current toxicological data requirements, the database relative to pre- and post-natal effects in children is complete. Further, in the developmental toxicity study in the rabbit and the 2-generation reproduction study in the rat, the NOEL's are already an additional 30X and an average (male/female) of 9X, respectively, above the NOEL on which the RfD was established (5.0 mg/kg/day from a one-year feeding study in dogs). Based on all the above information, Monsanto concludes that an additional uncertainty factor is not warranted and that the RfD of 0.05 mg/kg/day is appropriate for assessing risk to infants and children.

Using the conservative dietary exposure assumptions described above, EPA has concluded that the percent of the RfD that will be utilized by aggregate exposure to residues of clofencet by children aged <1 (nursing) to age 12, ranges from 10.5 percent for children 7 to 12 years old up to 22.7 percent for non-nursing infants (<1 year old). Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Monsanto concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to clofencet residues.

8. *Estrogenic effects.* No specific tests have been conducted with clofencet to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occuring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity tests, i.e., teratology and multi-generation reproduction studies, which would suggest that clofencet produces these kinds of effects.

9. *Chemical residue.* The metabolism of clofencet in plants and animals is adequately understood for the purposes of these tolerances. There are no Codex maximum residues levels established for residues of clofencet on wheat or indicated rotational crops. There is a practical analytical method for detecting and measuring levels of clofencet in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. EPA will provide information on this method to the Food and Drug Administration (FDA). The method is available to anyone who is interested in pesticide residue enforcement from: By mail: Calvin Furlow, Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Crystal Mall #2, Rm. 1128, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703) 305-5805.

Residues of clofencet have been found to concentrate slightly (<2×) in wheat shorts and bran, and in soybean hulls and meal. The EPA examined all relevant data and after consideration of the restricted use of the chemical for seed production only, the limited opportunity for this seed to enter commerce as grain and the dilution factors involved in making all of the above processed fractions (with the exception of wheat bran) ''ready to eat'', the EPA determined that no additional tolerances were necessary to cover these processed fractions. All of the proposed tolerance levels are adequate to cover residues likely to be present from the proposed use of clofencet. Therefore, no special processing to reduce the residues will be necessary

10. *Environmental fate.* Laboratory studies indicate that clofencet has the potential to persist in soil and be mobile. However, the results of field dissipation studies indicate that downward movement of clofencet is limited. In addition, the limited use of clofencet for hybrid wheat seed production only, the current practice of never using the same seed production field in two consecutive years and label mitigation measures agreed upon by Monsanto and the EPA, will further reduce the likelihood of clofencet appearing in ground or surface water.

II. Administrative Matters

Interested persons are invited to submit written comments on this notice of filing. Comments must bear a notation indicating the document control number, [PF–678]. All written comments filed in response to this petition will be available, in the Public Response and Program Resources Branch, at the address given above from 8:30 a.m. to 4:00 p.m., Monday through Friday, except legal holidays.

A record has been established for this notice of filing under docket number [PF–678] (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 4:00 p.m., Monday through Friday, excluding legal holidays. The public record is located in Room 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments can be sent directly to EPA at:

opp=Docket@epamail.epa.gov

Electronic comments must be submitted as as ASCII file avoiding the use of special characters and any form of encryption.

The official record for this filing of notice, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer all comments received electronically into printed, paper form as they are received and will place the paper copies in the official record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the address in ''ADDRESSES'' at the beginning of this document.

List of Subjects

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: December 4, 1996.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 96–31555 Filed 12–11–96; 8:45 am]

BILLING CODE 6560–50–F

[PF–677; FRL–5576–1]

Valent U.S.A. Corporation; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of filing.

SUMMARY: This notice is a summary of a pesticide petition proposing to renew a time-limited tolerance for residues of the herbicide lactofen, 1-(carboethoxy)ethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, and its associated metabolites containing the diphenyl ether linkage on the raw agricultural commodity (RAC) cottonseed at 0.05 part per million (ppm). This summary was prepared by the petitioner, Valent U.S.A. Corporation (Valent).

