Dated: December 18, 1998.

Jack E. Housenger,

Director, Special Review and Reregistration Division, Office of Pesticide Programs.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–849; FRL–6047–7]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–849, must be received on or before January 22, 1999.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
James Tompkins	Rm. 239, CM #2, 703–305–5697, e-mail:tompkins.jimepamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Amelia M. Acierto	Rm. 707A, CM #2, 703–308–8377, e-mail:acrieto.ameliaepamail.epa.gov.	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–849] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–849] and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: December 15, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Monsanto Company

PP 7F4840

EPA has received a pesticide petition (PP 7F4840) from Monsanto Company, 600 13th Street, N.W., Suite 600, Washington, D.C., proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of sulfosulfuron; 1-(4,6-dimethoxypyrimidin-2-yl)-3-(2-ethanesulfonyl-imidazo 1,2-a pyridine-3-yl)sulfonylurea, and its metabolites converted to 2-(ethylsulfonyl)-imidaazol 1,2-a pyridine and calculated as sulfosulfuron in or on the following raw agricultural commodities and animal products:

Commodity	Part per million (ppm)
Wheat.	
Grain	0.02
Straw	0.1
Hay	0.3
Forage	4.0
Animal Products.	
Milk	0.006

Commodity	Part per million (ppm)
Fat (cattle, goats, horses, hogs, sheep).	0.005
Meat (cattle, goats, horses, hogs, sheep).	0.005
Meat by-products (cattle, goats, horses, hogs, sheep).	0.05

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data maybe needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Metabolism of sulfosulfuron in plants is negligible. The nature of the major sulfosulfuron residues in wheat matrices depends primarily on the mode of application with a reliance upon metabolism in the soil.

Postemergence applications result in residues that are mostly made up of parent compound, with small amounts of five to six metabolites that together make up less than 15% of the total radioactive residue (TRR).

Preemergence application result in soil degradation of the parent compound followed by uptake primarily of the imidazopyridine ring-containing metabolites and small amounts of the parent compound. The pyrimidine ring-containing metabolites under these conditions are tightly bound to the soil, resulting in negligible uptake of these residues. Little further metabolism of the imidazopyridine metabolites takes place in the plant. The predominant residues resulting from preemergence applications were sulfonamide (22% TRR) and guanidine (18.3% TRR).

In both cases, translocation of residue to the grain is negligible. The highest residues are observed following postemergence applications and the residues are primarily parent compound.

In rotational crops, residues were low, with the TRR's not exceeding 0.01 ppm in most crops. The most abundant metabolite was sulfonamide, with low levels of a sulfonamide-sugar conjugate and parent compound also observed.

2. *Analytical method*. The primary crop (wheat) residue and the secondary (animal products) residues are analyzed as total residue by hydrolyzing sulfosulfuron and its imadazopyrimidine-containing metabolites under acidic conditions to the common chemophore, ethyl sulfone. Ethyl sulfone is then separated and quantitated by High Performance Liquid Chromatography (HPLC) with fluorescence detection.

3. *Magnitude of residues*. Field residue trials at 25 locations were made in winter and spring wheat as preplant incorporated (PPI), preemergent (PRE) and, postemergent (POST) applications at a target application rate of 0.035 lb a.i./acre. Residues in grain from all modes of application were < 0.008 ppm; residues in the other RACs in PRE and PPI applications did not exceed 0.016 ppm. Residues in forage samples from POST applications taken on the day of and 2-weeks after application showed maximum residues of 3.04 ppm and 0.70 ppm, respectively.

Spring and winter wheat treated with an exaggerated rate of 10x the anticipated use rate resulted in grain residues below the analytical limit of quantitation. Since no quantifiable residue were detected at rates greater than the maximum theoretical concentration (9x for wheat), processing studies were not required.

