[PF–720; FRL–5592–6]

BASF Corporation; Pesticide Tolerance Petition Filing

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of filing.

SUMMARY: This notice announces the filing of a pesticide petition proposing regulations establishing tolerances for residues of vinclozolin [3-(3,5-dichlorophenyl)-5-methyl-5-vinyl-1,3-oxazolidine-2,4-dione)] and metabolites containing the 3.5-dichloroanaline moiety at 5.0 ppm to control Botrytis gray mold and Scelertinia white mold on succulent beans. In conjunction with this petition, BASF is requesting that the tolerances for prunes, plums, tomatoes grapes (excluding grapes grown for wine production) and raisins be withdrawn by the Agency. This notice includes a summary of the petition that was prepared by the petitioner, BASF Corporation.

DATES: Comments, identified by the docket number [PF–720], must be received on or before April 18, 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 be 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 5.1 file format or in ASCII file format. All comments and data in electronic form must be identified by docket control number [PF–720]. Electronic comments on this notice may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found below 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: Connie Welch, Product Manager (PM) 21, 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. 227, CM#2, 1921 Jefferson Davis Highway, Arlington, VA 703–305–6226. e-mail: welch.connie @epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petition (PP) 9F3762 from BASF Corporation, Agricultural Products, PO Box 13528, Research Triangle Park, NC 27709, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C section 346a (d), to amend 40 CFR part 180 by establishing tolerances for residues of vinclozolin [3-(3,5-dichlorophenyl)-5-methyl-5-vinyl-1,3-oxazolidine-2,4-dione) and metabolites containing the 3,5-dichloroanaline moiety when used as a fungicide in or on raw agricultural commodity succulent beans at 5.0 ppm. EPA has determined that the petition contains data or information 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 these data support granting of the petition. Additional data may be needed before EPA rules on the petition. The proposed analytical method is gas chromatography using Electron Capture detection.

As required by section 408(d) of the FFDCA, as recently amended by the Food Quality Protection Act, BASF Corporation 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 BASF; EPA, as mentioned above, 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. Plant Metabolism

BASF Corporation notes that metabolism in plants is understood, the residues of concern are vinclozolin [3-(3,5-dichlorophenyl)-5-methyl-5-vinyl-1,3-oxazolidine-2,4-dione) and metabolites containing the 3,5-dichloroanaline moiety.

B. Analytical Method

The proposed analytical method involves extraction, hydrolysis, distillation, partition, and deriviatization followed by detection of residues by gc/ecd. An enforcement method has been published in FDA's **Pesticide Analytical Methods**, Volume II pg. 876–887.

C. Magnitude of the Residues

Sixteen residue trials were carried out in 7 succulent bean producing states; CA, FL, MI, NY, NC, OR, and WI. Residue in the succulent beans ranged from 0.38 to 2.40 ppm and averaged 0.83 ppm.

D. Toxicological Profile

1. *Acute Toxicity*. The acute toxicity studies place technical vinclozolin in acute toxicity category IV for acute oral (LD_{50} of >15,000 mg/kg), and inhalation (LD_{50} of 29.1 mg/l) and acute toxicity category III for acute dermal (LD50 of >5,000 mg/kg) , eye (minimal) and dermal (minimal) irritation and the technical material is a positive skin sensitizer.

2. *Chronic Toxicity Testing.*

a. Chronic feeding Nonrodent. A 1-year feeding study in dogs fed dosages of 0, 1.1 , 2.4, 4.9, and 48.7 mg/kg/day with a No-Observed Adverse-Effect Level (NOAEL) of 2.4 mg/kg/day based on the following effects: (*1*) slight decrease in hematological and increase clinical chemistry values in the 48.7 mg/kg/day dose group (highest dose tested - (HDT)); (*2*) increased absolute and/or relative weights for the testes (male only), adrenals, liver, spleen, and thyroids in the either the 4.9 or 48.7 mg/kg/day dose groups; and (*3*) a dose-related atrophy of the prostate in the 4.9 or 48.7 mg/kg/day dose groups; and (*4*) microscopic findings in the adrenal and testes (males) in the 48.7 mg/kg/day dose group and liver findings for both male and female dogs in the 48.7 mg/kg/day dose groups and in the females in the 4.9 mg/kg/day dose group, only.

b. Chronic feeding/Oncogenicity - Rats. A combination of 2 chronic feeding and one carcinogenicity studies that were performed separately, resulted in rats being fed combined dosages of 0, 1.2, 2.4, 7.0, 23, 71, 143, and 221 mg/kg/day (males) and 0, 1.6, 3.1, 7.0, 23, 71, 180, and 221 mg/kg/day (females) with a NOAEL of 1.2 mg/kg/day (males) and 1.6 mg/kg/day (females) based on the following effects: (*1*) decreased body weights in both males and female rat at

