Rule 71.2 Storage of Reactive Organic Compound Liquids (Adopted 9/26/89)
Rule 71.3 Transfer of Reactive Organic Compound Liquids (Adopted 6/16/92)
Rule 71.4 Petroleum Sumps, Pits, Ponds, and Well Cellars (Adopted 6/8/93)
Rule 72 New Source Performance Standards (NSPS) (Adopted 6/28/94)
Rule 74 Specific Source Standards (Adopted 7/6/76)
Rule 74.1 Abrasive Blasting (Adopted 11/12/91)
Rule 74.2 Architectural Coatings (Adopted 08/11/92)
Rule 74.6 Surface Cleaning and Degreasing (Adopted 5/8/90)
Rule 74.6.1 Cold Cleaning Operations (Adopted 9/12/89)
Rule 74.6.2 Batch Loaded Vapor Degreasing Operations (Adopted 9/12/89)
Rule 74.7 Fugitive Emissions of Reactive Organic Compounds at Petroleum Refineries and Chemical Plants (Adopted 1/10/89)
Rule 74.8 Refinery Vacuum Producing Systems, Waste-water Separators and Process Turnarounds (Adopted 7/5/83)
Rule 74.9 Stationary Internal Combustion Engines (Adopted 12/21/93)
Rule 74.10 Components at Crude Oil Production Facilities and Natural Gas Production and Processing Facilities (Adopted 6/16/92)
Rule 74.11 Natural Gas-Fired Residential Water Heaters-Control of NO_X (Adopted 4/9/85)
Rule 74.12 Surface Coating of Metal Parts and Products (Adopted 11/17/92)
Rule 74.15 Boilers, Steam Generators and Process Heaters (5MM BTUs and greater) (Adopted 12/3/91)
Rule 74.15.1 Boilers, Steam Generators and Process Heaters (1–5MM BTUs) (Adopted 5/11/93)
Rule 74.16 Oil Field Drilling Operations (Adopted 1/8/91)
Rule 74.20 Adhesives and Sealants (Adopted 6/8/93)
Rule 74.24 Marine Coating Operations (Adopted 3/8/94)
Rule 74.28 Asphalt Roofing Operations (Adopted 5/10/94)
Rule 74.30 Wood Products Coatings (Adopted 5/17/94)
Rule 75 Circumvention (Adopted 11/27/78)
Appendix IV–A Soap Bubble Tests (Adopted 12/86)
Rule 100 Analytical Methods (Adopted 7/18/72)
Rule 101 Sampling and Testing Facilities (Adopted 5/23/72)
Rule 102 Source Tests (Adopted 11/21/78)
Rule 103 Stack Monitoring (Adopted 6/4/91)
Rule 154 Stage 1 Episode Actions (Adopted 9/17/91)
Rule 155 Stage 2 Episode Actions (Adopted 9/17/91)
Rule 156 Stage 3 Episode Actions (Adopted 9/17/91)
Rule 158 Source Abatement Plans (Adopted 9/17/91)
Rule 159 Traffic Abatement Procedures (Adopted 9/17/91)

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[FR Doc. 95–1185 Filed 1–17–95; 8:45 am]
BILLING CODE 6050–50–P

40 CFR Part 185

[OPP–300360; FRL–4910–8]

RIN 2070–AC18

Acephate, Triadimefon, Iprodione, and Imazalil; Revocation of Food Additive Regulations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to revoke food additive regulations for the pesticides acephate, triadimefon (1-(4-chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone), iprodione, and imazalil, which EPA has determined ''induce cancer'' within the meaning of the Delaney clause of section 409 of the Federal Food, Drug and Cosmetic Act (FFDCA). As a result of a 1992 court decision regarding the Delaney clause, EPA has initiated the process of revoking those section 409 tolerances for pesticides found to ''induce cancer.'' This proposed rule is the second in a series of proposals to revoke affected regulations under section 409 of the FFDCA.

DATES: Written comments, identified by the document control number [OPP-300360], must be received on or before April 18, 1995.

ADDRESSES: By mail, submit comments to: Public Response Section, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring comments to: OPP Docket, Public Information Branch, Field Operations Division, Rm. 1132, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. The telephone number for the OPP docket is (703)-305-5805.

