1,000 mg/kg/day, and the NOEL for reproductive toxicity was greater than 5,000 mg/kg/day. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Uniroyal concludes that there is reasonable certainty that no harm will result in infants and children from aggregate exposure to residues of diflubenzuron and its conversion products containing the p-chloroaniline moiety.

E. Residues in the Raw Agricultural Commodity and Processed Food/Feed

1. *Nature of residues in plants and livestock.* The nature of the residue in plants and livestock is adequately understood. In plants, the metabolism of diflubenzuron was investigated in soybeans, oranges and rice. The main component of residues in rice was CPU; levels of PCA were negligible to non-detectible. The main component of the residues in soybeans and oranges was the parent diflubenzuron (DFB). A considerable portion of the residues were bound. DFB showed very limited absorption and translocation in plants with most of the residues remaining on the surface.

In livestock, goats treated for three days at about 1X (10 ppm feeding level) the dietary burden of ^{C}14 DFB gave DFB equivalent of ^{C}14 = 7-9 ppb in milk, 217-262 ppb in liver, 16-19 ppb in kidney, about 1 ppb in muscle, and about 4 ppb in fat. Milk residues were mainly CPU and DFBAM. PCA was not detectable. Liver residues were DFB, 2-hydroxy DFB, CPU, and DFBAM. Again, PCA was not detected at this dose however, it was detected in studies conducted at about 22X dose. Chickens were dosed with ^{C}14 DFB at 5 ppm level for 1-28 days. Residues in tissues as DFB equivalent were highest in liver and kidney. The main residues in tissues and eggs were DFB and DFBA. Trace amount of PCA and its acetanilide were detected, but not confirmed, in liver kidney and egg white.

2. *Magnitude of residues and proposed tolerances.* An adequate number of separate residue trials have been conducted with diflubenzuron on rice. Analyses of these trials show that the maximum total residue for diflubenzuron and its conversion products PCA and CPU will be at or below 0.01 ppm.

A tolerance has been requested for the combined residues of diflubenzuron and metabolites convertible to p-chloroaniline expressed as diflubenzuron on rice at 0.01 ppm. The proposed tolerance is adequate to cover residues likely to be present from the use of diflubenzuron on rice. Therefore, no special processing to reduce the residues will be necessary.

The meat by-products tolerances are adequate to cover residues resulting from the rice use. Uniroyal Chemical has submitted calculations from a goat metabolism study which supports the 0.05 ppm tolerance in meat by-products. Therefore, no increase in the meat by-products tolerances should be necessary.

F. Practical Analytical Method

Practical analytical methods for detecting levels of DFB, CPU and PCA, in or on food with a limit of detection that allows monitoring of the residue at or above the level set in the tolerance was used to determine residues in rice and its respective processed fractions.

Residues of the individual analytes are detectable and quantifiable using three separate analytical methods. Residues of DFB are extracted from rice with dichloromethane. Extracts are purified with deactivated florisil. An aliquot of the extract is hydrolyzed with phosphoric acid and the DFB is partitioned into hexane. The resulting extract is derivatized in heptafluorobutyric anhydride (HFBA). Quantification of DFB is accompanied by gas chromatography using an electron capture detector.

The analytical method for quantitation of the 4-chlorophenylurea requires ethyl acetate extraction of the residue from the matrix. Column chromatography is utilized for clean-up of the extract immediately prior to derivitization with HFBA. Derivatized extracts are analyzed by gas chromatography equipped with an electron capture detector.

The analysis for the determination of PCA residues in rice matrices utilizes an internal standard method. Samples of matrix to be analyzed are fortified with the internal standard. Residues of 12C-PCA and the internal standard are subjected to acid and base hydrolysis. The final extract is passed through florisil column for clean-up and derivatized with HFBA in hexane. An aliquot of the derivatized extract is analyzed by gas chromatography using a mass spectrometry detector in the selective ion monitoring mode. Recovery of PCA is determined by the combined peak areas for the two mass spectral ions obtained from the derivatized 12C-PCA relative to the response factor derived from the combined areas of the corresponding two mass spectral ions from the internal standard.

G. List of All Pending Tolerances and Exemptions

A tolerance for diflubenzuron on range grass at 4.0 ppm is pending. There are no exemptions from tolerance for diflubenzuron.

H. List International Tolerances (Code MRLs)

There are no Codex Alimentarius Commission maximum residue levels for residues of diflubenzuron on rice. The Codex MRL on citrus is 1.0 mg/kg vs. 0.05 ppm for U.S. tolerance. The Codex MRL for mushrooms is 0.1 mg/kg vs. 0.2 ppm for U.S. tolerance. The Codex MRL for soybeans is 0.1 mg/kg/ vs. 0.05 ppm for the U.S. The Codex MRL is 1 mg/kg for apples, Brussels sprouts, cabbage, pears, plums and tomatoes for which there are no U.S. tolerances. The Codex MRL for meat, milk and eggs is 0.05 mg/kg/ which is the same as the established U.S. tolerances.

