. 1977, as reported in Goyer et al. 1981, p. 123).

After application of 100 mg of an alcohol ethoxylate ($C_{12}EO_6$) to human skin, most (81% average) was recovered from swabbing the skin after 144 hours (Drotman 1980, as reported in Goyer et al. 1981, p. 180).

6.0 CONCLUSIONS

6.1 Toxicity to Non-Mammalian Organisms

Certain structure-activity relationships have been delineated for the alcohol-derived surfactants in aquatic systems (see Table 1). As the number of carbon atoms in the alkyl chain of straight-chain alcohols increases, the toxicity of the alcohol decreases. When the (EO) chain length of alkyl ethoxylates remains the same, an increase in the alkyl chain length increases toxicity. Conversely, when the alkyl chain remains the same, an increase in the EO chain length decreases toxicity (as opposed to the response of laboratory rodents). The sulfation of the end EO group reduces the acute toxicity of these compounds by a factor of more than 20 compared to the parent ethoxylate compound. Alcohol sulfates also appear to be less acutely toxic to aquatic organisms than are the alcohol ethoxylates. Although anionic surfactants are less acutely toxic than nonionic surfactants, fish have a greater ability to recover after exposure to nonionic surfactants than to anionic surfactants.

Surfactants have been shown to cause a variety of sublethal effects in aquatic organisms, such as changes in ventilation rates, inhibition of larval development and immobilization.

The limited data available on the chronic effects of surfactants (mainly the ethoxylates) indicate that growth inhibition and altered respiratory rates in crustaceans can be caused by long-term exposures. In general, exposure for several months to concentrations exceeding 0.2 mg/L can cause adverse effects in aquatic animals.

Surfactants have been shown to inhibit the growth and development of aquatic microflora and higher plants such as barley and rye.

Due to dissolution of the waterproofing oils on their feathers, waterfowl may be at increased risk of hyperthermia if exposed to surfactants.

NEODOLs and the other alcohol-based surfactants do not persist in aquatic environments, and are readily biodegraded to apparently non-toxic intermediates, then to carbon dioxide and water. Short of a direct spill, concentrations of surfactants reaching waterways would be substantially lower than those that are acutely toxic to aquatic organisms.

Effects of repetitive exposures to surfactants have not been adequately studied. Additional toxicity tests should focus on the effects of continuous exposure of early life stages of test organisms to low concentrations of surfactant, a situation such as might exist near a sewage outfall or drainage/ overflow conduit.

The effects of 1,4-dioxane contamination of ethoxysulfates on aquatic organisms cannot be determined from available data.

6.2 Mammalian Toxicity

In general, NEODOL products exhibit a low order of toxicity to mammals in toxicity tests (see Table 2). At worst, acute toxicity can only be labeled moderate, except in the cases of skin or eye irritations which are often severe for undiluted derivitized NEODOL products. However, dilutions of 0.1% are generally non-irritating, and according to Shell, use concentrations are only $\leq$0.04%.

NEODOL alcohols, which are the least acutely toxic to mammalian systems, become more toxic with either sulfation or ethoxylation. Sulfation of an ethoxylate, however, decreases toxicity. Length of the alkyl chain does not appear to play a significant role in acute toxicity of alcohol ethoxylates.

Subchronic and chronic dietary tests resulted in reduced body weights and increased organ to body weight ratios for some organs. There was no evidence of carcinogenicity or mutagenicity for any NEODOL product tested.

