UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

August 2, 2006

ACTION MEMORANDUM

SUBJECT: Inert Reassessments: Five Exemptions from the Requirement of a Tolerance for Petroleum Hydrocarbons

FROM: Pauline Wagner, Chief Pauline Wagner 8/2/06
Inert Ingredient Assessment Branch
Registration Division

TO: Lois A. Rossi, Director
Registration Division

I. FQPA REASSESSMENT ACTION

Action: Reassessment of five inert ingredient exemptions from the requirement of a tolerance. Current exemptions are to be maintained.

Chemicals: See Table 1

Table 1. Tolerance Exemptions Expression

40 CFR	Inert Ingredients	Limits	Uses	CAS Reg. No. and Names
180.910[a]	Petroleum hydrocarbons, light odorless conforming to 21 CFR 172.884	None	Solvent, diluent	See Appendix A
	Petroleum hydrocarbons, synthetic isoparaffinic, conforming to 21 CFR 172.882		Solvent, diluent	See Appendix A
	Petroleum naphtha, conforming to 21 CFR 172.250(d)		Component of coating agent	See Appendix A

40 CFR	Inert Ingredients	Limits	Uses	CAS Reg. No. and Names
180.930[b]	Petroleum hydrocarbons, light, odorless, conforming to 21 CFR 172.884 or 172.3650	None	Solvent, diluent	See Appendix A
	Petroleum hydrocarbons, synthetic isoparaffinic, conforming to 21 CFR 172.882 or 178.3530		Solvent, diluent	See Appendix A

[a] Residues listed in 40 CFR 180.910 are exempted from the requirement of a tolerance when used in accordance with good agricultural practice as inert (or occasionally active) ingredients in pesticide formulations applied to growing crops or to raw agricultural commodities after harvest.
[b] Residues listed in 40 CFR 180.930 are exempted from the requirement of a tolerance when used in accordance with good agricultural practice as inert (or occasionally active) ingredients in pesticide formulations applied to animals.

Background: A risk assessment for petroleum hydrocarbons (Risk Assessment for Tolerance Exemption Reassessment for C_8-C_{20} Aliphatic Hydrocarbon Fluids, Memorandum, R. Daiss to P Wagner, August 1, 2006) (see Appendix B) and the July 12, 2006, Reregistration Eligibility Decision (RED) Document for Aliphatic Solvents (Mineral Oil and Aliphatic Petroleum Hydrocarbons) provide risk assessments for the petroleum hydrocarbon inert ingredients that are described in Table 1 above. The following sections provide the FQPA safety finding information.

Special Considerations for Infants and Children: Petroleum hydrocarbons, as given in Table 1 above, are of low toxicological concern for developmental and reproductive effects based on the available toxicity data. Therefore, there is no concern, at this time, for increased sensitivity to infants and children to petroleum hydrocarbons (as given in Table 1) when used as an inert ingredient in pesticide formulations and the additional tenfold safety factor for the protection of infants and children has been reduced to 1X for these risk assessments.

Human Health Risk Characterization: The risk assessments conclude that: "[Petroleum hydrocarbons as given in Table 1] exhibit low acute toxicity by oral, inhalation and dermal routes (toxicity Category III or IV by all exposure routes). These compounds are minimally irritating to eyes and skin and negative for dermal sensitization effects" and that screening level assessments of dietary (food and drinking water) and residential (inhalation and dermal) exposures indicate "no risks of concern" for these chemicals.

Taking into consideration the available information Petroleum hydrocarbons as given in Table 1, there is a reasonable certainty that no harm to any population subgroup will result from aggregate exposure when considering dietary exposure and all other non-occupational sources for which there is reliable information. Therefore, it is recommended that the five exemptions from the requirement of a tolerance established for residues of Petroleum hydrocarbons as given in Table 1 when used under 40 CFR 180.910 and 40 CFR 180.930 can be considered reassessed as safe under section 408(q) of the FFDCA.

List Classification Determination: Because EPA has determined that there is a reasonable certainty that no harm to any population subgroup will result from aggregate exposure to these

chemicals when used as inert ingredients in pesticide formulations, the List Classification for the petroleum hydrocarbons (as defined in Table 1) will be List 4B.

II. MANAGEMENT CONCURRENCE

I concur with the reassessment of the five exemptions from the requirement of a tolerance for the petroleum hydrocarbons (as defined in Table 1), as well as the List Classification determination described above. I consider the three exemptions from the requirement of a tolerance established in 40 CFR 180.910 and the two exemptions from the requirement of a tolerance established in 40 CFR 180.930 to be reassessed for purposes of FFDCA's section 408(q) as of the date of my signature, below. A Federal Register Notice regarding this tolerance exemption reassessment decision will be published in the near future.

Lois A. Rossi

Lois A. Rossi, Director
Registration Division

August 2, 2006
Date:

cc: Debbie Edwards, SRRD
Joe Nevola, SRRD

APPENDIX A

Chemical Names and CAS Reg. Nos. for Petroleum Hydrocarbons Chloride Compounds

Chemical Name (CAS 9th Collective Index Name)	CAS Reg. No.
40 CFR 180.910	
Petroleum hydrocarbons, light odorless conforming to 21 CFR 172.884	
Naphtha (petroleum), light alkylate	64741-66-8
Distillates (petroleum), solvent-refined heavy paraffinic	64741-88-4
Distillates (petroleum), solvent-refined light paraffinic	64741-89-5
Distillates (petroleum), hydrotreated middle	64742-46-7
Distillates (petroleum), hydrotreated light	64742-47-8
Naphtha (petroleum), hydrotreated heavy	64742-48-9
Distillates (petroleum), hydrotreated light paraffinic	64742-55-8
Distillates (petroleum), solvent-dewaxed light paraffinic	64742-56-9
Paraffins (petroleum), normal C5-20	64771-72-8
Petroleum hydrocarbons, synthetic isoparaffinic, conforming to 21 CFR 172.882	
Distillates (petroleum), hydrotreated middle	64742-46-7
Distillates (petroleum), hydrotreated light	64742-47-8
Naphtha (petroleum), hydrotreated heavy	64742-48-9
Petroleum naphtha, conforming to 21 CFR 172.250(d)	
Naphtha (petroleum), light alkylate	64741-66-8
40 CFR 180.930	
Petroleum hydrocarbons, light, odorless, conforming to 21 CFR 172.884 or 172.3650	
Naphtha (petroleum), light alkylate	64741-66-8
Distillates (petroleum), solvent-refined heavy paraffinic	64741-88-4
Distillates (petroleum), solvent-refined light paraffinic	64741-89-5
Distillates (petroleum), hydrotreated middle	64742-46-7
Distillates (petroleum), hydrotreated light	64742-47-8
Naphtha (petroleum), hydrotreated heavy	64742-48-9
Distillates (petroleum), hydrotreated light paraffinic	64742-55-8
Distillates (petroleum), solvent-dewaxed light paraffinic	64742-56-9
Paraffins (petroleum), normal C5-20	64771-72-8
Petroleum hydrocarbons, synthetic isoparaffinic, conforming to 21 CFR 172.882 or 178.3530	
Distillates (petroleum), hydrotreated middle	64742-46-7
Distillates (petroleum), hydrotreated light	64742-47-8

Chemical Name (CAS 9th Collective Index Name)	CAS Reg. No.
Naphtha (petroleum), hydrotreated heavy	64742-48-9

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460
Office of Prevention, Pesticides and Toxic Substances

August 1, 2006

MEMORANDUM

SUBJECT: Risk Assessment for Tolerance Exemption Reassessment for C_8-C_{20} Aliphatic Hydrocarbon Fluids

FROM: Becky Daiss, Environmental Health Scientist
Reregistration Branch 4
Health Effects Division (7509P)

THRU: Susan V. Hummel, Branch Senior Scientist
Reregistration Branch 4
Health Effects Division (7509P)

TO: Pauline Wagner, Branch Chief
Inert Ingredient Assessment Branch
Registration Division (7505P)

This provides a health assessment for the C_8-C_{20} aliphatic hydrocarbon fluids based on data submitted for the corresponding ExxonMobil trade name products. The following trade name products were included in this assessment; Exxsol™ D40 Fluid, Exxsol™ D60 Fluid, Exxsol™ D80 Fluid, Exxsol™ D95 Fluid Exxsol™ D100 Fluid, Exxsol™ D100s Fluid, Exxsol™ D110 Fluid, Exxsol™ D110S Fluid, Exxsol™ D120 Fluid, Exxsol™ D130 Fluid, Exxsol™ D140 Fluid, Isopar™ C Fluid, Isopar™ E Fluid, Isopar™ G Fluid, Isopar™ H Fluid, Isopar™ K Fluid, Isopar™ L Fluid, Isopar™ M Fluid, Isopar™ N Fluid, and Isopar™ V Fluid.

The attached assessment summarizes available information on the use, physical/chemical properties, toxicological effects, exposure profile, and environmental fate of these pesticide inert ingredients. The purpose of this document is to reassess existing tolerance exemptions for residues of these inert ingredients as required under the Food Quality Protection Act (FQPA) section 408. This provides a screening level risk assessment in which high-end assumptions were used for most key parameters. HED is confident that this analysis does not underestimate risks associated with exposure to C_8-C_{20} aliphatic hydrocarbon fluids.

1.0 EXECUTIVE SUMMARY

This assessment evaluates potential risks from use of C_8-C_{20} aliphatic hydrocarbon fluids as inert ingredients in pesticides used for agricultural and consumer product applications. ExxonMobil has submitted dietary and residential exposure/risk assessments for the trade name products included in this group of compounds in support of a tolerance exemption reassessment. HED has evaluated ExxonMobil's submissions and has incorporated information from those assessments into its risk assessment. Toxicological data submitted by ExxonMobil provide the primary basis for HED's hazard identification evaluation.

Exxsol™ D Fluids and Isopar™ Fluids are ExxonMobil's trade names for the company's brand of related aliphatic hydrocarbon fluids between C_8 and C_{20} carbon length that are used as pesticide inert ingredients. The aliphatic hydrocarbon fluids consist of compounds that contain normal paraffins, branched (iso) paraffins, and cycloparaffins within a carbon number range of C_8-C_{20}. They are dearomatized and therefore contain less than 1.5% aromatics. These products are manufactured as part of the crude oil refining process, and they are considered to be hydrotreated petroleum distillates. Their manufacture involves three basic processes. First, petroleum (crude oil) distillation provides hydrocarbon feedstocks with boiling ranges that are close to those of the final aliphatic hydrocarbon fluid products. Next, hydrofining removes sulfur and nitrogen impurities and hydrotreating converts most aromatic molecules to cycloparaffins (also called naphthenes). Finally, additional distillation (fractionation) is usually employed to complete the separation of the dearomatized aliphatic products into their final boiling ranges.

Sufficient toxicity data and information on Exxsol™ D Fluids and Isopar™ Fluids are available from the ExxonMobil. OPP agrees with ExxonMobil's argument that the Exxsol™ D and Isopar™ Fluids are compositionally similar such that data from some aliphatic hydrocarbon fluids can be used to assess the potential toxicity of other C_8-C_{20} fluids. Based on common functional substructure, common metabolic pathways/kinetics of metabolism, and comparable molecular properties, the C_8-C_{20} aliphatic hydrocarbon fluids can be considered valid analogues of each other for purposes of predicting toxicity (Personal Communication Rebecca Jones, OPPT/OPPTS, 6/30/06).