DATES: Comments, identified by the docket number [PF–677], must be received on or before, January 13, 1997.

ADDRESSES: By mail, submit written comments to Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW.,

Washington, DC 20460. In person, bring comments to Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA. Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: 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 docket number [PF–677]. Electronic comments on this notice may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found below in this document.

Information submitted as comments 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: Joanne I. Miller, Product Manager (PM 23), Rm. 237, CM#2, 1921 Jefferson Davis Highway, Arlington, VA 22202; (703) 305–6224. e-mail: miller.joanne@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the Federal Register of June 14, 1990, (55 FR 24084), EPA established a time-limited tolerance under section 408 of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 346a) for residues of the herbicide lactofen, 1-(carboethoxy)ethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, and its associated metabolites containing the diphenyl ether linkage in or on the raw agricultural commodity cottonseed at 0.05 ppm. The time-limited tolerance expires on December 31, 1996. This tolerance was requested in pesticide petition (PP) 9F3798 by Valent U.S.A. Corporation, 1333 N. California Blvd., Walnut Creek, CA 94596, and establishes the maximum permissible level for residues of the herbicide in or on this RAC. The tolerance was issued as a time-limited tolerance because EPA required additional residue chemistry data. The petitioner proposes to renew the time-limited tolerance for a one–year period. Valent requested this tolerance extension pursuant to the Federal Food, Drug, and Cosmetic Act (FFDCA) as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170). The request addresses the requirements of the new FFDCA Section 408(d)(2). The time-limited tolerance would expire on December 31, 1997. The proposed analytical method is RM–28D, a gas chromatography method.

Pursuant to the Section 408(d)(2)(A)(i) of the FFDCA, as amended, Valent has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by Valent and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioner's and not necessarily EPA's and to remove certain extraneous material.

I. Valent Petition Summary

A. Residue Chemistry

1. *Plant metabolism*. Lactofen is used to control broad leaved weeds in crops by preemergent (soybean, peanut), or early postemergent (soybean, cotton, peanut) applications with extended pre-harvest intervals (45 to 70 days). Plant metabolism protocols (soybean, peanut, and tomato) have been designed to mimic the field applications with respect to timing, but have been applied at rates exceeding normal application to facilitate identification of metabolites.

The lactofen molecule is rapidly degraded in the environment and in plants. Therefore, the consistent result of all plant metabolism studies using lactofen has been: radiocarbon is distributed throughout the plant; much of the radiocarbon is irreversibly bound; little radiocarbon is found in the RAC (seeds, fruit); and very little terminal residue is identified as finite metabolites due to extensive degradation.

To demonstrate plant metabolic pathways and to prove the analytical methods can isolate, recover, and identify lactofen and its metabolites, plant samples were analyzed soon after application and well before normal harvest. It is from these early samples that the definition of the residue has been obtained. The regulated residue is defined as parent and four metabolites containing the diphenyl ether moiety. Parent lactofen is identified as PPG–844 and the metabolites are identified as PPG–847, PPG–947, PPG–1576, and PPG–2597. The regulated residue as defined has never been found in a RAC sample either from plant metabolism or from crop field studies. At maximum treatment rates in crop field trials, only one soybean seed sample was found to have residues of lactofen greater than the limit of detection, but less than the limit of quantitation. Even at exaggerated rates in metabolism or crop residue studies, residues are rarely above the limit of detection for any analyte. In addition, more than analyte has never been found above the limit of detection in a single RAC sample from crop field trials. See further discussion in the Magnitude of Residue section.

2. *Analytical method*. Adequate analytical methodology (gas chromatography) is available for detecting and measuring levels of lactofen and its metabolites in or on food with a limit of detection that allows monitoring of food with residues at or above the level set in the time-limited tolerance on cotton. The current method, RM–28D, has been validated by an independent laboratory on both cottonseed and peanuts and is still undergoing PMV trials at the EPA. In general, the analytical method has a limit of detection of 0.005 ppm and limit of quantitation of 0.01 ppm in crops.