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with an LD_{50} of >5,000 milligrams/kilogram (mg/kg), EPA Category IV.

i. A rabbit acute dermal study with an LD_{50} of >5,000 mg/kg, EPA Category IV.

ii. A rat inhalation study with an LC_{50} of >3.0 mg/l, the highest concentration generated, EPA Category IV.

iii. A primary eye irritation study in the rabbit showing moderate eye irritation, EPA Category III.

iv. A primary dermal irritation study in the rabbit showing essentially no irritation, EPA Category IV.

A dermal sensitization study in the guinea pig showing no potential for sensitization. Acute and subchronic neurotoxicity studies in rats demonstrating no neurotoxicity potential. Sulfosulfuron has a low order of acute toxicity.

2. *Genotoxicity*—i. An *in vitro Ames/Salmonella* mutagenicity assay in five commonly used strains was negative for mutagenic potential. An *in vitro* CHO/HGPRT Gene Mutation assay was negativefor mutagenicity up to the limit of solubility.

ii. An *in vitro* chromosomal aberration test in cultured mammalian cells demonstrated the induction of chromosomal aberrations only under conditions of prolonged incubation at high dose levels that exceeded the solubility of the test material. The mechanism responsible for this induction and the biological relevance of the effect is not clear. Other, more relevant, chromosomal aberration tests were negative.

iii. An *in vitro* chromosome aberration study in human lymphocytes was negative for chromosomal aberrations.

iv. An *in vivo* bone marrow micronucleus assay in the mouse was negative for chromosomal effects. The weight of evidence demonstrates that sulfosulfuron does not produce significant genotoxic or mutagenic effects.

3. *Reproductive and developmental toxicity*. A developmental study in the rat demonstrated no signs of maternal or developmental toxicity up to the maximum dose level of 1,000 mg/kg/day. The no-observed adverse effect level (NOAEL) was considered to be 1,000 mg/kg/day. A developmental study in the rabbit demonstrated no signs of maternal or developmental toxicity up to the maximum dose level of 1,000 milligram/kilogram/day (mg/kg/day). The NOAEL was considered to be 1,000 mg/kg/day. A 2-generation reproduction study in the rat demonstrated a subchronic toxicity NOAEL of 5,000 ppm based on body weight and food consumption decreases, urinary bladder calculi formation and minor bladder and kidney pathology. There were no effects on reproduction or fertility up to 20,000 ppm, the highest dose tested (HDT). Sulfosulfuron demonstrates no reproductive effects in rats and no teratogenic or developmental effects in rats, and rabbits.

4. *Subchronic toxicity*. A 28 day dermal study in the rat with a NOAEL of at least 1,000 mg/kg/day, HDT. A 90 day feeding study in the rat resulted in only mild body weight/weight gain effects at 20,000 ppm, the HDT. The NOAEL for both males and females was considered to be 6,000 ppm. A 90 day feeding study in the dog demonstrated subchronic toxicity, primarily in the urinary bladder, secondary to urinary crystal formation and, urolithiasis at dose levels of 300 and, 1,000 mg/kg/day in females and, at 1,000 mg/kg/day in males. The NOAEL was considered to be 100 mg/kg/day in females and, 300 mg/kg/day in males. Sulfosulfuron has a low order of subchronic toxicity, related only to the precipitation of test material in the urinary bladder of dogs at high doses.

5. *Chronic toxicity*. A 1 year study in the dog demonstrated toxicity in the urinary bladder secondary to urinary crystal and calculus formation at 500 mg/kg/day in a single male animal. Urinary crystal formation was observed in females at 500 mg/kg/day with no

subsequent pathology. The NOAEL was considered to be 100 mg/kg/day for male and female dogs.

A combined chronic toxicity/oncogenicity study in the rat demonstrated chronic toxicity, primarily in the urinary bladder, in males and females at 5,000 and females at 20,000 mg/kg/day. The NOAEL for chronic toxicity was considered to be 500 ppm or 24.4 mg/kg/day. This is the lowest NOAEL and is used in the calculation of the Reference Dose (RfD).

An 18 month oncogenicity study in the mouse demonstrated chronic toxicity, primarily in the urinary bladder, of male mice at 3,000 and 7,000 ppm. No chronic toxicity was observed in females. The NOAEL for chronic toxicity was considered to be 700 ppm for male mice, and 7,000 ppm for female mice.