Based on data submitted by ExxonMobil on the trade name products Exxsol™ D and Isopar™ Fluids, C_8-C_{20} aliphatic hydrocarbon fluids exhibit low acute toxicity by oral, inhalation and dermal routes (Toxicity Category III or IV by all exposure routes). They are minimally irritating to eyes and are negative for skin sensitization. Subchronic oral and inhalation toxicity studies indicate these aliphatic hydrocarbon fluids to be relatively non-toxic. Kidney effects were consistently observed in male rats. However, these effects are considered to be indicative of alpha-2u-globulin nephropathy; they are specific to male rats and are not considered to be of biological relevance to humans. Depressed body weight and clinical signs were reported at mid- and high doses in subchronic oral toxicity studies in rats. Developmental oral and inhalation studies in rats show no evidence of developmental effects or any adverse effects in maternal animals at the highest doses tested. Neurobehavioral effects were observed at the high dose in short-term (3 day) inhalation neurotoxicity studies conducted in rats. There

are no substance-specific absorption, metabolism, distribution and excretion studies done specifically on aliphatic hydrocarbon fluids. However, ExxonMobil submitted information which indicates these compounds are typically well absorbed, widely distributed between tissues, extensively metabolized and rapidly excreted. Based on available data, C_8-C_{20} aliphatic hydrocarbons are not likely to be carcinogenic.

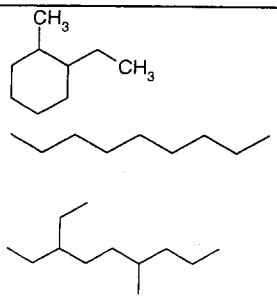
The aliphatic hydrocarbon fluids are of low toxicological concern for developmental and reproductive effects based on the available toxicity data. Therefore, it is recommended that the Food Quality Protection Act (FQPA) tenfold safety factor be reduced to 1X for this risk assessment.

The C_8-C_{20} aliphatic hydrocarbon fluids have been assessed together in this document because they are determined to be toxicologically equivalent and share similar use patterns and routes of exposure. HED conducted screening level dietary and residential risk assessments for the aliphatic hydrocarbon fluids. A screening level quantitative assessment of dietary exposure indicates no risks of concern. Based on the high volatility of the aliphatic hydrocarbons in this group and aeration sequences used in many drinking water treatment utilities, it is unlikely that most of these compounds will be found in treated water. Screening level assessments of incidental oral, inhalation, and dermal exposures from residential application and post-application exposures indicate no risks of concern. Based on the available environmental fate and effects data, application of pesticides formulations containing these inerts to terrestrial environments at label maximum application rates will not result in exceedance of the Agency's level of concern for endangered species.

2.0 USE INFORMATION

Exxsol™ D Fluids and Isopar™ Fluids have many uses as solvents. They are commonly used in lubricants, processing aids, household and consumer products, reaction diluents, cleaning agents, extraction fluids, printing inks, food-related applications, combustion fluids, and as pesticide inert ingredients in agricultural formulations. Compounds included in this risk assessment are provided in Table 1.

Table 1. Aliphatic Hydrocarbons Included in Risk Assessment

Descriptive Name	Trade Name	CAS No	Predominant Carbon No	Representative Structures
Distillates, petroleum, hydrotreated light	Exxsol™ D40, D60, D80, D95, D100, D110, Isopar M, N, P	64742-47-8	C_9-C_{16}	
Hydrotreated heavy naphtha	Isopar™ G, H, J, K, L	64742-48-9	C_6-C_{13}	
Distillates, petroleum, hydrotreated middle	Exxsol™ D120, D130, Isopar V	64742-46-7	C_{11}-C_{20}	
Naphtha, petroleum, light alkylate (alkanes)	Isopar™ C, E	64741-66-8	C_7-C_{10}	

3.0 PHYSICAL AND CHEMICAL PROPERTIES

Table 2. Key Chemical/Physical Properties and Environmental Fate of Representative Exxsol™ D Fluids*						
Parameter	**Exxsol™ D40**	**Exxsol™ D60**	**Exxsol™ D80**	**Exxsol™ D95**	**Exxsol™ D110**	**Exxsol™ D130**
CAS No.	64742-47-8	64742-47-8	64742-47-8	64742-47-8	64742-47-8	64742-46-7
Predominant Carbon No.	$C_9 - C_{12}$	$C_{11} - C_{12}$	$C_{12} - C_{14}$	$C_{13} - C_{14}$	$C_{14} - C_{16}$	$C_{16} - C_{18}$
Chemical Name:	Distillate Petroleum, Hydrotreated Light, Middle, Heavy; Naphtha (Petroleum) Hydrotreated Heavy, Light					
Average Molecular Weight	143	158	171	181	200	229
Vapor Pressure (mm Hg)[1]	2.03E+00	4.50E-01	1.70E-01	7.00E-02	2.00E-02	3.00E-03
Distillation Range (°C)	161 – 202	188 – 210	208 – 234	249 – 268	249 – 268	282 – 311
Relative Evaporation Rate[2]	15	6	1	< 1	< 1	< 1
Water Solubility (mg/L)	< 1.0 – 2.0	< 1.0 – 2.0	< 1	< 1	4.8 - 55.0	< 1
Log K_{ow}	> 3.0	> 3.0	> 3.0	> 3.0	> 3.0	> 3.0
Atmospheric Half-life (hours)	6.4 – 9.5	6.4 – 14	5.6 – 10.7	5.6 – 10.7	10.2 – 15.3	8.5 – 14.1
Biological Degradation (% in 28 days)	70	67	64	64	63	62
Fugacity Modeling (Mackay Level 1)						
Air (%)	99.7 – 100	99.6 – 99.9	96.4 – 99.9	96.4 – 99.9	71.3 – 84.6	1.9 – 58
Soil (%)	0 – 0.1	0.01 – 0.2	0.05 – 1.8	0.05 – 1.8	15.0 – 28.0	41 – 95.9
Sediment (%)	0 – 0.1	0.01 – 0.2	0.05 – 1.7	0.05 – 1.7	0.3 – 0.6	0.9 – 2.1

*The data in this table for the Exxsol™ D Fluids sold in the U.S. subsume the range of data for Exxsol™ D Fluids sold in Europe (Exxsol™ D100, D100S, D110S, D120, D140 Fluids)

[1] hPa = 0.75 mm Hg; http//www.paroscientific.com/convtable.htm

[2] As compared to n-butyl acetate = 100; vapor pressure = 11.5 mm Hg at 25 C

Table 3. Key Chemical/Physical Properties and Environmental Fate of Representative Isopar™ Fluids		
CAS No.	64742-47-8, 64741-66-8, 64742-48-9	64742-46-7
Predominant Carbon Range	C_6-C_{13}	C_{11}-C_{20}
Chemical Name:	Distillate Petroleum, Hydrotreated Light, Middle, Heavy; Naphtha (Petroleum) Hydrotreated Heavy, Light	
Distillation Range, °C (°F)	150-515 (300-420)	218-288 (425-550)
Specific Gravity @ 16/16 C (60.60 F)	0.70 – 0.80	0.81 – 0.85
Aromatics (%)	0.0-2.0	≤2
Benzene (ppmv)	0-10	<1

Table 4. Typical Aliphatic Hydrocarbons*		
CAS No.	64742-47-8	64742-46-7
Typical n-Paraffins	n-Nonane n-Tridecane	n-Tetradecane n-Hexadecane
Typical Isoparaffins	2-Methyloctane 2,3,5-Trimethylhexane 2,4-Dimethylnonane 2,5,8-Trimethyldecane	2-Methyltridecane 3-Ethyldodecane 2,5,6,9-Tetramethyldecane 2,5,8-Trimethyltridecane
Typical Cycloparaffins (Naphthenics)	1,2,4-Trimethylcyclohexane Decalin 2,3,6-Trimethyldecalin	2,3,6,7-Tetramethyldecalin 1,5-Diethyldecalin 1-Nonylcyclohexane 1,6-Di-n-propyledecalin

* Typical constituents representing category members were selected on the basis of carbon number, chemistry/structure, measured distillation ranges, and hydrocarbon process (distillation) knowledge.

4.0 HAZARD ASSESSMENT

4.1. Hazard Profile

This hazard assessment was developed using toxicity data for the trade name products Exxsol™ D Fluids and Isopar™ Fluids provided by ExxonMobil. The toxicity data base is adequate for the selection of doses and endpoints for use in risk assessment of C_8-C_{20} aliphatic hydrocarbon fluids. Toxicological data for C_8-C_{20} aliphatic hydrocarbon fluids are summarized in Tables 5 and 6.

Table 5. Acute Toxicity Aliphatic Hydrocarbons

Compound	Study	Acute Toxicity
Distillates, petroleum, hydrotreated light	Oral Rat	LD50 > 15 g/kg (Exxsol™ D40, D60, D80) LD50 > 10 g/kg (Isopar™ M)
	Inhalation Rat	LC50 > 6100 mg/m3 (Exxsol™ D40) LC50 > 5266 mg/m3; 7 mg/L (Exxsol™ D80, D110) LC50 > 5991 mg/m3 (Isopar M)
	Dermal Rabbit	LD50 > 3160 mg/kg (Exxsol™ D40)
	Eye Irritation	Slight irritant (Exxsol™ D100, D140)
	Dermal Irritation*	Mild irritant (Exxsol™ D140)
Hydrotreated heavy naphtha	Oral Rat	LD50 > 10000 µL/kg (Isopar™ H, L)
	Inhalation Rat	LC50 > 5.6 mg/L (Isopar™ H) LC50 > 4.6 mg/L (Isopar™ L) LC50 > 12.4 mg/L (Isopar™ G)
	Dermal Rabbit	LD50 > 3160 mg/kg (Isopar™ H, G, L)
Distillates, petroleum, hydrotreated middle	Oral Rat	LD50 > 5000 mg/kg (Isopar™ V)
	Inhalation Rat	LC50 > 1.97 mg/L (Isopar™ V)
	Dermal Rabbit	LD50 > 2000 mg/kg (Isopar™ V)
Naphtha, petroleum, light alkylate (alkanes)	Oral Rat	LD50 > 10000 µL/kg (Isopar™ C)
	Inhalation Rat	LC50 > 21 mg/L (Isopar™ C, E)
	Dermal Rabbit	LD50 > 3160 µL/kg (Isopar™ C, E)

* Similar to other hydrocarbon solvents, when evaporation is impeded, these compounds may cause defatting of the skin and associated irritation in some sensitive individuals.