3. *Magnitude of residues*. Lactofen is the active ingredient in COBRA Herbicide (EPA Reg. No. 59639–34) and STELLAR Herbicide (EPA Reg. No. 59639–92). Tolerances have been established for lactofen on cotton, soybeans, and snap beans. A tolerance is also pending for peanuts. Lactofen is a broad-spectrum broadleaf herbicide with the following use patterns:

Soybeans: pre-emergence and/or post-emergence, broadcast application with a PHI of 45 days.

Cotton: post-emergence, directed spray application with a PHI of 70 days.

Snap Beans: pre-emergence, soil application with a PHI of 55 days.

Peanuts: (pending) pre-emergence and/or post-emergence, broadcast application with a PHI of 70 days.

Due to relatively long pre-harvest intervals and extensive metabolism by plants, lactofen residues are rarely found in treated raw agricultural or processed commodities. Consequently, tolerances have been established based on the limit of quantitation for lactofen and its metabolites containing the diphenyl ether linkage. To date, tolerances have been established at 0.05 ppm based on a limit of quantitation of 0.01 ppm for lactofen and four plant metabolites.

B. Toxicological Profile

1. *Acute toxicity*. Lactofen (PPG–844) Technical has been placed in EPA Toxicity Category III for dermal toxicity and Category IV for the other four acute toxicity tests. It has also been found to be a weak skin sensitizer. Teratology and reproduction studies indicate that adverse effects, including embryotoxicity, occur only at doses that are also maternally toxic. This chemical therefore represents a minimal acute toxicity risk.

2. *Genotoxicity*. Lactofen Technical has been tested and produced negative results in a number of genotoxicity tests including unscheduled DNA synthesis in rat hepatocytes, DNA covalent binding in mouse liver, chromosomal aberration in CHO cells, and an Ames assay. In a second Ames assay lactofen was positive without metabolic activation at 5000 ug/plate and above. Overall lactofen is not considered a genetic hazard.

3. *Reproductive and developmental toxicity*. Pregnant rats were administered oral doses of 0, 15, 50 and 150 mg/kg/day Lactofen Technical on days 6–19 of gestation. Maternal toxicity (death, abortion and reduced body weight gain) was observed at 150 mg/kg/day. Developmental toxicity (reduced fetal weight, slightly reduced ossification, bent ribs and bent limb bones) was also observed at 150 mg/kg/day. The NOEL for this study was 50 mg/kg/day.

Two developmental toxicity studies were conducted in rabbits with Lactofen Technical. In the first study, pregnant rabbits were administered oral doses of 0, 5, 15 or 50 mg/kg/day Lactofen Technical on days 6–18 of gestation. Maternal toxicity (clinical signs and reduced weight gain) and developmental effects (increased embryonic death, decreased litter size and increased post-implantation loss) were reported at 15 and 50 mg/kg, however EPA concluded that the data were insufficient to establish a clear NOEL. The study was classified as core-supplementary. In the second rabbit developmental toxicity study, pregnant rabbits were exposed to 0, 1, 4 or 20 mg/kg/day oral doses on days 6–18 of gestation. Maternal toxicity (reduced food consumption) was observed at 20 mg/kg/day, while no developmental effects were observed at any dose. Therefore, the maternal NOEL was 4 mg/kg/day and the developmental NOEL was greater than 20 mg/kg/day.

Groups of male and female rats were administered 0, 50, 500 or 2000 ppm of Lactofen Technical for two generations. Adult systemic toxicity (mortality, reduced body weight, increased liver and spleen weight, decreased kidney weight and histological changes in the liver and testes) was observed at levels of 500 ppm and greater. Reproductive toxicity (lower pup survival rates, reduced pup weight and pup organ weight effects) was also observed at levels of 500 ppm and greater. The NOEL for both systemic and reproductive toxicity was 50 ppm (2.5 mg/kg).