Sulfosulfuron demonstrates chronic toxicity related only to the formation of crystals and calculi of the compound in the urinary bladders of mice, rats, and, dogs.

An 18 month oncogenicity study in the mouse demonstrated a small increase in the incidence of benign mesenchymal tumors of the urinary bladder submucosa in male mice with urinary bladder calculi at 7,000 ppm. However, these tumors are reportedly unique to Swiss-derived mice and were considered to be of biological relevance only to the mouse by an Independent Working Group on Mouse Mesenchymal Tumors convened by the International Life Sciences Institute (ILSI).

A combined chronic toxicity/oncogenicity study in the rat (same as above) demonstrated a urinary bladder transitional cell carcinoma and, a urinary bladder transitional cell papilloma in two females at 5,000 mg/kg/day, probably secondary to urinary system calculi formation and, (chronic) irritation.

The low incidences of oncogenicity observed in the oncogenicity studies conducted with sulfosulfuron are either considered to be relevant to the mouse only or a secondary threshold effect related to chronic irritation resulting from bladder stone formation at high doses. Sulfosulfuron is not considered to be a primary oncogen.

Using the Guidelines for Carcinogenic Risk Assessment published September 24, 1986, Monsanto believes that the EPA would classify sulfosulfuron as a Group C carcinogen, without quantitative risk assessment, i.e., using the margin of exposure (MOE) approach for risk assessment. Under the proposed guidelines published April 10, 1996, however, Monsanto believes that sulfosulfuron should be included in the ''Not Likely Human Carcinogen'' category based upon mechanistic considerations. To quote the 1996 EPA guideline document discussing a similar effect in a rat study.

A major uncertainty is whether the profound effects of (substance 5) may be unique to the rat. Even if (substance 5) produced stones in humans, there is only limited evidence that humans with bladder stones develop cancer. Most often human bladder stones are either passed in the urine or lead to symptoms resulting in their removal.

In either case, a MOE assessment or RfD approach would be utilized. Since the chronic NOAEL for male rats is lower than the oncogenic NOAEL for female rats (24 mg/kg/day vs 30 mg/kg/day), the male rat chronic NOAEL was used with a 100 fold safety factor for a RfD of 0.24 mg/kg/day, for the quantitation of human risk.

6. *Animal metabolism*. An animal metabolism study was conducted in the rat using sulfosulfuron radio labeled in both the pyrimidine and iminodazopyridine rings to detect possible cleavage of the sulfonylurea bond. Following oral dosing of sulfosulfuron, absorption was found to be greater at low doses (>90%) than at the higher doses (40%). Sulfosulfuron was readily excreted, mostly unchanged, with urinary excretion the major route of elimination at low doses and fecal excretion the major route at high doses. Greater than 90% of the dose was excreted 3-days after administration. Expiration as carbon dioxide or volatiles was not a significant route of elimination. Metabolism of sulfosulfuron in the rat occurred to only a limited extent with demethylation and pyrimidinering hydroxylation as the major metabolic routes, yielding desmethyl-sulfosulfuron and 5-hydroxy-sulfosulfuron as the major metabolites. There was no evidence of bio-retention of sulfosulfuron or its metabolites; tissue and blood levels were negligible, with no individual tissue showing levels exceeding 0.2% of the doses.

7. *Metabolite toxicology*. Dietary residues are comprised almost entirely of parent sulfosulfuron and the imidazopyridine-containing metabolites sulfonamide and guanidine. Specific toxicology data is not available on these metabolites, but the structures do not suggest any specific toxicologic concern and the level of dietary exposure is low. These metabolites are not considered to present a significant toxicological risk.

