food or drinking water is anticipated by using *Pseudomonas fluorescens* PRA-25 as a seed treatment.

2. Non-dietary exposure such as lawn care, topical insect repellents, etc. is not anticipated since this microbial pesticide does not have these uses.

3. Occupational exposure will be mitigated through the use of proper personal protective equipment.

E. Cumulative Exposure

Biological control agents of this type generally work by out competing disease organisms, thus, not having a toxic mode of action that can be shared. Other exposure can occur since other strains of *Pseudomonas fluorescens* are registered as microbial pesticides. Good Bugs, Inc. believes that human exposure from use of *Pseudomonas fluorescens* PRA-25 as a seed treatment is expected to be negligible.

F. Safety Determination

Good Bugs, Inc. believes that the safety of the U.S. population and that of infants and children will not be adversely affected by the use of *Pseudomonas cepacia* PRA-25 as a vegetable seed treatment. Strain PRA-25 is a naturally occurring strain originally isolated from the rhizosphere of a pea.

G. Existing Tolerances

1. Tolerance exemptions have been granted for other strains of *Pseudomonas fluorescens*.

2. International tolerance exemptions have been granted for other strains of Pseudomonas fluorescens.

II. Public Record

Interested persons are invited to submit comments on the notice of filing. Comments must bear a notation indicating the document control number, [PF-711].

A record has been established for this notice under docket number [PF-711] including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in Room 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

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

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

List of subjects

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

Dated: February 11, 1997.

Janet L. Andersen,

Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 97–4630 Filed 2–25–97; 8:45 am]

BILLING CODE 6560–50–F

[PF–701; FRL–5585–2]

Rhone-Poulenc Ag Company; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of filing.

SUMMARY: This notice announces the filing of a pesticide petition proposing the establishment of a tolerance for residues of isoxaflutole in or on field corn. This notice contains a summary of the petition prepared by the petitioner, Rhone-Poulenc Ag Company.

DATES: Comments, identified by the docket control number [PF–701], must be received on or before, March 28, 1997.

ADDRESSES: By mail, submit written comments to Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St. SW., Washington, DC 20460. In person, bring comments to Rm. 1132, CM#2, 1921 Jefferson Davis Highway, Arlington, VA 22202.

Comments and data may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on disks in WordPerfect in 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–701]. Electronic comments on this notice may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found in Unit II of this document.

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

FOR FURTHER INFORMATION CONTACT: Joanne Miller, Product Manager (PM) 23, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC. Office location, telephone number and e-mail address: Rm. 237, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, 703–305–6224, e-mail: miller.joanne@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition (PP 6F4664) from Rhone-Poulenc Ag Company, P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for the combined residues of the herbicide isoxaflutole [5-cyclopropyl-4-(2-methylsulfonyl-4-trifluoromethyl benzoyl) isoxazole] and its metabolites 1-(2-methylsulfonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropylpropan-1,3-dione and 2-methylsulphonyl-4-trifluoromethyl benzoic acid, calculated as the parent compound, in or on the raw agricultural commodity field corn at 0.20 parts per million (ppm), field corn, fodder, at 0.50 ppm, field corn, forage at 1.0 ppm; and

establishing a tolerance for combined residues of the herbicide isoxaflutole [5-cyclopropyl-4-(2-methylsulfonyl-4-trifluoromethyl benzoyl)isoxazole] and its metabolite 1-(2-methylsulfonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropylpropan-1,3-dione, calculated as the parent compound, in or on the liver of cattle, goat, hogs, horses, poultry and sheep at 0.40 ppm, meat byproducts (except liver) of cattle, goat, hogs, horses, and sheep at 0.2 ppm and milk at 0.02 ppm. The proposed analytical method is gas chromatography. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petitions. Additional data may be needed before EPA rules on the petitions.

As required by section 408(d) of the FFDCA, as recently amended by the Food Quality Protection Act (FQPA) Rhone-Poulenc included in the petition a summary of the petition and authorization for the summary to be published in the Federal Register in a notice of receipt of the petition. The summary represents the views of Rhone-Poulenc; EPA is in the process of evaluating the petition. As required by section 408(d)(3) EPA is including the summary as a part of this notice of filing. EPA may have made minor edits to the summary for the purpose of clarity.