Since lactofen causes teratogenic and reproductive effects only at levels which also produce systemic toxicity it is not considered a reproductive hazard.

4. *Subchronic toxicity*. In a 4–week oral toxicity study of Lactofen Technical in rats, a slight increase in spleen weight was the basis for a LOEL of 200 ppm (lowest dose tested). At doses of 1000 ppm or higher the following findings were reported: clinical signs of toxicity; decreased RBC, hemoglobin, hematocrit, and increased WBC; increased relative liver and spleen weights; and necrosis and pigmentation of hepatocytes. At 10,000 ppm severe toxic signs were observed by day 7 and all animals were dead or killed in extremis by day 11. Hypocellularity of the spleen, thymus and bone marrow was also observed in animals exposed to 10,000 ppm.

Histopathological changes in the liver and significant changes in clinical chemistry associated with the liver were observed in rats exposed to 1000 ppm Lactofen Technical in the diet for 90 days. Decreased RBC, hemoglobin and hematocrit values were also observed at 1000 ppm. The NOEL in this study was 200 ppm.

In a 90–day study in mice, the LOEL for Lactofen Technical was 200 ppm based on: increased WBC; decreased hematocrit, hemoglobin and RBC; increased alkaline phosphatase, SGOT, SGPT, cholesterol and total serum protein levels; increased weights or enlargement of the spleen, liver, adrenals, heart and kidney; histopathological changes of the liver, kidney, thymus, spleen, ovaries and testes observed at 1000 ppm.

Butler et al (1988) studied the effects of lactofen on peroxisome proliferation in mice exposed for seven weeks to dietary concentrations of 2, 10, 50 and 250 ppm. Liver-weight to body-weight ratio, liver catalase, liver acyl-CoA oxidase, liver cell cytoplasmic eosinophilia, nuclear and cellular size, and peroxisomal staining were increased by the tumorigenic dose of lactofen, i.e. 250 ppm. Lower doses of lactofen had little to no effect on these parameters. Thus, this study indicates that lactofen induces peroxisome proliferation and further, that 50 ppm, a dose which is not tumorigenic, would be considered a threshold dose for peroxisome proliferation produced by lactofen.

As noted in the study by Butler et al (1989), the NOEL for peroxisome proliferation in mice following a seven week exposure period is 50 ppm (7 mg/kg/day) and the LOEL is 250 ppm (36 mg/kg/day). A subchronic study conducted in chimpanzees (Couch and Erickson, 1986), indicated no effect on clinical chemistry or histological endpoints that would suggest liver toxicity or peroxisome proliferation at doses up to 75 mg/kg/day administered for 93 days. Therefore, Valent believes that 75 mg/kg/day is a clear NOEL for peroxisome proliferation observed in a species closely related to man.

5. *Chronic toxicity*. In an 18–month oncogenicity study in mice at doses of 10, 50 and 250 ppm Lactofen Technical, an increase in liver adenomas and carcinomas, cataracts and liver pigmentation was observed at 250 ppm. The lowest dose, 10 ppm, was the LOEL based on increased liver weight and hepatocytomegaly.

In a 2-year chronic feeding/oncogenicity study of Lactofen Technical in rats at doses of 500, 1000 or 2000 ppm in the diet, an increase in liver neoplastic nodules and foci of cellular alteration was observed in both sexes at 2000 ppm. The NOEL for systemic toxicity is 500 ppm based on kidney and liver pigmentation.

In a 1–year study in dogs exposed to 40, 200, or 1000(wk1–17)/3000 ppm(wk 18–52) ppm of Lactofen Technical, the NOEL was determined to be 200 ppm based on renal dysfunction and decreased RBC, hemoglobin hematocrit and cholesterol observed at 1000/3000 ppm.

Lactofen (PPG–844) Technical causes adverse health effects when administered to animals for extended periods of time. The effects include proliferative changes in the liver, spleen, and kidney; hematological changes; and blood biochemistry changes. Based on the Lowest Effect Level (LEL) of 1.5 mg/kg/day in the 18–month mouse feeding study and an uncertainty factor of 1000, a reference dose (RfD) of 0.002 mg/kg/day has been established for lactofen. An uncertainty factor of 1000 was used since a NOEL was not be established.