8. *Endocrine disruption*. There was no evidence that exposure to sulfosulfuron had any effect on reproduction, fertility or mating indices, development or maturation of embryos, or development, growth and survival of offspring in the battery of short-term, chronic, reproductive and, developmental mammalian, avian and aquatic studies conducted. There were no gross or microscopic pathologic effects in endocrine organs or endocrine-sensitive tissues, or in any reproductive organs, tissues or endpoints that were considered related to exposure to sulfosulfuron. With no evidence of bioaccumulation and low environmental concentrations, there is negligible risk of endocrine disruption in humans or wildlife

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Estimates of dietary exposure to residues of sulfosulfuron utilized the proposed tolerance-level residues for wheat grain (0.01 ppm) and for the following animal products: milk (0.004 ppm), fat (0.004 ppm), meat (0.004 ppm), and meat by-products (0.1 ppm, including kidney, and liver). 100% market share was assumed as well as the assumption that no loss of residue would occur due to processing and cooking. A RfD of 0.24 mg/kg/day was assumed based on the low NOAEL from the chronic/oncogenicity study in rats (24 mg/kg/day) with a safety factor of 100. Since the present label lists only wheat or fallow as approved rotations, no residues were entered for rotational crops. Using these conservative assumptions, dietary residues of sulfosulfuron contribute only 0.000149 mg/kg/day (0.006% of the RfD) for children 1-6 years, the most sensitive sub-population. For the U.S. population as a whole, the exposure was only 0.000048 mg/kg/day (0.02% of the RfD).

ii. *Drinking water*. Given the low use rates, rapid soil degradation, strong soil binding characteristics and low soil mobility of sulfosulfuron, the risk of significant ground and surface water contamination and exposure via drinking water is considered to be negligible. Assuming that 10% of the RfD is allocated to drinking water exposure (0.024 mg/kg/day), and the average, 70 kg human consumes 2 liters of water per day, a Maximum Allowable Concentration (MAC) value for drinking water of 0.84 mg/l is proposed for sulfosulfuron.

iii. *Non-dietary exposure*. Sulfosulfuron is proposed for a variety of non-crop uses including roadsides, fence rows,industrial sites, parks, apartment complexes, schools and, other public areas. Exposure assessments have been made for mixer/loaders and applicators in these situations (occupational exposure) and, the cumulative (amortized) daily

exposure from both these activities has been estimated to be less than 0.5 mg/kg/day, or approximately 0.2% of the RfD. The non-occupational exposure in these locations to the casual passer-by would be expected to be orders of magnitudeless. The exposure in either instance does not present a significant exposure risk.

D. Cumulative Effects

Sulfosulfuron falls into the common category of sulfonylurea SU herbicides; however, there is no information to suggest that any of the SU s have a common mechanism of mammalian toxicity or even produce similar effects. It is not appropriate to combine exposures in this case, and Monsanto is considering only the potential risk of sulfosulfuron in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. As presented above, the exposure of the U.S. General population to sulfosulfuron is low, and the risks, based on comparisons to the reference dose, are negligible. Margins of safety are expected to be considerable. Monsanto concludes that there is a reasonable certainty that no harm will result to the U.S. population from aggregate exposure to sulfosulfuron residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of sulfosulfuron, Monsanto considered data from developmental toxicity studies in the rat, and rabbit and a 2-generation reproduction study in rats. No developmental or reproductive effects were observed up to the HDT in each of the three studies. The NOAELs were 1,000 mg/kg/day, 1,000 mg/kg/day and 20,000 ppm, respectively. Using the same conservative assumptions that were made previously for the dietary exposure analysis for the U.S. general population, the percent of the RfD utilized by pre-adult sub-populations are: all infants-0.03%;, nursing infants-0.005%;, and non-nursing infants-0.04%; children, 1-6 years-0.06%; children, 7-12 years-0.04%. Monsanto concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to sulfosulfuron residues.

F. International Tolerances

There are currently no international (Codex) tolerances established for sulfosulfuron.

Sulfosulfuron is currently registered on wheat in Ireland, Switzerland, Poland, the Czech Republic, Slovakia and, South Africa. There are no harmonized MRL's at the European Union level at present. Petitions for tolerances for sulfosulfuron in/on wheat have been submitted in Canada, Australia and, in other countries in the European Union.