I. Petition Summary

A. Isoxaflutole Uses

Isoxaflutole is the first compound in a new class of isoxazole herbicides. Weeds found resistant to other herbicides are not cross resistant to isoxaflutole. The unique mode of action, which disrupts pigment biosynthesis in susceptible plants, of isoxaflutole provides excellent selective control for a wide spectrum of grass and broadleaf weeds at low use rates.

Isoxaflutole will be used on field corn to control broadleafs (including Kochia, lambsquarters, mallow, mustard, nightshade, pigweed, ragweed, smartweed, velvetleaf, and waterhemp); grasses (including barnyardgrass, cupgrass, foxtails, Panicum and wild proso millet).

Isoxaflutole will be applied in either conventional, conservation tillage, or no-till crop management systems and may be applied either pre-plant, pre-plant incorporated or preemergence for use in field corn production. The product controls emerging weeds and also has postemergent burn-down activity to small exposed weeds. Application rates for isoxaflutole alone range from 0.035 to 0.14 pounds active ingredient per acre dependent on soil texture. Combinations of isoxaflutole with up to one-half rates of other herbicides improves control of several annual grasses and dramatically reduces total herbicide volume usage in comparison with current agronomic practices. Applications can be made up to 14 days before planting field corn in either conventional or no-till situations. Isoxaflutole is formulated as a 75 percent water dispersible granule and will be marketed under the trade name of ''BALANCE''.

B. Isoxaflutole Safety

Rhone-Poulenc Ag Company has submitted 41 separate toxicology studies in support of tolerances for isoxaflutole. According to Rhone-Poulenc, isoxaflutole is not acutely toxic and produces minimal skin and eye irritation. Further, isoxaflutole is not genotoxic, teratogenic nor a reproductive toxin.

The following mammalian toxicity studies have been conducted to support the tolerance of isoxaflutole:

A rat acute oral study with an LD_{50} of greater than 5,000 milligrams/kilogram (mg/kg).

A rabbit acute dermal LD_{50} of greater than 2,000 mg/kg.

A rat acute inhalation of LC_{50} of greater than 5.23 milligram/litre (mg/L).

A primary eye irritation study in the rabbit which showed minimal irritation.

A primary dermal irritation study in the rabbit which showed minimal irritation.

A primary dermal sensitization study in the guinea pig which showed no sensitization.

An acute neurotoxicity study conducted in rats administered a single dose at 0, 125, 500 or 2,000 mg/kg with a no observed effect level (NOEL) of 2,000 mg/kg (limit dose) and no treatment-related effects at any dose.

A 90–day subchronic neurotoxicity study in rats administered at dose levels of 0, 25, 250 or 750 milligrams/kilogram of body weight per day (mg/kg bwt/day) with NOEL of 750 mg/kg/day. This dose is also the Lowest Effect Level (LEL) for non-neurotoxic effects based on a significant decrease in mean body weight gain.

A 12–month feeding study in dogs administered at levels of 0, 240, 1,200, 12,000 or 30,000 ppm with NOEL of 1,200 ppm based on slight changes in liver and kidney weights in the absence of any associated histopathological changes.

A 24–month chronic feeding/oncogenicity study in rats administered at levels of 0.5, 2, 20 or 500 mg/kg bwt/day) with an overall NOEL of 2.0 mg/kg/day based on non-neoplastic changes in the cornea, sciatic nerve, thigh muscle, thyroid and liver observed at 20 mg/kg/day. An increased incidence of hepatocellular adenomas and carcinomas was observed at 500 mg/kg bwt/day for males and females. In addition, most of the 500 mg/kg/day males with liver tumors also had follicular cell adenomas in the thyroid.

An oncogenicity study in mice administered 0, 25, 500 and 7,000 ppm with a NOEL of 25 ppm based on a slight effect on liver weight and body weight gain at the LEL of 500 ppm. An increased incidence of hepatocellular adenomas and carcinomas was observed at 7,000 ppm in both sexes. Increased liver weight, non-neoplastic cellular changes in the liver, and amyloidosis in the duodenum, ileum, jejunum, kidneys, heart ventricle, mesenteric lymph node, and thyroid were also observed at 7,000 ppm.

