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**HIGH PRODUCTION VOLUME (HPV)
CHEMICAL CHALLENGE PROGRAM**

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TEST PLAN

For

Glydexx[®] N10

Prepared by:

ExxonMobil Chemical Company

December 17, 2003

EXECUTIVE SUMMARY

Under the U.S. Environmental Protection Agency (EPA) High Production Volume (HPV) Chemical Challenge Program (Program), ExxonMobil Chemical Company committed to voluntarily compile a Screening Information Data Set (SIDS) that can be used in an initial assessment to characterize the hazard of Glydexx® N-10 (neodecanoic acid, 1,2-epoxypropyl ester; CAS No. 26761-45-5). The data for this assessment include selected physicochemical, environmental fate, and human and environmental effect endpoints identified by the U.S. HPV Program.

A search for existing studies/information and their review identified adequate data to characterize all endpoints except developmental and reproductive toxicity. However, data from the repeated dose toxicity study suggest that Glydexx N-10 may not be developmental or reproductive toxicant. At the time of this submission, discovery efforts have not confirmed the existence of a definitive study for this endpoint.

Data suggest that Glydexx N-10 generally presents a low order of hazard for human health although it has been identified as a skin sensitizer. In the environment, it is expected to rapidly hydrolyze to form the diol, upon which the environmental assessment is based. Glydexx N-10 presents a moderate order of hazard for environmental health. In the environment, Glydexx N-10 is calculated to partition primarily to the aqueous phase, where biological and physical processes can mediate its degradation, which is expected to occur at a slow rate.

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TEST PLAN FOR GLYDEXX® N-10

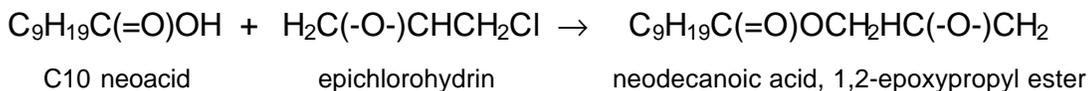
I. INTRODUCTION

Under the U.S. Environmental Protection Agency (EPA) High Production Volume (HPV) Chemical Challenge Program (Program), ExxonMobil Chemical Company committed to voluntarily compile a Screening Information Data Set (SIDS) for Glydexx N-10. This substance is supported by selected screening data needed for an initial assessment of physicochemical properties, environmental fate, and human and environmental effects as defined by the Organization for Economic Cooperation and Development (OECD). Information cited within this test plan comes from existing data.

Procedures to assess the reliability of selected data for inclusion in this test plan were based on guidelines described by Klimisch *et al.* (1997) and identified within the US EPA (1999a) document titled Determining the Adequacy of Existing Data.

II. CHEMICAL PROCESS AND DESCRIPTION

For purposes of the HPV Program, the chemical name for Glydexx N-10 is neodecanoic acid, 1,2-epoxypropyl ester (CAS No. 26761-45-5). The production of Glydexx N-10 includes the reaction of a C10 neoacid with epichlorohydrin to form the corresponding neoacid epoxypropyl ester.



III. TEST PLAN RATIONALE AND DATA SUMMARY

All data identified for Glydexx N-10 (neodecanoic acid, 1,2-epoxypropyl ester) were developed using the parent substance. However, an appropriate assessment for this substance must consider that the parent form has the potential to hydrolyze to form the diol in aqueous conditions. Therefore, for some endpoints, the data were identified for the hydrolysis product. For example, the water solubility is calculated for neodecanoic acid, 1,2-propyldiol ester rather than for neodecanoic acid, 1,2-epoxypropyl ester. Also, the aquatic toxicity data represent the epoxy form, but characterizing toxicity requires caveating that the parent substance hydrolyzes and that results more accurately characterize the hydrolysis product. Note of this reaction should also be included when interpreting human health data.

A. Physicochemical Data

Physicochemical data (Table 1) include measured data from the material safety data sheet (Exxon, 1998). Calculated data are also provided from the EPIWIN® model (EPIWIN, 1999), as discussed in the EPA document titled The Use of Structure-Activity Relationships (SAR) in the High Production Volume Chemicals Challenge Program (US EPA, 1999b). The values for two endpoints, water solubility and log Kow, were calculated for the diol form because the epoxide is subject to hydrolysis in aqueous

solution at a sufficiently rapid rate such that the epoxide form would not be found in solution. Hydrolysis of this substance must also be taken into consideration when discussing fate and toxicity in aquatic systems (see relative sections below).