2. Whitmire Micro-Gen Research Laboratories, Inc.

PP 5E4442

EPA has received a pesticide petition (PP 5E4442) from Whitmire Micro-Gen Research Laboratories, Inc., 3568 Tree Court Industrial Bvd., St. Louis, MO 63122-6682, proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for Dibasic esters (DBE). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

DBE is a colorless liquid that consists of a mixture of dimethyl glutarate (55-75%), dimethyl adipate (10-25%), and dimethyl succinate (19-26%). The identity and properties of each component of DBE is summarized in the table below.

DBE Component	CAS	Formula	MW	Density
Dimethyl succinate	106-65-0	$CH_3OOC(CH_2)_2COOCH_3$	146.14	1.12
Dimethyl glutarate	1119-40-0	$CH_3OOC(CH_2)_3COOCH_3$	160.17	1.09
Dimethyl adipate	627-93-0	$CH_3OOC(CH_2)_4COOCH_3$	174.20	1.06

Analytical method. DBE vapors may be detected by gas chromatography using a flame ionization detector, for which a detection limit of 0.7 µg/L has been reported (Morris *et al.* 1991). In aqueous media, DBE may be detected by high pressure liquid chromatography using a diode ray detector, for which no detection limit was reported (Bogdanffy *et al.* 1991).

B. Toxicological Profile

1. *Acute toxicity*. Acute (24 hours) dermal contact with DBE produced mild to severe erythema and mild edema in rabbits exposed to undiluted DBE (Sarver, 1989). Fourteen day dietary exposure to large concentrations of DBE in feed (10,000, 20,000, or 50,000 ppm) did not produce any gross or microscopic pathological changes in rats (Henry, 1981). Body weight gain was slightly reduced in a dose-dependent manner at the end of the exposure period. This study identified a no-observed adverse effect level (NOAEL) of 10,000 ppm (842 milligrams/kilogram/day (mg/kg-day)). Similarly, body weight gains were significantly reduced in rats exposed via inhalation to concentrations of 0.4 and 1.0 milligram/liter (mg/L) DBE for 6 hours/day, 5 days/week for 2 weeks (Alvarez, 1988). In both studies, however, decreases in body weight gain appear to be attributable to a dose-dependent decreases in feed consumption, rather than a pathological change caused by treatment.

2. *Genotoxicity*. DBE was not mutagenic in a *Salmonella typhimurium* assay in the presence or absence of a rat liver activation system (Koops, 1977; Arce, 1988). A significant increase in chromosomal aberrations was observed *in vitro* in human lymphocytes when metabolically activated (using a rat liver S-9 fraction), but not in the absence of metabolic activation (Vlachos, 1987). However, in an *in vivo* mouse bone marrow micronucleus assay, no significant increase in micronucleated cells were observed (Rickard, 1987).

3. *Reproductive and developmental toxicity*. No effects on fetal survival, fetal weight, litter size, implantation, or the incidence of terata were observed in rats exposed via inhalation to concentrations 0.16, 0.4, or 1.0 mg/L DBE on days 7-16 of gestation (Alvarez, 1988). In addition, no treatment-related effects were observed for various reproduction indices (male fertility, female fertility, born alive, viability, gestation, and lactation) in rats exposed via inhalation to 0.16, 0.4, or 1.0 mg/L DBE for 14 weeks prior to mating, and continuing through breeding (15 days), gestation (21 days), and lactation (21

days). Pup weights were significantly reduced at concentrations of 1.0 mg/L DBE, however, this appears to be attributable to decreased food intake and body weight gain in maternal animals, which were significantly depressed at concentrations of 0.4 mg/L and higher (Kelly, 1988).