Table 6. Toxicity Profile for Aliphatic Hydrocarbons

Study Type	Publication	Doses	Results
870.3700a developmental inhalation rat (Exxsol™ D40 Fluid, Isopar™ G Fluid)	EMBSI 1978 MRID 46719024 MRID 46543526 Acceptable/Guideline	300, 900 ppm	Maternal LOAEL = NA Maternal NOAEL = > 900 ppm - HDT Developmental LOAEL = NA Developmental NOAEL = > 900 ppm - HDT
870.3700a developmental inhalation rat (Isopar™ C Fluid)	EMBSI 1979 MRID 46719023 Unacceptable/Guide-line/Upgradeable	0, 400, 1200 ppm	Maternal LOAEL = NA Maternal NOAEL = > 1200 ppm - HDT Developmental LOAEL = NA Developmental NOAEL = > 1200 ppm - HDT
870.3700a developmental oral gavage rat (Exxsol™ D130 Fluid)	EMBSI 1996 MRID 46569210 Acceptable/Guideline	0, 400, 800, 1000 mg/kg/day	Maternal LOAEL = NA Maternal NOAEL = >1000 mg/kg/day – HDT Developmental LOAEL = NA Developmental NOAEL = >1000 mg/kg/day - HDT

Table 6. Toxicity Profile for Aliphatic Hydrocarbons			
Study Type	**Publication**	**Doses**	**Results**
3 day neurotoxicity inhalation rat (Nappar 10)	TNO Nutrition Food Research 2001 MRID 46543518 Acceptable/Non-guideline	0, 170, 430, 860 ppm	LOAEL = 860 ppm changes in gait and lower body temperature NOAEL = 430 ppm
3 day neurotoxicity inhalation rat (Isane IP 155)	TNO Nutrition Food Research 2001 MRID 4543519 Acceptable/Non-guideline	0, 85, 260, 860 ppm	LOAEL = 860 ppm increased latency to make a correct response in visual discrimination task NOAEL = 260 ppm
3 day neurotoxicity inhalation rat (n-decane)	TNO Nutrition Food Research 1999 MRID 46569207 Acceptable/Non-guideline	0, 85, 260, 860 ppm	LOAEL = 860 ppm decrease in forelimb grip strength observed in FOB and increase in the number of initial responses with a latency of > 6 seconds in the visual discretion test NOAEL = 260 ppm
Sub-chronic Toxicity			
870-3456 8 week inhalation rat, mouse (Isopar™ G Fluid)	EMBSI 1981 MRID 46719017 Acceptable/Non-guideline	0, 300, 900 ppm	LOAEL = 300 ppm altered clinical chemistry and urinalysis parameters related to kidney function in male and female rats NOAEL = NA effects seen at LDT
870-3456 12 week inhalation rat, mouse (Exxsol™ D40 Fluid, Isopar™ G Fluid)	EMBSI 1978 MRID 46543515 Acceptable/Non-guideline	0, 300, 900 ppm	LOAEL = NA NOAEL = > 900 ppm - HDT
870.3100 90 day oral gavage rat (Isopar™ M Fluid)	EMBSI 1990 MRID 46719018 Unacceptable/non-guideline/Upgradeable (pgs missing)	0, 100, 500, 1000 mg/kg/day	LOAEL = NA NOAEL = > 1000 mg/kg/day HDT
870.3100 90 day oral gavage rat (Exxsol™ D60 Fluid)	EMBSI 1991 MRID 46543517 Acceptable/Guideline	0, 500, 2500, 5000 mg/kg/day	LOAEL = 500 mg/kg/day depressed body weight, clinical signs NOAEL = NA effects seen at LDT
870.3100 90 day oral gavage rat (Exxsol™ D80 Fluid)	EMBSI 1991 MRID 46569206 Acceptable/Guideline	0, 100, 500, 1000 mg/kg/day	LOAEL = NA NOAEL = 1000 mg/kg/day - HDT
Chronic Toxicity			
870.4300 Chronic/Cancer - No Studies available	NA	NA	Not likely to be carcinogenic in humans
Genetic Toxicity			
870.5450 Dominant lethal inhalation assay (Exxsol™ D40 Fluid)	Schroeder et al 1978	0, 300, 900, ppm	No biologically significant difference between the control group and the treated group with respect to pregnancy rate or any of parameters indicative of dominant lethality
870.5450 Dominant lethal inhalation assay (Isopar™ G Fluid)	EMBSI 1978	0, 300, 900, ppm	Under the conditions of this test, this test substance administered by inhalation is not genotoxic in the germ cells of treated male rats
in vivo mouse bone marrow	EMBSI 1991	1.25, 2.5, 5.0 g/kg	Non-cytotoxic, non-clastogenic

Table 6. Toxicity Profile for Aliphatic Hydrocarbons			
Study Type	**Publication**	**Doses**	**Results**
cytogenetics assay (Exxsol™ D60 Fluid, Isopar™ M)			
Microbial Mutagenesis Ames Assay (Exxsol™ D60 Fluid)	EMBSI 1991	100, 320, 1000, 3200, 10000 µg/plate	Negative and without metabolic activation
Microbial Mutagenesis Ames Assay (Exxsol™ D100S Fluid)	EMBSI 1987	50, 150, 1500, 5000 µg/plate	Negative and without metabolic activation
Mammalian chromosome aberration test (Exxsol™ D100S Fluid)	EMBSI 1991	3.13 to 750 µg/mL	Negative and without metabolic activation
in vivo mouse bone marrow cytogenetics assay (Isopar™ G Fluid)	Jrnl of Applied Tox 1991	25 mL/kg	negative
Bacterial reverse mutation (Ames) assay (Exxsol™ D100, D140 Fluid, Isopar™ G)	EMBSI 1991 HRC 1990	Not stated in reference	not mutagenic
Bacterial reverse mutation (Isopar™ G Fluid)	Jrnl of Applied Tox 1990	Not stated in reference	negative
Microbial mutagenesis Ames assay (Isopar™ M Fluid)	EMBSI 1991	100, 320, 1000, 3200, 10000 µg/plate	negative
In vitro cytogenetic Assay (CHO) (Exxsol™ D100)	HLI 1991	Not stated in reference	negative
DNA Repair Test (Isopar™ G Fluid)	Jrnl of Applied Tox 1991	Not stated in reference	negative

4.2 Hazard Characterization

Aliphatic hydrocarbon fluids exhibit low acute toxicity by oral, inhalation and dermal routes (toxicity Category III or IV by all exposure routes). These compounds are minimally irritating to eyes and skin and negative for dermal sensitization effects.

The Exxsol™ D and Isopar™ aliphatic hydrocarbons are compositionally similar. Therefore, the toxicological data provided on a number of representative aliphatic hydrocarbons can be used to assess the potential toxicity of structurally-related compounds in the family of aliphatic hydrocarbon fluids between C_8 and C_{20} carbon length (Personal Communication Rebecca Jones, OPPTS, 6/30/06).

Subchronic oral and inhalation exposure studies indicate that Exxsol™ D and Isopar™ aliphatic hydrocarbon fluids exhibit low subchronic toxicity by the inhalation and oral routes of exposure. Depressed body weight and clinical signs (e.g., stomach abnormalities) were reported at the mid and high doses in a subchronic oral toxicity study in rats. C_8-C_{20} aliphatic hydrocarbons are not likely to be carcinogenic based on available data. Neither evidence of

developmental effects nor evidence of adverse effects in maternal animals was observed in oral and inhalation developmental studies in rats. However, altered behaviorial effects were observed at the HDT in short-term neurotoxicity studies on C_{10}-C_{11} mixed isoaliphatics. Aliphatic hydrocarbon fluids are of low toxicity for endpoints of concern for developmental and reproductive effects, based on the available information. Therefore, the tenfold FQPA safety factor for the protection of infants and children may be reduced to 1 for these compounds.

4.3 Summary of Toxicity Studies

4.3.1 Developmental and Reproductive Toxicity

Oral

In a developmental toxicity study (MRID 46569210), Exxsol™ D130 Fluid (% inert ingredient not stated) was administered to 25 Crl:CDBR female rats/dose by gavage at dose levels of 0, 400, 800, or 1000 mg/kg bw/day from days 6 through 15 of gestation. On gestation day (GD) 21, dams were sacrificed, subjected to gross necropsy, and all fetuses examined externally. The total number of fetuses examined (number of litters) were 334(23), 351(24), 361(25), and 386(25) in the 0, 400, 800, and 1000 mg/kg bw/day groups, respectively. Approximately one-half of the fetuses were examined viscerally, and the other one-half of the fetuses were examined for skeletal malformations/variations. No adverse effects were noted in dams. All animals survived to study termination and no treatment-related effects were observed in clinical signs, mean body weight or body weight gain, mean feed consumption, or gross pathological findings. No statistically significant adverse effects on pregnancy rate, number of corpora lutea, pre- or postimplantation losses, resorptions/dam, fetuses/litter, fetal body weight, or fetal sex ratio were observed in the treated groups compared with the controls. No exposure-related external, visceral, or skeletal malformations/variations were observed in any fetus. The maternal toxicity LOAEL for Exxsol™ D130 in rats could not be established. The maternal NOAEL is $\geq$ 1000 mg/kg bw/day. The developmental toxicity LOAEL in rats could not be established. The developmental NOAEL is $\geq$ 1000 mg/kg bw/day.

Inhalation

In a developmental toxicity study (MRID 46719023), 20 female Sprague-Dawley rats/group were exposed to air only, 400 or 1200 ppm of Isopar™ C Fluid. Exposures were in whole-body, dynamic inhalation chambers for 6 hours/day on gestation days (GDs) 6-15. On GD 21, dams were sacrificed and examined grossly. Each fetus was tagged, weighed, measured for crown-rump length, and examined for external malformations/variations and sex determination. Approximately two-thirds of the fetuses in each litter were examined viscerally by gross dissection then processed for skeletal examination. The remaining one-third of fetuses in each litter were fixed in Bouin's solution and examined viscerally by serial sectioning. Internal sex determination was made on all fetuses during visceral examination. No evidence of maternal or fetal toxicity was noted at either exposure level tested. The maternal inhalation toxicity NOAEL for this study is 1200 ppm and the maternal toxicity LOAEL is not identified.

The developmental inhalation toxicity NOAEL in rats is 1200 ppm and the developmental toxicity LOAEL is not identified.

In a developmental toxicity study (MRID 46719024, MRID 46543526), 20-21 female Sprague-Dawley rats/group were exposed to air only, 300 or 900 ppm of Exxsol D40 Fluid, or 300 or 900 ppm of Isopar G Fluid. Percent purity was not given for either test article. Exposures were in whole-body, dynamic inhalation chambers for 6 hours/day on gestation days (GDs) 6-15. On GD 21, dams were sacrificed and examined grossly. Each fetus was tagged, weighed, measured for crown-rump length, and examined for external malformations/variations and sex determination. Approximately two-thirds of the fetuses in each litter were examined viscerally by gross dissection then processed for skeletal examination. There was no evidence of maternal or fetal toxicity, nor any malformations noted at either exposure level tested. The maternal inhalation toxicity NOAEL for this study is 900 ppm and the maternal toxicity LOAEL is not identified. The developmental inhalation toxicity NOAEL in rats is 900 ppm and the developmental toxicity LOAEL is not identified.

The aliphatic hydrocarbon fluids have not been tested for reproductive toxicity. However, based on the following information on reproductive toxicity submitted by ExxonMobil, HED agrees that C_8-C_{20} aliphatic hydrocarbons are likely to be of low concern for reproductive toxicity.