[FR Doc. 98–4812 Filed 2-24-98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–789; FRL–5767–5]

Notice of Filing of Pesticide Petition

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–789, must be received on or before March 27, 1998.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (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. 119 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, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 237, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–6224; e-mail: miller.joanne@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical 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 this petition contains 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–789] (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. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number (PF–789) and appropriate petition number. Electronic comments on this 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: February 11, 1998

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the views of the petitioner. EPA is publishing the petition summary verbatim without editing it 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.

Valent U.S.A Corporation

PP 9F3798

EPA has received a pesticide petition (PP 9F3798) from Valent U.S.A Corporation, 1333 North California Blvd., Suite 600, Walnut Creek, California 94596-8025 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 extending a time-limited tolerance for residues of 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 parts per million (ppm). The tolerance would expire on December 31, 1999. The time limitation on the tolerance would allow Valent to complete, and EPA to evaluate, additional prospective groundwater study data. 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

1. *Plant metabolism*. Lactofen, formulated as COBRA Herbicide, is used to control broadleaf weeds in soybeans by pre- and/or post-emergent application and in cotton by post-directed application. Pre-harvest intervals are extended, 45 to 70 days. Plant metabolism protocols (cotton, peanut, soybean and tomato) have been designed to mimic the field applications with respect to application methods and timing. In the studies, plant material has been treated at rates exceeding normal field application to facilitate identification of metabolites. Postdirected application to cotton was simulated in the field using radiocarbon labeled lactofen and demonstrated that no radioactivity (> 0.001 ppm lactofen equivalent) was detected in the bolls.

The lactofen molecule is rapidly degraded in the environment and in plants. Therefore, the consistent result of all detailed plant metabolism studies using radiolabeled lactofen has been:

i. Low concentrations of radiocarbon are distributed throughout the plant,

ii. Much of the radiocarbon is irreversibly bound and unextractible,

iii. Very low concentrations of radiocarbon is found in the RAC (seeds), and

iv. Very little of the terminal residue is identifiable as finite metabolites as a result of the extensive degradation and binding.

To demonstrate plant metabolic pathways and to validate that the residue analytical methodology can extract, identify and quantitate lactofen and its metabolites as aged residues, plant samples from radiocarbon metabolism studies were analyzed soon after application, well before normal harvest. It is from these early samples that the definition of the regulated residue in RAC has been obtained. The residue of concern is defined by the Agency as parent and four degradates containing the intact diphenyl ether moiety. Parent lactofen (PPG-844) is degraded hydrolytically to corresponding carboxylic acid-lactate ester (PPG-947), and further to the benzoic acid (PPG-847). In a separate pathway, the esters remain intact and the aromatic nitro group is reduced to the corresponding aniline (PPG-1576) and the aniline is formylated (PPG-2597). Further, there is good evidence that these lactofen metabolites are further degraded by cleavage of the

diphenyl ether. The sodium salt of the benzoic acid (PPG-847) is the commercial herbicide acifluorfen. All five of the compounds in the regulated residue as defined have never been found in a single RAC sample either from plant metabolism or from crop field studies. For example, at maximum treatment rates in crop field trials, only one soybean seed sample was found to have a residue of parent lactofen greater than the limit of detection, but less than the limit of quantitation and only a single cotton gin trash sample was found to contain a finite residue of lactofen. Even at exaggerated rates in metabolism or crop residue studies, residues are rarely above the limit of detection for any analyte. In fact, more than one analyte has never been found above the limit of detection in a single RAC sample from crop field trials.

2. *Analytical method*. Adequate analytical methodology is available for detecting and measuring levels of lactofen and regulated 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 method involves extraction with triethylamine/aqueous ethanol, partitioning, methylation of the carboxylic acids, column clean-up, and separation and quantitation by gas chromatography with electron capture detection. The method, RM-28D, has been validated by an independent laboratory on both cottonseed and peanuts and was found to be acceptable with comments for enforcement in cottonseed by the EPA Analytical Chemistry Laboratory. 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). There are existing tolerances for lactofen on soybeans, and snap beans. A time limited tolerance supported use on cotton, and a tolerance is pending for peanuts. Lactofen is a broad-spectrum broadleaf herbicide with the following use pattern on cotton:

Post-emergence directed spray applications with a single application maximum of 0.2 lb. a.i./acre, a seasonal maximum total application of 0.4 lb. a.i./acre, and a PHI of 70 days.

Because of relatively long pre-harvest interval, post-directed applications, and extensive degradation, finite lactofen residues have not been found in cottonseed or processed cottonseed commodities. Reports covering field residue trials from twenty-one sites in all cotton growing states, several at exaggerated rates, along with processing studies have failed to show detectable residues of lactofen or its regulated degradates in any sample. Consequently, a tolerance on cottonseed is proposed at 0.05 ppm, based on the sum of the 0.01 ppm limits of quantitation for lactofen and its four regulated metabolites containing the diphenyl ether linkage. Field residue data for cotton gin trash has recently been submitted. All other lactofen tolerances to date have been established similarly at 0.05 ppm.

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. This chemical therefore represents a minimal acute toxicity risk.