A developmental toxicity study in rats administered at doses of 0, 10, 100 or 500 mg/kg bwt/day on gestation days 6 through 15 with a maternal NOEL of 100 mg/kg/day based on salivation and lower body weight, body weight gain and food consumption observed at 500 mg/kg/day and a fetal NOEL of 10 mg/kg/day based on growth retardation and increased incidences of vertebral and rib anomalies and subcutaneous edema observed at 100 mg/kg/day.

A developmental toxicity study in rabbits administered at levels of 0, 5, 20 or 100 mg/kg bwt/day on gestation days 6 through 19 with a maternal NOEL of 20 mg/kg/day based on no weight gain and decreased food consumption observed at 100 mg/kg/day and fetal NOEL of 5 mg/kg/day based on growth retardation and increased incidences of rib and vertebral anomalies noted at 20 mg/kg/day.

A 2 generation reproduction study in rats fed at dose levels of 0, 0.5, 2, 20 or 500 mg/kg bwt/day with a NOEL for postnatal development and parental toxicity of 2 mg/kg/day based on increased liver weight and hepatocellular hypertrophy in F0 and F1 adults and a slightly lower viability index for F1 pups at 20 mg/kg/day. No adverse effects on mating or fertility indices and gestation, live birth or weaning indices were noted in any generation.

Mutagenicity—Ames Assay. Negative with and without metabolic activation.

Mouse lymphoma. Negative with and without metabolic activation.

In-vivo Mouse Micronucleus Assay. Negative.

In-vitro Cytogenetics Human Lymphocyte Assay. Negative in the presence and absence of metabolic activation.

A metabolism study in the rat which demonstrates that the majority of the total radioactivity (TRR) is excreted within 24 to 48 hours through the urine and feces. Isoxaflutole is metabolized primarily via hydrolysis to the 1-(2-methylsulfonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropylpropan-1,3-dione (RPA 202248) followed by either reduction of the cyanonitrile group to form RPA 205834 or further hydrolysis to 2-methylsulphonyl-4-trifluoromethyl benzoic acid (RPA 203328). The RPA 202248 is the major metabolite excreted while RPA 203328 is the most polar. Thus, the acute oral toxicity and mutagenic potential of these two metabolites were assessed.

In the acute oral toxicity studies, RPA 203328 had an oral LD_{50} greater than 5,000 mg/kg while RPA 202248 had an oral LD_{50} greater than 2,000 mg/kg in fasted rats. At 5,000 mg/kg, RPA 202248 produced 40 percent mortality in both male and female rats. In Ames assays, both RPA 202248 and RPA 203328 were found to be devoid of mutagenic activity in the absence and presence of metabolic activation.

In the 28-day rat study, RPA 203328 was administered continuously in the diet at levels of 0, 150, 500, 5,000, and 15,000 ppm (10 rats/sex/group). No mortalities or treatment-related clinical signs were observed during the study. No effects were observed on body weight, food consumption, hematology, clinical chemistry, urinalysis, or ophthalmoscopy. Further, no changes in organ weight or histopathology were noted at any level. The NOEL of 15,000 ppm is equivalent to 1,120 mg/kg/day in males and 1,270 mg/kg/day in females.

C. Chronic Dietary Effects

Based upon all available data, the lowest NOEL of 2.0 mg/kg/day was observed in the chronic rat study. Using this NOEL and a safety factor of 100, a theoretical Reference Dose (RfD) of 0.02 mg/kg/day is obtained. The only pending registration for isoxaflutole is for use in/on field corn. A chronic dietary risk assessment using the maximum residue limits proposed in this petition, and a 100 percent crop treated shows that this use represents 1.8, 4.8, 5.3, and 3.3 percent of the RfD for the whole U.S. population, for non-nursing infants less than 1 year old, for children aged 1 to 6 years, and for children aged 7 to 12 years, respectively. Realizing that isoxaflutole is likely to achieve only a 25 percent market share at maturity, less than 1.5 percent of the RfD is reached for all segments of the population. Thus, Rhone-Poulenc believes that the anticipated dietary exposure to isoxaflutole is well below the theoretical RfD of 0.02 mg/kg/day and is negligible for all segments of the population including infants and children.