> OECD SIDS guidelines expressly provide that chemicals such as Exxsol™ D Fluids need not be tested for reproductive toxicity to conclude that they are not likely to be reproductive toxicants: "For health effects testing the reproduction toxicity requirements may be satisfied through the use of data from several studies. . . . Requirements are met if existing data on the chemical include a developmental toxicity study and a 90-day repeated dose study that sufficiently documents that reproductive organs were examined histologically and indicate no effects." OECD, *Manual for Investigation of HPV Chemicals, Chapter 2: SIDS, The SIDS Plan and the SIDS Dossier*, p.11 (2002). Here, developmental toxicity studies representing the range of Exxsol™ D Fluids were conducted on Exxsol™ D40 and Exxsol™ D130 (BDI, 1978c; EMBSI, 1996). In addition, 90-day repeated-dose studies conducted on Exxsol™ D60 and Exxsol™ D80 showed no histopathological effects on the reproductive organs of rats (EMBSI, 1991f; EMBSI, 1991g). Though not a 90-day study, an 84-day repeated dose study on Exxsol™ D40 also showed no histopathological effects on rat reproductive organs. Together, these studies demonstrate that Exxsol™ D Fluids are of low concern for reproductive toxicity.

4.3.2 Neurotoxicity

A neurobehavioral testing program on aliphatic, cycloaliphatic and aromatic hydrocarbons was conducted by the Hydrocarbon Solvent Producers Association (HSPA) (TNO Nutrition and Food Research Institute 1999, 2001). Twelve representative constituents of complex hydrocarbon solvents, with carbon chain lengths ranging from C_5- C_{11}, were evaluated. Most representative for purposes of evaluating the toxicity of aliphatic hydrocarbon fluids were

the tests on C_{10}-C_{11} Mixed Isoaliphatics, C_{10} Cycloaliphatics, and *n*-decane. Male rats were exposed by inhalation, 8 hours per day for 3 consecutive days and tested for effects on motor activity, functional observation measures, and learned performance of a visual discrimination task.

In the TNO study on Isane IP 155 (MRID 46543519), male WAG/RijCrlBR rats (16/dose) were exposed by inhalation to the compound for 8-hours/day for 3 consecutive days at exposure levels of 0 g/m^3 (air), 0.5 g/m^3 (85 ppm), 1.5 g/m^3 (260 ppm), or 5.0 g/m^3 (860 ppm) in two separate cohorts and observed daily. There were no treatment related effects on mortality, clinical signs or body temperature. Slightly decreased body weight was observed in exposed groups during the 3-day exposure period. Functional observation battery and motor activity testing revealed no treatment-related effects. Some gait abnormalities and an overall difference for forelimb grip strength were observed, but these were not considered related to exposure. In the visual discrimination task, high-level (5.0 g/m^3) exposure induced mild non-persistent effects on measures of learned performance, including slightly increased latency to make a correct response, and increased variability in the speed of responding. Significant increases were found in mean number of long (>6 second) response latency in the 0.5 and 5.0 g/m^3 groups. However, the effects on the lower dose group should be considered biologically insignificant since differences were within the range of variation seen in this and other studies with solvents. Drink response latency, as a measure of single-choice response speed, was not significantly changed by exposure. Measures of discrimination accuracy and stimulus control were not affected. The inhalation LOAEL was 5 g Isane IP 155 /m^3 (~860 ppm)/day (based on increased latency to make a correct response in the visual discrimination task), with a NOAEL of 1.5 g Isane IP 155 /m^3 (~260 ppm)/day.

In a second TNO study (MRID 46543518), groups of 16 male WAG/RijCrlBR rats were exposed whole-body to atmospheres of Nappar 10 at concentrations of 0, 1, 2.5, or 5 g/m^3 (0, 170, 430, or 860 ppm, respectively), 8 hours/day, for three consecutive days. A single 8-hour exposure to Nappar 10 had no toxicologically significant effect on body weight or clinical signs. Bloody exudate around the nose and mouth was observed in the 2.5 and 5 g/m^3 groups after two and three days of 8-hour exposures. During the FOB, changes in gait (tip-toe walking and ataxia in 2/8 and 1/8 rats, respectively) and lowered body temperature ($p<0.05$) were observed in the 5 g/m^3 group after one 8-hour exposure. Visual discrimination results were variable and largely inconsistent and, therefore, generally inconclusive. No effect on response was apparent after one day of exposure. Three days of exposure to 5 g/m^3 may have decreased the number of very short latency responses (<1 sec) to the correct choice and increased the number of long latency responses (>6 sec). However, the toxicological relevance of these differences is questionable due to the small increase in numbers of long latency responses and the absence of a dose-response relationship. Based on the effects seen in this study (changes in gait and lower body temperature), the acute inhalation LOAEL for Nappar 10 in male rats is 860 ppm. The NOAEL is 430 ppm.

For the TNO study on n-decane, three separate inhalation experiments were performed in a neurobehavioral/toxicokinetic study (MRID 46569207) exposing 3-8 male WAG/RijCrlBR rats

(>99% inert ingredient) vapor at concentrations of 0 (air only), 0.5, 1.5 or 5 g/m^3 (0, 85, 260 or 860 ppm, respectively. In the functional observation battery, a statistically significant ($p<0.05$) decrease in the forelimb grip strength was observed after the third exposure period in the 5.0 g/m^3 group. This parameter was decreased compared to controls (21%) and compared to the pre-test value (29%). After the first 8-hour exposure, this same parameter was decreased when compared to controls (15%) and compared to the pre-test values (21%) in the 5.0 g/m^3 group, although it was not statistically significant. In the visual discrimination test, a statistically significant ($p< 0.05$) increase in the number of initial correct responses with a latency of > 6 seconds was observed in the 5.0 g/m^3 rats after the third exposure day. This number was increased 23% compared to controls and 18% compared to the same group in the pre-test. Although not indicated to be statistically significant in the study report, this trend was also observed in the same group of rats after the first and second exposure days. The post-exposure values were comparable to the controls and pre-test values indicating the effects were reversible. The test substance was found at higher concentrations in the brain than the blood and did accumulate at higher amounts with the increased exposure concentration. The test substance did not accumulate as the values were very similar in both the brain and blood when compared after a single 8 hour exposure and after three days of consecutive 8 hour exposures. The LOAEL for *n*-decane in rats was 860 ppm based on the decrease in forelimb grip strength observed in the FOB and an increase in the number of initial responses with a latency of > 6 seconds in the visual discrimination test. The NOAEL for n-decane in rats was 260 ppm.

4.3.3 Subchronic Toxicity

Oral

In a 90-day oral toxicity study (MRID 46543517) Exxsol D60 Fluid was administered by oral gavage to Sprague Dawley rats, ten/sex/dose at dose levels of 0, 500, 2500, and 5000 mg/kg bw/day, 7 days/week. Ten additional rats/sex, administered the test material at 5000 mg/kg bw/day, were maintained on control diet for a further four weeks to determine the reversibility of any effects seen. Clinical signs of toxicity were observed in the 5000 mg/kg dose group and to a lesser extent in the mid dose group. The most consistent findings were swollen anus, anogenital staining, and alopecia. No treatment-related mortality was observed. Statistically significant lower body weight compared to controls was observed in mid- and high-dose males ($p<0.01$), and in mid and high dose females ($p<0.05$). The lower body weight could not be attributed to decreased food consumption. The most significant treatment-related effects were swollen anus in the high dose group and the high incidence (≥70%) of stomach abnormalities seen histologically in both sexes from the mid- and high-dose groups. Some of the hyperplasia and hyperkeratosis of the stomach squamous mucosa was still evident in male rats from the high dose group after the recovery period. In females, there were no effects on kidneys at any dose, but dose-related kidney effects consistent with alpha-2μ-globulin nephropathy (hyaline droplet formation) were observed at all dose levels in males. HED agrees with ExxonMobil that these kidney effects are specific to male rats and should not be considered to be of biological relevance to humans. Based on the results of this study, the LOAEL is 500 mg/kg/day based on depressed body weight and clinical signs.

In a 90-day oral toxicity study (MRID 46569206) Exxsol D80 Fluid (purity not reported) was administered to 10 HSD:SD(CD) rats/sex/dose by gavage at dose solution concentrations of 0 (vehicle only), 2, 10 or 20%, 7 days/week for 90 days. The respective nominal dosages were 0, 100, 500 and 1000 mg/kg/day. Additional groups of 10 rats/sex, designated as satellite groups, were administered 1000 mg/kg/day for 90 days and observed for an additional 28 days (recovery period) before being sacrificed. The dose volume was 5 ml/kg for each group. There were no toxicologically significant effects based on the assessment of mortality, clinical signs, body weight, food consumption, eyes, hematology, clinical chemistry, organ weights or gross and histologic pathology. Histopathologic changes observed in kidneys at all dosage levels in males included hyaline droplets in the cytoplasm of proximal tubules of the cortex, dilated medullary tubules with granular casts and an increased incidence and severity of multifocal cortical tubular basophilia. These changes are indicative of alpha-2μ-globulin nephropathy which has been observed only in male rats and which is not relevant to humans. Compound-related changes in the liver included minimal centrilobular hepatocellular hypertrophy (reversed 28 days after termination of dosing) at 500 mg/kg/day in females and 1000 mg/kg/day in males and females. Slightly increased liver weights were detected at 500 and 1000 mg/kg/day in both sexes, however, there were no other findings that were supportive of hepatotoxicity. Based on these findings, the NOAEL for this 90-day oral study is 1000 mg/kg/day (the highest dose tested). A LOAEL was not determined.

In a 90-day oral toxicity study (MRID 46719018), Isopar M Fluid (100% inert ingredient.) was administered to Crl: CDBR Sprague-Dawley rats (10-20 male and 10 female rats/dose group) by gavage at dose levels of 0, 0.1, 0.5, or 1.0 g/kg/day (equivalent to 0, 100, 500, or 1000 [limit dose] mg/kg/day), 7 days/week for 13 weeks. Additionally, a satellite group (observed for at least 28 days post treatment) received the high dose, 7 days/week for 13 weeks. There were no compound related effects on survival, clinical signs, body weight, or food consumption. No biologically significant differences were found in hematology and clinical chemistry parameters between the treated and control groups. The dose-related increase in absolute and relative liver weights in male and female rats was considered to be an adaptive response because these increases were not supported by changes in gross or microscopic findings in the liver. The LOAEL for this compound is not established because of lack of significant toxicity at the limit dose. The NOAEL is 1000 mg/kg/day.