Table 1. Selected Physico-Chemical Properties for Glydexx N-10.

DATA SOURCE	MELTING POINT (° C)	BOILING POINT (° C)	VAPOR PRESSURE (Pa)	WATER SOLUBILITY (mg/L)	LOG K _{ow}
EPIWIN	-	-	-	156.3 ^a	2.58 ^a
MSDS	<-68	484 - 491	14.67 @ 20° C 74.66 @ 50° C	na	na

a calculated value representing the diol, which is the form that would be present in an aqueous solution; the epoxide would not present because it rapidly hydrolyzes to form the diol

na not applicable

B. Human Health Effects Data

Glydexx N-10 has a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure. It is mildly irritating to the eyes and non-irritating to the skin. Dermal sensitization has been observed in guinea pigs and has been reported in humans following occupational exposure. *In vitro* genotoxicity testing indicated weak mutagenic activity in point mutation assays with metabolic activation using *Salmonella*, but not in *E. coli* or yeast. Mutagenic activity was not observed in an *in vitro* mammalian cell assay. A weak ability to produce chromosomal damage was observed in cultured rat liver cells, but no DNA damage was produced in an *in vivo* rat liver assay. A low order of toxicity was observed in subchronic dietary testing with a No Observed Adverse Effect Level (NOAEL) of 1000 ppm in the diet. At high concentrations of 5000 and 10000 ppm in the diet, kidney effects were observed that were more prominent in males than in females. No effects were noted in reproductive organs of either sex. Further testing to evaluate potential developmental or reproductive effects has not been identified.

Acute Toxicity

Glydexx N-10 has a low order of acute toxicity by the oral, dermal, and inhalation routes of exposure based on testing conducted on the same chemical, but not the ExxonMobil Chemical manufactured substance. In rats, the oral LD₅₀ was greater than 10 ml/kg (approximately 10 g/kg) and the dermal LD₅₀ was greater than 4 ml/kg (approximately 4 g/kg), (Shell Toxicology Laboratory (Tunstall, 1977)). The rat 4-hour inhalation LC₅₀ was greater than 0.24 mg/L (approximately 240 mg/m³), a concentration exceeding the saturated vapor pressure, (Gardiner, 1992). Due to the low vapor pressure resulting in a low level of maximal attainable vapor concentration, inhalation exposure is expected to pose a negligible hazard.

Genotoxicity

Multiple studies have been conducted to evaluate the mutagenic activity of Glydexx N-10 using the same chemical, but not the ExxonMobil Chemical manufactured substance. In point mutation studies without metabolic activation, no mutagenic activity was observed in *Salmonella*, *E. coli*/WP2, or JD1 yeast cells. Testing with S9 activation showed positive results in *Salmonella* strains TA100, TA1535, TA1538, but not in *Salmonella* strains TA97, TA98, TA 1537 or in *E. coli* or yeast (Canter, 1986; Gardiner, 1992; Shell Toxicology Laboratory (Tunstall), 1979a). No significant effects on mutagenic frequency were detected below 100 ug of material per plate suggesting that the mutagenic activity in bacteria was relatively weak. The range of bacterial strains in which activity was demonstrated indicates the mutagenicity was expressed both by base-pair substitution and frameshift mechanisms.

The clastogenicity of Glydexx N-10 was tested in an *in vitro* chromosomal aberration assay conducted in rat liver cells (RL1 Assay) using the same chemical, but not the ExxonMobil Chemical manufactured substance. A small, but consistent number of chromatid aberrations were seen under test conditions without metabolic activation. The assay was not conducted with S9 activation. Thus, the test material was a weak chromosome-damaging agent in the cultured rat liver cells, (Shell Toxicology Laboratory (Tunstall), 1979a; Gardiner, 1992).

Other genotoxicity assays have been conducted on Glydexx N-10 using the same chemical, but not the ExxonMobil Chemical manufactured substance. In an *in vivo* rat liver DNA integrity assay, no measurable DNA single-strand damage was seen following administration of this test material to rats as a single dose of 5 ml/kg. Additionally, in an *in vitro* mammalian cell gene mutation assay using Syrian hamster BHK cells, no increased frequency of transformed cells was observed at concentrations up to 350 ug/ml, (Shell Toxicology Laboratory (Tunstall), 1982; Shell Toxicology Laboratory (Tunstall), 1979b; Gardiner, 1992).