The Toxicology Branch Peer Review Committee in EPA's Office of Pesticide Programs has determined that lactofen meets the criterion for a B2 (possible human) carcinogen since it caused an increase in liver tumors (adenomas and/or carcinomas) in two species. Based on

the mouse oncogenicity study, a human upper-bound potency estimate (Q1*) was calculated as 0.17 (mg/kg/day)–1.

The calculated human Q1* was based on the standard interspecies scaling factor of BW0.67 and recent EPA guidance indicates that BW0.75 is a more appropriate factor for general use. This change alone would result in a reduction of the calculated human potency factor and a reduction in the calculated carcinogenic risk by about 20%. In addition, evidence suggests that carcinogenic effects caused by lactofen in rodent livers may be due to peroxisomal proliferation as opposed to a direct genotoxic effect. This mechanism of action would more appropriately be regulated as a threshold effect (similar to RfD comparisons) as opposed to a non-threshold effect with a quantitative potency factor derived from low dose extrapolations. These changes in the hazard assessment process for lactofen would have a profound effect on the exposure and risk assessments for this chemical.

6. *Animal metabolism*. Rat metabolism studies have been conducted for lactofen and demonstrate that lactofen is almost completely eliminated (>95%) in excreta within three days of oral dosing. Generally about 60% of orally administered radioactivity (14C–lactofen) is found in the feces with lactofen itself being the major component. About 40% of radioactivity is recovered in urine and PPG–847 (hydrolyzed side chain) is the major metabolite. Other metabolites include PPG–947, PPG–1576, and PPG–2053.

C. Aggregate Exposure

Complete information to perform an aggregate exposure assessment may be available to the Agency, but is not available to Valent, and an extension of the lactofen cotton tolerance has been requested by Valent in order to allow EPA time to perform a complete aggregate exposure assessment. As discussed below, lactofen contributes insignificant chronic toxicity and carcinogenic risks as compared to the other diphenyl ethers.

1. *Dietary exposure*. (a) *Food*. Lactofen is approved for use in the production of commercial agricultural crops including soybeans, cotton, snap beans, and pine seedlings. Dietary exposures are expected to represent the major route of exposure to the public.

A chronic dietary assessment for lactofen has been conducted by the registrant using Anticipated Residue Contributions (ARC) for existing and proposed uses of lactofen. Since crop field trial data indicate that quantifiable residues of lactofen are rarely found in raw agricultural and processed commodities , ARCs were estimated based on the analytical method limit of detection (LOD) for each commodity. When available, analytical results for control samples were used to determine the method LOD for lactofen and its related metabolites. When all control samples contained no detectable residues, the limit of detection was determined to be 0.005 ppm. Mean anticipated residues were determined based on the sum of residues found above the LOD, or when no detectable residues were present for lactofen or any metabolite, one-half the greatest LOD for any analyte was used as the anticipated residue level. Anticipated residue levels also considered the percent of crop treated with lactofen as follows: 5% of soybeans, 2.5% of cotton, 4.5% of snap beans, and 5% of peanuts. The soybean and cotton values are based on 1995 marketing research data (Maritz) and the snap bean and peanut values are estimates for the future from the registrant. Note that a lactofen peanut tolerance is still pending at the Agency and no lactofen is used on this crop even though peanuts are included in the dietary exposure assessment. The assessment results are summarized below in the Safety Determination section.

EPA has performed chronic dietary exposure assessments for the related diphenyl ethers mentioned above in conjunction with tolerance approvals. For acifluorfen and fomesafen, recent assessments were performed with anticipated residues, but did not consider percent of crop treated. For oxyfluorfen, anticipated residues were considered for only some crops and the same is true for percent of crop treated. And for diclofop, neither anticipated residues nor percent of crop treated were considered. Therefore, the current dietary assessments performed by EPA are highly conservative, but not worst case. Additional time is necessary for the Agency to perform a consistent and integrated dietary exposure assessment for these related chemicals. The assessment results are summarized below in the Safety Determination section.