TABLE 2. Effects of NEODOL(R) products in laboratory mammals

	Alcohol Sulfates	Alcohol Ethoxylates	Alcohol Ethoxysulfates
Acute LD_{50} (g/kg)	Oral or dermal, rats, or rabbits, >1; commercial use dilutions between 5 and 15.	Oral, rats, 0.87 to >10; dermal, rats or rabbits, >2; inhalation (4 hrs. exposure) rats, between 1.5 and 3 mg/L.	Oral, rats, 1.7 to 5; dermal, rabbits, 4.7 to 12.9.
Skin irritation (rabbits)	0.1% dilution, non-irritating; ≥10% dilution, severe.	0.1%, non-irritating to mild; ≥10%, mild to severe.	0.1%, non-irritating to minimal; undiluted, mild to severe.
Eye irritation (rabbits)	Undiluted, severe to extreme.	≥10%, practically non-irritating to extreme; 0.1% non-irritating.	Undiluted, severe; 0.1% non-irritating.
Subchronic	4% given in diet for 16 weeks reduced body weight of rats; cumulative skin irritation; daily ingestion of 250 mg/kg for two months slightly inhibited progression of cholesterol-induced atherosclerosis in rabbits.	1% given in diet for 13 weeks reduced body weight, increased liver weight of rats.	0.5% given in diet for 13 weeks increased kidney, liver, and heart weights in female and kidney weights in male rats; repeated skin (guinea pigs and rabbits) exposure to 10% dilutions, severe irritation; 1% no reaction.

TABLE 2. Effects of NEODOL(R) products in laboratory mammals (Continued)

	Alcohol Sulfates	Alcohol Ethoxylates	Alcohol Ethoxysulfates
Chronic	1% in diet, rats, one year, no adverse effects.	1% in diet, rats, two years, reduced body weight, elevated organ to body weight ratios, increased incidence of focal myocarditis. 5% dermal application to mice for 1.5 years, no notable results.	0.5% in diet, rats, two years, no adverse effects.
Carcinogenic	No evidence from long-term feeding studies in rats or skin-painting tests in mice.	No evidence from long-term feeding tests in rats or from long-term percutaneous administration to mice.	No evidence from two-year feeding (0.5%), drinking water (0.1%), or skin-painting (5.0%) studies.
Mutagenic	No effects on chromosomes of rat bone marrow cells from 90 day diet of maximum tolerated dose (1.13% active ingredient).	No evidence from in vitro and host-mediated mutagenicity tests.	No effects on choromosomes of rat bone marrow cells from 90 day diet of maximum tolerated dose (1.13% active ingredient). No evidence from hamster embryo cell culture or yeast or bacteria studies.

TABLE 2. Effects of NEODOL(R) products in laboratory mammals (Continued)

	Alcohol Sulfates	Alcohol Ethoxylates	Alcohol Ethoxysulfates
Teratogenic/ Reproductive	No evidence from ingestion of up to 300 mg/kg during gestation. Daily skin application of 20% to pregnant mice on days 1 to 10 interfered with embryonic development; 10% 2 times/day, pregnant mice, days 0 to 3, elevated incidence of deformed embryos. Doses severely toxic to dams reduced litter size and caused fetal loss in mice but not in rats or rabbits.	No evidence from feeding (up to 0.5%) studies in rats or rabbits.	No data on AES administered alone. No evidence from oral administration of formulations containing AES to mice, rats, or rabbits. No adverse reproductive effects from 0.1% in the diets of rats for two generations.

Data are generally lacking on teratogenic/reproductive effects. Data available from a limited number of feeding studies indicate no teratogenicity for any of the NEODOL products tested. However, repeated dermal exposure of mice to high concentrations of an alcohol sulfate during early gestation interrupted cleavage of eggs and retarded fetal development. Further studies should be performed to clarify the teratogenic potential and/or reproductive effects of NEODOL products.

Animal studies show that, in general, NEODOL products administered orally are readily absorbed, metabolized, and primarily excreted in the urine. Cutaneous exposure, the usual route of exposure to most surfactants, results in slower absorption of alcohol ethoxylates.