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'' (or CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2 and in section 10 of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). For questions related to disclosure of materials, contact the OPP Docket at the telephone number given above. 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 the OPP Docket, Rm. 1132 at the Virginia address given above, from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Niloufar Nazmi or Lisa Nisenson, Special Review and Reregistration Division (7508W), Environmental Protection Agency, 401 M St. SW., Washington, DC, 20460. Office location and telephone number: Crystal Station #1, 2800 Crystal Drive, Arlington, VA. Telephone 703-308-8010.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. Statutory Background

The Federal Food, Drug and Cosmetic Act (FFDCA) (21 U.S.C. 301 et seq.) authorizes the establishment of maximum permissible levels of pesticides in foods, which are referred to as ''tolerances'' (21 U.S.C. 346a, 348). Without such a tolerance or an exemption from a tolerance, a food containing a pesticide residue is ''adulterated'' under section 402 of the FFDCA and may not be legally moved in interstate commerce (21 U.S.C. 342). Monitoring and enforcement of pesticide residues are carried out by the U.S. Food and Drug Administration (FDA) and the United States Department of Agriculture (USDA).

The FFDCA governs tolerances for raw agricultural commodities (RACs) and processed foods separately. For pesticide residues in or on RACs, EPA establishes tolerances, or exemptions from tolerances when appropriate, under section 408. In processed foods, food additive regulations setting maximum permissible levels of pesticide residues are established under section 409. Section 409 tolerances are needed, however, only for certain pesticide residues in processed food. Under section 402(a)(2) of the FFDCA, no section 409 tolerance is required if any pesticide residue in a processed food is equal to or below the tolerance for that pesticide in or on the RAC from which it was derived and all other conditions of section 402(a)(2) are met. This exemption in section 402(a)(2) is commonly referred to as the ''flow-through'' provision because it allows the section 408 raw food tolerance to flow through to the processed food form. Thus, a section 409 tolerance is necessary to prevent foods from being deemed adulterated when the concentration of the pesticide residue in a processed food is greater than the tolerance prescribed for the RAC, or if

the processed food itself is treated or comes in contact with a pesticide.

If a food additive regulation must be established, section 409 of the FFDCA requires that the use of the pesticide will be ''safe'' (21 U.S.C. 348(c)(3)). Relevant factors in this safety determination include: (1) the probable consumption of the pesticide or its metabolites; (2) the cumulative effect of the pesticide in the diet of man or animals, taking into account any related substances in the diet; and (3) appropriate safety factors to relate the animal data to the human risk evaluation. Section 409 also contains the Delaney clause, which specifically provides that, with little exception, ''no additive shall be deemed safe if it has been found to induce cancer when ingested by man or animal'' (21 U.S.C. 348(c)(3)).

Before a pesticide may be sold or distributed, it must be registered under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). To qualify for registration, a pesticide must, among other things, perform its intended function without causing ''unreasonable adverse effects on the environment'' (7 U.S.C. 136a(c)(5)). The term ''unreasonable adverse effects on the environment'' is defined as ''any unreasonable risk to man or the environment taking into account the economic, social and environmental costs and benefits of the use of any pesticide'' (7 U.S.C. 136(bb)).

B. Regulatory Background

On May 25, 1989, the State of California, the Natural Resources Defense Council, Public Citizen, the AFL-CIO, and several individuals filed a petition requesting that EPA revoke several food additive regulations and challenging EPA's de minimis interpretation of the Delaney clause. The petition, which sought a ''zero-risk'' interpretation of the Delaney clause, requested that EPA revoke certain food additive regulations. The petitioners argued that these food additive regulations should be revoked because they violate the Delaney clause.

EPA responded to the petition by revoking certain food additive regulations, but retained several others on the grounds that the Delaney clause provides an exception for pesticide residues posing de minimis risk; EPA denied the petition for the food additive regulations determined to fall under this exception.

EPA's response was challenged by the petitioners in the U.S. Court of Appeals, Ninth Circuit. On July 8, 1992, the court ruled in *Les v. Reilly*, 968 F.2d 985 (9th Cir.), cert. denied, 113 S.Ct. 1361 (1993), that the Delaney clause barred the establishment of a food additive regulation for pesticides which ''induce cancer'' no matter how infinitesimal the risk.

On July 14, 1993, EPA issued a revised response to the petition taking into account the court's ruling. That revised response granted the original petition and revoked the food additive regulations named in the petition. The food additive regulations for two of the four affected pesticides, benomyl and trifluralin, have been reinstated pending judicial review by the Court of Appeals, District of Columbia Circuit, of several registrants' challenge to the revocation.

