C. Mammalian Toxicological Profile

Bacillus thuringiensis proteins have been used commercially for more than 30 years without any evidence for adverse health effects. *Bacillus thuringiensis* mode-of-action can be divided into a series of critical steps: ingestion by the insect, specific binding to brush border membrane receptors, membrane insertion, and pore formation thus destroying the midgut lining and causing death of the insect. *Bacillus thuringiensis* proteins do not bind or cause these types of effects to mammalian gut membranes. The extensive mammalian toxicity studies performed to support the safety of *Bacillus thuringiensis* - containing pesticides clearly demonstrate that the tested isolates are not toxic or pathogenic (McClintock, *et al.*, 1995, Pestic. Sci. 45:95–105). Although *Bacillus thuringiensis* strains have been used for decades as sprayable microbial products, no confirmed cases of allergic reactions have been documented, despite dermal, oral and inhalation exposures. A reference to this is made by the EPA in a **Federal Register** notice, dated August 16, 1995 (60 FR 42443) (FRL–4971–3).

The Cry9C protein insecticidal mode-of-action is apparently similar to that of the well known Cry1A proteins. In addition to the safe history of *Bacillus thuringiensis* proteins outlined above, several other studies were performed to evaluate mammalian safety of the Cry9C protein. An acute toxicological study was performed with mice, which demonstrated that the Cry9C protein had an LD_{50} >6,500 mg/kg. A test for *in vitro* digestibility under simulated gastric conditions showed that the Cry9C protein found in bacteria and the protein produced in plants was stable for 4 hours when exposed to simulated gastric juice. However, an amino acid sequence homology search performed using three different data banks (against 135,867 sequences) only found homology to other related *Bacillus thuringiensis* proteins. To determine possible short stretch homology, an 8-amino acid homology search was also performed. Except with the *Bacillus thuringiensis* proteins, no identical 8-amino acid peptide sequences could be detected in the searches. Therefore, it is unlikely that Cry9C protein would have significant allergenic potential.

The Cry9C protein or metabolites of the protein are not expected to interact with the immune or endocrine system, since the protein sequence does not match any known allergens or hormones. Since proteins, in general, are not known to be carcinogenic it is unlikely that the Cry9C protein would have carcinogenic properties.

All living organisms contain DNA and there are no examples of nucleic acids causing any toxicological effects from dietary consumption. The genetic material necessary for the production of the Cry9C protein in plants includes the genetic construct that encodes the Cry9C protein and all other necessary genetic elements for it's expression. These elements include: a promotor, polylinker sequences, leader sequences and terminators and none of which are expected to cause any toxicological effects.

Taken together, the data supports the lack of mammalian toxicological effects for the plant-pesticide *Bacillus thuringiensis* subsp. *tolworthi* Cry9C protein and the genetic material necessary for the production of this protein in corn for feed use only.

D. Aggregate Exposure

Since the Cry9C protein is expressed in plant tissues, dermal or inhalation will be negligible to non-existent. Drinking water is unlikely to be contaminated with Cry9C protein due to the rapid degradation of plant materials in the soil. Furthermore, no direct human dietary exposure to Cry9C protein will occur since this request is for animal feed use only.

E. Cumulative Effects

The unique mode-of-action of Bt proteins in general, coupled with the lack of mammalian toxicity for the Cry9C protein provides no basis for the expectation of cumulative effects with other compounds.

F. Safety Determination

Bt microbial pesticides containing Cry proteins have been applied for more than 30 years to food and feed crops consumed by the US population. There have been no human safety problems attributed to Cry proteins. The extensive mammalian toxicity studies performed to support the safety of *Bacillus thuringiensis* - containing pesticides clearly demonstrate that the tested isolates are not toxic or pathogenic (McClintock, et al., 1995, Pestic. Sci. 45:95–105). The lack of mammalian toxicity of the Cry9C protein provides support for our request of a temporary exemption from the requirement of a tolerance set forth in this petition. Non-dietary exposure of infants, children or the US population in general, to the Cry9C protein expressed in corn plant materials, are not expected due to the uses of this product for animal feed use only.