2. *Genotoxicty*. Lactofen Technical has been tested and produced negative results in genotoxicity tests including unscheduled DNA synthesis in rat hepatocytes, DNA covalent binding in mouse liver, chromosomal aberration in CHO cells. Lactofen technical was also negative in an Ames assay. In repeat Ames assays, lactofen was shown to be positive without metabolic activation at 5,000 µg/plate and above. Overall lactofen is not a genetic hazard.

3. *Reproductive and developmental toxicity*. Reproduction and teratology studies indicate that adverse effects, including embryotoxicity, occur only at doses that are also maternally toxic. Since lactofen causes effects only at levels which also produce systemic toxicity the compound is not a reproductive hazard.

4. *Reproduction— Rats*. Groups of male and female rats were fed 0, 50, 500 or 2,000 ppm of Lactofen Technical continuously in their diets for 2–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 No-Observed Effect-Level (NOEL) for both systemic and reproductive toxicity was 50 ppm (2.5 milligram/kilogram/day (mg/kg/day).

5. *Developmental toxicity— Rats*. 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.

6. *Developmental toxicity— Rabbits*. 2 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 milligram/kilogram (mg/kg). The Agency concluded that the data were insufficient to establish a clear NOEL. 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.

C. Subchronic Toxicity

1. *Subchronic feeding— Rat— 4-week*. Male and female rats were fed diets containing Lactofen Technical at concentrations of 0, 200, 1,000, 5,000, and 10,000 ppm for four weeks. A slight increase in spleen weight was the basis for a Lowest-Observed Effect-Level (LOEL) of 200 ppm (lowest dose tested). At doses of 1,000 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.

2. *Subchronic feeding— Rat— 3-month*. Lactofen Technical was fed to male and female rats at dietary concentrations of 0. 40, 200, and 1,000 ppm for 13–weeks. Histopathological changes in the liver and significant changes in clinical chemistry associated with the liver were observed in rats exposed to 1,000 ppm Lactofen Technical dosage. Decreased RBC, hemoglobin and hematocrit values were also observed at 1,000 ppm. The NOEL in this study was 200 ppm.

3. *Subchronic feeding— Dog— 4–week*. In a range finding study Lactofen Technical was fed in the diet of dogs at 0. 1,000, 3,000, and 10,000 ppm for 4–weeks. Toxic effects noted in dogs fed 10,000 ppm included decreased rbc count and hemocrit, and increased BUN and SGPT. Food palatability problems led to greatly decreased feed consumption at higher dosages. The NOEL appeared to be 1,000 ppm.

4. *Subchronic feeding— Mice— 3–month*. Groups of Male and female mice were fed diets containing Lactofen Technical at concentrations of 0, 40, 200, 1,000, 5,000, and 10,000 for 13–weeks. At week 5, the dosage of the 40 ppm groups was increased to 2,000 ppm. Treatment related mortality occurred at dosages above 1,000 ppm. The LOEL 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. In general, effects were slight in the 200 ppm groups, and moderate to severe in the 1,000 ppm groups.

5. *Peroxisome proliferation— Mice— 7–weeks*. Butler et al (1988) studied the effects of lactofen on peroxisome proliferation in mice exposed for 7–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 (7 mg/kg/day), a dose which is not tumorigenic, would be considered a threshold dose in mice for peroxisome proliferation produced by lactofen. Peroxisome Proliferation -- Chimpanzees 14-weeks: 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.

D. Chronic Toxicity

A complete chronic data base supported by appropriate subchronic studies for lactofen is available to the Agency. Lactofen Technical causes adverse health effects when administered to animals for extended periods of time. These 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 1,000, a reference dose (RfD) of 0.002 mg/kg/day has been established for lactofen. An uncertainty factor of 1,000 was used since a clear NOEL was not established.

1. *Chronic/carcinogenicity feeding study— Mouse— 24-month*. In a dietary 18-month oncogenicity study in mice at dosages 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.

2. *Chronic/carcinogenicity feeding study— Rat— 24-month*. In a 2-year chronic feeding/oncogenicity study of Lactofen Technical in rats at dosages of 0, 500, 1,000 and 2,000 ppm in the diet, an increase in liver neoplastic nodules and foci of cellular alteration was observed in both sexes at 2,000 ppm. The NOEL for systemic toxicity is 500 ppm based on kidney and liver pigmentation.

3 *Oral toxicity study— Dog— 12-month*. In a 1–year study in dogs exposed to 40, 200, and 1,000 (wk.1-17) or 3,000 ppm (wk 18-52) Lactofen Technical in their diet, the NOEL was determined to be 200 ppm based on renal dysfunction and decreased RBC, hemoglobin hematocrit and cholesterol observed at 1,000/3,000 ppm.

4. *Carcinogenicity*. The Toxicology Branch Peer Review Committee 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 (Q_1*) was calculated as 0.17 (mg/kg/day).

The calculated human Q_1* is based on the standard interspecies scaling factor of $BW^{0.67}$. Recent EPA guidance indicates that $BW^{0.75}$ 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%.

More importantly, evidence summarized above suggest that carcinogenic effects observed in rodent liver related to long term lactofen consumption are attributable 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. This change in the hazard assessment process for lactofen would have a profound effect on the exposure and risk assessments for this chemical.