In implementing the court's decision in *Les v. Reilly*, EPA has taken steps to identify and revoke all section 409 tolerances for pesticides which have been found to ''induce cancer.'' EPA has issued two lists of pesticide uses which would likely be affected by the court's decision. The first list contains affected food and feed additive regulations, and the second identifies uses for pesticides that have either been found to induce cancer or are likely to be so classified where data show a food or feed additive regulation needs to be established. Both lists have been updated to reflect changes in data reviews and other regulatory actions (see 59 FR 14980, March 30, 1994). The first proposed revocation, which included 26 food additive regulations for seven pesticides classified as ''B'', probable human carcinogens or ''C'', possible human carcinogens subject to quantification by a linear low-dose extrapolation model, was published in the **Federal Register** of July 1, 1994 (59 FR 33941).

II. Proposed Revocation of Section 409 Tolerances Which are Inconsistent with the Delaney Clause

EPA intends to revoke all food and feed additive regulations which are inconsistent with the Delaney clause. This notice proposes revocation of all food additive regulations published in the March 30, 1994 **Federal Register** notice which have not previously been proposed for revocation. EPA expects to publish additional proposed revocations for feed additive regulations in the near future.

A. Basis for Proposing Revocation

As a result of the court's 1992 decision, the only issue to be considered for these proposed revocations is whether acephate, triadimefon, imazalil, and iprodione qualify under the Delaney clause as having been ''found to induce cancer when ingested by man or animals, or it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal.'' 21 U.S.C. 348(c)(3)(A). If EPA finds they are human or animal carcinogens within the meaning of the Delaney clause, the food additive regulations must be revoked.

In construing the ''induce cancer'' standard as to animals, EPA follows a weight-of-the-evidence approach which is guided, where appropriate, by the principles in EPA's Cancer Assessment Guidelines. In regard to animal carcinogenicity, EPA, in general, interprets ''induces cancer'' to mean:

> The carcinogenicity of a substance in animals is established when administration in adequately designed and conducted study or studies results in an increase in the incidence of one or more types of malignant (or, where appropriate, benign or a combination of benign and malignant) neoplasms in treated animals compared to untreated animals maintained under identical conditions except for exposure to the test compound. Determination that the incidence of neoplasms increases as the result of exposure to the test compound requires a full biological, pathological, and statistical evaluation. Statistics assist in evaluating the biological significance of the observed responses, but a conclusion on carcinogenicity is not determined on the basis of statistics alone. Under this approach, a substance may be found to ''induce cancer'' in animals despite the fact that increased tumor incidence occurs only at high doses, or that only benign tumors occur, and despite negative results in other animal feeding studies. (See 58 FR 37863, July 14, 1993; 53 FR 41108, October 19, 1988; and 52 FR 49577, December 31, 1987).

Acephate, triadimefon, imazalil, and iprodione all qualify as animal carcinogens under this test.

Summarized below is the information supporting EPA's determination that these pesticides ''induce cancer.'' Full copies of each of these reviews and other references in this notice are available in the OPP Docket, the location of which is given under ''ADDRESSES'' above.

Acephate

After a full evaluation of all the data and supporting information regarding animal carcinogenicity, EPA has concluded that exposure to acephate results in the induction of malignant hepatocellular carcinomas in female CD-1 mice.

Male and female CD-1 mice were fed 0, 50, 250, or 1,000 parts per million (ppm) of acephate for 105 weeks. Although fewer low-dose and mid-dose female mice survived to the end of the study compared with controls, the survival of the highest dose tested (HDT) female mice and all male mice was higher than that with the controls. Decreases in body weight gain ranged

from 8 to 11 percent for males and 6 to 14 percent for females at the mid-dose, and about 24 percent for males and 29percent for females at the HDT. Dose-related increasing levels of liver toxicity, including regenerative changes, were observed. In the female mice at the HDT, the incidence of malignant hepatocellular carcinomas and hyperplastic nodules was significantly increased in comparison with controls. The increased incidence of carcinomas exceeds the testing laboratory's historical control range. There were no increases in tumors in the two lower dosed female groups or any of the male groups.

Male and female Charles River (CD) Sprague-Dawley rats were fed 0, 5, 50, and 700 ppm of acephate for 28 months. There was no dose-related effect on mortality, although there was significant cholinesterase inhibition in the mid- and high-dose male and female rats. There was a 4 to 8 percent weight loss in the HDT males.