6.3 1,4-Dioxane Contamination

There is concern because 1,4-dioxane is a contaminant of some NEODOL products. Shell (May 7, 1980 memorandum to G.T. Youngblood) claims that 1,4-dioxane ($OCH_2CH_2OCH_2CH_2$) is present only in their EOS products, and postulates that it is formed during sulfation of the EO and that the presence of a polyoxyethylene chain and a highly acidic agent, such as sulfur trioxide, are required. The typical potential exposure for an adult female is estimated to be 1.65×10^{-8} g/kg/d from hair shampoo and 7.56×10^{-10} g/kg/d from light duty liquid (Shell c 1980, Appendix B-3). Worst case estimates are 1.12×10^{-7} g/kg/d for shampoo and 6.68×10^{-9} g/kg/d for light duty liquids.

It is impossible to determine from the data provided whether dioxane contributes to the observed EOS toxicity. However, dioxane contamination does not alter the significance of the toxicity of NEODOL products, for it appears that it is often the dioxane-contaminated products (EOS) to which environmental species are exposed. It would be useful to compare the toxicities of contaminated samples with purified samples.

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APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Daphnia magna			
24	$C_{12-13}EO_{6.5}$	0.57	Shell Internal Report 1974. ECO 1 Program
	$C_{12-18}EO_{14}$	1.1	Arthur D. Little, Inc. 1978
	$C_{12-15}EO_{9}$	1.71	Shell Internal Report 1974. ECO 1 Program
	$C_{14}EO_{8}$	2.0	Arthur D. Little, Inc. 1978
	$C_{12-14}EO_{7.4}$	2.3	Arthur D. Little, Inc. 1978
	$C_{12-14}EO_{6.3}$	2.5	Arthur D. Little, Inc. 1978
	$C_{12-15}EO_3S40$ (39%)	3.3	Shell Internal Report 1974. ECO 1 Program
	$C_{12}EO_3S$	5.0	Lundahl et al. 1972
	$C_{12-14}EO_{11}$	5.1	Arthur D. Little, Inc. 1978
	$NaC_{12-14}AS$	6.3	Arthur D. Little, Inc. 1978
	NaC_{12}aveAS (Ziegler derivative)	13.5	Lundahl et al. 1972
	$C_{12-14}EO_3S$ (Ammonium salt)	16.3	Continental Oil Co., unpublished data
	$C_{12-14}EO_3S$ (Sodium salt)	18.9	Continental Oil Co., unpublished data

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Daphnia magna			
24	$C_{11-16}EO_3S$	19.6 (average)	Unilever Research Laboratories, unpublished data
48	$C_{14-15}EO_7$	0.36 (average)	Goyer et al. 1981
	$C_{12-14}EO_{2.2}S$ (Natural alcohol derived)	21	Lundahl et al. 1972
	$C_{12}EO_3S$ (Ziegler derived)	37	Lundahl et al. 1972
96	$C_{14}EO_3$	0.73	U.S. Food and Drug Administration
	$C_{14}EO_1$	0.83	U.S. Food and Drug Administration
	$C_{14}EO_2$	1.53	U.S. Food and Drug Administration
	$C_{14}EO_4$	1.76	U.S. Food and Drug Administration
	$C_{14}EO_6$	4.17	U.S. Food and Drug Administration
	$C_{14}EO_9$	10.07	U.S. Food and Drug Administration
Bluegill sunfish (*Leopomis macrochirus*)			
24	$C_{15.9}EO_{2.1}S$	0.3	Procter and Gamble Company, unpublished data
	$C_{12-15}EO_3$	1.8	U.S. Dept. of Interior 1968

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Bluegill sunfish (Leopomis macrochirus)			
	$C_{12-15}EO_9$	1.87	Shell Internal Report 1974. ECO 1 Program
		2.4 (average)	U.S. Dept. of Interior. 1968
	$C_{14.7}EO_1S$	1.9	Procter and Gamble Co., unpublished data
	$C_{15}EO_{2.6}S$	<2.1; <2.4	Procter and Gamble Co., unpublished data
	$C_{12-13}EO_{6.5}$	2.45; 2.36	Shell Internal Report. 1974. ECO 1 Program
	$C_{14}EO_{1.9}S$	4.3	Procter and Gamble Co., unpublished data
	$C_{14}EO_{2.6}$	<5.7; <7.5	Procter and Gamble Co., unpublished data
	$C_{14}EO_3S$	7.1	Procter and Gamble Co., unpublished data
	$C_{12-15}EO_9$ (75% linear primary)	8.0	Cook Research Laboratories 1966
	$C_{14-18}EO_9$	10	Cook Research Laboratories 1966