Isoxaflutole presents a minimal acute hazard. The acute oral NOEL is at least 1,000–fold higher than lowest chronic NOEL of 2 mg/kg/day indicating that acute exposure is unlikely to constitute any significant dietary risk. Further, as field corn is generally not directly consumed, no significant acute dietary exposure is likely to occur.

D. Aggregate Exposure

The FQPA of 1996 lists three other potential sources of exposure to the general population that must be addressed. These are pesticides in drinking water, exposure from non-occupational sources, and the potential cumulative effect of pesticides with similar toxicological modes of action. These exposures for isoxaflutole are discussed below.

1. *Drinking water.* There is no established maximum contaminant level (MCL) or health advisory level (HAL) for isoxaflutole nor its primary metabolite, 1-(2-methylsulphonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropane-1,3-dione. In the field dissipation study, the half-life for isoxaflutole was up to 3.0 days and for 1-(2-methylsulphonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropane-1,3-dione was 16 days under actual field conditions. Residues were only found in the uppermost depths (above 12 inches). Based upon the data generated by this study, isoxaflutole and its primary metabolite have a low potential for reaching groundwater. Under actual use conditions, neither compound is expected to be present at toxicologically significant concentrations in ground water due to the low application rate (maximum use rate 0.14 lbs. per acre) and the low acute toxicity of each compound. Therefore, Rhone-Poulenc does not anticipate the presence of isoxaflutole residues in drinking water.

2. *Non-occupational exposure.* Isoxaflutole is being proposed for use on field corn only at this time. Thus, non-occupational exposure to isoxaflutole via dermal or inhalation routes does not exist and dietary exposure is the only consideration for risk assessment purposes.

3. *Common mechanism of action.* No other pesticides have been identified which inhibit 4-HPPDase. The thyroid and liver tumors observed with isoxaflutole in the rodent studies are most likely indirectly related to a significant induction of the hepatic microsomal enzymes PROD, BROD, and UDPGT. While hepatic microsomal enzyme induction in rodents is likely to be produced by many other pesticides, there is no data to indicate that these effects would be cumulative with any other pesticide. Considering the rapid elimination of isoxaflutole in the animal metabolism study, the effects associated with isoxaflutole are unlikely to be cumulative with any other compound. Further, considering the known sensitivity of the rat to the development of thyroid lesions in response to an imbalance of thyroid hormones and rodents to the development of liver tumors in response to the induction of microsomal enzymes, occurrence of these tumors via these mechanisms in rodent studies have little if any practical relevance for human cancer or risk assessment. Epidemiological studies support the position that neither thyroid tumors observed in rats due to an imbalance of thyroid hormones or liver tumors observed in rodents exposed to inducers of microsomal enzyme activity are likely to occur in humans.

Therefore, only the potential risks associated with exposure to isoxaflutole are considered for this assessment.

E. Determination of Safety for Infants and Children

Developmental toxicity (delayed ossification and rib and vertebral anomalies) were observed in the developmental toxicity studies. The NOELs were 10 mg/kg/day in rats and 5 mg/kg/day in rabbits. In a 2-generation reproduction study, pups from the high dose group of 500 mg/kg/day had significantly lower weights and a slightly lower viability index for both F1 and F2 litters and corneal lesions for F2 litters. Parental systemic toxicity for this dose group consisted of lower weight gain and food consumption, corneal lesions, increased liver weight, and hepatocellular hypertrophy. In addition, a slightly lower viability index was noted for F1 pups from the 20 mg/kg/day dose group but not for F2 pups. Parental systemic toxicity at 20 mg/kg/day included increased liver weight and hepatocellular hypertrophy.

Considering the conservative exposure assumptions in setting the tolerances and the dietary risk assessment assuming 100 percent crop treated, less than 5.5 percent of the RfD is utilized for non-nursing infants,

children 1 to 6 years old, and children 7 to 12 years old. No non-occupational sources of exposure exist for isoxaflutole. Therefore, based upon the completeness and reliability of the toxicity data and the conservative exposure assessment, Rhone-Poulenc believes that there is a reasonable certainty that no harm will result to infants and children from exposure to the residues of isoxaflutole and no additional uncertainty factor is warranted.

