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. Despite the potential for exposure to S-metolachlor in drinking water and from non-dietary, non-occupational exposures, the assessment presented above demonstrates that the high levels of safety exist for current and proposed uses of S-metolachlor; it is not expected that aggregate exposure from all sources will exceed 100% of the RfD. Therefore, one can conclude there is a reasonable certainty that no harm will result from aggregate exposure to S-metolachlor.

2. *Infants and children*. FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to prenatal and postnatal effects for children is complete. A full consideration of the available reproductive toxicity data supporting S-metolachlor demonstrates no increased sensitivity to infants and children. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that the cRfD at 0.1 mg/kg/day is appropriate for assessing aggregate risk to infants and children from use of S-metolachlor.

Based on the aggregate assessment described above, the percent of the cRfD that will be utilized by aggregate exposure to residues of S-metolachlor is less than 0.2% for non-nursing infants and children 1 to 6 years old, and 0.1% for children 7 to 12 years old. 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. Despite the potential for exposure to S-metolachlor in drinking water and from non-dietary, non-occuptional exposure, the assessment described above demonstrates that it is not expected that aggregate exposure from all sources provides for a large margin of safety and will exceed 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the exposure assessment, it is concluded there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to S-metolachlor residues.

F. International Tolerances

There are no Codex Alimentarius Commission maximum residue levels (MRL's) established for residues of S-metolachlor in or on raw agricultural commodities.

[FR Doc. 03–2019 Filed 1–28–03; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0001; FRL–7287–6]

Lactofen; Notice of Filing Pesticide Petitions to Establish Tolerances for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket ID number OPP–2003–0001, must be received on or before February 28, 2003.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Joanne I. Miller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6224; e-mail address: *miller.joanne@epamail.epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0001. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in EPA's Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper

form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2003–0001. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to *opp-docket@epa.gov*, Attention: Docket ID Number OPP–2003–0001. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM*. You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) 7502C, Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID Number OPP–2003–0001.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, Attention: Docket ID Number OPP–2003–0001. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI to the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

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

List of Subjects

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

Dated: January 17, 2003.

Debra Edwards,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The petitioner's summaries of the pesticide petitions are printed below as required by FFDCA section 408(d)(3). The summaries of the petitions were prepared by the petitioner and represent the views of the petitioner. The petitions summaries announce the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemicals residues or an explanation of why no such method is needed.

Valent U.S.A. Corporation

PP 8F3591 and PP 9F3798

EPA has received pesticide petitions (8F3591 and 9F3798) from Valent U.S.A. Corporation, 1333 North California Boulevard, Suite 600, Walnut Creek, California 94596–8025 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR 180.432 by establishing tolerances for residues of the herbicide lactofen, 1-(carboethoxy)ethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, in or on the raw agricultural commodities (RACs) cottonseed at 0.01 part per million (ppm), cotton gin byproducts at 0.02 ppm, and peanut nutmeats at 0.01 ppm. EPA has determined that the petitions contain 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 support granting of the petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants is adequately understood based on plant metabolism studies on cotton, peanut, soybean, and tomato. The Health Effects Division (HED) Metabolism Assessment Review Committee (MARC) met on April 4, 2000, considered all of the metabolism studies submitted to date and concluded that only the parent compound needs to be regulated for plant commodities, provided that pre-harvest intervals exceed 45 days.

2. *Analytical method*. Adequate analytical methodology is available for detecting and measuring levels of lactofen in or on RACs with a limit of detection (LOD) that allows monitoring of food with residues at or above the level of the proposed tolerances. The method, RM–28D, has been successfully radio validated in conjunction with a tomato metabolism study and has undergone a successful independent laboratory validation trial. This method was also successfully validated by EPA's Analytical Chemistry Laboratory using peanut nutmeats and cottonseed. In general, the analytical method has a LOD of 0.005 ppm and limit of quantitation (LOQ) of 0.01 ppm in crops.