4. *Subchronic toxicity*. In rats exposed via inhalation to 0.02, 0.08, or 0.40 mg/L DBE for 6 hours/day, 5 days/week, for 14 weeks, the only histopathological change of significance included mild squamous metaplasiain the olfactory epithelium (Kelly, 1987). Slight changes in liver weight, body weight, blood calcium, and sodium levels were also reported, however, these were considered to be of minimal biologic significance. A no effect concentration was not identified for nasal effects. However, for systemic effects, the highest concentration tested (0.4 mg/L) was considered to be a NOAEL.

5. *Chronic toxicity*. In rats exposed via inhalation to 0.16, 0.4, or 1.0 mg/L DBE for 22 weeks, the only histopathological change of significance included squamous metaplasia in the olfactory epithelium (Kelly, 1988). The incidence and severity of the nasal lesions was greater in this study in comparison to the 14 week study discussed above. A no effect concentration was not identified for nasal effects.

6. *Animal metabolism*. The compounds that comprise DBE are derivatives of three naturally occurring dicarboxylic acids (adipic, glutaric, and succinic acids). Specifically, DBE consists of dimethyl esters of these three acids. Due to the presence of carboxylesterases and other diesterases in mammalian tissues, these dimethyl esters are rapidly cleaved in the body to form their corresponding dicarboxylic acids: adipic, glutaric, and succinic acids.

7. *Metabolite toxicology*. By the oral route, the toxicity of DBE metabolites is low. The principle metabolites of DBE are naturally occurring dicarboxylic acids: succinic, glutaric, and adipic acids. Adipic, and succinic acids are classified as Generally Recognized As Safe (GRAS) by the U.S. FDA for substances directly added to human food (21 CFR 184.1009 and 21 CFR 184.1091 respectively). Although glutaric acidis not classified as GRAS, its relative safety can be inferred since its carbon chain length (5) is intermediate of adipic (6) and succinic (4) acids. The dicarboxylic acids are substrates for glycolytic and gluconeogenic reactions in the cell, and as such, the components of DBE possess nutritional value (Ladriere *et al*. 1996).

By the inhalation route, the metabolites of DBE are irritants to the nasal mucosa, and are likely responsible for the metaplasia of the olfactory epithelia observed in exposed rats. *In vitro* studies indicate that inhibition of nasal carboxylase activity reduces the toxicity in rat nasal explants (Trela and Bogdanffy, 1991). In the rat, carboxylesterases appear to be preferentially localized in cells of the Bowman's gland and sustentacular epithelial cells which are immediately adjacent to olfactory nerve cells (Olson *et al*. 1993).

8. *Endocrine disruption*. Mono- and dimethyl esters of succinic acid are capable of stimulating insulin release in rats (Vicent et al. 1994;, Ladriere et al. 1996). However, rather than evidence of endocrine disruption, this observation is likely attributable to the nutritional value of DBE.

C. Aggregate Exposure

1. *Dietary exposure*. Dietary exposure due to use of DBE as an antifreeze agent is believed to be minimal, as is discussed for food and drinking water below.

2. *Food*. DBE is not intended to be directly applied to foods. Rather, the use of DBE in pesticide formulations for food handling areas will be limited to sprays and aerosols for crack/crevice applications. Any incidental dietary exposure to DBE from such uses will be minimal in comparison to the currently permitted use of DBE component, dimethyl succinate, as a food additive in beverages, ice cream, candy, and baked goods (21 CFR 172.515). Furthermore, the levels of dimethyl esters present in food as a result of DBE application in food areas are likely to be far less, on a molar equivalent basis, than the levels of naturally occurring dicarboxylic acids present in foods.

3. *Drinking water*. Because DBE-containing pesticide formulations are not applied to agricultural crops, its migration to groundwater aquifers or to surface water bodies that may serve as suitable sources of drinking water is not anticipated.

4. *Non-dietary exposure*. The greatest potential for exposure to DBE is to pesticide applicators, who may be exposed via inhalation or dermal routes. USEPA's Pilot Inter disciplinary Risk Assessment Team (PIRAT,1997) evaluated potential exposures to workers using a handwand applicator or a backpack applicator.