Repeated Dose Toxicity

A low order of toxicity was observed in rats following five-week dietary testing of Glydexx N-10 using the same chemical, but not the ExxonMobil Chemical manufactured substance. Treatment-related effects were limited to the upper two dietary dose levels of 5,000 and 10,000 ppm (approximately 478 and 888 mg/kg/day body weight, respectively). Dose-related effects at these two dietary levels included: decreased food intake and body weights, minor changes in hematology and clinical chemistry, increased liver and kidney weights and nephrotoxicity to the proximal tubules of the kidneys that was more pronounced in males than in females. The Lowest Observed Adverse Effect Level (LOAEL) was 5,000 ppm in the diet (approximately 478 mg/kg/day body weight) and the No Observed Adverse Effect Level (NOAEL) was 1,000 ppm in the diet (approximately 96 mg/kg/day body weight), (Sittingbourne Research Centre, 1981; Gardiner, 1992).

Developmental and Reproductive Toxicity

Data were not identified for the evaluation of developmental and reproductive toxicity of Glydexx N-10. However, in the five week repeated dose toxicity study of the same chemical, but not the ExxonMobil Chemical manufactured substance, reproductive organs were examined and no toxic effects were observed. Testicular organ weights in treated animals showed no significant differences when compared to control animals and microscopic histopathology examinations were unremarkable. No significant differences between treated and control animals were noted for the other reproductive organs that were examined: prostate, ovaries and uterus.

Non-SIDS Endpoints

Eye Irritation

Glydexx N-10 was non-irritating to the eyes based on rabbit data for the same chemical, but not the ExxonMobil Chemical manufactured substance, (Shell Toxicology Laboratory (Tunstall), 1977; Gardiner, 1992).

Skin Irritation

Glydexx N-10 was a mild irritant to the skin based on rabbit data for the same chemical, but not the ExxonMobil Chemical manufactured substance. A single 24-hour application of the test material to intact, occluded rabbit skin was mildly irritating. The test material is not expected to be irritating when applied to intact skin in the EEC/OECD 4-hour semi-occluded test, (Shell Toxicology Laboratory (Tunstall), 1977; Gardiner, 1992).

Skin Sensitization

Glydexx N-10 has been shown to be skin sensitizer based on guinea pig data for this substance and the same chemical, but not the ExxonMobil Chemical manufactured substance. Two skin sensitization studies have been conducted in guinea pigs using the Magnusson and Kligman procedure. Both studies showed severe sensitization effects, (Shell Toxicology Laboratory (Tunstall), 1977; Gardiner, 1992; Exxon Biomedical Sciences, Inc., 1990). Skin sensitization has been reported in humans from repeated contact in the occupational setting, (Dalquist, 1979).

Table 2. Mammalian Toxicity Studies for Glydexx N-10

ENDPOINT	RESULT
ACUTE	
Oral ^{1,2} - Rat	LD ₅₀ >10 ml/kg (approx. 10 g/kg)
Dermal ^{1,2} - Rat	LD ₅₀ >4 ml/kg (approx. 4 g/kg)
Inhalation ¹ - Rat	LC ₅₀ >0.24 mg/L (240 mg/m ³) (saturated vapor pressure)
GENOTOXICITY	
Point Mutation ^{1,2}	<ul style="list-style-type: none"> ▪ Weakly positive with S9, negative w/o (TA100, TA1535, TA1538); ▪ Negative with and w/o S9 (TA97, TA98, TA 1537); ▪ Negative with and w/o S9 (<i>E. coli</i> WP2); ▪ Negative with and w/o S9 (JD1 yeast)
Chromosome Aberration ^{1,2}	Weakly positive w/o S9 (RL1)
REPEATED DOSE	
Oral ^{1,2} - Rat	NOAEL = 1000 ppm in the diet (approx. 96 mg/kg/day bw) LOAEL = 5000 ppm in the diet (approx. 478 mg/kg/day bw)
REPRODUCTIVE / DEVELOPMENTAL	
Developmental Toxicity	NI
Reproductive Toxicity	NI
IRRITATION / SENSITIZATION	
Ocular Irritation ^{1,2} -Rabbit	Non-irritant
Dermal Irritation ^{1,2} -Rabbit (occluded)	Mild irritant
Dermal Sensitization ² -Guinea Pig	Positive (sensitizer)

¹ Based on data for the same chemical, but not the ExxonMobil Chemical manufactured substance

² Robust summary provided

NI - data not identified

C. Aquatic Toxicity Data

Data are available to characterize the fish and invertebrate acute toxicity and alga toxicity of Glydexx N-10. Although the data are associated with the parent substance, neodecanoic acid, 1,2-epoxypropyl ester, the results are interpreted to characterize the

hydrolyzed form of this substance because the parent epoxide rapidly forms the diol in aqueous systems.