G. Existing Tolerances

No tolerances or tolerance exemptions have been granted for the *Bacillus thuringiensis* subsp. *tolworthi* Cry9C and the genetic material necessary for the production of this protein in corn for feed use only. (Michael Mendelsohn)

[FR Doc. 97–31131 Filed 11–25–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–779; FRL–5755–6]

Notice of Filing of Pesticide Petition

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–779, must be received on or before December 26, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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

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

Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: James Tompkins, Registration Division (7505C) Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 265, CM #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305-7801; e-mail: tompkins.james@epamail.epa.gov.

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–779] (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 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–779] and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: November 4, 1997

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

Rhone-Poulenc Ag Company

PP 3F4233

EPA has received a pesticide petition (PP 3F4233) from Rhone-Poulenc Ag Company, 2 Alexander Drive, Research Triangle Park, NC 27709, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to extend the current time-limited tolerances for bromoxynil and its metabolite DBHA (3,5-dibromo-4-hydroxybenzoic acid) resulting from the application of octanoic and heptanoic acid esters of bromoxynil to cotton in or on the raw agricultural commodities undelinted cottonseed at 7 parts per million (ppm), cotton gin byproducts at 50 ppm, and cotton hulls at 21 ppm for a 1–year period and to increase the current acreage limitation from 3% to 10% of the U. S. cotton acreage (1,300,000 acres). 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*. The nature of the bromoxynil residue in bromoxynil-tolerant cotton is considered to be adequately understood. The two major components of the terminal residue are parent bromoxynil and the metabolite 3,5-dibromo-4-hydroxybenzoic acid (DBHA).

2. *Analytical method*. Adequate analytical methodologies for both parent bromoxynil and the DBHA are available for enforcement purposes. The method involves sample reflux in methanolic KOH, partitioning with ether/hexane and analysis by Gas Chromatography. Limits of quantitation allow monitoring of residues in cotton commodities at or above tolerance levels. Multiresidue testing with DBHA has been conducted and submitted to FDA.

3. *Magnitude of residues*. Available magnitude of the residue data from a 60 day phi crop field residue study conducted at a maximum application rate of 4.5 lb active ingredient/acre indicate that the currently established time-limited tolerances for bromoxynil and DBHA will not be exceeded when Buctril 4EC herbicide is used according to approved label directions.

B. Toxicological Profile

1. *Acute toxicity*. A complete battery of acute toxicity studies for bromoxynil (phenol) has been conducted. The acute oral toxicity study in rats resulted in a LD_{50} of 81 milligrams/kilogram (mg/kg) (males) and a LD_{50} of 93 mg/kg (females). The acute dermal toxicity study in rabbits resulted in a LD_{50} of >2,000 mg/kg for both males and females. The acute inhalation study in rats resulted in a LC_{50} of 0.269 milligram/liter (mg/L) for males and 0.150 for females. The primary eye irritation study showed corneal opacity resolved within 3 days, iritis resolved within 4 days and conjuctival irritation which persisted for 10 days. There was no irritation in the primary dermal irritation study and the dermal sensitization study in guinea pigs was negative.

2. *Genotoxicty*. Mutagenicity studies conducted include an unscheduled DNA synthesis study-rat primary hepatocytes (negative); *in vitro* transformation assay-mouse cells (negative); sister chromosomal exchange study-CHO cells (negative); forward mutation study-mouse lymphoma cells (negative without activation and positive with activation); DNA repair test-*E. Coli* (positive); *in vitro* chromosomal aberration (negative without activation and positive with activation); two separate micronucleus assays (both negative); forward mutation-CHO cells (negative); and *Salmonella typhimurium* reverse mutation assay (negative with and without activation). Rhone-Poulenc considers bromoxynil (phenol) and DBHA to be non-mutagenic.