5. *Animal metabolism*. Single high, single low, and repeated low dose radiocarbon labeled lactofen metabolism studies have been performed in male and female rats. Radiocarbon is almost completely eliminated (>95%) in excreta within 3–days of oral dosing. Generally about 60% of orally administered radioactivity (^{14}C-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. Except for the formyl derivative (PPG-2597), a minor plant metabolite, there were no plant metabolites detected that were not also produced in mammals.

Additional pharmacokinetic studies using both radiocarbon labeled and unlabeled lactofen were performed in rats, mice, rhesus monkeys, and chimpanzees. Little parent was seen in the plasma of any species tested. At steady state, the primary metabolite in the circulation of rodents was PPG-847. In the primates, PP-2053 was the primary circulating metabolite. Mice appeared to be least efficient in clearing PPG-844 and other lactofen metabolites from the circulation, while rats, and especially primates appeared to be more efficient.

6. *Metabolite toxicology*. A major hydrolytic metabolite of lactofen is PPG-847, the benzoic acid. The sodium salt of this benzoic acid, sodium 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, is the registered herbicide acifluorfen. This product has a complete data base supporting registration with a RfD of 0.013 mg/kg/day and a Cancer Potency Factor of 0.107 $(mg/kg/day)^{-1}$. Exposure to acifluorfen from all sources must be evaluated to perform a cumulative risk analysis.

7. *Endocrine disruption*. No special studies to investigate the potential for estrogenic or other endocrine effects of lactofen have been performed. However, as summarized above, a large and

detailed toxicology data base exists for the compound including studies acceptable to the Agency in all required categories. These studies include evaluations of reproduction and reproductive toxicity and detailed pathology and histology of endocrine organs following repeated or long term exposure. These studies are considered capable of revealing endocrine effects and no such effects were observed.

E. Aggregate Exposure

1. *Dietary exposure*. A chronic dietary toxicity endpoint of concern, RfD, has been identified by the Agency 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 1,000. The RfD is 0.002 mg/kg/day for lactofen. An uncertainty factor of 1,000 was used since a clear NOEL was not established. The Toxicology Branch Peer Review Committee 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 (Q_1*) was calculated as 0.17 $(mg/kg/day)^{-1}$. An acute or short term dietary endpoint of concern has not been established by the Agency. Valent has chosen to use the maternal NOEL for systemic toxicity of 4 mg/kg/day from the rabbit developmental toxicity study for acute and short term dietary risk analyses. Lactofen has no uses not associated with commercial agriculture. Therefore the only potential exposure possible to the U.S. Population is through the diet in food and drinking water. Risk analyses via other exposure routs, inhalation, dermal, are not necessary. Thus, only chronic and acute dietary exposure and risk analyses are necessary.

2. *Food*. Lactofen is registered for use in the production of commercial agricultural crops including soybeans, cotton, snap beans, and conifer seedlings. Dietary exposures are expected to represent the major route of exposure to the public.

3. *Chronic*. A chronic dietary assessment for lactofen has been conducted using Anticipated Residue Contributions (ARC) for existing and proposed uses of lactofen. This exposure/risk analysis has been submitted to the Agency along with a detailed description of the methodology and assumptions used. 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. The chronic exposure analysis 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 of future market penetration. 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 Dietary exposure was calculated for the U.S. population and 26 population subgroups. Chronic dietary exposure was less than 0.1% of the RfD for all subpopulations.

4. *Acute*. A first tier acute exposure and risk analysis was performed for lactofen assuming tolerance level residues in soybeans, snapbeans, cotton, and peanuts (0.05 ppm) and 0.02 ppm in all meat and milk commodities. Using the acute dietary endpoint of 4.0 mg/kg/day, the NOEL from the rabbit developmental toxicity study, the calculated exposures and margins of exposure (MOE) for the higher exposed proportions of the subgroups are listed below. It should be noted that the population sizes are small at the lower probability exposures (e.g. 99th and 99.9th percentiles) oftentimes leading to unrealistically high calculated exposures. In all cases, margins of exposure exceed 1,000.

Calculated Acute Dietary Exposures to Lactofen Residues in Food

Population Subgroup	99th Percentile		99.9th Percentile	
	Exposure (mg/kg bw/day)	MOE	Exposure (mg/kg bw/day)	MOE
U.S. Population	0.001199	3,337	0.002211	1,809
Females 13-50	0.000464	8,619	0.000712	5,616
Children 1-6	0.001911	2,094	0.002781	1,438
Children 7-12	0.001019	3,927	0.001472	2,717
All Infants	0.002887	1,385	0.003870	1,034
Non-Nursing Infants(<1)	0.002956	1,353	0.003901	1,025

5. *Drinking water*. Drinking water represents a potential route of acute or chronic dietary exposure for lactofen and should be considered in an aggregate exposure assessment. Since lactofen is applied outdoors to growing agricultural crops, the potential exists for lactofen or its metabolites to leach into ground water or reach surface water that are used for drinking. There is no established Maximum Concentration Level for residues of lactofen in drinking water under the Safe Drinking Water Act.