Glydexx N-10 demonstrated a 96-hour rainbow trout (*Oncorhynchus mykiss*) LC₅₀ toxicity value of 9.61 mg/L (Exxon Biomedical Sciences, Inc., 1998). Data developed for the same chemical, but not the ExxonMobil Chemical manufactured substance, demonstrated a 48-hour invertebrate (*Daphnia magna*) EC₅₀ toxicity value of 4.8 mg/L and an alga (*Selenastrum capricornutum*) EC₅₀ toxicity value of 3.5 mg/L, based on biomass.

D. Environmental Fate Data

Biodegradation

Biodegradation of an organic substance by bacteria can provide energy and carbon for microbial growth. This process results in a structural change of an organic substance and can lead to the complete degradation of that substance, producing carbon dioxide and water.

The test guideline used to assess the biodegradability of Glydexx N-10 was OECD 301F (Manometric Respirometry Test). The test system applied to this guideline used a continuously stirred, closed system and assessed biodegradability based on oxygen consumption. These procedures are recommended when assessing the biodegradability of poorly water soluble, volatile substances like Glydexx N-10. The source of the microbial inoculum used in this study was a domestic wastewater treatment facility. The inoculum was not acclimated.

Glydexx N-10 biodegraded to 11.6% after 28 days (Exxon Biomedical Sciences, Inc., 1996). Although the data are associated with the parent substance, neodecanoic acid, 1,2-epoxypropyl ester, the results are interpreted to characterize the hydrolyzed form of this substance because the parent epoxide rapidly forms the diol in aqueous systems, which is the case for biodegradation tests.

Additional data were developed for the same chemical, but not the ExxonMobil Chemical manufactured substance, using the OECD 302A (Modified SCAS Test) guideline (Stephenson, 1983). Dissolved organic carbon (DOC) was monitored over a 36-day period. Results between days 22 and 36 of the study (after acclimation may have occurred) showed 68% DOC removal, which suggests that Glydexx N-10 can be removed in a wastewater treatment facility.

Photodegradation – Photolysis

Direct photochemical degradation occurs through the absorbance of solar radiation by a chemical substance in aqueous solution. If the absorbed energy is high enough, then the resultant excited state of the chemical may undergo a transformation. A prerequisite for direct photodegradation is the ability of one or more bonds within a chemical to absorb ultraviolet (UV)/visible light in the 290 to 750 nm range. Light wavelengths longer than 750 nm do not contain sufficient energy to break chemical bonds, and wavelengths

below 290 nm are shielded from the earth by the stratospheric ozone layer (Harris, 1982a).

An approach to assessing the potential for a substance to undergo photochemical degradation is to assume that degradation will occur in proportion to the amount of light wavelengths >290 nm absorbed by constituent molecules (Zepp and Cline, 1977).

Assessing the potential for Glydexx N-10 to photolyze requires consideration of the hydrolyzed form of this substance because this substance has a relatively short hydrolysis half-life and rapidly forms the diol in water. Saturated hydrocarbons and R-OH groups do not absorb light above 290 nm (Harris J, 1982a). Therefore, these moieties are stable in regard to direct photolytic processes. Esters are also stable as this group absorbs UV light in the far UV region, below 220 nm (Mill T, 2000). Consequently, Glydexx N-10 is not subject to photolytic processes in the aqueous environment.

Photodegradation – Atmospheric Oxidation

Photodegradation can be measured (US EPA, 1999a) or estimated using an atmospheric oxidation potential (AOP) model accepted by the EPA (US EPA, 1999b). Atmospheric oxidation as a result of hydroxyl radical attack is not direct photochemical degradation, but rather indirect degradation.

Glydexx N-10 has the potential to volatilize to air, based on a vapor pressure of 14.67 @ 20° C, where it is subject to atmospheric oxidation. In air, Glydexx N-10 can react with photosensitized oxygen in the form of hydroxyl radicals (OH⁻). The computer program AOPWIN (atmospheric oxidation program for Microsoft Windows) (EPIWIN, 1999) calculates a chemical half-life for a 12-hour day (the 12-hour day half-life value normalizes degradation to a standard day light period during which hydroxyl radicals needed for degradation are generated), based on an OH⁻ reaction rate constant and a defined OH⁻ concentration.