(b) *Drinking water*. Since lactofen is applied outdoors to growing agricultural crops, the potential exists for lactofen or its metabolites to leach into groundwater. Drinking water, therefore represents a potential route of exposure for lactofen and should be considered in an aggregate exposure assessment.

Based on available lactofen studies used in EPA's assessment of environmental risk, EPA required a prospective groundwater study for lactofen. Valent conducted a study using the maximum application rate applied to a site which was extremely vulnerable to leaching to a shallow aquifer. The water table was at a depth of 6 to 9 feet, the top two feet of soil were classified as loamy sand (78 - 82% sand), and the deeper soil was classified as sand (88 - 94% sand).

A final report was submitted in 1994 which indicates that lactofen degrades rapidly without downward movement in soil and will not contaminate even shallow groundwater beneath light, sandy soils. There were no reported or possible detections of lactofen (< 1 ppb) in lysimeter or monitoring well water samples with the exception of apparent detections (1.4 - 1.6 ppb) in two well water samples which were determined to be due to matrix interferences. Reanalysis to resolve the interference problem indicated that lactofen was not present at the 1 ppb level. Lactofen degrades to acifluorfen, which was also monitored in the study. Although acifluorfen was found to degrade somewhat more slowly than lactofen, it did not leach to groundwater during the study. Since acifluorfen results from lactofen degradation, but is not the only degradation product, concentrations are expected to be lower for acifluorfen than for lactofen. In fact, there were no reported or possible detections of acifluorfen (< 1 ppb) in lysimeter or monitoring well samples. This report has been placed in review at EPA, but a review has not been completed.

There is no established Maximum Concentration Level for residues of lactofen in drinking water under the Safe Drinking Water Act.

Based on this information, lactofen appears to represent an insignificant risk for exposure through drinking water.

2. *Non-dietary exposure*. Lactofen is currently approved only for the commercial production of agricultural crops including cotton, soybeans, snap beans, and pine seedlings. The potential for non-occupational exposure to the general public, other than through the diet or drinking water, is therefore insignificant.

D. Cumulative Effects.

There are several other pesticide compounds which are structurally related and may have similar effects on animals. Specifically, lactofen, acifluorfen, fomesafen, oxyfluorfen, and diclofop methyl are all diphenyl ethers which have caused liver tumors in

rodents. These chemicals are approved for food uses in the U.S. and could be considered in an aggregate exposure assessment. Dietary exposures to these other diphenyl ethers are expected to represent the major route of exposure to the public. It is premature to add the risk from these chemicals since exposure considerations as well as endpoint, pharmacokinetic, and pharmacodynamic considerations may indicate that it is inappropriate to add the risks. However, to meet the requirements of the FQPA of 1996, it is prudent to consider if it is likely that these chemicals violate the provisions of the new law. The information presented below indicates that while more study is necessary, it is unlikely that these materials violate the provisions of the act.

Summaries of the established reference doses, quantitative cancer potency factors, and cancer sites in animals for these structurally related chemicals are presented below.

Chemical	Reference Dose (mg/kg/day)	Cancer Potency Factor (mg/kg/day)$^{-1}$	Cancer Site
Lactofen	0.002	0.17	Liver
Acifluorfen	0.013	0.107	Liver, Stomach
Fomesafen	0.0025	0.19	Liver
Oxyfluorfen	0.003	0.13	Liver
Diclofop Methyl	0.002	0.231	Liver

This comparison indicates that reference doses determined from chronic toxicity studies and cancer potency factors for these related chemicals are on the same order of magnitude as for lactofen.

It should be noted that these related chemicals would benefit from the use of the EPA's new interspecies scaling factor as well as lactofen, and that the rodent liver tumor effects may also be due to peroxisome proliferation which would more appropriately be regulated as a threshold effect. The carcinogenic risk assessments performed to date are, therefore, highly conservative.