3. *Magnitude of residues*. Adequate lactofen residue data are available for cotton and peanuts. An adequate number of field trials distributed throughout cotton and peanut growing areas of the United States have been conducted on these crops to determine lactofen residues resulting from the application of lactofen at the maximum labeled or proposed use rate.

i. *Cotton*. Residues of lactofen were each <0.01 ppm, in/on cottonseed (n=14) harvested 59–127 days following a single postemergence soil-directed application of lactofen at 0.4 lb active ingredient per acre (lb active ingredient/acre) (2x the single application rate) and in/on cottonseed (n=10) harvested 23–108 days following the last of two postemergence directed applications at 0.4 lb active ingredient/acre application (2x the maximum seasonal rate). With one exception, residues of lactofen were also each <0.01 ppm, in/on cotton gin byproducts (gin trash) (n=11) derived from cotton harvested 69–108 days following two applications at 0.2 lb active ingredient/acre. One gin trash sample bore residues of lactofen at 0.03 ppm, but confirmatory analyses of this sample detected lactofen at <0.01–0.02 ppm, and residues of lactofen were <0.01 ppm, in the duplicate treated sample from the same trial.

In a single processing study, residues of lactofen were <0.01 ppm, in/on cottonseed harvested 76 days following the last of two directed applications of lactofen at 0.6 lb active ingredient/acre application (1.2 lb active ingredient/acre/season, 3x rate). Residues of lactofen were <0.01 ppm in samples of meal, hulls, oil, (crude and refined) and soapstock.

All these data support proposed tolerance for lactofen in/on cottonseed at 0.01 ppm, and in/on cotton, gin byproducts at 0.02 ppm. No separate tolerances are needed for cotton processed commodities.

ii. *Peanuts*. In 8 field trials, residues of lactofen were each <0.01 ppm, in/on 16 samples of peanut nutmeats and hulls harvested 65–71 days following the last of 2 broadcast applications of lactofen totaling 0.45 lb active ingredient/acre (1x the maximum proposed rate). Residues of lactofen were also <0.01 ppm, in/on peanut nutmeats and hulls from 2 trials conducted at 2x and 5x the maximum seasonal rate.

In a processing study, residues of lactofen were <0.01 ppm in meal, oil, crude and refined, and soapstock processed from nutmeats treated at 3x and 5x the maximum proposed use rates.

All these data support proposed tolerance for lactofen in/on peanut nutmeats at 0.01 ppm. No separate tolerances are needed for peanut processed commodities.

B. Toxicological Profile

1. *Acute toxicity*. Lactofen has very low acute toxicity. The acute oral LD_{50} is 5.96 gram/kilogram/body weight (g/kg/bwt) toxicity category IV, the acute dermal LD_{50} is >2.0 g/kg/bwt toxicity category III and the acute inhalation LD_{50} is >6.3 milligrams/liter (mg/L) toxicity category IV. Lactofen is not a

skin sensitizer but is a very slight dermal irritant.

2. *Genotoxicity*. Lactofen has very little mutagenic or genotoxic activity. While a positive mutagenic response was reported in one trial of a *Salmonella typhimurium*/mammalian microsome mutagenicity assay, this response was not repeated in the second assay conducted. In addition, lactofen did not appear to induce chromosomal aberrations, unscheduled deoxyribonucleic acid (DNA) synthesis or inhibit DNA repair.

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.

In a 2–generation reproduction study in rats, decreased pup weight and decreased absolute and relative weights of the spleen were first reported at approximately 26.2 milligrams/kilogram/day (mg/kg/day) (based on dose administered to the parental group). The same dose level elicited mortality and decreased male fertility in the parental groups. The no observed adversed effect level (NOAEL) for both systemic and reproductive toxicity in this study was 2.6 mg/kg/day.

In the developmental toxicity study in rats, effects were observed at the 150 mg/kg/day dose level consisting of decreases in fetal weight as well as skeletal abnormalities. This dose level also elicited signs of toxicity in the parental group. The NOAEL for this study was 50 mg/kg/day. Based on this NOAEL and an uncertainty factor (UF) of 100, the acute reference dose (RfD) for lactofen has been set at 0.50 mg/kg/day.

Two developmental toxicity studies were conducted in rabbits. In the first study, pregnant rabbits were administered oral doses of 0, 5, 15, or 50 mg/kg bwt/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. The Agency concluded that the data were insufficient to establish a clear NOAEL. In the second rabbit developmental toxicity study, pregnant rabbits were exposed to 0, 1, 4, or 20 mg/kg bwt/day oral doses on days 6–18 of gestation. Maternal toxicity (reduced food consumption) was observed at 20 mg/kg bwt/day, but no developmental effects were observed at any dose. Therefore, the maternal NOAEL was 4 mg/kg bwt/day and the developmental NOAEL was greater than 20 mg/kg bwt/day.