Glydexx N-10 has a calculated half-life in air of 13.3 hours or 1.1 days, based on a rate constant of 23.71E-12 cm³/molecule·sec and an OH⁻ concentration of 1.5E6 OH⁻/cm³.

Stability in Water (Hydrolysis)

Hydrolysis of an organic chemical is the transformation process in which a water molecule or hydroxide ion reacts to form a new carbon-oxygen bond. Chemicals that have a potential to hydrolyze include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters (Neely, 1985).

Glydexx N-10 is an epoxide, neodecanoic acid, 1,2-epoxypropyl ester, which is subject to hydrolysis to form neodecanoic acid, 1,2-propyldiol ester. Hydrolysis is estimated to occur at a relatively rapid rate for Glydexx N-10, based on data for 14 epoxides (Mabey and Mill, 1978), as summarized by Harris (1982b), that ranged in half-life from approximately 1 minute to 8 days at pH 7.

Chemical Distribution In The Environment (Fugacity Modeling)

Fugacity-based multimedia modeling provides basic information on the relative distribution of a chemical between selected environmental compartments (i.e., air, soil, water, sediment, suspended sediment, and biota). A widely used fugacity model is the EQC (Equilibrium Criterion) Level I model (Mackay, 1996; Mackay, 1998).

The EPA guidance document (US EPA, 1999a) states that EPA accepts Level I fugacity data as an estimate of chemical distribution values. The input data required to run a Level I model include basic physicochemical parameters; distribution is calculated as percent of chemical partitioned to 6 compartments (air, soil, water, suspended sediment, sediment, biota) within a unit world. Level I data are basic partitioning data that allow for comparisons between chemicals and indicate the compartment(s) to which a chemical may partition, based on selected physical parameters.

Results of the Mackay Level I environmental distribution model (Table 3) suggest that Glydexx N-10 will partition primarily (>99%) to air. These results can be explained by its vapor pressure, 14.67 hPa at 20°C (Exxon, 1998). However, in the environment Glydexx N-10 can undergo hydrolysis to the diol and the distribution of this form is significantly different from the parent. Because hydrolysis for this substance occurs rapidly, it is also appropriate to consider the potential environmental distribution of this form. The data in Table 3 show that the hydrolyzed form would be expected to partition largely to water, 74%, and soil, 25%.

Table 3. Environmental distribution as calculated by the Mackay (1998) Level I fugacity model

Environmental Compartment	Glydexx N-10 Percent Distribution*	Hydrolyzed Form (Diol) Percent Distribution**
Air	99.69	0.00
Water	0.23	74.38
Soil	0.08	25.04
Sediment	0.00	0.56
Suspended Sediment	0.00	0.02
Biota	0.00	0.00

*Distribution is based on the following model input parameters for neodecanoic acid,

1,2-epoxypropyl ester:

Molecular Weight	228.33	
Temperature	20°C	
Log K _{ow}	2.58	
Water Solubility	156.3g/m ³	
Vapor Pressure	1467 Pa	
Melting Point	-68°C	(the EPIWIN value was not used as it was felt the estimated value is incorrect)

**Distribution is based on the following model input parameters for neodecanoic acid,

1,2-propyldiol ester:

Molecular Weight	246.35	
Temperature	20°C	
Log K _{ow}	2.58	
Water Solubility	156.3g/m ³	
Vapor Pressure	9.3E-5 Pa	
Melting Point	-68°C	(the EPIWIN value was not used as it was felt the estimated value is incorrect)

IV. TEST PLAN SUMMARY

A search for existing studies/information and their review identified adequate data to characterize all endpoints for Glydexx N-10 except developmental and reproductive toxicity. At the time of this submission, discovery efforts have not identified the existence of a definitive study for this endpoint. However, testing is not planned until the lack of these data has been confirmed.

A dossier containing the robust summaries of the data presented in this test plan is attached.