E. Safety Determination

1. *U.S. population.* Using the dietary exposure assessment procedures described above (and performed by Valent) for lactofen, and recent EPA assessments for related chemicals, chronic dietary exposures resulting from existing and proposed uses of lactofen and related chemicals were compared to the reference dose (RfD) for each chemical. The following contributions to the RfD were found for the U.S. Population and all of the subpopulations for which dietary consumption data are available:

Lactofen: less than 0.1% for all subpopulations.

Acifluorfen: less than 1% for all subpopulations.

Fomesafen: less than 1% for all subpopulations.

Oxyfluorfen: less than 1% for all subpopulations.

Diclofop: not available to Valent.

EPA generally has no concern for exposures below 100 percent of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. The current and proposed uses of these chemicals, even when considered collectively, represent a minimal chronic toxicological risk to the general public.

Carcinogenic risks were calculated by Valent using a potency factor (Q1*) for lactofen of 0.17 (mg/kg/day)$^{-1}$. The dietary carcinogenic risk resulting from existing and proposed uses of lactofen is calculated at 1.54 X 10^{-8} or less for several lifetime population groups. This is approximately 65 times lower than the acceptable level of one-in-a-million additional lifetime cancers. It should be noted that the proposed use on peanuts, which is not being considered in the current action, accounts for more than a third of the exposure contributing to the calculated carcinogenic risk. Therefore, these estimates of carcinogenic risk are conservative and are well within acceptable levels.

EPA has performed dietary carcinogenic risk assessments for the related diphenyl ethers mentioned above in conjunction with tolerance approvals. The following table summarizes the dietary risk assessment made by Valent for lactofen and the most recent dietary risk assessments performed by EPA for related chemicals.

Chemical	Data Source	Date	Carcinogenic Risk
Lactofen	Valent Report	8/20/96	1.54 X 10^{-8}
Acifluorfen	61 FR 16740	4/17/96	5.8 X 10^{-7}
Fomesafen	61 FR 31057	6/19/96	1.56 X 10^{-6}
Oxyfluorfen	60 FR 49816	9/27/95	1.8 X 10^{-6}
Diclofop methyl	51 FR 19176	5/28/86	1 X 10^{-5}

Regarding drinking water exposures, groundwater monitoring studies have been required for acifluorfen, fomesafen, and diclofop methyl as well as for lactofen. Detections in groundwater have been reported for acifluorfen and fomesafen. Complete information may be available to the Agency, but is not to available to Valent, and additional time is requested to allow time for EPA to adequately address the drinking water exposure issue. However, based on the lactofen groundwater study, lactofen exposures to the public through drinking water are expected to be insignificant compared to these other chemicals.

Regarding non-dietary exposures, the other diphenyl ethers are also used primarily for commercial agricultural production. However, some of these chemicals may involve some uses around the home which could lead to non-occupational exposure. Information about this small potential exposure is not available to Valent, but if a significant potential exists for non-occupational exposure, is should be considered in an aggregate risk assessment by EPA. Some exposures to residential pesticides are being evaluated by an industry task force, the Outdoor Residential Exposure Task Force (ORETF), of which Valent is a member.

In summary, this comparison shows that lactofen's contribution to aggregate cancer risk is insignificant compared to the other diphenyl ethers, based on

current registrant and EPA assessments. In addition, the conservative risks calculated by EPA for fomesafen and oxyfluorfen are slightly above the new standard set by FQPA and for diclofop methyl is significantly above the new standard. Valent believes that when these other diphenyl ethers are evaluated using anticipated residues, percent of crop treated, revised cancer potency factors, and up-to-date exposure methodology the projected risks will be much lower than 1 X 10^{-6} for all of these chemicals. Industry and EPA are also developing methodology for determining whether or not multiple exposures will occur and with what frequency for these and other chemicals. If multiple exposures do not occur, or occur with a low frequency, it is not appropriate to add risks. For these reasons, additional time will be necessary for the Agency to address the aggregate risk to the U.S. population for this group of related chemicals.