Acephate has been tested in a wide array of genotoxicity assays. The evidence indicates that acephate produced positive responses in gene mutation *in vitro* assays with *Salmonella*, *E. coli*, and *S. cerevisiae*. Acephate has been reported to produce mutations in mouse lymphoma cells, sister chromatid exchanges (SCEs) in Chinese hamster ovary (CHO) cells, and mitotic recombination in *Saccharomyces*. Several *in vivo* assays for SCEs and cytogenetic endpoints have been negative.

Based on this information regarding animal carcinogenicity, the Agency concludes that exposure to acephate results in the induction of malignant hepatocellular carcinomas in female CD-1 mice. The incidence exceeded the historical control range of the testing laboratory. There is evidence that acephate is genotoxic based on *in vitro* studies, but this activity may be difficult to detect *in vivo*. The relevance of these data to an evaluation of acephate's potential for human carcinogenicity is discussed in the Peer Review document of Acephate (May 8, 1985).

Triadimefon

After a full evaluation of all the data and supporting information regarding animal carcinogenicity, EPA has concluded that exposure to triadimefon results in the induction of hepatocellular adenomas in both male and female NMRI mice. Male and female NMRI mice were fed 0, 50, 300, or 1,800 ppm of triadimefon for 21 months. At the HDT, the incidence of hepatocellular adenomas was increased in both male and female mice by pair-wise comparison between the HDT and controls. A positive dose-related significant trend for adenomas was found in both sexes. The incidence of hepatocellular adenomas for each sex exceeded the testing laboratory's historical control range for adenomas in NMRI mice.

In another study, male and female CF1-W74 mice were fed 0, 50, 300, or 1,800 ppm of triadimefon for 24 months. The HDT was considered appropriate for assessing carcinogenicity based on increased hematological changes; statistically significant increases in liver weights accompanied by histopathological changes and weight gains at the HDT were significantly lower than in controls.

Initially, the tumor profile was thought to provide no indication that triadimefon had an influence on total tumor incidence, on the number of mice with tumors or on incidence of single tumor types; however, the pathology report indicated that more mice had hyperplastic liver nodules at the HDT than mice in the other treated groups or the controls. The Peer Review Committee recommended that in light of the NMRI study results outlined above, and that the original analysis of the study results was performed before the current criteria were put into place, the liver nodules should be re-read with updated criteria.

The new histopathological information for the CF1-W74 mouse study was submitted subsequent to the completion of the latest Triadimefon Peer Review document. Only a small number of slides were available for reexamination, and the results were deemed inconclusive. However, they are suggestive of an effect on tumor incidence in the liver and are consistent with the findings in the NMRI study that the liver is a principal site for tumor induction. Lesions which were originally classified as hyperplastic or regenerative nodules were reclassified as either hepatocellular adenomas or carcinomas. In males, 3, 3, 2, and 3 adenomas and 1, 4, 4, and 4 carcinomas were found out of 6, 8, 7, and 13 liver samples examined at doses of 0, 50, 300, and 1,800 ppm, respectively. This suggests that triadimefon may contribute to the induction of liver tumors and there may be a carcinoma component.

In a 104-week study, male and female Wistar rats were fed 0, 50, 300, or 1,800 ppm of triadimefon. Triadimefon induced a positive dose-related trend in the incidence of thyroid follicular cell adenomas/adenomas multiple in male Wistar rats. Positive dose-related trends were achieved in both sexes for combined incidences of thyroid follicular cell cystic hyperplasia and adenomas/adenomas multiple.

Hepatocellular adenomas are considered to be evidence of cancer because hepatocellular adenomas can progress to hepatocellular carcinomas. Malignancy (carcinoma) implies a more extensive disease process. Thus, hepatocellular adenomas represent an earlier stage than carcinomas in the progression of cancer induction. This is one of the major reasons that the National Toxicology Program (NTP) has used to justify combining these two tumor types for an overall analysis of carcinogenicity (in addition to analyzing them separately). For triadimefon, the possible progression to carcinoma was suggested in the CF1-W74 mouse study and is strongly supported by carcinoma induction in close structural analogues, e.g., etaconazole, uniconazole, cyproconazole, tebuconazole, and fenbuconazole.