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Bluegill sunfish (Leopomis macrochirus)			
	$C_{17.9}EO_{1.9}S$	10.8	Procter and Gamble Co., unpublished data
	$C_{12-15}EO_9$ (98% linear primary)	11.0	Cook Research Laboratories 1966
24	$C_{19.6}EO_{1.1}S$	15	Procter and Gamble Co., unpublished data
	$C_{13}EO_3S$	24	Procter and Gamble Co., unpublished data
	$C_{12-15}EO_3A$	32	Shell Internal Report 1971
	$C_{12-15}EO_3S$	32	Shell Internal Report 1971
	$C_{12}EO_3S$	37	Procter and Gamble Co., unpublished data
	$C_{12}EO_3S$	73	Procter and Gamble Co., unpublished data
	$C_{12}EO_{2.1}S$	87	Procter and Gamble Co., unpublished data
	C_8EO_3S	>250	Procter and Gamble Co., unpublished data

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Bluegill sunfish (Leopomis macrochirus)			
	$C_{10}EO_{2.1}S$	375	Procter and Gamble Co., unpublished data
96	$NH_4C_{15}AS$, branched	2.13 (1.37-3.31)	Procter and Gamble Co., unpublished data
	$NH_4C_{12-14}AS$	3.2 (2.8-3.7)	Procter and Gamble Co., unpublished data
	$NH_4C_{15}AS$	3.39 (2.59-4.43)	Procter and Gamble Co., unpublished data
	$NaC_{12}AS$	4.5	Bishop and Perry 1979
	$NaC_{12}AS$	4.83 (4.06-5.75)	Procter and Gamble Co., unpublished data
	$NH_4C_{15}AS$	5.19 (3.97-6.77)	Procter and Gamble Co., unpublished data
	$NH_4C_{11}AS$, branched	16.5 (13.1-21.0)	Procter and Gamble Co., unpublished data
	$NH_4C_{13}AS$, branched	18.4 (15.2-22.2)	Procter and Gamble Co., unpublished data
	$NH_4C_{12}AS$	20.3 (16.0-25.7)	Procter and Gamble Co., unpublished data

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Bluegill sunfish (Leopomis macrochirus)			
	$NH_4C_{16}AS$	21.7 (16.7-28.1)	Procter and Gamble Co., unpublished data
	$NH_4C_{11}AS$	26.0 (19.0-35.4)	Procter and Gamble Co., unpublished data.
	$C_{16-18}AS$	76.0 (50-116)	Procter and Gamble Co., unpublished data.
	NaC_1AS	1000	Procter and Gamble Co., unpublished data.
Rainbow trout (Salmo gairdneri)			
96	$C_{14-15}EO_7$	0.8	Abram et al. 1977
		0.9	Reiff 1976
	$C_{12-15}EO_9$	1.2	U.S. Dept. of Interior. 1968
	$C_{12-14}EO_{10.5}$	1.8 and 0.8	Reiff et al. 1979
	$C_{12-15}EO_3$	1.3 and 1.7	Shell Internal Report, TLGR 113.78
	$C_{12-13}EO_2$	1 - 2	Shell Internal Report, TLGR 0115.78
	$C_{14-15}EO_{11}$	1.8 - 2.5	Reiff 1976