6. *Ground water*. Based on available lactofen studies used in EPA's assessment of environmental risk, EPA required a small scale prospective ground water 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). The final report demonstrated that lactofen degrades rapidly without downward movement in soil and did not contaminate even shallow ground water beneath light, sandy soils. There were no detections of lactofen (< 1 ppb) in lysimeter or monitoring well water samples. Lactofen degrades to acifluorfen, which was also monitored in 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. Acifluorfen was found to degrade

somewhat more slowly than lactofen, and it did not leach to ground water during the study. There were no detections of acifluorfen (> 1 ppb) in lysimeter or monitoring well samples.

Assuming that all ground water contains lactofen at one-half the limit of quantitation from this study, 0.005 ppm, is non-determinate, and overly conservative. SCI-GROW modeling, using the same environmental fate parameters utilized below gave a Ground Water Screening Concentration of 0.002 ppb.

7. *Surface water.* Potential surface water concentrations for lactofen were estimated using GENEEC and the following conservative use, physical property, and environmental fate parameters: use rate, 0.2 lb a.i./a; applications, 2 aerial broadcast; application interval, 14 days; K_{OC}, 6,600; water solubility 0.945 ppm; aerobic soil half-life, 2.2–days; hydrolysis (pH 7) half-life 11–days; and photolysis in water half-life, 2.75–days. The maximum concentration predicted in the hypothetical small stagnant farm pond water was 1.05 ppb and 0.17 ppb for the 4 and 56 day average GEEC, respectively.

Potential lactofen concentrations in actual drinking water would be much lower than one-half of the quantitation limit in the ground water study or the concentration modeled in ground water from the SCI-GROW Ground Water Screening Concentration or the concentration modeled by GENEEC in the hypothetical small stagnant farm pond. For this risk analyses, the finite concentrations modeled by GENEEC are selected. Based on this analyses, the lactofen exposure contribution from drinking water to realistic dietary risk analyses is negligible.

Exposure to Lactofen from Drinking waterfor Adults and Children from GENEEC Modeling

Exposure	Exposure (mg/kg bw/day)	
	Adult (70 kg, 2 liter/day)	Child (10 kg, 1 liter/day)
Acute (4-day average)	0.000030	0.000105
Chronic (56-day average)	0.0000049	0.000017

1. *Summary— Aggregate chronic dietary exposure.* Aggregate chronic dietary exposure to lactofen is the sum of the contributions from food and water as shown in the table below. It can be seen that the total potential chronic exposure to lactofen to two representative population subgroups is dominated by the conservative estimation of residues in water, but even so, there is no cause for concern.

Aggregate Chronic Exposure to Lactofenfor Two Representative U.S. Populations

Exposure Medium	Exposure (mg/kg bw/day)	
	U.S. Population (all seasons)	Non-Nursing Infant (less than 1 year)
Food	0.0000001	0.0000001
Drinking Water	0.0000049	0.000017
Sum of Chronic Exposures	0.000005	0.000017
Occupancy of RfD(percent)	0.25	0.85

2. *Summary— Aggregate acute exposure.* It is possible to sum calculated acute exposures from dietary sources as shown in the table below. However, summation is exceedingly conservative because the approach assumes that two low probability events occur simultaneously. For example, it is highly unlikely that an individual in a single day consumes the 99.9th percentile dietary exposure (one-in-a-thousand), and also consumes all the daily drinking water from a pond surrounded by treated cotton fields. Even so, the acute exposures shown below that sum exposures from food and drinking water gives MOE values at or above 1,000. These calculated acute and short term exposures are very conservative, and are small enough to be of little significance.

Aggregate Acute Exposure to Lactofenfor Two Representative U.S. Populations(summation of low probability maximum values)

Exposure Medium	Exposure (mg/kg bw/day)	
	U.S. Population (all seasons)	Non-Nursing Infant (less than 1 year)
Food	0.002211	0.003901
Drinking Water	0.000030	0.000105
Sum of Acute Exposures	0.002241	0.004006
Margin of Exposure	1785	999

3. *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.

F. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' ''Available information'' in this context includes not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk

assessments. Valent will submit information for EPA to consider concerning potential cumulative effects of lactofen consistent with the schedule established by EPA at (62 FR 42020; August 4, 1997) (FRL 5734-6) and other EPA publications pursuant to the Food Quality Protection Act.

There are several other pesticide compounds which are structurally related to lactofen and may have similar effects on animals. Specifically, lactofen, acifluorfen, fomesafen, oxyfluorfen, and diclofop methyl are all diphenyl ethers and all have caused liver tumors in rodents. These chemicals are approved for food uses in the U.S. and could be considered in a cumulative exposure assessment. It is premature to simply add the risk from all these chemicals. Exposure considerations as well as toxicity endpoint, pharmacokinetic, and pharmacodynamic considerations may indicate that it is inappropriate to add the risks. Dietary exposures to these other diphenyl ethers are expected to represent the major route of exposure to the public.