4. *Subchronic toxicity*—i. *Rats 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 4 weeks. A slight increase in spleen weight was the basis for a lowest observed adversed effect level (LOAEL) of 200 ppm, lowest dose tested (LDT). At doses of 1,000 ppm, or higher, the following findings were reported: clinical signs of toxicity; decreased red blood cell (RBC), hemoglobin, hematocrit, and increased white blood cell (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.

ii. *Rats 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, dosage. Decreased RBC, hemoglobin and hematocrit values were also observed at 1,000 ppm. The NOAEL in this study was 200 ppm, 14.1 mg/kg/day.

iii. *Dogs 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 blood urea nitrogen (BUN) and serum glutamic-pyruvic transaminase (SGPT). Food palatability problems led to greatly decreased feed consumption at higher dosages. The NOAEL appeared to be 1,000 ppm.

iv. *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 LOAEL was 200 ppm, 28.6 mg/kg/day based on:

- Increased WBC; decreased hematocrit, hemoglobin and RBC.
- Increased alkaline phosphatase, serum glutamic-oxloacetic transaminase (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.

v. *Peroxisome proliferation*. 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. 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. 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 bwt/day administered for 93 days. Therefore, Valent believes that 75 mg/kg bwt/day is a clear NOAEL for peroxisome proliferation observed in a species closely related to man. On January 17, 2001, the Mechanism of Toxicity Assessment Review Committee (MTARC) reviewed the merits of the toxicological data supporting peroxisome proliferation as the proposed mode of action for lactofen. Based on the weight-of-evidence from guideline, as well as mechanistic studies, the MTARC concluded that there are sufficient data to classify lactofen as a non-genotoxic hepatocarcinogen in rodents with peroxisome proliferation being a plausible mode of action.

5. *Chronic toxicity*. Lactofen 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.

i. *Mouse 18–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, a dose that clearly exceeded the maximum tolerance dose (MTD). The lowest dose, 10 ppm, 1.4 mg/kg/day, was the LOAEL based on increased liver weight and hepatocytomegaly.

ii. *Rat 24–month*. In a 2–year chronic feeding/oncogenicity study of lactofen technical in rats at dosages of 0, 500, 1,000 ppm; 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 NOAEL for systemic toxicity is 500 ppm, 2 mg/kg/day based on kidney and liver pigmentation.

iii. *Dog 12–month*. In a 1–year study in dogs exposed to 40, 200, and 1,000 ppm; week 1–17 or 3,000 ppm; week 18–52 lactofen technical in their diet, the NOAEL was determined to be 200 ppm, (0.79 mg/kg/day) based on renal dysfunction and decreased RBC, hemoglobin hematocrit and cholesterol observed at 1,000/3,000 ppm. Based on this NOAEL and an uncertainty factor (UF) of 100, the chronic RfD for lactofen has been set at 0.008 mg/kg/day.

iv. *Carcinogenicity*. As a member of the diphenyl ether chemical family, lactofen is structurally related to four other chemicals that are oncogenic in rodents:

- Sodium acifluorfen (acifluorfen is a lactofen metabolite), nitrofen, oxyfluorfen, and fomesafen.
- Sodium acifluorfen produces hepatocellular adenomas and carcinomas in mice but is negative in rats.
- Nitrofen produces hepatocellular carcinomas in mice and pancreatic carcinomas in rats.
- Oxyfluorfen produces marginally positive liver tumors in mice but is negative in rats.
- Fomesafen produces hepatocellular adenomas and carcinomas in mice.