Inhalation

In an 8 week inhalation toxicity study (MRID 46719017), Isopar G Fluid (100% inert ingredient) was administered to 62 Fischer 344 rats/sex/dose and 20 B6C3F1 mice/sex/dose via inhalation at dose levels of 0, 300, or 900 ppm for 6 hours/day, 5 days/week. Ten rats/sex/dose were sacrificed at the end of weeks 1, 4, and 8 (after doses 5, 20, and 40) and after a 4-week recovery period and samples were taken for hematology and clinical chemistry. Gross pathological examinations were performed and samples were taken for possible histopathological examination. Three additional rats/sex/dose were designated to provide kidney samples for electron microscopy and an additional 10 rats/sex/dose were utilized for urinalysis at the same

time periods. All B6C3F1 mice were sacrificed after the final dose and no laboratory studies were performed. No treatment-related mortality was observed. In rats, absolute and relative liver weights were increased ($p \leq 0.01$) at 900 ppm in both sexes, relative kidney weight was increased ($p \leq 0.01$) at both dose levels in male rats throughout the treatment period but returned to control values during the recovery period. In mice, absolute and relative liver weight was increased at both dose levels ($p \leq 0.01$) in both sexes at terminal sacrifice. In male rats, decreases in RBC, hematocrit, hemoglobin, and reticulocyte values were observed at both dose levels during the treatment and recovery periods. Increases in clinical chemistry parameters were detected in males and females at various times throughout the treatment period. All clinical chemistry parameters returned to control levels during the recovery period. Results of urinalysis also indicated kidney function effects during the treatment period but urinalysis values returned to control levels during the recovery period. Results of electron microscopy on kidney samples of high-dose male rats after 5 exposures showed the formation of large, angular-shaped phagolysosomes in the cells of the proximal convoluted tubules. Acid phosphatase content of these phagolysosomes was reduced and limited to the periphery of the cells. All other cellular structures were comparable to controls. The inhalation LOAEL for this study is 300 ppm based on altered clinical chemistry and urinalysis parameters related to kidney function in both male and female rats. The inhalation NOAEL was not established in this study.

In a subchronic inhalation toxicity study (MRID 46543515), Exxsol™ D40 Fluid (purity and lot # not provided) was administered to 70 Sprague Dawley rats/sex/concentration by dynamic whole body exposure at nominal concentrations of 0, 300, or 900 ppm (analytical concentrations of 0, 312 ± 24, and 890 ± 33 ppm, respectively) for 6 hours per day, 5 days/week for a total of 12 weeks. Twenty rats/group were sacrificed after 4 and 8 weeks of treatment; the remaining 30 rats/group were sacrificed at study end. There was no mortality in the treated rats. Slight dry rales occurred in both the control and treated groups, likely related to chronic pneumonia found at microscopic examination. Ano-genital staining occurred almost exclusively in the treated groups during most weeks of the study. Body weight of the 900 ppm males was slightly decreased (7%, $p \leq 0.05$) after 8 and 12 weeks. No treatment-related changes were seen in hematology or clinical chemistry parameters for either sex. The liver/body weight ratios of both sexes in the 900 ppm group were slightly increased (9-12%, $p \leq 0.05$ or 0.01) at all sacrifice times, likely due to an adaptive response and/or decreased body weight. The kidney/body weight ratio in the 900 ppm males was increased 11-18% ($p \leq 0.01$) at all sacrifice times, and may have reflected decreased body weight and/or resulted from kidney lesions seen at the microscopic examination. Gross pathology was unremarkable. Microscopic examination revealed mild to moderate tubular injury in the kidneys of 20% of the 300 ppm males after 8 and 12 weeks, and 20%, 50%, and 50% of the 900 ppm males after 4, 8, and 12 weeks, respectively. The kidney injury was characterized by multifocal tubular degeneration, necrosis, and microcystic dilatation. The kidney effects observed in male rats are indicative of alpha-2μ-globulin nephropathy. These kidney effects are specific to male rats and are not considered to be of biological relevance to humans. The LOAEL is not established and the NOAEL is $\geq$ 900 ppm.

4.3.4 Chronic Toxicity/Carcinogenicity

C_8-C_{20} aliphatic hydrocarbon fluids have not been tested specifically for chronic toxicity/carcinogenicity. However, submitted data on the structure and metabolism, subchronic health effects, and genotoxicity of these compounds indicate that they are not likely to have carcinogenic properties. The submitted data indicate that these aliphatic hydrocarbon fluids do not belong to a class of chemicals known to react with DNA, nor are they metabolized to materials that are likely to react with DNA. The data available for aliphatic hydrocarbon fluids indicate that these compounds also do not produce significant cumulative toxicity. Based on the available information, HED agrees that aliphatic hydrocarbon fluids are unlikely to be carcinogenic.

4.3.5 Metabolism

There are no absorption, metabolism, distribution or excretion studies done specifically on C_8-C_{20} aliphatic hydrocarbon fluids. However, ExxonMobil described the absorption, distribution, metabolism and excretion aliphatic hydrocarbon fluids as follows based on data provided in Snyder 1987.

Typically, aliphatic hydrocarbons are well absorbed, widely distributed between tissues, extensively metabolized and rapidly excreted. Aliphatic hydrocarbons are absorbed into the blood predominantly from oral and inhalation routes of exposure, with respiratory absorption being the predominant route for the lower molecular weight aliphatics. Dermal absorption of aliphatic hydrocarbons is generally low and the efficiency of dermal absorption depends on the molecular weight and branching structure of the compounds. Typically, the solvents will be found at higher levels in the organs of metabolism and excretion, although they can distribute to other tissues as well, particularly those with high lipid content.

For most aliphatic hydrocarbons, hydroxylation at the penultimate carbon atom is the major metabolic pathway. Cytochrome P450 catalyzes the oxidation of the solvents to alcohol or acidic forms. Glucuronidation and sulfation are both common Phase II reactions in the metabolism of aliphatic hydrocarbons, and these reactions typically occur in the liver. Other conjugation reactions also may occur. This conjugation typically serves to detoxify the metabolites, and the conjugates often can be found in the urine.

Aliphatic hydrocarbons are rapidly excreted as water-soluble metabolites in urine or by exhalation of the parent material. Both rodents and humans show similar clearance kinetics of hydrocarbons from blood. In the urine, glucuronide conjugates are the predominant metabolites, although other conjugates and some parent compound may still be present. Some lower molecular weight aliphatic hydrocarbons – including some of the constituents in Exxsol™ D Fluids – may also be excreted through the lung. In radiotracer experiments, most aliphatic hydrocarbons are almost completely eliminated from the body within 72 hours, although small amounts may reside in organs with high lipid content for slightly longer periods of time.

4.4 Special Considerations for Infants and Children

C_8-C_{20} aliphatic hydrocarbon fluids exhibit low toxicity for developmental and reproductive effects based on the currently available information. Therefore an additional tenfold safety factor for the protection of infants and children is determined to be unnecessary.

4.5 Endpoint Selection

Acute RfD

An acute RfD for the general population and/or all population subgroups was not selected because no effect attributable to a single (or few) day(s) oral exposure was observed in animal studies.

Chronic RfD

For chronic dietary exposure for all populations, the oral NOAEL of 100 mg/kg/day was selected based on two 90-day oral toxicity studies conducted with Exxsol D80 and Isopar M Fluids. The LOAEL is 500 mg/kg/day based on depressed body weight and clinical signs (e.g., stomach abnormalities) observed in a 90-day oral toxicity study conducted with Exxsol D60 Fluid. An uncertainty factor (UF) of 100x (10x for interspecies variation and 10x for intraspecies is extrapolation) was applied which results in an RfD of 1 mg/kg/day. The study and the end point are considered as the most appropriate for chronic dietary exposure based on the available toxicological data. An additional UF for use of a subchronic study for selection of a chronic exposure endpoint is not required because effects do not tend to become more severe with higher exposure.

$$\text{Chronic RfD} = \frac{100 \text{ mg/kg/day}}{100 \text{ (UF)}} = 1 \text{ mg/kg/day}$$

Short Term Inhalation

For short-term inhalation, the toxicology endpoint was selected from studies conducted as part of a 3 day inhalation neurobehavioral testing program in rats on aliphatic, cycloaliphatic and aromatic hydrocarbons. The NOAEL for studies on Isane IP 155 and n-decane was 260 ppm (400 mg/kg) based on decrease in forelimb grip strength observed in FOB and increase in the number of initial responses with a latency of > 6 seconds in the visual discretion test (n-decane) and increased latency to make a correct response in visual discrimination task (Isane IP) at the HDT of 860 ppm (1300 mg/kg). These inhalation studies are considered the most appropriate for endpoint selection based on the expected duration of exposure (short-term). The level of concern (LOC) or target margin of exposure (MOE) for inhalation exposures is 100 based on the conventional uncertainty factor of 100X (10x for interspecies and 10x for intraspecies is extrapolation).

Short Term Dermal

For short term dermal exposure, the oral NOAEL of 100 mg/kg/day was selected from the 90-day oral toxicity study based depressed body weight and clinical signs at the LOAEL of 500 mg/kg/day. The LOC or MOE for dermal exposures is 100 based on the conventional uncertainty factor of 100X.

Dermal Absorption

A dermal absorption estimate of 0.5% was selected based on a dermal absorption study conducted in weanling pigs with selected components of JP-8 jet fuel (Singh et al., 2003). In this study, radiolabeled heptane and hexadecane were applied to the skin. In addition to penetration, transepidermal water loss (TEWL) was measured to assess damage to the stratum corneum. Clearly, TEWL was enhanced by heptane but not by hexadecane. Results indicated that heptane and hexadecane were absorbed 0.14 and 0.43%, respectively. Heptane (C_7) and Hexadecane (C_{16}) are at the low-end and high-end of the class.

Incidental Oral

For incidental oral exposure, the oral NOAEL of 100 mg/kg/day was selected from the 90-day oral toxicity study based depressed body weight and clinical signs at the LOAEL of 500 mg/kg/day. The LOC or MOE for incidental oral exposures is 100 based on the conventional uncertainty factor of 100X.

Table 7. Summary of Toxicological Doses & Endpoints for C_8-C_{20} Aliphatic Hydrocarbons for Risk Assessment

Exposure Scenario	Dose Used in Risk Assessment, UF	FQPA SF and Level of Concern for Risk Assessment	Study and Toxicological Effects
Acute Dietary (general population)	An acute RfD for the general population and/or all population subgroups was not selected because no effect attributable to a single (or few) day(s) oral exposure was observed in animal studies.		
Chronic Dietary (all populations)	NOAEL = 100 mg/kg/day UF =100 Chronic RfD = 1 mg/kg/day	FQPA SF = 1X cPAD = chronic RfD / FQPA SF = 1 mg/kg/day	LOAEL male = 500 mg/kg/day depressed body weight, clinical signs
Incidental Oral Exposure, Short-Term (1 - 30 days)	NOAEL = 100 mg/kg/day UF =100	MOE = 100	LOAEL male = 500 mg/kg/day depressed body weight, clinical signs
Dermal Exposure Short-term DAF = 0.005	NOAEL = 100 mg/kg/day UF = 100	MOE = 100	LOAEL male = 500 mg/kg/day depressed body weight, clinical signs
Inhalation Exposure Short-term	NOAEL of 260 ppm (400 mg/kg/day) UF=100	MOE = 100	LOAEL = 860 ppm (1300 mg/kg/day) based on increased latency to make a correct response in visual discrimination task decrease in forelimb grip strength observed in FOB and increase in the number of initial responses with

Table 7. Summary of Toxicological Doses & Endpoints for C_8-C_{20} Aliphatic Hydrocarbons for Risk Assessment			
Exposure Scenario	**Dose Used in Risk Assessment, UF**	**FQPA SF and Level of Concern for Risk Assessment**	**Study and Toxicological Effects**
			a latency of > 6 seconds in the visual discretion test
Cancer (oral, dermal, inhalation)	Not likely to be carcinogenic in humans		

5.0 Exposure Assessment

5.1 Dietary Exposure and Risk Assessment

To assess whether C_8-C_{20} aliphatic hydrocarbon fluids meet the standard for reissuance of a tolerance exemption, HED conducted a chronic dietary exposure and risk assessment using the Screening-Level Dietary Exposure Model for Inert Ingredients developed jointly by the Inerts Team and residue chemists in HED. An acute exposure assessment was not conducted for this analysis because no effect attributable to a single (or few) day(s) oral exposure was observed in animal studies. For the chronic assessment, anticipated residues of C_8-C_{20} aliphatic hydrocarbon fluids were compared to modeled anticipated residues for inerts which were derived based on the inert ingredients screening model.