3. *Reproductive and developmental toxicity*. A teratology study was conducted with rats administered (orally) bromoxynil phenol at dose levels of 0, 4, 12.5, or 40 mg/kg/day. The maternal no-observed-effect level (NOEL) and lowest-observed-effect level

(LEL) are 12.5 and 40 mg/kg/day respectively. The developmental NOEL and LEL are 4.0 and 12.5 mg/kg/day, respectively. Maternal body weights and food consumption were reduced in the high dose group. Fetal effects observed were reduced body weight, with associtaed decreases in ossification. An increase in 14th ribs was observed in the mid and high dose levels. A teratology study was conducted with rats administered (orally) bromoxynil phenol at dose levels of 0, 5, 15, or 35 mg/kg/day. The maternal NOEL and LEL are 5.0 and 15 mg/kg/day, respectively. The fetotoxicity and developmental NOEL and LEL are less than 5 and 5 mg/kg/day, respectively. Significant maternal mortality and decreased body weight gain were associated with the high dose, indicating that the maximum tolerance dose was exceeded. Decreases in maternal body weight gain were also observed in the mid and low dose levels. At the mid-dose level a statistically significant increase in the number of fetuses with supernumerary ribs, a common fetal variant was observed. A teratology study was conducted with rats administered (orally) bromoxynil phenol at dose levels of 0, 1.7, 5, or 15 mg/kg/day. The maternal NOEL and LEL are 5 and 15 mg/kg/day, respectively. The developmental NOEL and LEL are 5 and 15 mg/kg/day, respectively. This study was classified as unacceptable, primarily due to reporting deficiendies. A teratology study was conducted with rabbits administered (orally) bromoxynil phenol at dose levels of 0, 15, 30, or 60 mg/kg/day. The maternal NOEL and LEL are 15 and 30 mg/kg/day, respectively. The developmental NOEL and LEL are less than 15 and 15 mg/kg/day, respectively. Significant body weight gain decrements were reported at the two highest dose levels along with observed decreases in food consumption. The severe maternal toxicity among high dose dams was associated with fetoxicity and teratogenicity. A slight, nonsignificant increase in supernumerary ribs was reported at the mid and low dose levels. A teratology study was conducted with mice administered (orally) bromoxynil phenol at dose levels of 0, 11, 32, or 96 mg/kg/day. Maternal mortality was observed at 32 and 96 mg/kg/day. Fetal body weight was decreased at the top dose level, associated with a decrease in caudal vertebral ossification and an increase in supernumerary ribs. The maternal NOEL and LEL are 11 and 32 mg/kg/day respectivel. The developmental NOEL and LEL are 32 and 96 mg/kg/day, respectively.

A reproduction study was conducted with rats administered (orally) bromoxynil phenol at dose levels of 0. 0.8, 4, or 21 mg/kg/day in the diet. The systemic adult rat NOEL is 4 mg/kg/day and the LEL is 21 mg/kg/day. The reproductive NOEL is 21 mg/kg/day, and the LEL is greater than 21 mg/kg/day. The postnatal developmental NOEL is 4 mg/kg/day, and the LEL is 21 mg/kg/day. Body weight gain decrements were reported. However, no adverse effects on fertility, fecundity, reproductive performance or pre and postnatal development were observed. A reproduction study was conducted with rats administered (orally) bromoxynil phenol at dose levels of 0, 1.5, 5, or 15 mg/kg/day in the diet. The systemic rat NOEL is 1.5 mg/kg/day, and the LEL is is 5 mg/kg/day. The reproductive NOEL is 15 mg/kg/day, and the LEL is greater than 15 mg/kg/day. The offspring developmental NOEL is 5 mg/kg/day and the LEL is 15 mg/kg/day. Body weight gain decrements were reported. However, no adverse effects on fertility, fecundity, reproductive performance or pre and postnatal development were observed.

Based on the studies discussed above, it is concluded that bromoxynil is not teratogenic at doses that are not maternally toxic. In addition, bromoxynil is not considered a reproductive toxicant and shows no evidence of endocrine effects.