For the handwand applicator scenario, assuming a unit exposure of 29.178 milligrams/pound (mg/lb) handled for the dermal pathway and a unit exposure of 1.063 mg/lb handled for the inhalation pathway, average daily doses of 0.03 and 0.001 mg/kg-day were calculated for dermal and inhalation exposures, respectively. In their calculations, USEPA conservatively assumed 100% absorption via both routes, a 70 kilogram/body/weight (kg/bwt), an application rate of 0.08 lbs DBE/day for product containing 4.2% (w/w) DBE yielding a finish spray containing 0.065% DBE.

For the backpack applicator scenario, assuming a unit exposure of 482.581 mg/lb handled for the dermal pathway and a unit exposure of 0.329 mg/lb handled for the inhalation pathway, average daily doses of 1.0 and 0.007 mg/kg/day were calculated for dermal and inhalation exposures, respectively. In their calculations, USEPA conservatively assumed 100% absorption via both routes, a 70 kilogram/body/weight, an application rate of 0.14 lbs DBE/day for product containing 4.2% (w/w) DBE yielding a finish spray containing no more than 1% DBE.

D. Cumulative Effects

Since exposures to DBE from food and drinking water are believed to be minimal, the potential for cumulative exposures (i.e., summed across multiple routes of exposure) exceeding those estimated for pesticide applicators is very small. Furthermore, because the components of DBE are readily metabolized to polar, water-soluble metabolite, DBE is not expected to be persistent in biological tissues. Because DBE is irritating to the skin and nasal passages, any exposures are expected to be self-limiting. For these reasons, the potential for cumulative effects from exposure to DBE is low.

E. Safety Determination

1. *U.S. population*. Potential dietary exposures to DBE are not likely to pose a significant risk to the general U.S. population. The components of DBE are dimethyl esters of three naturally occurring dicarboxylic acids (adipate, succinate, and glutarate), two of which are currently classified as GRAS by the U.S. FDA for direct addition to human foods. It should be noted that the presence of methyl groups does not increase the toxicity of DBE. To the contrary, methylation is one of the metabolic pathways by which the body attempts to detoxify xenobiotics (Hodgson and Levi, 1987). As such, dimethyl succinate, dimethyl glutarate, and dimethyl adipateare likely to be less toxic than succinate, glutarate, and adipate, respectively. In support of this statement, Trela and Bogdanffy (1991)

reported that succinate, glutarate, and adipate produced concentration-dependent increases in cytotoxicity in a rat nasal explant system. The cytotoxicity of DBE in the same system, however, was greatly diminished by a carboxylesterase inhibitor which effectively blocks the conversion of DBE to the dicarboxylic acids.

The potential hazards posed by DBE to pesticide applicators exposed via inhalation and dermal routes are low. For the handwand applicator, the average daily dermal and inhalation doses of 0.03 mg/kg/day, and 0.001 mg/kg/day, respectively, are well below exposures which are believed to be without risk of deleterious effects (8.42 mg/kg/day for dermal exposures, and 0.38 mg/kg/day for inhalation exposures). Specifically, USEPA conservative assumptions for a worker applying a DBE-containing (4.2% w/w) product with a handwand maintain margin of exposures (MOEs) of 280 and 380 for dermal, and inhalation exposures, respectively. Based on these MOEs workers applying a hypothetical formulation containing 100% DBE would still be adequately protected. For the backpack applicator, the average dermal and inhalation doses of 1 and 0.007 mg/kg/day, are also below exposures which are believed to be without risk of deleterious effects. USEPA's conservative assumptions for a backpack applicator maintain a MOE of 8, and 54 for dermal and inhalation exposures, respectively. Based on these MOEs, workers applying a hypothetical formulation containing 33% DBE would still be adequately protected. As this percentage far exceeds the levels anticipated for DBE-containing products, no concentration limit need be specified for DBE.

2. *Infants and children*. There is no information available which suggests that infants and children are more highly exposed or are more susceptible to the effects of DBE. The lack of any significant toxicity in reproductive/developmental studies on DBE suggests that growing organisms are not at increased risk. Since potential dietary exposures to infants and children are minimal based on anticipated use patterns, and since the toxicity of DBE by the oral route is very low, it is unlikely that these types exposures will result in any deleterious effects. Direct exposures to infants and children via the inhalation and dermal routes are not anticipated for the intended use of DBE.