5.1.1 Inert Ingredient Screening Model

The Tier 1 Inert Ingredient Model assessment is based on the following assumptions: actual crop-specific residue data for active ingredients can be utilized as surrogate data for inert ingredient residue levels (including secondary residues in meat, milk, poultry and eggs); inert ingredients are used on all crops and 100% of all crops are "treated" with inert ingredients; no adjustment made for percent of inert in formulation, application rate, or multiple applications of different active ingredient formulations; and only preharvest applications are considered.

The Inert Ingredient Model exposure estimates are based on highest tolerance level residues of high-use active ingredients for all food forms, including meat, milk, poultry, and eggs. A group of 57 of the most "significant" active ingredients were considered. These active ingredients included substances in the insecticide, fungicide, and herbicide class and were selected based on a overall ranking scheme that included the following components. Overall use from 1999 data for active ingredient use (in lbs/yr) – all herbicides at >5 million lbs/yr and all fungicides and insecticides at > 1 million lbs/yr were included. All active ingredients used on crops that are significant contributors to diet were included (i.e., all which had substantial use on crops that make up the "Top 25" children's diet). Crop-by-crop pesticide use information was evaluated to identify the most frequently used active ingredients. Data from actual residue monitoring studies from active ingredients with the highest frequency of detection were used. Tolerances for the 57 active ingredients were examined for each of the representative crops in the Agency's crop group designations [40 CFR 180.41] and for all crops not included in a crop group. Where there were multiple tolerances for a given crop or commodity, the highest

tolerance was chosen as the residue level for the model. Non-representative crops within each crop group were matched to their most-closely related representative crop based on OPP/HED's standard operating procedure 2000.1 (USEPA, 2000).

Tier 1 generic inert ingredient acute and chronic dietary exposure assessments were conducted using the Dietary Exposure Evaluation Model software with the Food Commodity Intake Database (DEEM-FCID™, Version 1.3), which incorporates consumption data from USDA's Continuing Surveys of Food Intakes by Individuals (CSFII), 1994-1996 and 1998. The 1994-96, 98 data are based on the reported consumption of more than 20,000 individuals over two non-consecutive survey days. Foods "as consumed" (e.g., apple pie) are linked to EPA-defined food commodities (e.g. apples, peeled fruit - cooked; fresh or N/S; baked; or wheat flour - cooked; fresh or N/S, baked) using publicly available recipe translation files developed jointly by USDA/ARS and EPA. Consumption data are averaged for the entire U.S. population and within population subgroups for chronic exposure assessment, but are retained as individual consumption events for acute exposure assessment.

For chronic exposure and risk assessment, an estimate of the residue level in each food or food-form (e.g., orange or orange juice) on the food commodity residue list is multiplied by the average daily consumption estimate for that food/food form. The resulting residue consumption estimate for each food/food form is summed with the residue consumption estimates for all other food/food forms on the commodity residue list to arrive at the total average estimated exposure. Exposure is expressed in mg/kg body weight/day. This procedure is performed for each population subgroup. A DEEM™-type analysis was performed utilizing the highest established tolerance level residue for each commodity. In those cases where DEEM listed a commodity for which a published tolerance did not exist, the input value was selected based on representative crops or other "default" values (e.g., use of standard processing factors). DEEM-FCID™, Version 1.3 analyses were performed for chronic dietary exposure scenario. Results are given in Table 8. The results of this Inert Ingredient Screening Model should represent an upper-bound estimate of likely potential dietary exposure to an inert ingredient resulting from preharvest use. For this assessment of C_8-C_{20} aliphatic hydrocarbon fluids, these values were compared to the selected toxicity endpoints using the percent of Population Adjusted Dose (%PAD) approach.

Table 8. Estimated Chronic Dietary Exposure[1] for a Generic Inert.

Population Subgroup	Estimated Chronic Exposure (mg/kg/day) Average
U.S. Population (total)	0.120
All infants (< 1 year)	0.245
Children (1-2 years)	0.422
Children (3-5 years)	0.310
Children (6 -12 years)	0.174
Youth (13-19 years)	0.100
Adults (20-49 years)	0.087
Adults (50+ years)	0.086
Females (13-49 years)	0.087

[1] Exposure estimates are based on highest-tolerance-level residues of high-use active ingredients for all food forms, including meat, milk, poultry, and eggs.

5.1.2 Chronic Dietary Exposure and Risk

The Tier 1 Inert Ingredient Model screening assessment does not account for evaporative loss. To assess the impact of evaporative loss on dietary exposures to C_8-C_{20} aliphatic hydrocarbon fluids, ExxonMobil conducted an assessment of the evaporative loss of Exxsol D Fluids using ASTM Method D3539. Based on the results reported by ExxonMobil there is a significant potential for evaporative loss of Aromatic Hydrocarbons from treated agricultural surfaces (e.g., foliage). According to ExxonMobil, these results indicate a significant potential for evaporative loss of aliphatic hydrocarbon fluids from treated agricultural surfaces i.e., the evaporative loss of neat Exxsol™ D Fluids are expected to be < 1 to 10 days. The company further notes that this would also be expected with aliphatic hydrocarbon fluids in aqueous-based end-use formulations to a somewhat greater or lesser degree than neat material due to influences of co-volatilization and mixture effects, respectively and that field evaporative loss will also be influenced by environmental conditions such as variable temperature and air movement. ExxonMobil estimates that an evaporative loss factor of 95% loss and 99.9% can be applied to the chronic "no loss" exposure estimates presented in Table 7. ExxonMobil also notes C_8-C_{20} aliphatic hydrocarbon fluids being evaluated in this assessment are applied in the 1 to 4 lbs of inert per acre range, and the tolerance-based residue data used in the Tier 1 assessment are based on application rates also ranging from 1 to 5 lbs a.i. per acre.

Application of a 95% loss factor to the Tier 1 Inerts Model residue values, as proposed by ExxonMobil based on the ASTM data, results in exposures significantly below OPP's level of concern for chronic exposures as shown in Table 9. Significantly, an highly conservative assumption of no evaporative loss would still result in exposures below the level of concern.

Table 9. Estimated Chronic Dietary Exposure and Risk for C_8-C_{20} Aliphatic Hydrocarbon Fluids

Population Subgroup	Chronic Dietary Exposure		
	cPAD (mg/kg/day)	Exposure (mg/kg/day)	Mean % cPAD
U.S. Population (total)	1.0	0.0060	0.6
All infants (< 1 year)	1.0	0.0123	1.2
Children (1-2 years)	1.0	0.0211	2.1
Children (3-5 years)	1.0	0.0155	1.6
Children (6-12 years)	1.0	0.0087	0.9
Youth (13-19 years)	1.0	0.0050	0.5
Adults (20-49 years)	1.0	0.0044	0.4
Adults (50+ years)	1.0	0.0043	0.4
Females (13-49 years)	1.0	0.0044	0.4

The results of this assessment indicate that chronic dietary risks are well below OPP's level of concern. This assessment likely represents an upper-bound estimate of likely potential dietary exposure to C_8-C_{20} aliphatic hydrocarbon fluids resulting from preharvest application of these inert ingredients. As stated in the documentation for the Inert Screening Model, in cases where this model would yield dietary risk values below the level of concern, no further

refinements are necessary, and the potential dietary exposure and risk are considered adequately characterized.

5.2 Environmental Fate and Drinking Water Characterization

The Environmental Fate and Effects Division (EFED) conducted the following qualitative assessment of the likely fate and exposure associated with the use of aliphatic hydrocarbon fluids pesticide inert ingredients (Personal Communication, Sid Abel, EFED, 7/17/06). The compounds included in this review are identified in Table 1. Information summarized was obtained from a number of sources, including Structural Activity Relationships (SARs) for representative compounds. Representative compounds include several intermediate-, substituted-, and cyclo-paraffins. The SAR class selected for estimating physical-chemical properties, environmental behavior, and environmental toxicity is the neutral organic compounds class. The compounds subject to this review are generally classified by aliphatic chain length or number of carbons.

A review of the readily available information and use of SARs on representative compounds that make up the C_7 through C_{25} aliphatic hydrocarbons is sufficient to conduct a qualitative assessment of the likely fate, exposures and environmental toxicity associated with their use as pesticide inert ingredients. Environmental loadings attributed to use as an inert in pesticide formulations is likely to be overwhelmed by other anthropogenic sources.

Available data and SAR indicate that the aliphatic hydrocarbons will, as discrete chemicals or mixtures, undergo primary biologically mediated degradation in a matter of days to weeks and ultimate degradation (mineralization) in a matter of weeks to months (half-lives will be shorter than reported for ultimate degradation) for most chains lengths. Longer chain aliphatic hydrocarbons (C_{12} through C_{25}) tend to degrade at a slower rate than the shorter chain molecules. Under anaerobic conditions, the C_7 through C_{25} compounds are expected to biodegrade somewhat slower than under aerobic conditions. Where available, literature data are in good agreement with SAR estimates.

Based on vapor pressure, these compounds are expected to partition to the atmosphere fairly rapidly, shorter chains molecules ($<C_{18}$) having a greater likelihood of volatilization than the longer ($>C_{18}$) chain molecules. Once in the atmosphere, they are available for long range transport and deposition via washout during precipitation. Likewise, they are subject to atmospheric photo-oxidation. Estimated indirect atmospheric photo-oxidation is expected to occur for all compounds in this group. Reaction rates (half-life) range from hours to several days based on representative compound analyses.

The short- and intermediate-chained (C_7-C_{12}) compounds are expected to be predominantly found in the non-sorbed state, while compounds greater than C_{12} will likely be found sorbed to sediments and organic material in terrestrial and aquatic environments based on fugacity modeling. Transformation and/or degradation via hydrolysis and direct soil and water

photolysis are not important dissipation pathways based on a lack of hydrolizable functional groups and the absorption range for these compounds outside the visible range, respectively.

Transport to surface water in the dissolved phase is expected to dominate the non-degradation and volatilization pathways of dissipation for the C_7- C_{12} compounds based on water solubility and low sorption coefficients. Longer chain compounds, C_{13}- C_{25} are likely to move to surface water in the dissolved phase and in association with sediments and other particulate matter when runoff producing rainfall occurs within days of application to terrestrial environments. Bioconcentration is not expected to be significant for the shorter chain molecules, while longer chain molecules will exhibit greater propensity to bioconcentrate.

Shallow aquifer ground water contamination of the short- and intermediate-chain compounds may occur; however, biologically mediated degradation in both aerobic and anaerobic conditions will limit loadings, thus, concentrations. Based on the high volatility of the aliphatic hydrocarbons in this group and aeration sequences used in many drinking water treatment utilities, it is unlikely that most of these compounds will be found in treated water at concentrations equivalent to those found in the environment. Concentrations of longer chain compounds (C_{13}-C_{25}) will be limited by solubility, volatility and biodegradation prior to transport to surface waters. There are no ambient water quality criteria or drinking water maximum contaminant or health advisory levels for these compounds.