A major hydrolytic metabolite of lactofen representing perhaps 50% of the applied dose in animal and environmental fate studies, is PPG-847, the benzoic acid. The sodium salt of this benzoic acid, sodium 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, is the registered herbicide acifluorfen. This product has a complete data base supporting registration with a RfD of 0.013 mg/kg/day and a Cancer Potency Factor of 0.107 $(mg/kg/day)^{-1}$. Because lactofen and acifluorfen have a ''common metabolite'', exposure to both acifluorfen and lactofen from all sources must be evaluated to perform a cumulative risk analysis.

It should be noted that acifluorfen, and the other related diphenyl ethers, would benefit from the use of the larger interspecies scaling factor as well as lactofen. Further, the rodent liver tumor effects of these other diphenyl ethers may 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.

G. Safety Determination

The Food Quality Protection Act introduces a new standard of safety, a reasonable certainty of no harm. To make this determination exposure and consequent risk to both acifluorfen and lactofen from all sources must be evaluated.

In evaluating chronic dietary exposures, the food and water consumed for a lifetime is assumed to contain a baseline amount of residues. Chronic risks are evaluated by comparing a conservatively calculated baseline exposure to the RfD. A long term exposure in mg/kg bw/day is compared to a NOEL from an appropriate long term animal exposure study adjusted by a safety factor. It is quite reasonable to suppose that daily baseline exposures to two or more compounds could occur simultaneously. That is, a consumer could have chronic dietary exposure to lactofen residues and acifluorfen residues at the same time, and because acifluorfen is a metabolite of lactofen, a cumulative risk analysis is appropriate. The situation is very different for acute dietary exposures. In an acute dietary risk analysis, exposures to residues are related to the probability of occurrence of a daily diet containing the residues. At its most simplified, the probability of consuming a diet simultaneously containing both lactofen and acifluorfen at the 99.9 th percentile diet is one in one-million. A simple, additive cumulative risk analysis cannot take the probability of simultaneous exposure into account and is not appropriate.

1. *U.S. population* —i. *Chronic*— Food. Using the dietary exposure assessment procedures described above (and performed by Valent) for lactofen, and a recent assessment for acifluorfen published in the 61 FR 16740; (April 17, 1996) (FRL 5356-6) chronic dietary exposures resulting from existing and proposed uses of lactofen and acifluorfen were compared to their respective reference doses. 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:

ii. *Lactofen*. Exposure 0.0000001 (mg/kg bw/day) less than 0.01% for all subpopulations.

iii. *Acifluorfen*. Exposure 0.0000052 (mg/kg bw/day, 61 FR 16740) less than 0.04 % for all subpopulations.

iv. *Chronic*— Drinking water— Lactofen. Using the conservative assumption that all drinking water contains lactofen at levels calculated by GENEEC for a small farm pond surrounded by lactofen treated fields, a very conservative estimate of risk can be made. Using standard assumptions about body weight and water consumption, the adult chronic exposure from this drinking water would be 4.9×10^{-6} mg/kg bw/day, 0.25% of the RfD.

2. *Acifluorfen*. Acifluorfen that may be in drinking water can be derived directly from acifluorfen applied to crops, or may be acifluorfen derived from degradation of lactofen. The physical properties and soil stability of acifluorfen indicate that the compound may dissolve in surface water, or leach to groundwater that may be used for drinking water.

The U.S. Geological Survey is engaged in a National Water Quality Assessment (NAWQA). This program samples both ground and surface water and analyzes the samples for 75 pesticides and metabolites including acifluorfen, but not lactofen. The data through August 1997 are available from USGS, on the internet at http://water.wr.usgs.govpnsp/gwsw1.html. The NAWQA sampling program was designed to provide an overview of pesticide occurrence in water that could be used for drinking water. Specific types of agriculture or specific products, including acifluorfen, were not targeted. While the program is not exhaustive, it probably provides a reasonably unbiased estimate of the occurrence of agricultural chemical contaminants in potential drinking water. A table summarizing the data for acifluorfen is presented below.

USGS NAWQA data on Acifluorfen

Water Type	Number of Samples		Maximum Concentration (ppb)
	Total	>0.05 ppb	
Agricultural Streams	1148	10	2.2
Urban Streams	418	ND	-
Large Streams	282	6	0.44
Total Surface Water	1848	16	
Agricultural Shallow Ground Water	1069	ND	--
Urban Shallow Ground Water	314	1	0.070
Major Ground Water Aquifer	965	1	0.190

USGS NAWQA data on Acifluorfen

Water Type	Number of Samples		Maximum Concentration (ppb)
	Total	>0.05 ppb	
Total Groundwater	2348	2	

It is noteworthy that there were only 18 detections of acifluorfen in the nearly 4,200 samples analyzed for acifluorfen. More detections and highest concentrations were found in surface water than in groundwater. In light of all these monitoring data, it is unreasonable to choose the single highest concentration value from a small agricultural stream as representative of all drinking water. Accordingly, using the conservative assumption that all drinking water contains acifluorfen at 0.00044 ppm, the highest value in the USGS NAWQA data on acifluorfen from large streams, a very conservative estimate of risk can be made. Using standard assumptions about body weight and water consumption, the chronic exposure from this drinking water would be 1.26 x 10^{-5} mg/kg bw/day for adults, 0.1% of the RfD of 0.013 mg/kg bw/day.