2. *Infants and children.* As stated above, dietary exposure assessments utilize less than 1% of the RfD for all subpopulations including infants and children. Reproduction and developmental effects have been found in toxicology studies for lactofen, however, the adverse effects were seen at levels that were also maternally toxic. This indicates that developing animals are not more sensitive than adults. FQPA requires an additional safety factor of up to 10 for chemicals which present special risks to infants or children. Lactofen does not meet the criterion for application of an additional safety factor for infants and children.

Information on the reproduction and developmental effects caused by the other diphenyl ethers is not available to Valent. Additional time is necessary for the Agency to evaluate the need for an additional safety factor related to these other chemicals. However, even if an additional safety factor were deemed necessary, the dietary exposures are still expected to be well below the established reference doses.

F. International Tolerances

There are no Codex Maximum Residue Limits (MRL) established for lactofen on cotton commodities, so there is not conflict between this proposed action and international residue limits.

II. Administrative Matters

Interested persons are invited to submit comments on this notice of filing. Comments must bear a notation indicating the document control number, [PF–677]. All written comments filed in response to this petition will be available in the Public Response and Program Resources Branch, at the address give above from 8:30 a.m. to 4 p.m., Monday through Friday, except legal holidays.

A record has been established for this notice under docket number [PF–677] 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 public record is located in Rm. 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments can be sent directly to EPA at:

opp-Docket@epamail.epa.gov

Electronic comments must be submitted as ASCII file avoiding the use of special characters and any form of encryption.

The official record for this rulemaking, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer all comments received electronically into printed, paper form as they are received and will place the paper copies in the official rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the address in ''ADDRESSES'' at the beginning of this document.

List of Subjects

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: December 4, 1996.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 96–31556 Filed 12–11–96; 8:45 am]

BILLING CODE 6560–50–F

[FRL–5663–2]

Proposed De Minimis Settlement Pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as Amended by the Superfund Amendments and Reauthorization Act—Golden, CO

AGENCY: Environmental Protection Agency.

ACTION: Correction to original notice and request for public comment.

SUMMARY: The original notice of proposed de minimis settlement published on September 17, 1996 (61 FR 48951) is corrected by adjusting the settlement figure for Energy Fuels Nuclear, Inc. from $326,800.73 to $184,800.41 and is hereby submitted for public comment. In accordance with the requirements of section 122(I)(1) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended (CERCLA), notice is hereby given of a proposed de minimis settlement under section 122(g), concerning the Colorado School of Mines Research Institute site in Golden, Colorado (Site). The proposed Administration Order on Consent (AOC) requires five (5) Potentially Responsible Parties to Pay an aggregate total of $215,640.36 to address their liability to the United States Environmental Protection Agency (EPA) related to response actions taken or to be taken at the Site.

OPPORTUNITY FOR COMMENT: Comments must be submitted on or before January 13, 1997.

ADDRESSES: The proposed settlement is available for public inspection at the EPA Superfund Record Center, 999 18th Street, 5th Floor, North Tower, Denver, Colorado. Comments should be addressed to Kelcey Land, Enforcement Specialist (8ENF–T), U.S. Environmental Protection Agency, 999 18th Street, Suite 500, Denver, Colorado, 80202–2405, and should reference the Colorado School of Mines Research Institute site de minimis settlement (EPA Docket No. CERCLA–VIII–96–17).

FOR FURTHER INFORMATION CONTACT: Kelcey Land, Enforcement Specialist, at (303) 312–6393.

SUPPLEMENTARY INFORMATION: Notice of section 122(g) de minimis settlement: In accordance with section 122(I)(1) of CERCLA, notice is hereby given that the terms of an Administrative Order on Consent (AOC) have been agreed to by the following five (5) parties, for the following amounts:

Energy Fuels Nuclear, Inc.....$184,800.41