4. *Subchronic toxicity*. In a 12–week range-finding study, bromoxynil (phenol) was administered in the diets of male and female CD-1 mice at dose levels of 0, 1.3, 3.9, 13, 39, 130, or 390 mg/kg/day. For male mice, the NOEL is 3.9 mg/kg/day and the LOEL is 13 mg/kg/day based on increased liver weights and hepatocellular hypertrophy. In female mice, the NOEL is 13 mg/kg/day and the LOEL is 39 mg/kg/day based on increased liver weights, hepatocellular hypertrophy, hepatocellular degeneration, and hepatocellular vacuolization. In a 13–week subchronic feeding study, bromoxynil (phenol) was administered in the diet to male and female Sprague-Dawley rats at dose levels of 0, 28, 58, or 168 mg/kg/day. For male rats, the NOEL is 28 mg/kg/day and the LOEL is 58 mg/kg/day based on decreased body weight gain, increased ALT and increased alkaline phosphatase. For female rats, no NOEL was determined in this study and the LOEL is 35 mg/kg/day based on decreased body weight gain. In a 13–week range-finding study, bromoxynil (phenol) was administered orally to male and female dogs at doses of 0, 1, 5, 8, 12, 16, 20, 30, 40, or 50 mg/kg/day. For males, no NOEL was determined and the LOEL is 1 mg/kg/day based on decreased body weight gain. For females, the NOEL is 1 mg/kg/day and the the LOEL is 5 mg/kg/day based on decreased body weight gain, panting and liquid feces. In a 21 day subchronic dermal study, bromoxynil (phenol) was applied to skin of male and female New Zealand white rabbits at doses of 0, 30, 300, or 1,000 mg/kg/day for 6 hours/day, 5 days/week. Treatment produced no observable dermal or systemic toxicity, therefore the NOEL is 1,000 mg/kg/day.

5. *Chronic toxicity*. A 1–year oral study was conducted with dogs administered bromoxynil (phenol) at dose levels of 0, 0.1, 0.3, 1.5, and 7.5 mg/kg/day in capsules. The NOEL/LEL is 1.5 mg/kg/day for both females and males based on decreased body weight gain, decreased RBC count, decreased hemoglobin, decreased PCV, and increased liver weights. The chronic dog study was determined by Rhone-Poulenc to be the most appropriate study for setting the Reference Dose (RfD) of 0.015 mg/kg/day (includes a hundredfold safety factor).

A 2–year combined chronic toxicity/carcinogenicity study was conducted with rats administered (oral) dosages of 0, 60, 190, or 600 ppm (0, 2.6, 8.2, or 28 mg/kg/day in males; 0, 3.3, 11.0, or 41 mg/kg/day in females) bromoxynil phenol in the diet. In males the no-observed-effect-level (NOEL) for systemic toxicity is 2.6 mg/kg/day, and the lowest-effect-level (LEL) is 8.2 mg/kg/day. In females, the NOEL is 3.3 mg/kg/day, and the LEL is 11.0 mg/kg/day. This study did not demonstrate any increase in tumor incidences in either male or female rats.

A 2–year combined feeding/carcinogenicity study was conducted with rats administered bromoxynil phenol in the diet at dose levels of 0, 10, 30, or 100 ppm (0, 0.5, 1.5, or 5 mg/kg/day). In both males and females, the NOEL and LOEL for systemic toxicity was 5 mg/kg/day and >5 mg/kg/day, respectively. At the highest dose tested, increased liver weights were observed at 12 months, but not at 24 months. This study was considered negative for carcinogenicity. An 18 month carcinogenicity study was conducted with mice administered bromoxynil phenol at dose levels of 0, 10, 30, or 100 ppm (0, 1.3, 3.9, or 13 mg/kg/day) in the diet. For males, dose related increases in hyperplastic nodules and liver adenomas/carcinomas were observed which were statistically significant at the 100 ppm. Increased relative liver weights were also observed. In females,