Based on the above data and supporting information regarding animal carcinogenicity, it is concluded that exposure to triadimefon results in the induction of hepatocellular adenomas in both male and female NMRI mice. A positive dose-related significant trend for adenomas was also found in both sexes. This conclusion is bolstered by the extensive structural activity support from closely structurally related triazole compounds tested in many mouse studies that showed increased incidences of not only adenomas but carcinomas as well. It is also noted that although the analysis was inconclusive, there was a carcinoma response by triadimefon in the CF1-W74 mouse study. In addition, triadimefon induced a positive dose-related trend in the incidence of thyroid follicular cell adenomas/adenomas multiple in male Wistar rats. Positive dose-related trends were achieved in both sexes for combined incidences of thyroid follicular cell cystic hyperplasia and adenomas/adenomas multiple.

The relevance of these data to an evaluation of triadimefon's potential for human carcinogenicity is discussed in the Peer Review document of Triadimefon (September 26, 1990).

Iprodione

After a full evaluation of the data and supporting information regarding animal carcinogenicity, EPA concludes that exposure to iprodione resulted in an increased incidence of hepatocellular malignant carcinomas in male mice and combined hepatocellular adenomas/carcinomas in both sexes of mice, ovarian lutenomas in female mice, and

testicular interstitial cell tumors in male rats.

Iprodione was administered to CD-1 mice (50/sex/group) at levels of 0, 160, 800, or 1,400 ppm for at least 99 weeks (or until the 52-week interim sacrifice of 15 additional mice/sex/group). At the terminal sacrifice, there was a significantly increased incidence of benign and malignant liver cell tumors in both sexes compared to the control. Analysis indicates that male mice had significant difference in the pair-wise comparisons of the 1,400-ppm dose group with the controls for liver adenomas, carcinomas and combined adenomas and/or carcinomas. Female mice had significant increasing trends in liver adenomas, carcinomas, and combined adenomas and/or carcinomas. All males in all dose groups (including concurrent controls) displayed a higher incidence of carcinomas than observed in historical controls. Although there was no increase in the incidence of testicular tumors in the male mice, there was a dose-related increase in the incidence of interstitial cell hyperplasia at the 800- and 1,400-ppm dose levels.

In female mice, iprodione was associated with significant dose-related increasing trends in liver adenomas, carcinomas and combined adenomas and/or carcinomas; there were significant differences in pair-wise comparisons with the high-dose level with controls for liver adenomas and combined adenomas and/or carcinomas. The increased incidences of hepatocellular tumors at the 1,400-ppm level generally exceeded the available historical control data for these tumor types in mice of this strain. Additionally, iprodione was associated with a significant increasing trend in ovarian lutenomas, and there was a significant difference in the pair-wise comparison of the 1,400-ppm dose group with the control group and historical controls. EPA considers the dose levels used in this study to be adequate for testing the carcinogenicity of iprodione in mice.

Iprodione was administered in the diet to 60 Sprague-Dawley rats/sex/group for 2 years at dose levels of 0, 150, 300, or 1,600 ppm. There was a 52-week interim sacrifice of 10 additional rats/sex/group. At the interim sacrifice, males at the high-dose level displayed an increase in the incidence of lesions in the adrenals, and there was an increase in the incidence of centrilobular hepatocyte enlargement in males at the 300 and 600 dose levels; females displayed an increased incidence of centrilobular hepatocyte enlargement at the highest dose tested.

In male rats fed iprodione for 2 years, there was a significant dose-related increasing trend and a significant difference in the pair-wise comparison of the 1,600-ppm dose group with the controls for testicular interstitial cell benign tumors. The incidence of both unilateral and bilateral benign interstitial cell tumors was increased at this dose level compared to historical control data. In addition to the neoplastic lesions, interstitial cell hyperplasia in the testes, reduced spermatozoa in the epididymis, and absent/empty secretory colloid cells or reduced secretion in the seminal vesicles were observed at the 300- and 1,600-ppm dose levels. Atrophy of the seminiferous tubules in the testes, with atrophy of the prostate and absence of spermatozoa in the epididymis, were observed at 1,600 ppm. Centrilobular hepatocyte enlargement was increased in males at the high-dose level. Adrenal lesions were observed in both sexes at the 300- and 1,600- ppm dose levels, although males displayed more lesions than females.

In females rats fed iprodione at the high-dose level for 2 years, there were no significant compound-related tumors observed, although there was an increased incidence of tubular hyperplasia in the ovaries and increased sciatic nerve fiber degeneration compared to the controls. The dose levels chosen for this study were considered appropriate for assessing the carcinogenicity of iprodione in rats.