F. Estrogenic Effects

No evidence of estrogenic or androgenic effects were noted in any study. No adverse effects on mating or fertility indices and gestation, live birth, or weaning indices were noted in the 2-generation rat reproduction study. An imbalance of thyroid hormones related to the induction of UDPGT was noted in rats. However, considering species differences in the half-life of thyroid hormones in rodent versus primates (12 to 24 hours in rat compared to 5 to 9 days in humans) and differences in the responsiveness of thyroid cells to TSH, thyroid hormone levels in humans are unlikely to be affected by the extremely low levels of isoxaflutole residues that might be present in food. Therefore, Rhone-Poulenc believes that isoxaflutole is not likely to cause any endocrine effects in most species including humans.

G. Chemical Residue

The nature of the residue of isoxaflutole in plants and animals is considered understood. In plants, the metabolism proceeds through the hydrolysis of the isoxazole ring to form the primary degradate, 1-(2-methylsulphonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropane-1,3-dione, and further hydrolysis yields the second metabolite, 2-methylsulphonyl-4-trifluromethyl benzoic acid. In animals the metabolic pathway is very similar and the metabolites formed are primarily the 1-(2-methylsulphonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropane-1,3-dione with two other toxicologically insignificant minor degradates.

An analytical method is available for detecting and measuring levels of isoxaflutole in field corn with a limit of quantitation of 0.01 ppm. The method involves hydrolysis of isoxaflutole to a methyl ester for gas chromatography analysis.

A total of 32 field corn trials were conducted in 13 different states. The maximum residues were 0.88 ppm in forage, 1.1 ppm in silage, 0.40 ppm in fodder and 0.11 ppm in grain. Based on these data, the proposed tolerance levels are adequate to cover residues likely to be present from the proposed use of isoxaflutole. Isoxaflutole residues do not appear to concentrate in corn processed commodities. Therefore, no food additive tolerances are being proposed for these processed commodities.

In animal feeding studies, quantifiable residues in the cow were observed only in liver (up to 0.8 ppm), kidney (up to 0.2 ppm) and milk (up to 0.03 ppm) at the 46 ppm (10X) dietary burden level. No residues were observed in fat or muscle. In poultry, quantifiable residues were observed only in the liver (up to 0.6 ppm) at the highest dose level of 1.8 ppm (10X dietary burden). No residues of isoxaflutole nor its primary metabolite, 1-(2-methylsulphonyl-4-trifluoromethylphenyl)-2-cyano-3-cyclopropane-1,3-dione, were observed in eggs, meat, fat or muscle. Based on these data and the expected (1X) dietary burden in animal feed, the proposed tolerance levels are adequate to cover residues likely to be present in animal tissues resulting from the corn feed items of the animal's diet.

II. Public Record

EPA invites interested persons to submit comments on this notice of filing. Comments must bear a notification indicating the docket control number [PF–701].

A record has been established for this notice under docket control numbers [PF–701] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in Rm. 1132 of the Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments can be sent directly to EPA at:

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

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

Authority: 21 U.S.C. 346a.

List of Subjects

Environmental Protection, Administrative practice and procedure, Agricultural commodities, Pesticide and pest, Reporting and recordkeeping requirements.

Dated: February 11, 1997.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 97–4628 Filed 2–25–97; 8:45 am]

BILLING CODE 6560–50–F

[OPP–00468; FRL–5587–4]

Pesticide Product Label System; Notice of Availability

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the availability of the new Pesticide Product Label System on CD ROM which supersedes and replaces the Compact Label File on microfiche.

FOR FURTHER INFORMATION CONTACT: By mail: BeWanda Alexander, Office of Pesticide Programs (7502C), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location for commercial courier delivery and telephone number: Rm. 700N, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, (703) 305–5259.

ADDRESSES: For specific address and price information, refer to Unit II. of this document.

SUPPLEMENTARY INFORMATION:

I. Introduction

The Pesticide Product Label System (PPLS), a software product developed by EPA's Office of Pesticide Programs (OPP), contains images of registered pesticide product labels submitted by pesticide registrants and accepted by OPP since 1971.

The label images have been indexed by company, product, and date. The retrieval program allows the user to search by registration number, which is a combination of company number and product number. Searches can be conducted based on partial numbers if