Chronic exposure to drinking water:

i. *Lactofen*. Less than 0.25% for the U.S. Population.

ii. *Acifluorfen*. Less than 0.1% for the U.S. Population.

1. *Summary- cumulative aggregate chronic dietary risk*— i. U.S. population. The aggregate chronic dietary risks from both food and drinking water exposure expressed as a percentage of their respective RfD values is presented below for both lactofen and acifluorfen. It is noteworthy that the calculated exposures and consequent risks are very small, yet dominated by the very conservative estimates of residues in water.

ii. *Lactofen*. Exposure 0.000005 (mg/kg bw/day) less than 0.25% for all subpopulations.

iii. *Acifluorfen*. Exposure 0.0000178 (mg/kg bw/day) less than 0.14 % for all subpopulations.

EPA generally has no concern for exposures below 100% 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 two chemicals, even when considered collectively, represent a minimal chronic toxicological risk to the general public and it can be concluded that there is reasonable certainty of no harm from chronic exposures..

2. *Acute*. Assessment of aggregate acute exposure to food and drinking water residues of lactofen to the U.S. Population has demonstrated that exposures are small. MOE values using very conservative exposure assumptions and a conservative toxicity endpoint are all greater than 1,000 and it can be concluded that there is reasonable certainty of no harm to the U.S. Population from acute dietary exposures to lactofen residues.

3. *Carcinogenicity*. Carcinogenic risks for both lactofen and acifluorfen can be calculated from the aggregate chronic dietary exposures presented above. Because both products are only used in agriculture, the exposure to the general population is exclusively dietary from potential residues in food and drinking water.

4. *Food*. For lactofen, carcinogenic risks from exposure to residues in food were calculated by Valent using a potency factor (Q_1*) of 0.17 (mg/kg/day) $^{-1}$. The resulting carcinogenic risk from existing and proposed uses of lactofen was calculated at 1.54×10^{-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 from lactofen residues in food are conservative and are well within acceptable levels.

For acifluorfen, carcinogenic risks from exposure to residues in food were published by EPA (61 FR 16740; April 17, 1996) (FRL-5356-6) using a Q_1* value of 0.107 (mg/kg/day)$^{-1}$. The resulting carcinogenic risk from existing and proposed uses of acifluorfen is calculated at 5.6×10^{-7} or less. This is lower than the generally acceptable level of one-in-a-million additional lifetime cancers.

5. *Drinking water*. In the discussions above, very conservative estimates of lactofen and acifluorfen residues in potential drinking water have been presented. The estimates are conservative in that common concentrations of the compounds in real drinking water are zero, or orders of magnitude below the estimates. Using the conservative exposure estimates and the corresponding cancer potency factors, the cancer risk from drinking water is 8.5×10^{-7} and 6.7×10^{-6} or less for lactofen and acifluorfen, respectively.

6. *Summary- cumulative aggregate chronic cancer risk*— i. *U.S. population*. The aggregate chronic dietary risks of cancer from exposure to food and drinking water residues is presented below for both lactofen and acifluorfen.

ii. *Lactofen*. Chronic Exposure less than 0.000005 mg/kg bw/day Q* 0.17 (mg/kg bw/day)$^{-1}$ Cancer Risk: 8.5×10^{-7}.

iii. *Acifluorfen chronic exposure*. Less than 0.0000178 mg/kg bw/day Q* 0.107 (mg/kg bw/day)$^{-1}$ Cancer Risk 1.9×10^{-6}.

It is noteworthy that the calculated exposures and consequent risks are dominated by the very conservative estimates of potential residues in water. The Agency has expressed concern about the potential for excess oncogenic risk of acifluorfen in drinking water. To evaluate drinking water exposures, groundwater monitoring studies have been required for both acifluorfen and lactofen. Additional time is required to allow registrants to complete the studies, to present real data in potential drinking water, and for EPA to evaluate the information and adequately address the drinking water exposure issue. The calculated cancer risks are for lifetime exposure to levels of all potential acifluorfen in drinking water little of which could possibly be attributable to lactofen use on cotton. There is a reasonable certainty of no harm during the time necessary to obtain and evaluate real exposure data.

7. *Non-dietary exposure*. Lactofen and acifluorfen are currently approved only for the commercial production of agricultural crops. The potential for non-occupational exposure to the general public, other than through the diet or drinking water, is therefore insignificant.

8. *Infants and children — Safety factor for infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of lactofen, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to 10-fold, for added protection for infants and children in the case of threshold

effects unless EPA determines that a different margin of safety will be safe for infants and children. The toxicological data base for evaluating pre- and post-natal toxicity for lactofen is complete with respect to current data requirements. There are no special pre- or post-natal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies and the reproductive toxicity study in rats. Systemic toxicity effects, and not reproductive or developmental toxicity determined the no effect levels for these studies of 50, 4, and 2.5 mg/kg bw/day, respectively. Valent concludes that reliable data support use of the standard 100-fold uncertainty factor with respect to protection of infants and children, and that an additional uncertainty factor is not needed to be further protective.