Table 10. Summary of Qualitative Environmental Characteristics of Aliphatic Hydrocarbons

CASN	Solubility (mg/L)	Vapor Pressure (mm Hg)	Log K_{ow}	Biodegradability	Atmospheric Half-life	Fugacity
64742-47-8	<100	>0.02	>3	Inherently Biodegradable	<1 day	Air and Soils
64742-48-9	<100	>0.02	>3	Inherently Biodegradable	<1 – 2 days	Mostly Air
64742-46-7	<10	~0.003	>3	Not Readily Biodegradable	<1 day	Air and Soils
64741-66-8	<100	>0.02	>3	Readily Biodegradable	1 – 3 days	Mostly Air

5.3 Residential Exposure and Risk Assessment

ExxonMobil submitted a screening level quantitative residential exposure risk assessment for aliphatic hydrocarbons to EPA in support of a tolerance exemption reassessment. The ExxonMobil assessment used the Residential Exposure Assessment Model (REx Version 4.0) for the majority of exposure scenarios. HED conducted an independent quantitative residential exposure and risk assessment for the aliphatic hydrocarbons using OPP established SOPs and available scenario specific exposure data. High end use and exposure assumptions were used for the residential exposure assessment (e.g., maximum application rate, no evaporative loss). Therefore, this analysis is considered to be a screening level analysis and is likely to overestimate risk.

5.3.1 Exposure Scenarios

Based on the information provided by ExxonMobil on use patterns for C_8-C_{20} aliphatic hydrocarbon fluids applied as inert ingredients, HED assessed the following residential exposure scenarios.

1) Mixing, loading, and applying liquid spray formulation to lawns and ornamentals by low-pressure handwand.
2) Mixing, loading, and applying liquid spray formulation to lawns and ornamentals by hose-end sprayer.
3) Toddler incidental ingestion of residue from exposed turf grass via hand-to-mouth and object-to-mouth activities.
4) Dermal Exposure to adults and children reentering treated lawns.
5) Toddler incidental ingestion of residues deposited on carpet and vinyl via hand-to-mouth activities after use of total release foggers.
6) Toddler incidental ingestion of residues on pets via hand-to-mouth activities and dermal exposure after pet treatment.
7) Inhalation exposure to by adult applicator to aerosol spray during and after space spray application and post-application inhalation exposure to aerosol spray by child.
8) Direct application to humans of insect repellents containing aliphatic hydrocarbon fluids.

5.3.2 Exposure Assumptions

Only short-term residential exposures are expected based on the anticipated use patterns. In accordance with HED policy, data from the Pesticide Handlers Exposure Database (PHED) and/or Outdoor Residential Exposure Task Force was used handler exposures in the absence of chemical-specific monitoring data (USEPA, 1998, USEPA, 2000). Assumptions regarding application rates and percent inert ingredient are based on information provided by ExxonMobil. Scenario specific data from the Non-Dietary Exposure Task Force (NDETF) was used to estimate indoor residential exposures. Data from a DEET Joint Venture/Chemical Specialties Manufacturers Association 1990 survey was used to estimate exposures from personal use insecticide repellent.

5.3.2.1 Outdoor Residential Application and Post-Application

- average body weight of an adult handler is 70 kg
- average body weight of a toddler is 15 kg
- maximum application rate is 2.2 lb inert per acre based on information submitted by ExxonMobil
- area treated is 0.5 acres per day
- estimated turf transferable residue is assumed to be 5% of the maximum application rate for sprays
- saliva extraction factor is 50 percent
- surface portion of hand put in mouth is 20 cm^2

- hand-to-mouth exposure frequency is 20 times per hour
- object to mouth transfer efficiency is equal to 20% of the application rate
- ingestion rate of residues from mouthing turf or a small object is 25 cm^2
- exposure duration is 2 hours
- dermal absorption is 0.5%

5.3.2.2 Indoor Residential Application and Post-Application

Scenario specific data on from the Non-Dietary Exposure Task Force (NDETF) was used to conservatively estimate deposition on vinyl and carpet flooring following use of a total release indoor fogger and indoor air concentrations from use of aerosol sprays (MRID Numbers: 46188602, 46188613, 46188623, 46188629 and 46188618)

- indoor surface residue from use of indoor foggers is 200 µg/cm2 based on NDETF study data and a maximum inert ingredient concentration of 45% based on ExxonMobil data
- hand transfer efficiency is 13% for carpet; 10% for vinyl based on NDETF data
- saliva extraction factor is 50 percent
- surface portion of hand put in mouth is 20 cm^2
- hand-to-mouth exposure frequency is 20 times per hour
- for indoor aerosol spray, one 16 oz spray can containing maximum of 35% inert ingredient (based on ExxonMobil data) is used per application;
- Exposure duration is 2 hours

5.3.2.3 Pet Application

- ½ of 16 oz spray container per 6000 cm^2/animal with maximum of 25% inert ingredient (% inert based on information provided by ExxonMobil)
- transferable residue from a treated pet is assumed to be 20% of the maximum application rate for sprays
- surface area of a treated (30 lb) dog is 6000 cm2 (EPA 1993 Wildlife Exposure Factors Handbook - carbaryl)
- saliva extraction factor is 50 percent
- surface portion of hand put in mouth is 20 cm^2
- transferable residue from pet is 10 percent
- frequency of hand-to-mouth/dermal events is one per day (frequency modified to reflect transferable residue assumption which is based on a 5 minute heavy rubbing/petting technique that would lead to significantly higher concentrations than would result from a single contact)

5.3.2.4 Personal Use Insect Repellant

Use frequency and quantity data were obtained from the 1990 survey study conducted for the insecticide repellent DEET and submitted by a joint group of registrants, the DEET Joint Venture/Chemical Specialties Manufacturers Association (MRID 41968001).

- adult Male - 70 kg
- adult Female - 60 kg
- child 6-12 and under - 33 kg
- child 13-17 - 58 kg
  Note: The body weights and age ranges used for this assessment correspond to the age groupings for which exposure data were provided in the DEET survey. Body weights are from the USEPA Exposure Factors Handbook (1997)
- Mean amount of product applied to skin & clothing per application (DEET Survey):
  - Adult Male - 5.2 g
  - Adult Female - 4.3 g
  - Child 6-12 years - 4.8 g
  - Child 13 to17 years - 5.2 g
- Based on information provided by the ExxonMobil, the maximum concentration of aliphatic hydrocarbon fluids in a product formulation intended for human application is 65%.
- Dermal Absorption is 0.5%

5.3.3 Residential Exposure and Risk Estimates

Results of the residential exposure assessment are provided in Tables 11-17. A target MOE of 100 for the inhalation and dermal routes is considered adequate for the residential exposure and risk assessment. Estimated inhalation and dermal MOEs for both handler scenarios are greater than the target MOE of 100 and not of concern. Again, the residential exposure assessment is a screening level analysis based on high end use and exposure assumptions and is therefore likely to overestimate risk.

Table 11. Estimated Inhalation Exposure & MOEs for Residential Handlers – Lawn Application Target MOE = 100

Exposure Scenario	Dermal Unit Exposure (mg/lb inert)[1]	Inhal Unit Exposure (µg/lb inert)[1]	Use[2]	Max App Rate[3] (lb/acre)	Daily Area Treated[4] (Acre/day)	Dermal Dose (m/k/d)[5]	Inhal Dose (m/k/d)[6]	Dermal MOE[7]	Inhal MOE[8]
Mixing/Loading/Applying Liquids									
Low Pressure Handwand	100	30	Lawn	2.2	0.5	0.008	0.0005	13000	800000
Hose-end Sprayer	17	11				0.001	0.0002	75000	>1000000

[1]Baseline inhalation unit exposures represent no respirator. Values are reported in the PHED Surrogate Exposure Guide dated August 1998 or are from data submitted by the Outdoor Residential Exposure Task Force dated May 2000.
Baseline dermal unit exposures represent long pants, long sleeved shirts, shoes, and socks. Values are reported in the PHED Surrogate Exposure Guide dated August 1998 or are from data submitted by the Outdoor Residential Exposure Task Force dated May 2000.
[2] Use patterns are from information provided by the registrant and product labels

[3]Application rates are based on maximum values submitted by the registrant and verified by an HED cursory label review. In most scenarios, a range of maximum application rates is used to represent the range of rates for different crops/sites/uses. Application rates upon which the analysis is based are presented as lb ai/A.

[4]Amount treated is based on the area or gallons that can be reasonably applied in a single day for each exposure scenario of concern based on the application method and formulation/packaging type. (Standard EPA/OPP/HED values).

[5]Dermal dose (mg/kg/day) = [unit exposure (mg/lb ai) * Dermal absorption (0.5%) * Application rate (lb ai/acre or lb ai/gallon) * Daily area treated (acres or gallons)] / Body weight (70 kg).

[6] Inhalation dose (mg/kg/day) = [unit exposure (ug/lb ai) * 0.001 mg/ g unit conversion * Inhalation absorption (100%) * Application rate (lb ai/acre or lb ai/gallon) * Daily area treated (acres or gallons)] / Body weight (70 kg).

[7] Dermal MOE = short-term endpoint for dermal - dermal LOAEL (mg/kg/day) / Daily Dermal Dose.

[8] Inhalation MOE = short-term endpoint for inhalation - oral NOAEL (mg/kg/day) / Daily Inhalation Dose.

Table 12. Estimated Post-application Incidental Ingestion Risks to Toddlers Reentering Treated Lawns Short-Term Target MOE = 100

	Hand to Mouth			Object to Mouth			Aggregate
Max AR (lb inert/A)	Hand Transfer (µg/cm2)	Daily Oral Dose (m/k/d)	MOE	Dislogeable Foliar Residue (ug/cm2)	Daily Oral Dose (m/k/d)	MOE	MOE
2.2	1.1	0.029	1400	4.3	0.007	5600	1100

1 DOD(mg/kg/day) = Daily Oral Dose (PDR/ BW)
BW = 15 kg for toddler

Hand To Mouth Calculation

$PDR_{(t)}$ (mg/day) = ($HTF_{(t)}$ (µg/cm^2) * SEF * SA * Freq * ED/1000 (µg/mg)

where:

PDR = Potential Dose Rate at time (t) attributable for activity in a previously treated area (mg/day)
$HTE_{(t)}$ = Hand Transfer Efficiency at time t = 5% of Application Rate (µg/cm^2)
SEF = Saliva Extraction Factor (50%)
SA = Surface Area of Two Fingers (20 cm^2)
Freq = Frequency of Hand to Mouth Events (20)
ED = Exposure Duration in hours (2 hr/day)
t = Postapplication Day on which exposure is being assessed (day 0)
MOE = Short Term Oral NOAEL /Daily Oral Dose (mg/kg/day)

Object to Mouth Calculation

$PDR_{(t)}$ (mg/day) = ($DFR_{(t)}$ (µg/cm^2) * SA/1000 (µg/mg)

where:

PDR = Potential Dose Rate at time (t) attributable for activity in a previously treated area (mg/day)
$DFR_{(t)}$= Dislogeable Foliar Residue at time t = 20% of Application Rate (µg/cm^2)
SA = Surface Area of grass or toy mouthed by toddler (25 cm^2 day)
t = Postapplication day on which exposure is being assessed (day 0)
MOE = Short Term Oral NOAEL/[Daily Oral Dose (mg/kg/day) MOEs are reported to two significant figures

Table 13. Estimated Post-Application Risks to Persons Reentering Treated Lawns: Target MOE = 100

Exposed Individual	Maximum AR (lb inert/A)	TTR (µg/cm2)	DDD (mg/kg/day) [1]	MOE [2]
Adult	2.2	1	0.002	46000
Child	2.2	1	0.004	28000

1 DDD(mg/kg/day) = Daily Dermal Dose (DDE / BW)
BW = 70 kg for adult; 15 kg for toddler

where

$DDE_{(t)}$ (mg/day) = ($TTR_{(t)}$ (µg/cm^2) x TC (cm^2/hr) x Hr/Day)/1000 (µg/mg)

where:

DDE = Daily Dermal Exposure at time (t) attributable for activity in a previously treated area (mg/day);
TTR = 5% of AR (µg/cm^2)
TC = Transfer Coefficient (500 cm^2/hour for adult golfer; 14,500 cm^2/hour for adults; 5200 cm^2/hour for toddler)
Hr = Exposure duration in hours (2 hr/day for adult & toddler)
TTR_t = TTR_0 * (Max AR/StudyAR) * $e^{(TTRslope * t)}$

where:

AR = application rate (lbs ai/ft^2 or lb ai/acre)
t = postapplication day on which exposure is being assessed = day 0

2 Dermal MOE = Dermal NOAEL (mg/kg/day)/[Daily Dermal Dose (mg/kg/day) x Dermal Absorption Value 0.5%].

Table 14. Estimated Post-application Incidental Ingestion Risks To Toddlers Playing on Vinyl Floor and Carpet after Treatment with Fogger Formulation - Non-Cancer Short-Term Target MOE = 100

Indoor Surface	Application Rate (lb ai/1000 ft2)	Indoor Surface Residue (ug/cm2)	Hand Transfer Efficiency (%)	Daily Oral Dose (mg/kg/day)1	MOE
carpet	0.07	200	13	0.38	150
vinyl	0.07	200	10	0.71	200

1 DOD(mg/kg/day) = Daily Oral Dose = PDR/ BW

$PDR_{(t)}$ (mg/day) = $(ISR_{(t)}$ (µg/cm^2) * TE * SEF * SA * Freq * ED/1000 (µg/mg)

where:

PDR = Potential Dose Rate on day of application (mg/day)
ISR = Indoor Surface Residue (µg/cm^2) at maximum AR of 0.07 lbs inert ingred/1000 ft^2
HTE = Hand Transfer Efficiency - transfer of (13% for carpet; 8% for vinyl)
SEF = Saliva Extraction Factor (50%)
SA = Surface Area of Two Fingers (20 cm^2)
Freq = Frequency of Hand to Mouth Events (20)
ED = Exposure Duration in hours = 2 hr/day
t = Postapplication Day on which exposure is being assessed (day 0)
BW = 15

Table 15. Estimated Post-application Incidental Ingestion And Dermal Risks To Toddlers Playing with Pets after Treatment with Spray Formulation - Non-Cancer - Short-Term Target MOE = 100

Application Method	AR (g inert/animal)	Transferable Residue (%)	Daily Oral Dose (mg/kg/day)[1]	Daily Dermal Dose (mg/kg/day)[2]	Oral MOE	Dermal MOE
Aerosol Spray	40	20	0.9	0.00005	110	>1000000

1 DOD(mg/kg/day) = Daily Oral Dose = PDR/ BW

$PDR_{(t)}$ (mg/day) = $((AR_t$ (mg ai/animal) * F)/$SA_{pet})$ * SEF * SA_{hands} * Freq

where:

PDR = Potential Dose Rate - nondietary ingestion dose from contact with treated pets (mg/day)
AR = Application Rate or amount applied to animal in a single treatment (mg ai/animal) = ½ of 16 oz spray container with maximum of 25% inert ingredient per 6000 cm^2/animal
F_{AR} = Fraction of Application Rate available contact as dislogeable residue (20%)
SA_{pet} = Surface Area of a treated dog (6000 cm^2/animal)
t = Time After Application (0 days)
SEF = Saliva Extraction Factor (50%)
SA_{hands} = Surface Area of the hands (20 cm^2)
Freq = Hand-to-Mouth Events (1 event/day)
BW = 15 kg for toddler
MOE = Short Term Oral NOAEL/Daily Oral Dose (mg/kg/day) MOEs are reported to two significant figures.

2 DDD(mg/kg/day) = Daily Dermal Dose = PDR/ BW

$PDR_{(t)}$ (mg/day) = $((AR_t$ (mg ai/animal) * F)/$SA_{pet})$ * ATR * DAF

where:

PDR = Potential Dose Rate - dermal dose from contact with treated pets (mg/day)
AR = Application Rate or amount applied to animal in a single treatment (mg ai/animal) = ½ of 16 oz spray container with maximum of 25% ai per 6000 cm^2/animal
F_{AR} = Fraction of Application Rate available for contact as dislogeable residue (20%)
SA_{pet} = Surface Area of a treated dog (6000 cm^2/animal)
t = Time After Application (0 days)

Table 16. Estimated Inhalation Risks To Adults and Children During and After Indoor Aerosol Space Spray Application - Target MOE = 100

Application Method	Exposed Individual	Breathing Zone Conc (mg/m3)	Inhalation Dose (mg/kg/day)1	MOE
Aerosol Spray	Adult Application & Post Application	0.3	0.004	48000
	Child Post-Application	0.13	0.014	30000

$PDR_{(t)}$ (mg/day) = $((AR_t$ (lb ai/A)- BZC * BR * ED

where:

PDR = Potential Dose Rate - inhalation dose in breathing zone after spray application (mg/m^3)
AR = application rate - 1 16 oz can containing 25% ai applied to a 16 x 16 x 8 ft room

BZC = Breathing Zone Concentration (mg/m^3) - measured air concentration from NDETF study adjusted to reflect a likely maximum application rate
BR = Breathing rate for adult or child (m^3/hr) (1.0 m^3/hr adult, 0.8 m^3/hr child)
BW = 70 kg for adult; 15 kg for toddler
ED = Exposure Duration (2 hr/day)
MOE = Inhalation NOAEL/Inhalation Dose (mg/kg/day) MOEs are reported to two significant figures.

Table 17. Estimated Dermal Exposure and Risk from Direct Application of Insect Repellant

Age Group	Applied Dose (mg/kg/day)	Body Weight (kg)	Daily Dose (mg/kg/day)	MOE [1]
Child 6-12 years	3120	30	0.24	210
Child 13-17 years	3380	58	0.12	340
Adult Female	2795	60	0.09	430
Adult Male	3380	70	0.08	410

[1] MOE = Oral NOAEL(mg/kg/day) / Daily Dermal Dose (mg/kg/day)

where:

Daily Dermal Dose = (applied dose (mg) * dermal absorption factor) ÷ body weight (kg)
Applied Dose = Applied Dose of Repellant Product from DEET Survey x % Inert Ingredient in Product (65%)
Dermal Absorption Factor = 0.5%

5.4 Aggregate Exposures

In examining aggregate exposure, FFDCA section 408 directs EPA to consider available information concerning exposures from the pesticide residue in food and all other non-occupational exposures, including drinking water from ground water or surface water and exposure through pesticide use in gardens, lawns, or buildings (residential and other indoor uses). Only dietary, dermal and inhalation routes of exposure have been assessed for this analysis for reasons explained above. Inhalation and oral exposures cannot be aggregated for this assessment because the toxicity endpoints selected for the chronic dietary route of exposure and those selected for the inhalation route are not based on common effects. Inhalation and dermal exposures cannot be aggregated for the same reason. Dietary, dermal, and inhalation exposures can be aggregated because the toxicity endpoints selected for these exposure routes are based on common effects. However, given that highly conservative, screening level assessments do not present exposures of concern for any of these exposure routes, aggregate risks are also not likely to be of concern. HED did not conduct an aggregate assessment of risk from the C_8-C_{20} aliphatic hydrocarbon fluids because co-occurrence of these compounds is not expected.

6.0 Cumulative Exposure

Section 408(b)(2)(D)(v) of the FFDCA requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider "available information" concerning the cumulative effects of a particular pesticide's residues and "other substances that have a common mechanism of toxicity."

Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, EPA has not made a common mechanism of toxicity finding as to C_8-C_{20} aliphatic hydrocarbon fluids and any other substances, and these materials do not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that C_8-C_{20} aliphatic hydrocarbon fluids have a common

mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the policy statements released by EPA's Office of Pesticide Programs concerning common mechanism determinations and procedures for cumulating effects from substances found to have a common mechanism on EPA's website at http://www.epa.gov/fedrgstr/EPA_PEST/2002/January/Day_16/.

7.0 Ecotoxicity and Ecological Risk Characterization

EFED conducted the following assessment of environmental toxicity associated with use of aliphatic hydrocarbon fluids as pesticide inert ingredients (Personal Communication, Sid Abel, EFED, 7/17/06). Based on the Agency's toxicity categories, as a group, these compounds would be classified as moderately toxic to aquatic. Terrestrial organism toxicity, using mammal data as a surrogate for the absence of avian data, indicates that these compounds would be classified as slightly toxic to practically non-toxic. Table 17 provides a summary of limited measured data obtained from the Agency's Ecotoxicity Database (http://cfpub.epa.gov/ecotox/) for the hydrotreated light distillates (CASN: 64742-47-8).

SAR using several analog structures with chain-lengths represented by chemicals in this assessment indicated predictive toxicity for fish to be up to two orders of magnitude lower (more toxic) than measured in the laboratory with the exception of several cycloparaffins whose estimates were within 2 fold of the measured toxicity. There were measured chronic toxicity data available. SAR estimated chronic toxicity was generally an order of magnitude lower than acute toxicity for fish.

Table 18. Summary of Measured Ecotox Data for Hydrotreated Light Distillates (CASN 64742-47-8)		
NALCO D-2303 (contained 10% 2,4,5-T)	Rainbow trout (Oncorhynchus mykiss)	96-h LC50 2.9 mg/L
NALCO-2088	Guppy (Poecilia reticulate)	48-h LC50 8.8 mg/L
NALCO-2088	Zebra danio (Danio rerio)	48-h LC50 7.5 mg/L
NALCO D-2303	Rainbow trout (Oncorhynchus mykiss)	96-h LC50 2.4 mg/L
NALCO D-2303	Bluegill sunfish (Lepomis macrochirus)	96-h LC50 5.9 mg/L
NALCO D-2303	Rainbow trout (Oncorhynchus mykiss)	96-h LC50 2.6 mg/L
NALCO D-2303	Bluegill sunfish (Lepomis macrochirus)	96-h LC50 2.2 mg/L

Based on the available environmental fate and effects data, application of pesticides formulations containing these inerts to terrestrial environments in excess of 3 pound /A may result in exceedance of the Agency's level of concern for endangered species. Based on information provided by ExxonMobil, the maximum application rate for aliphatic hydrocarbon fluids is 2.2 lb/A.

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