increased absolute liver weights and relative liver and kidney weights were observed. The study was considered negative for carcinogenicity for females. An 18 month carcinogenicity study was conducted with mice administered bromoxynil phenol at dose levels of 0, 20, 75, or 300 ppm (0, 3.1, 12 or 46 mg/kg/day in males and 0, 3.7, 14, or 53 mg/kg/day in females). Mice given 300 ppm had significantly increased absolute and relative liver weights. Histopathology of the liver revealed increased hepatocellular hypertrophy, hepatocellular degeneration, necrosis of individual hepatocytes, and pigment accumulation in hepatocytes and Kupffer cells. Male mice had statistically significant increased numbers of hepatocellular adenomas and carcinomas at 20 ppm, but not 75 ppm. In contrast, no significant increase in tumor incidence was observed for female mice by pair-wise analysis. The trend test was significant for adenomas or carcinomas in females, only at $p<0.05$, not $p<0.01$ as would be appropriate for this type of tumor. The trend is due entirely to the high dose group and therefore is of questionable validity. It is concluded that bromoxynil is a weak, single sex, single species, non-metastic, single target organ carcinogen, inducing hepatocellular tumors in male mice exposed to 300 ppm for 18 months. These tumors and associated histopathological findings are consistent with secondary mechanisms such as peroxisome proliferation, a mechanism known to have marked species differences and questionable relevance for humans. It is the opinion of Rhone-Poulenc that the data are not suitable for quantitative risk assessment. A threshold safety factor approach is more appropriate and is commonly used for single sex, single species carcinogens such as bromoxynil that are thought to work through secondary mechanisms. For the purposes of this tolerance petition, risk assessments have been performed using a low dose linear extrapolation model (Q_1* is 1.03×10^{-1}).

6. *Animal metabolism*. Results of a bromoxynil metabolism study with the rat (octanoate) demonstrated that 2 mg/kg of radiolabeled bromoxynil octanoate was rapidly absorbed, hydrolyzed to bromoxynil phenol, distributed, and excreted in rats following repeated oral administration. The urine was the major route of excretion, representing 80.24% of the administered dose in males and 67.91% in females at 7 days post-dosing. Tissue distribution was similar for both sexes with the highest radioactivity recovered in the liver and kidney. Similar results were obtained in a separate rat metabolism study conducted with bromoxynil heptanoate.

7. *Metabolite toxicology*. DBHA (3,5-dibromo-4-hydroxybenzoic acid) is a major plant metabolite of bromoxynil only in bromoxynil-resistant transgenic cotton. Acute oral toxicity testing with DBHA in rats resulted in an LD_{50} of >2,000 mg/kg. Acute dermal toxicity testing with DBHA in rabbits resulted in an LD_{50} of >2,000 mg/kg. The primary dermal irritation study with DBHA in rabbits indicated DBHA to be a slight irritant, and DBHA was not a dermal sensitizer in Guinea pigs. Mutagenicity studies conducted with DBHA include a Salmonella typhimurium reverse mutation assay (negative with and without activation); micronucleus assay (negative); and $TK^{+/-}$ mouse lymphoma assay (negative with and without metabolic activation). In subchronic feeding studies in the rat, DBHA was administered by oral gavage to groups of Sprague-Dawley rats for 28 days at dose levels of 25, 50, 100 and 250 mg/kg/day. No toxicologically meaningful changes were observed in any of the parameters measured in this study. The NOEL and LEL for this study were 250 and >250 mg/kg/day, respectively.

C. Aggregate Exposure

1. *Dietary (food) exposure*. For the purpose of estimating the potential human dietary exposure resulting from bromoxynil use on cotton under the existing tolerances, anticipated residues of bromoxynil and DBHA were used. Anticipated residue values of 1.44 ppm (cottonseed), 8.74 ppm (cotton gin trash), and 0.43 ppm (cottonseed meal) were derived by taking the mean residue values from available crop field trials conducted at the 4.5 lb/A broadcast rate and adjusting by a factor of 0.333 to extrapolate to the current 1.5 lb/A application rate. Adjusting these values for % dry matter and the proposed 10% of crop treated results in anticipated cotton feedstuff residue values of 0.14 ppm (cottonseed), 0.87 ppm (cotton gin trash), and 0.043 ppm (cottonseed meal). Based on the use of these exposure data and a unit risk (Q_1* (mg/kg/day)$^{-1}$, of bromoxynil of 1.03×10^{-1}, the upper-bound human risk estimate for the general (U.S.) population represented by all sources of bromoxynil exposure, including use on up to 10% of the U.S. treated acreage is approximately 2×10^{-6}.