F. International Tolerances

Whitmire is not aware of any tolerances for DBE outside of the United States.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6208–7]

Proposed Administrative Agreement for Collection of CERCLA Response and Oversight Costs

AGENCY: U.S. Environmental Protection Agency (U.S. ERA).

ACTION: Proposed CERCLA 122(h) Administrative Agreement.

SUMMARY: U.S. EPA is proposing to execute an Administrative Agreement (Agreement) under Section 122 of CERCLA for collection of a percentage of response and oversight costs at the West Roosevelt Drum Superfund Site. Respondent has agreed to pay $17,000 out of total response and oversight costs of Approximately $23,120, and in return will receive a covenant not to sue and contribution protection from U.S. EPA. U.S. EPA today is proposing to execute this Agreement because it achieves collection of a high percentage of total Site costs. (The Respondent at the Site previously performed a Superfund removal under a CERCLA Section 106 Unilateral Order, at a cost of approximately $50,000. Thus, the overall value of the clean up and settlement to U.S. EPA is $67,000 out of an approximate total of $73,120. This is 91% of total Site costs).

DATES: Comments on this proposed settlement must be received by January 22, 1999.

ADDRESSES: Copies of the proposed settlement are available at the following address for review: (It is recommended that you telephone Mr. Derrick Kimbrough at (312) 886–9789 before visiting the Region V Office). Mr. Derrick Kimbrough, OPA (P19–J), Coordinator, Office of Public Affairs, U.S. Environmental Protection Agency, Region V, 77 W. Jackson Boulevard (P–19J), Chicago, Illinois 60604, (312) 886–9789.

Comments on this proposed settlement should be addressed to: (Please submit an original and three copies, if possible) Mr. Derrick Kimbrough, Coordinator, Office of Public Affairs, U.S. Environmental Protection Agency, Region V, 77 W. Jackson Boulevard (P–19J), Chicago, Illinois 60604, (312) 886–9789.

FOR FURTHER INFORMATION CONTACT: Mr. Derrick Kimbrough, Office of Public Affairs, at (312) 886–9789.

SUPPLEMENTARY INFORMATION: The West Roosevelt Drum Superfund Site is located at 5728–32 W. Roosevelt Road, Chicago, Illinois (Cook County). In response to the release or threatened release of hazardous substances at or pursuant to Section 104 of CERCLA, 42 U.S.C. 9604. A January 27, 1995, EPA site assessment found the Site Buildings unsecured, and containing approximately 300 drums and other materials. On February 24, 1995, EPA issued a General Notice of Potential Liability to the Settling Party. The Settling party performed the clean up pursuant to the UAO. The removal was completed on August 8, 1995, and an EPA Completion of Work letter was issued by the EPA On-Scene Coordinator (OSC) on April 2, 1998.

Subsequent negotiations with the Settling party extended the Statute of Limitations for EPA to act upon or settle this matter until March 16, 1999. EPA has accrued Past Response Costs (including oversight costs) in connection with the Site of $23,120.

A 30-day period, beginning on the date of publication, is open pursuant to section 122(i) of CERCLA for comments on the proposed Administrative Agreement.

Comments should be sent to Mr. Derrick Kimbrough of the Office of Public Affairs (P–19J), U.S. Environmental Protection Agency, Region V, 77 W. Jackson Boulevard, Chicago, Illinois 60604.

Thomas Turner,

Assistant Regional Counsel,

United States Environmental Protection Agency.

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ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–51919; FRL–6051–5]

Certain Chemicals; Premanufacture Notices

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: Section 5 of the Toxic Substances Control Act (TSCA) requires any person who intends to manufacture or import a new chemical to notify EPA and comply with the statutory provisions pertaining to the manufacture or import of substances not on the TSCA Inventory. Section 5 of