Iprodione is structurally related to vinclozolin and procymidone. Procymidone has been associated with the appearance of tumors in both sexes in the reproductive organs and the liver, but did not have mutagenic activity in several tests. Vinclozolin, which is currently being tested for its carcinogenic potential, has been associated with adverse effects on the reproductive organs and liver. With the exception of the mouse lymphoma (forward mutation) assay, vinclozalin was negative for mutagenicity. In mutagenicity studies, iprodione was not mutagenic in the Ames assay, the CHO/HGPRT mammalian cell forwarded mutation assay, the in vitro chromosome aberration assay in CHO cells, the *in vitro* sister chromatid exchange assay in CHO cells and the dominant-lethal test in mice. However, iprodione was positive in the *Bacillus subtilis* assay for DNA damage without metabolic activation.

Imazalil

After a full evaluation of the data and supporting information regarding animal carcinogenicity, EPA concludes that exposure to imazalil is associated with an increased incidence of adenomas and combined adenomas/adenocarcinomas of the livers of male Swiss mice and with a significant dose-related increasing trend in hepatocellular adenomas and combined adenomas and/or carcinomas.

Imazalil base was administered in the diet to groups of 50 male and 50 female Swiss mice and treated for 100 to 101 weeks at levels of 0, 50, 200, or 600 ppm. Male mice had a significant dose-related increasing trends in hepatocellular adenomas and/or carcinomas. There was a significant difference in the pair-wise comparison of the 200-ppm dose group with the controls for hepatocellular adenomas. There were also significant differences in the pair-wise comparisons of the 600-ppm dose group with the controls for hepatocellular adenomas and combined adenomas and/or carcinomas. EPA has concluded that the malignant carcinoma response at the 600-ppm dose level was biologically relevant and related to imazalil exposure despite the lack of pair-wise statistical significance compared to controls. There was over a doubling of the concurrent control incidence and a positive trend for carcinomas. The male carcinoma incidence was also outside the historical control data provided by the submitting company. It was noted that about 50% of the significantly positive combined incidence was contributed by carcinomas. Also, there appears to be a progression towards malignancy across the dose groups.

Female mice had significant dose-related increasing trends in hepatocellular adenomas and combined adenomas and/or carcinomas. There were no significant differences in the pair-wise comparisons of the dosed groups with the controls.

Nonneoplastic changes in the liver were also observed in male mice at all dose levels. At the 200-ppm level, males had a significant increase in the incidence of focal cellular changes, large vacuoles, and swollen sinusoidal cells in the liver. At the highest dose tested, males also had a significantly increased incidence of pigmentation in the sinusoidal cells of the liver and focal cellular changes in the pancreas, increased absolute and relative liver weight, and decreased body weight and body weight gain. Female mice did not exhibit any cellular changes in the liver, although there was some effect on body weight at the 600-ppm dose and slight increases in liver weights at the highest dose tested as well.

There is extensive structure-activity relationship (SAR) support for the

tumor response associated with imazalil. Closely related compounds with the chlorinated benzene moiety, e.g., etaconazole, cyproconazole, tebuconazole, induced hepatocellular adenomas, and malignant carcinomas in both sexes of several strains of mice. The mutagenicity data for imazalil did not indicate genotoxic activity; however, a data gap was identified and additional testing is required.

B. Proposed Food Additive Revocations

Acephate. EPA is proposing to revoke the food additive regulation of 0.02 ppm for the combined residues of acephate (*O*,*S*-dimethyl acetylphosphoramidothioate) and its cholinesterase-inhibiting metabolite, methamidophos, set to cover use of the pesticide in food-handling establishments. This food additive regulation is codified at 40 CFR185.100. EPA is proposing to revoke this food additive regulation because the Agency has determined that acephate induces cancer in animals. Thus, the regulation violates the Delaney clause in section 409 of the FFDCA.

Triadimefon. EPA is proposing to revoke the food additive regulations for triadimefon (1-(4-chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone) and its metabolite *beta*-(4-chlorophenoxy)-*alpha*-(1,1-dimethylethyl)-1H-1,2,4triazole-1-ethanol set to cover residues in or on milled fractions of barley (except flour) and milled fractions of wheat (except flour). The food additive regulations, which are codified at 40 CFR 185.800, are set at 4 ppm. EPA is proposing to revoke these food additive regulations because the Agency has determined that triadimefon induces cancer in animals. Thus, the regulations violate the Delaney clause in section 409 of the FFDCA.