2. *Drinking water*. There is no Maximum Concentration Level or Health Advisory Level established for bromoxynil under the Safe Drinking Water Act. Based on field dissipation studies demonstrating a short half-life of bromoxynil in the environment (average half-life of 3–7 days), bromoxynil residues will degrade in soil before residues can move downward into ground water. Therefore, no significant potential exists for bromoxynil residues to be present in drinking water from ground water. Likewise, contamination of drinking water supplies from bromoxynil movement through agricultural surface runoff is considered highly unlikely due to relatively low application rates and rapid degradation rates in soil. As demonstrated by available monitoring data, normal dilution and degradation processes will greatly reduce concentrations in surface water during movement from agricultural ditches near fields into streams of adequate size for use as drinking water. It is the conclusion of Rhone-Poulenc that the potential bromoxynil exposure derived from any use through drinking water is insignificant and does not significantly increase the aggregate risk assessment above that estimated to occur through food exposure alone.

3. *Non-dietary exposure*. The potential for non-occupational exposure to bromxynil among the general public is insignificant. There are no residential lawn or garden uses for bromoxynil products where the general population might be exposed via inhalation or dermal routes. Turfgrass use is restricted to non-residential areas. Exposure to bromoxynil following application to non-residential turfgrass is not likely to be significant in either time or duration. This use will therefore not significantly add to the aggregate exposure.

D. Cumulative Effects

There are no reliable data suggesting that any toxic effect that might be caused by bromoxynil would be cumulative with those of any other compound. Further, bromoxynil does not appear to produce a toxic metabolite that is produced by other substances. Therefore, consideration of potential cumulative effects is not appropriate at this time.

E. Safety Determination

1. *U.S. population*. Using the present RfD for bromoxynil of 0.015 mg/kg/day, it has been determined that aggregate chronic exposure to bromoxynil from all uses, including cotton, represents <1% of the RfD for all population sub-groups. A unit risk, Q_1* (mg/kg/day)$^{-1}$, of bromoxynil of 1.03×10^{-1} in human equivalents, has been calculated based on mouse liver tumors. It is the opinion of Rhone-Poulenc that the bromoxynil data are not suitable for quantitative risk assessment. A threshold safety factor approach is more appropriate and is

commonly used for single sex, single species carcinogens such as bromoxynil that are thought to work through secondary mechanisms. Nevertheless, the risk assessments filed with this petition have been performed using quantitative risk assessment methodology. Accordingly, the upper-bound risk estimate for the general U.S. population represented by all sources of bromoxynil exposure, including use of bromoxynil on up to 10% of the U.S. treated acreage is approximately 2×10^{-6}.

2. *Infants and children*. To estimate acute dietary risk for systemic effects other than developmental from food sources, an MOE of 270 was calculated using 1–day dietary exposure for infants (the most highly exposed population group) and a NOEL of 8 mg/kg/day derived from a 13–week oral toxicity study in dogs. It is concluded that reliable data support use of the standard hundredfold margin of exposure/safety factor in assessing the risk to children. The general U.S. population and all population sub-groups are estimated to be exposed at a level less than 1 percent of the bromoxynil RfD of 0.015 mg/kg/day. Both chronic and acute assessments show no appreciable threshold risks to children and the non-threshold cancer risk is no greater than negligible. Therefore, there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to bromoxynil.

Two multi-generation rodent reproduction studies demonstrated that there were no adverse effects on reproductive performance, fertility, fecundity, pup survival, or pup development. Maternal and developmental NOELs and LOELs were comparable indicating no increase susceptibility of developing organisms. No evidence of endocrine effects were noted in any study. It is therefore concluded that bromoxynil poses no additional risk for infants and children and no additional uncertainty factor is warranted.