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Rainbow trout (Salmo gairdneri)			
	$C_{14-15}EO_{11}$	1.1	Abram et al. 1977
	$C_{12-15}EO_{7}$	2.7	Arthur D. Little, Inc. 1978
96	C_{12-13}	4 - 10	Shell Internal Report, TLGR 0161.78
	$C_{14-15}EO_{18}$	5 - 6.3	Shell Internal Report, TLGR 0064.77
	$C_{9-10}EO_{2.5}$	5 - 7	Shell Internal Report, TLGR 79.068
	C_{9-10}	6 - 10	Shell Internal Report, TLGR 0166.78
	$C_{9-10}EO_{5}$	8 - 9	Shell Internal Report, TLGR 0066.77
	$C_{12-15}EO_{3}S$	8.9 (7.3-10.3)	Shell Chemical Co., unpublished data
	$C_{12-13}EO_{3}S$	28 (23-35)	Shell Chemical Co., unpublished data
	C_{12-15}	45	Shell Internal Report, TLGR 0052.77
	$C_{9-10}EO_{2.5}S$	400 - 450	Shell Chemical Co., unpublished data
Fathead minnow (Pimephales promelas)			
24	$C_{16}EO_{6}S$	0.8	Monsanto Co., unpublished data
	$C_{16}EO_{4}S$	0.9	Monsanto Co., unpublished data

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Fathead minnow (Pimephales promelas)			
	$C_{16}EO_2S$	1.0	Monsanto Co., unpublished data
	$C_{12}EO_2S$	1.5	Monsanto Co., unpublished data
	$C_{14}EO_2S$	1.8	Monsanto Co., unpublished data
24	$C_{12-14}EO_{6.3}$	1.8	Arthur D. Little, Inc. 1978
	$C_{12-14}EO_{7.4}$	1.8	Arthur D. Little, Inc. 1978
	$C_{18}EO_6S$	2.1	Monsanto Co., unpublished data
	$C_{14}EO_4S$	4.0	Monsanto Co., unpublished data
	$C_{14}EO_6S$	9.3	Monsanto Co., unpublished data
	$C_{18}EO_4S$	15	Monsanto Co., unpublished data
	$C_{11}EO_4S$	17	Monsanto Co., unpublished data
	$C_{18}EO_2S$	80	Monsanto Co., unpublished data
Goldfish (Carassius auratus)			
6	$C_{12-14}EO_8$	1.8	Reiff et al. 1979
	$C_{15}EO_{3.2}S$, branched	3.7	Gafa 1974

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Goldfish (Carassius auratus)			
	$C_{12-14}EO_{10.5}$	4.3	Reiff et al. 1979
	$nC_{14}AS$ (92.4% AI)	5.0	Gafa 1974
	$C_{12}EO_4$	5.2	Marchetti 1964
	$C_{14}EO_3S$	6.0	Gafa 1974
	$nC_{14}AS$ (94.3% AI)	6.3	Gafa 1974
	$C_{14}AS$ (94% AI, branched)	7.8	Gafa 1974
	$nC_{12-15}AS$ (95.8% AI)	7.8	Gafa 1974
	$C_{12-15}AS$	7.8	Gafa and Lattanzi 1974
	$C_{11-15}AS$	8.1	Gafa and Lattanzi 1974
	$C_{14}EO_3S$ (5% branched)	8.1	Gafa 1974
	$C_{13}EO_5$	8.5	Shell Internal Report, TLGR 79.068
	$nC_{12-16}AS$ (94.3% AI)	12.0	Gafa 1974
	$nC_{13}AS$ (94.8% AI)	18.3	Gafa 1974
	$C_{16}EO_{3.4}S$	41.0	Gafa 1974