REFERENCES

- Battersby N and Turner S (1989). Cardura E10: An assessment of inherent biodegradability. Report No. SBGR.89.002. Sittingbourne Research Centre, Biotechnology and Toxicology Directorate, Kent, England. (Submitted to Federal Register, 47:38780-38799)
- Canter D, Zeiger E, Haworth S, Lawlor T, Mortelmans K and Speck W (1986). Comparative mutagenicity of aliphatic epoxides in Salmonella. *Mutation Research* **172**, 105-138.
- Dalquist I and Fregert S (1979). Contact allergy to Cardura E, an epoxy reactive diluent of the ester type. *Contact Dermatitis* **5**, 121-122.
- EPIWIN (1999). Estimation program interface for windows, version 3.04. Syracuse Research Corporation, Syracuse, NY, USA.
- Exxon Biomedical Sciences, Inc. (1990). Dermal sensitization test in the guinea pig. Study #259221. Unpublished report.
- Exxon Biomedical Sciences, Inc. (1996). Ready Biodegradability: "OECD Manometric Respirometry Test". Study #136894A. Unpublished report.
- Exxon Biomedical Sciences, Inc. (1998). Fish, acute toxicity test. Study #137258. Unpublished report.
- Exxon Chemical Company (1998). Glydexx N-10 Material Safety Data Sheet, March 21, 1998.
- Gardiner, TH, Waechter, JM, Wiedow, MA, and Solomon, WT, 1992. Glycidyoxy compounds used in epoxy resin systems: a toxicology review. *Regulatory Toxicology and Pharmacology* **15**, S1-S77.
- Harris J (1982a). Rate of aqueous photolysis. In: *Handbook of Chemical Property Estimation Methods*, Lyman W, Reehl W and Rosenblatt D (eds.), Chapter 8. McGraw-Hill Book Company, New York, USA.
- Harris J (1982b). Rate of hydrolysis. In: *Handbook of Chemical Property Estimation Methods*, Lyman W, Reehl W and Rosenblatt D (eds.), Chapter 7. McGraw-Hill Book Company, New York, USA.
- Klimisch H, Andreae M and Tillmann U (1997). A systematic approach for evaluating the quality of experimental toxicological and ecotoxicological data. *Regulatory Toxicol. Pharmacol.* **25**, 1-5.
- Mabey W and Mill T (1978). Critical review of hydrolysis of organic compounds in water under environmental conditions. *J. Phys. Chem. Ref. Data* **7**, 383-415.
- Mackay D, Di Guardo A, Paterson S and Cowan C (1996). Evaluating the environmental fate of a variety of types of chemicals using the EQC model. *Environ. Toxicol. Chem.* **15**, 1627-1637.
- Mackay D (1998). Level I Fugacity-Based Environmental Equilibrium Partitioning Model, Version 2.1 (16-bit). Environmental Modelling Centre, Trent University, Ontario, Canada.

Mill T (2000). Photoreactions in surface waters. In: Handbook of Property Estimation Methods for Chemicals, Boethling R and Mackay D (eds.) , Chapter 15. Lewis Publishers, New York, USA.

Neely W (1985). Hydrolysis. In: Environmental Exposure from Chemicals. Vol. 1. Neely W and Blau G (eds.), pp. 157-173. CRC Press, Boca Raton, FL, USA.

Shell Toxicology Laboratory (Tunstall), (1977). Toxicology of resins: Acute toxicity, skin and eye irritancy and skin sensitizing potential of CARDURA E10. Report number TLGR.0088.77.

Shell Toxicology Laboratory (Tunstall), (1979a). Toxicity studies with resins: In vitro mutation studies with CARDURA E10. Report No. TLGR.79.072

Shell Toxicology Laboratory (Tunstall), (1979b). Toxicity studies with resins: In vitro transformation studies with CARDURA E10. Report No. TLGR.80.137.

Shell Toxicology Laboratory (Tunstall), (1982). Studies on the effect of CARDURA E10 on the integrity of rat liver DNA in vivo. Report No. TLGR.80.102

Sittingbourne Research Centre, 1981. A five week feeding study of CARDURA E10 in rats. Report No. SBGR.81.248.

Stephenson R (1983). Cardura E10: Acute toxicity to *Salmo gairdneri*, *Daphnia magna* and *Selenastrum capricornutum*. Report No. SBGR.83.050. Sittingbourne Research Centre, Kent, England. (Submitted to Federal Register **47**, 38780-38799)

US EPA (1999a). Determining the Adequacy of Existing Data. OPPT, EPA, Washington, DC, USA.

US EPA (1999b). The Use of Structure-Activity Relationships (SAR) in the High Production Volume Chemicals Challenge Program. OPPT, EPA, Washington, DC, USA.

Zepp R and Cline D (1977). Rates of direct photolysis in the aqueous environment. *Environ. Sci. Technol.* **11**, 359.366.