The Cancer Peer Review Committee (CPRC) evaluated the relevant data on the carcinogenic potential of lactofen in 1987 and classified lactofen as a B2 carcinogen Probable Human Carcinogen and assigned a Cancer Potency Factor (Q_1*) of 1.7 x 10^{-1} mg/kg/day^{-1}, based on a interspecies scaling factor of 0.67. This Q_1* has since been reduced to 1.19 x 10^{-1} mg/kg/day^{-1} based on recent EPA guidance indicating that 0.75 is a more appropriate interspecies scaling factor. The B2 classification is based on an increase in the combined incidence of liver adenomas and carcinomas in mice and increases in liver neoplastic nodules and foci of cellular alteration (possible precursor of tumors) in rats. In 1996, and 1999, EPA proposed new cancer risk assessment guidelines which state that nonmutagenic carcinogens known to cause cancer via a threshold mechanism, such as peroxisome proliferation, could be assessed using a nonlinear margin of exposure (MOE) approach rather than the Q_1 * method. EPA has recently determined that lactofen acts via a peroxisome proliferation mechanism and is currently reevaluating its approach to the quantification of the cancer risk for lactofen.

6. *Animal metabolism*. In a rat metabolism study, lactofen was shown to metabolize to acifluorfen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate, which was eliminated via both urine and feces. While lactofen was the primary compound found in the feces, acifluorfen accounted for >90% of the radioactivity in the urine. Negligible amounts of the administered radioactivity were found in any tissue with less than 0.8% of the administered radioactivity being found in the liver one of the main target organs.

7. *Metabolite toxicology*. Acifluorfen is also a hydrolytic metabolite of lactofen. The sodium salt of this benzoic acid is the registered herbicide, sodium acifluorfen. This product has a complete data base supporting registration with a RfD of 0.013 mg/kg/day and a Q_1* of 5.30 x 10^{-2} mg/kg/day^{-1}. Because lactofen and its metabolites are not retained in the body, the potential for acute toxicity from *in situ* formed metabolites is low. The potential for chronic toxicity of lactofen metabolites has been adequately addressed by an extensive battery of lactofen chronic toxicity testing.

8. *Endocrine disruption*. No special studies to investigate the potential for estrogenic or other endocrine effects of lactofen have been performed. However, 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.

C. Aggregate Exposure

1. *Dietary exposure*. A full battery of toxicology testing, including studies of acute, chronic, oncogenicity, developmental, mutagenicity, and reproductive effects is available for lactofen. For the following risk assessments, the NOAEL from the chronic oral toxicity study in dogs, 0.79 mg/kg/day, was selected as the chronic oral toxicity endpoint. Based on this NOAEL, and an UF of 100, the chronic RfD and the chronic population adjusted dose (cPAD) for lactofen has been set at 0.008 mg/kg/day. The NOAEL from the rat developmental study, 50 mg/kg/day, was selected as the acute oral toxicity endpoint. Based on this NOAEL and an UF of 100, the acute RfD for lactofen has been set at 0.50 mg/kg/day. An acute adjusted dose (aPAD) of 0.17 mg/kg/day was calculated using this endpoint and an additional Food Quality Protection Act (FQPA) safety factor of 3. This aPAD will only be used to assess acute exposures to the females 13 to 50 year old population subgroup since it is derived from a developmental toxicity endpoint. No other acute endpoints were identified to assess acute exposures to other populations.

i. *Food*. Dietary risk was considered for the currently registered uses of lactofen on soybeans, snap beans, and cotton and for the pending use on peanuts. Dietary risk assessments were done using the Dietary Exposure Evaluation Model (DEEM™), which incorporates consumption data generated in U. S. Department of Agriculture (USDA) Continuing Surveys of Food Intakes by Individuals (CSFII), 1989–1992. For chronic dietary risk assessments, the 3–day average of consumption for each subpopulation is combined with residues in commodities to determine average exposure in mg/kg/day. For refined acute dietary risk assessments, the entire distribution of consumption events for individuals is multiplied by a distribution of residues to obtain a distribution of exposures in mg/kg/day. This is a probabilistic analysis, referred to as ''Monte Carlo,'' and the risk is reported at the 99.^{9th} percentile of exposure. Food monitoring data are not available from Food and Drug Administration (FDA) or USDA for residues of lactofen. Therefore, only field trial data were used. A value of one-half the LOQ, 0.005 ppm, was used to represent the residues in all treated commodities. Percent crop treated (PCT) were incorporated for soybeans and snap beans, as reliable usage information was available for these commodities. The estimated risk from food is presented in the following table:

TABLE 1.—DIETARY EXPOSURE AND RISK TO LACTOFEN FROM FOOD SOURCES

Population	Acute Endpoint		Chronic Endpoint		Cancer Endpoint[2]	
	Exposure mg/kg/day	%aPAD	Exposure mg/kg/day	%aPAD	Exposure mg/kg/day	Risk
U.S. population	NA[1]	NA	1×10^{-6}	<0.1	1×10^{-6}	8×10^{-8}
Females 13 to 50	2×10^{-6}	<0.1	$<1 \times 10^{-6}$	<0.1	NA	NA
Children 1 to 6	NA[1]	NA	2×10^{-6}	<0.1	NA	NA

[1]Acute endpoint applies only to females of childbearing age.
[2]Cancer risk is generally reported for the U.S. population.

ii. *Drinking water*. Environmental fate properties indicate that lactofen is not very persistent or mobile. Hydrolysis half-lives are 10.7, 4.6, and <1.0 days at pH 5, 7, and 9 at 40° C, respectively. This temperature most likely exceeds temperatures that lactofen would be expected to be exposed to under normal conditions, thus the hydrolysis rates are probably slower. Aerobic soil metabolism half-lives range from 1 to 3 days. Lactofen has a low probability to contaminate drinking water because it has a short half-life (3 days or less) and high binding potential(K_{oc}>1,000). Limited data suggest that lactofen conversion to acifluorfen in water is approximately 52%. The HED MARC has concluded that the residues of concern in drinking water are acifluorfen and amino acifluorfen. Insufficient information is available to estimate the amino acifluorfen concentration in water, but it is likely to be less than that of acifluorfen. Laboratory studies have shown that acifluorfen reaches its maximum concentration of 53.3% of applied lactofen at 7 days following application and it is most likely to form under the soil surface. Thus, the formed acifluorfen is not subject to drift, erosion, or runoff forces that contribute to surface water contamination. Surface water, however, could be contaminated with acifluorfen from lactofen applications via spray drift. The registrant also has conducted two prospective ground water studies which showed that neither lactofen nor acifluorfen from lactofen applications contaminate ground water. Therefore, in the following discussion, the potential exposure to lactofen from drinking water will address only potential surface water contamination with lactofen and acifluorfen.

The Tier II estimated environmental concentration (EEC) assessment in surface water uses a single site, or multiple single sites, which represents a high-end exposure scenario from pesticide use on a particular crop or non-crop use site. The EEC's for lactofen were generated for the standard Mississippi cotton scenario. The Agency has implemented the concept of index reservoirs (IR) and the PCT area to better estimate potential residue level in drinking water sources. The scenarios used with EPA pesticide root zone model (PRZM) and exposure analysis modeling systems (EXAMS) to estimate lactofen in the ''standard pond'' were rerun with the IR for the cotton and soybean scenarios. The Agency has estimated that the PCT area for the Mississippi cotton scenario is 0.20 (20%).

The Office of Pesticide Programs (OPP) has calculated drinking water levels of comparison (DWLOCs) for acute and chronic exposure to lactofen and acifluorfen from applications of lactofen in surface water. To calculate the DWLOC for acute exposure, the acute dietary food exposure from the DEEM™ analysis was subtracted from the aPAD. To calculate the DWLOC for chronic (non-cancer) exposure, the chronic dietary food exposure from the DEEM™ analysis was subtracted from the cPAD to obtain the acceptable chronic non-cancer exposure to lactofen and acifluorfen in drinking water. A DWLOC cancer was calculated in a similar manner, assuming a negligible risk of 1×10^{-6}. Assumptions used in calculating the DWLOCs include 70 kg bwt for the U.S. population, 60 kg bwt for adult females, 10 kg bwt for children, 2 liters of water consumption per day for adults, and 1 liter consumption for children.

TABLE 2.—DIETARY EXPOSURE AND RISK TO LACTOFEN FROM DRINKING WATER

Population	Acute Endpoint		Chronic Endpoint		Cancer Endpoint[2]	
	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L
U.S. population	NA[1]	NA	0.022	280	0.012	0.3
Females 13 to 50	0.62	5,100	0.022	240	-	-
Children 1 to 6	NA[1]	NA	0.022	80	-	-

[1] Acute endpoint applies only to females of childbearing age.
[2] Cancer risk is generally reported for the U.S. population.