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Goldfish (Carassius auratus)			
	$C_{14}AS$ (98% AI, branched)	49.1	Gafa 1974
	$C_{12}EO_{2.6}S$	55.0	Gafa 1974
	$nC_{12}AS$ (93% AI)	60.0	Gafa 1974
	$C_{12}EO_{2.6}S$ (5% branched)	66.5	Gafa 1974
	$nC_{16}AS$ (95.3% AI)	>300	Gafa 1974
1	$C_{12}EO_2$	2	Gloxhuber et al. 1968
	$C_{12}EO4$	4	Gloxhuber et al. 1968
	$C_{12}EO_6$	5	Gloxhuber et al. 1968
	$C_{12}EO_8$	7	Gloxhuber et al. 1968
	$C_{12}EO_{10}$	10	Gloxhuber et al. 1968
	$C_{12}EO_{12}$	20	Gloxhuber et al. 1968
	$C_{12}EO_{14}$	30	Gloxhuber et al. 1968
	$C_{12}EO_{16}$	40	Gloxhuber et al. 1968
	$C_{12}EO_{18}$	100	Gloxhuber et al. 1968

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Goldfish (Carassius auratus)			
48	$C_{12-15}EO_9$ (oxo-9™)	1.4	Kurata et al. 1977
	$C_{12-15}EO_9$ (LA-9™)	1.9	Kurata et al. 1977
	$C_{12-14}EO_7$	3.3	Kurata et al. 1977
	$C_{12-14}EO_9$	5.1	Kurata et al. 1977
	$C_{12-14}EO_{12}$	12.0	Kurata et al. 1977
Hermit crab			
48	$C_{12-15}EO_3$ (30%, kerosene solution)	85	Shell Internal Report, EMGR 0150.71
	$C_{12}EO_1$ (30%, isopropanol solution)	<<1000	U.S. Dept. of Interior 1968
	$C_{12}EO_3$ (30%, isopropanol solution)	<1000	Shell Internal Report, EMGR 0162.71
	$C_{12}EO_9$ (30%, isopropanol solution)	<<1000	Shell Internal Report, EMGR 0162.71
	$C_{14-15}EO_3$ (30%, isopropanol solution)	<1000	Shell Internal Report, EMGR 0162.71

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Hermit crab			
	$C_{14-15}EO_3$ (30%, kerosene solution)	<2000	Shell Internal Report, EMGR 0162.71
	$C_{14}EO_3$ (30%, isopropanol solution)	1500	Shell Internal Report, EMGR 0162.71
	$C_{16}EO_9$ (30%, isopropanol solution)	2000	Shell Internal Report, EMGR 0162.71
	$C_{14}EO_9$ (30% ?)	2500	Shell Internal Report, EMGR 0162.71
	$C_{16}EO_3$ (30%, isopropanol solution)	3500	Shell Internal Report, EMGR 0162.71
	$C_{14-15}EO_1$ (30%, isopropanol solution)	3000-6000	Shell Internal Report, EMGR 0162.71
	$C_{16-18}EO_6$ (30%, isopropanol solution)	4000	Shell Internal Report, EMGR 0162.71
	$C_{16-18}EO_9$ (30%, isopropanol solution)	4000	Shell Internal Report, EMGR 0162.71
Brown shrimp			
48	$C_{14-15}EO_3$ (30%, kerosene solution)	50	Shell Internal Report, EMGR 0162.71

APPENDIX A. Acute toxicity (LC_{50}) of alcohol surfactants to aquatic animals (continued)

Exposure (hours)	Surfactant	LC_{50} (mg/L)	Reference
Brown shrimp			
	$C_{12-15}EO_3$ (30%, kerosene solution)	20-30	Shell Internal Report, EMGR 0150.71
	$C_{12-15}EO_3$ (30%, isopropanol solution)	200	Shell Internal Report, EMGR 0162.71
	$C_{14-15}EO_3$ (30%, isopropanol solution)	200	Shell Internal Report, EMGR 0162.71
	$C_{14-15}EO_1$ (30%, isopropanol solution)	500	Shell Internal Report, EMGR 0162.71
	$C_{12-15}EO_9$ (30%, isopropanol solution)	>3300	Shell Internal Report, EMGR 0138.71