Furthermore, the chronic RfD for lactofen is based on the Lowest Effect Level (LEL) of 1.5 mg/kg/day in the 18-month mouse feeding study with an uncertainty factor of 1,000. An additional margin of safety, 10-fold, was used since a clear NOEL was not established in the mouse study. Thus, although an extra safety factor is not needed to further protect infants and children, an extra 10-fold uncertainty factor has been included because of the lack of a clear NOEL in the mouse study.

9. *Chronic— Food*. Using the dietary exposure assessment procedures described above (and performed by Valent) for lactofen, and a recent assessment for acifluorfen published in the **Federal Register** (61 FR 16740; April 17, 1996) total chronic dietary exposures resulting from existing and proposed uses of lactofen and acifluorfen were compared to their respective reference doses. The following contributions to the RfD were found for all of subpopulations including infants and children for which dietary consumption data are available:

i. *Lactofen*. Exposure 0.0000001 (mg/kg bw/day) less than 0.01% of RfD.

ii. *Acifluorfen*. Exposure 0.0000052 (mg/kg bw/day), (61 FR 16740; April 17, 1996) less than 0.04% of RfD.

10. *Chronic- drinking water- lactofen*. Using the conservative assumption that all drinking water contains lactofen at levels calculated by GENEEC for a small farm pond surrounded by lactofen treated fields, a very conservative estimate of risk can be made. Using standard assumptions about body weight and water consumption, the child chronic exposure from this drinking water would be 1.7×10^{-5} mg/kg bw/day, 0.85 percent of the RfD.

11. *Acifluorfen*. Using the very conservative assumption that all drinking water contains acifluorfen at 0.00044 ppm, from the USGS NAWQA data on acifluorfen, a very conservative estimate of risk can be made. Using standard assumptions about body weight and water consumption, the child chronic exposure from this drinking water would be 4.4×10^{-5} mg/kg bw/day, 0.34 percent of the RfD.

Summary - Cumulative aggregate chronic dietary risk— Infants and children. The aggregate chronic dietary risks from both food and drinking water exposure expressed as a percentage of their respective RfD values is presented below for children for both lactofen and acifluorfen. It is noteworthy that the calculated exposures and consequent risks are very small, yet dominated by the very conservative estimates of residues in water.

(a) *Lactofen*. Less than 0.86 % for all infant and children subpopulations.

(b) *Acifluorfen*. Less than 0.38 % for all infant and children subpopulations.

EPA generally has no concern for exposures below 100% 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 two chemicals, even when considered collectively, represent a minimal chronic toxicological risk to infants and children and it can be concluded that there is reasonable certainty of no harm from chronic exposures.

1. *Acute*. Assessment of aggregate acute exposure to food and drinking water residues of lactofen to non-nursing infants has demonstrated that exposures are small. MOE values using very conservative exposure assumptions and a conservative toxicity endpoint approximate 1,000. It can be concluded that there is reasonable certainty of no harm to infants and children from acute dietary exposures to lactofen residues.

G. International Tolerances

There are no Codex Maximum Residue Limits (MRL) established for lactofen on any commodity.

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ENVIRONMENTAL PROTECTION AGENCY

[OPPTS–00232; FRL–5770–1]

Lithographic Printing Industry Pollution Prevention and Risk Reduction Materials

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of Availability.

SUMMARY: The EPA's Design for the Environment (DfE) Program is announcing the availability of two documents providing pollution prevention and human health and environmental risk reduction information for the lithographic printing industry. The two documents being made available are:

The Cleaner Technologies Substitutes Assessment (CTSA): Lithographic Blanket Washes (document number EPA 744–R–97–006) is a comparison of 37 different blanket wash formulations in terms of performance, cost, risk, resource conservation and other aspects. The CTSA contains the technical data and analyses of the DfE Lithography Project. A draft of this report was released in September 1996 and comments have been addressed in this final version.

Solutions for Lithographic Printers: An Evaluation of Substitute Blanket Washes (document number EPA 744–F–96–003) is a simple, user friendly summary of the information developed through the DfE Lithography Project. This booklet will help printers to choose the best blanket wash for their facilities. The 35 page document describes how to identify, select and use substitute blanket washes and other ways to reduce pollution in a lithographic printing facility.

ADDRESSES: Both documents are available free of charge for a limited time from the Pollution Prevention Information Clearinghouse (PPIC), Environmental Protection Agency (7409), 401 M St., SW., Washington, DC 20460 telephone 202–260–1023, fax 202–260–4659 and e-mail at ppic@epamail.epa.gov. Also, both documents will be viewable and downloadable from the DfE Program web site at HTTP://www.epa.gov/dfe after March 14, 1998.

FOR FURTHER INFORMATION CONTACT: Karen Seeh, Economics, Exposure, and Technology Division, Office of Pollution Prevention and Toxics, (7406), Environmental Protection Agency, 401 M St. SW., Washington, DC 20460, telephone 202–260–1714, fax 202–260–