Iprodione. EPA is proposing to revoke the food additive regulations for iprodione [3-(3,5-dichlorophenyl)-*N*-(1-methylethyl)-2,4-dioxo-1-imidazolidinecarboxamide], its isomer [3-(1-methyl-ethyl)-*N*-(3,5-dichlorophenyl)-2,4-dioxo-1-imidazolidinecarboxamide], and its metabolite [3-(3,5-dichlorophenyl)-2,4-dioxo-1-imidazolidinecarboxamide] set to cover residues in dried ginseng at 4 ppm and raisins at 300 ppm. The food additive regulations for iprodione are codified at 40 CFR 185.3750. EPA is proposing to revoke these food additive regulations because the Agency has determined that iprodione induces cancer in animals. Thus, the regulation violates the Delaney clause in section 409 of the FFDCA.

Imazalil. EPA is proposing to revoke the food additive regulation for imazalil set to cover residues of the fungicide imazalil 1-[2-(2,4-dichlorophenyl)-2-(2-propenyloxy)ethyl]-1*H*-imidazole and its metabolite 1-(2,4-dichlorophenyl)-2-(1*H*-imidazole-1-yl)-1-ethanol in citrus oil at a level of 25 ppm. This food additive regulation is codified at 40 CFR 185.3650. EPA is proposing to revoke this food additive regulation because the Agency has determined that imazalil induces cancer in animals, and thus violates the Delaney clause in section 409 of the FFDCA.

III. Consideration of Comments

Any interested person may submit comments on this proposed action on or before April 18, 1995 at the address given in the section above entitled ''ADDRESSES.'' Before issuing final actions, EPA will consider all relevant comments. Comments should be limited only to the pesticides and food additive regulations subject to this proposed notice. After consideration of comments, EPA will issue a final order determining whether revocation of the regulations is appropriate and making a final finding on whether these pesticides induce cancer within the meaning of the Delaney clause. Such order will be subject to objections pursuant to section 409(f) (21 U.S.C. 348(f)). Failure to file an objection within the appointed period will constitute waiver of the right to raise issues resolved in the order in future proceedings.

IV. Executive Order 12866

Since this proposed action is being taken under the Delaney clause, which requires the Agency to act without considering the costs or benefits of the action, the Agency has not completed an evaluation of the economic impacts of this particular action. Nevertheless, pursuant to an agreement between EPA and OMB, this action was submitted to OMB for an informal 10-day review. As required by the Executive Order, any comments or changes made in response to OMB suggestions or recommendations have been documented in the public record. In addition, the Agency welcomes any comments and information regarding the impacts of this proposed action. These could contribute to an analysis of the impacts of similar future actions.

V. Regulatory Flexibility Act

The Regulatory Flexibility Act of 1980 (Pub. L. 96-354; 94 Stat. 1164, 5 U.S.C. 601 et seq.) requires EPA to analyze regulatory options to assess the economic impact on small businesses, small governments, and small organizations. As explained above, the Agency is compelled to take this action without regard to the economic impacts. Again, EPA welcomes any information on impacts to small businesses, governments, and organizations.

VI. Paperwork Reduction Act

This order does not contain any information collection requirements subject to review by Office of Management and Budget under the Paperwork Reduction Act of 1980, 44 U.S.C. 3501 et seq.

List of Subjects in 40 CFR Part 185

Environmental protection, Administrative practice and procedure, Agricultural commodities, Food additives, Pesticides and pests, Recording and recordkeeping requirements.

Dated: January 10, 1995.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

Therefore, it is proposed that 40 CFR part 185 be amended as follows:

PART 185—[AMENDED]

1. The authority citation for part 185 continues to read as follows:

Authority: 2l U.S.C. 346a and 348.

§ 185.100 [Removed]

2. By removing § 185.100.

§ 185.800 [Removed]

3. By removing § 185.800.

§ 185.3650 [Removed]

4. By removing § 185.3650.

§ 185.3750 [Removed]

5. By removing § 185.3750

[FR Doc. 95–1062 Filed 1–17–95; 8:45 am]
BILLING CODE 6560–50–F

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40 CFR Part 180

[OPP–300375; FRL–4926–6]

RIN 2070–AC18

Oryzalin; Revocation of Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to revoke tolerances for residues of the herbicide oryzalin in or on various raw agricultural commodities. EPA is taking this action because registered uses of oryzalin for cottonseed, barley grain,