TABLE 3.—DIETARY EXPOSURE AND RISK TO ACIFLUORFEN[1] FROM DRINKING WATER

Population	Acute Endpoint		Chronic Endpoint		Cancer Endpoint[3]	
	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L
U.S. population	NA[2]	NA	0.99	140	0.34	0.7
Females 13 to 50	4.9	600	0.99	120	-	-

TABLE 3.—DIETARY EXPOSURE AND RISK TO ACIFLUORFEN[1] FROM DRINKING WATER—Continued

Population	Acute Endpoint		Chronic Endpoint		Cancer Endpoint[3]	
	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L	Exposure µg/L	DWLOC µg/L
Children 1 to 6	NA[2]	NA	0.99	40	-	-

[1] Acifluorfen derived from applications of lactofen.
[2] Acute endpoint applies only to females of childbearing age.
[3] Cancer risk is generally reported for the U.S. population.

HED has a concern if the DWLOC for any scenario is below the estimated environmental concentration from the models. All of the DWLOCs shown in the tables above exceed the estimated EECs.

2. *Non-dietary exposure.* Lactofen is proposed only for agricultural uses and no home owner or turf uses. Thus, no non-dietary risk assessment is needed.

D. 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 include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way.

There are other pesticidal compounds that are structurally related to lactofen and have similar effects on animals. In consideration of potential cumulative effects of lactofen and other substances that may have a common mechanism of toxicity, there are currently no available data or other reliable information indicating that any toxic effects produced by lactofen would be cumulative with those of other chemical compounds. Thus, only the potential risks of lactofen have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of lactofen consistent with the schedule established by EPA in the **Federal Register** of August 4, 1997 (62 FR 42020) (FRL-5734–6), and other subsequent EPA publications pursuant to FQPA.

E. Safety Determination

1. *U.S. population.* Water is not expected to be a significant source of exposure for lactofen, as it degrades quickly in the environment to numerous degradates, including acifluorfen. EECs for lactofen and acifluorfen are well below the DWLOC for chronic, acute, and cancer risk. Therefore, the only significant source of human exposure to lactofen is in food. Residues of lactofen are generally non-detectable at a LOQ of 0.005 ppm, in all food forms. The exposure is <0.1% of the acute and chronic PAD for all population subgroups. Exposure is generally not of concern if it is less than 100% of the PAD. The estimated cancer risk for the U.S. population is 8×10^{-8}, which is more than an order of magnitude less than the risk that is generally considered negligible 1×10^{-6}.

2. *Infants and children.* As stated above, dietary exposure assessments, including drinking water, utilize less than 0.1% of the acute and chronic PADs for all population subgroups, including infants and children. Reproduction and developmental effects have been found in toxicology studies for lactofen but only 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. The FQPA Safety Factor Committee met on March 13, 2000 to evaluate the hazard and exposure data for lactofen and recommended that FQPA, safety factor for protection of infants and children should be reduced to 3x for lactofen. This safety factor was reduced to 3x by The FQPA, Safety Factor Committee because available data provide no indication of quantitative or qualitative increased susceptibility from *in utero* and/or postnatal exposure to lactofen in rats. Information on the reproduction and developmental effects caused by the other diphenyl ether herbicides is not available to Valent. Additional time is needed 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 established for lactofen on cotton or peanut commodities, so there is no conflict between this proposed action and international residue limits.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–7445–4; RCRA–2002–0029]

Land Disposal Restrictions: Treatment Standards for Mercury-Bearing Hazardous Waste; Notice of Data Availability

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of data availability.

SUMMARY: This notice of data availability (NODA) makes available to the public two studies conducted on the treatment of mercury wastes. The studies were initiated to help evaluate whether EPA could propose treatment and disposal alternatives to the current land disposal restriction (LDR) treatment standard of mercury retorting. The studies were performed to assess conditions that affect the stability of waste residues resulting from the treatment of high mercury (greater than 260 mg/kg total mercury) wastes. This NODA also makes available the results of the peer review of these studies. As a result of our investigation, we have concluded that changes to our national regulations are impractical at this time. Additionally, this notice also provides information on how to use the existing treatability variance procedures to make site-specific choices on alternatives to mercury recovery. The treatability studies and the results of the peer review are presented here only to provide information—we are not