F. International Tolerances

There are no Codex tolerances established for bromoxynil residues, therefore international compatibility is not considered to be an issue at this time.

[FR Doc. 97–30812 Filed 11–25–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[FRL–5927–7]

Proposed Settlement Pursuant to Section 122(g) of the Comprehensive Environmental Response, Compensation, and Liability Act, Regarding the Sealand Restoration Superfund Site, Lisbon, New York

AGENCY: Environmental Protection Agency.

ACTION: Notice of proposed administrative settlement and opportunity for public comment.

SUMMARY: In accordance with section 122(i) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended (CERCLA), 42 U.S.C. 9622(i), the U.S. Environmental Protection Agency (EPA), Region II, announces a proposed administrative *de minimis* settlement pursuant to section 122(g)(4) of CERCLA, 42 U.S.C. 9622(g)(4), relating to the Sealand Restoration Superfund Site (Site). The Site is located on Pray Road in the Town of Lisbon, St. Lawrence County, New York. This document is being published pursuant to section 122(i) of CERCLA to inform the public of the proposed settlement and give it the opportunity to comment. EPA will consider any comments received during the comment period and may withdraw or withhold consent to the proposed settlement if comments disclose facts or considerations which indicate that the proposed settlement is inappropriate, improper, or inadequate.

The proposed *de minimis* settlement between EPA and Westpoint Stevens Inc., on behalf of former Cluett, Peabody & Co. (Respondent) has been memorialized in an Administrative Order on Consent (Index Number CERCLA–97–0215). This Order will become effective after the close of the public comment period, unless comments received disclose facts or considerations which indicate the Agreement is inappropriate, improper, or inadequate, and EPA, in accordance with section 122(i)(3) of CERCLA, modifies or withdraws its consent to the Agreement. Under the Order, the Respondent will be obligated to make payments to the Hazardous Substance Superfund in reimbursement of EPA's response costs relating to the Site, plus a premium, based on documented volumes of substances in EPA's records associated with the Site, totaling $47,676.

Pursuant to CERCLA section 122(h)(1), the Order may not be issued without the prior written approval of the Attorney General or her designee. In accordance with that requirement, the Attorney General or her designee has approved the proposed administrative order in writing.

DATES: Comments must be provided on or before December 26, 1997.

ADDRESSES: Comments should be addressed to the U.S. Environmental Protection Agency, Office of Regional Counsel, New York/Caribbean Superfund Branch, 17th Floor, 290 Broadway, New York, New York 10007–1866, and should refer to: ''Sealand Restoration Superfund Site, U.S. EPA Index No. CERCLA–97–0215''. For a copy of the settlement document, contact the individual listed below.

FOR FURTHER INFORMATION CONTACT: Elizabeth Davis, Assistant Regional Counsel, New York/Caribbean Superfund Branch, Office of Regional Counsel, U.S. Environmental Protection Agency, 17th Floor, 290 Broadway, New York, New York 10007. Telephone: (212) 637–3165.

Dated: November 4, 1997.

William J. Muszynski,

Acting Regional Administrator.

[FR Doc. 97–31137 Filed 11–25–97; 8:45 am]

BILLING CODE 6560–50–P

COUNCIL ON ENVIRONMENTAL QUALITY

Notice of Meeting; Postponement

SUMMARY: The Council on Environmental Quality (CEQ) is postponing a public meeting it had previously scheduled for December 2, 1997, to discuss development of a memorandum of understanding on coordinating environmental response actions with natural resource restoration under the Comprehensive Environmental Response, Compensation, and Liability Act and other laws. 62 FR 51660 (October 2, 1997). CEQ intends to reschedule the meeting for late January or early February, 1998. CEQ will soon publish another **Federal Register** notice identifying the time, place, and agenda for the meeting.

FOR FURTHER INFORMATION CONTACT: Mary Morton at (202) 208–3302.

Bradley M. Campbell,

Associate Director.

[FR Doc. 97–31031 Filed 11–25–97; 8:45 am]

BILLING CODE 3125–01–M