dose levels ≥23 mg/kg/day dose groups with a progression of severity to the upper levels; (*2*) decreased food consumption in both males and female rats at dose levels ≥71 mg/kg/day dose groups with a progression of severity to the upper dose levels; (*3*) cataracts with associative histopathology at dose levels ≥23 mg/kg/day and lenticular changes at dose levels ≥7.0 mg/kg/day for male and female rats; (*4*) hematological and clinical chemistry value changes at dose levels ≥71 mg/kg/day dose groups with increase of severity at the higher doses tested; (*5*) increased absolute and/or relative weights for adrenals at dose levels ≥143 mg/kg/day, for the liver at dose levels ≥71 mg/kg/day, for the testes at dose levels ≥23 mg/kg/day, and for the ovaries at dose levels ≥143 mg/kg/day; (*6*) microscopic findings were observed in the liver, adrenal, pancreas, testes (males), ovaries and uterus (females) were seen in dose levels of ≥7.0 mg/kg/day with a progression of severity of histological effects in the upper dose levels; and (*7*) an increased incidence of neoplasms occurred at dose levels greater than the maximum tolerated dose (MTD) of 23 mg/kg/day in the liver, adrenals, pituitary, prostate (males), uterus (females), and ovaries (females) at dose levels ≥143 mg/kg/day. In the testes (males), neoplasms were seen slightly below the MTD at dose levels ≥7.0 mg/kg/day due the antiandrogenic nature of vinclozolin.

3. *Oncogenicity - Mice.* An oncogenicity study in mice fed dosages of 0, 2.1, 20.6, 432, and 1,225 (HDT) mg/kg/day (males) and 0, 2.8, 28.5, 557, and 1,411 (HDT) mg/kg/day (females) with a NOAEL of 20.6 mg/kg/day (males) and 28.5 mg/kg/day (females) based on the following effects: (*1) increased mortality in the highest dose tested (HTD) as compared to controls; (2*) decreased body weights and significant signs of clinical toxicity were observed in both males and female mice at the upper two dose levels with a progression of severity; (*3*) hematological and clinical chemistry value changes were observed at the highest dose tested; (*4*) increased absolute and/or relative weights for adrenals and liver were observed at the upper two dose levels, atrophic seminal vesicles and coagulation glands with reduction of the prostate (males) and atrophic uteri were observed at the upper two dose levels; (*5*) microscopic findings were observed in the liver, adrenal, testes (males), ovaries and uterus (females), and related sexual organs were seen in the upper two dose levels; (*6*) an increased incidence of neoplasms occurred at dose levels greater than the maximum tolerated dose (>28.5 mg/kg/day) in the liver of female mice.

4. *Developmental Toxicity Testing*

a. Teratology - Rat. A combination of four developmental studies in rats via oral gavage resulted in dosages of 0, 15, 50, 100, 150, 200, 400, 600, and 1000 (HDT) mg/kg/day with a development toxicity NOAEL of 15 mg/kg/day and a maternal toxicity NOAEL equal to or greater than 400 mg/kg/day based on the following: (*1*) no obvious signs of maternal toxicity were observed at dose levels less than or equal to 400 mg/kg/day; (*2*) an increased number of fetus with retarded ossification of thoracic vertebral bodies at dose levels greater than or equal to 200 mg/kg/day and increased number of fetus with soft tissue variations at dose levels greater than or equal to 400 mg/kg/day, both findings are regarded as unspecific embryo-/fetotoxic effects indicating transient delays in development but not indicative of a teratogenic effect; and (*3*) a statistical significant decrease or reduction of the anogenital index (AGI) in male was observed at levels greater than or equal to 50 mg/kg/day.

In a developmental study in rats via dermal exposure for six hours/day on intact skin with dosages of 0, 60, 180, and 360 mg/kg/day (HDT) with a development toxicity NOAEL of 60 mg/kg/day and a maternal toxicity NOAEL of 60 mg/kg/day based on the following: (*1*) increased absolute liver weights at dose levels >180 mg/kg/day; and (*2*) decreased anogenital distance and index at dose levels >180 mg/kg/day.

b. Teratology - Rabbits. A developmental study in rabbits via oral gavage resulted in dosages of 0, 20, 80, and 300 mg/kg/day (HDT) with a development toxicity NOAEL of 300 mg/kg/day and a maternal toxicity NOAEL of 300 mg/kg/day based on no signs of maternal or meaningful fetal toxicity were observed at any of the dose levels mentioned.

A second developmental study in rabbits via oral gavage resulted in dosages of 0, 50, 200, and 800 mg/kg/day (HDT) with a development toxicity NOAEL of 200 mg/kg/day and a maternal toxicity NOAEL of 50 mg/kg/day based on the following: (1) severe maternal toxicity with simultaneous change in hematological values changes and high number of abortions at the HDT; and (2) increased absolute and/or relative weights for adrenals in the mid and high dose groups.

5. *Reproductive Toxicity Testing*

a. Two-Generation Reproduction - Rat. A two-generation reproduction study (consisting of two studies: study A - dose levels of 0, 2.0 and 4.1 mg/kg/day; study B - dose levels of 0, 4.9, 29 100, and 307 mg/kg/day) with rats fed dosages of 0, 2.0, 4.1, 4.9, 29, 100, and 307 mg/kg/day with a reproductive NOAEL of 4.9 mg/kg/day based on feminization of male and the ability not to mate at dose levels >100 mg/kg/day and pup effects at 29 mg/kg/day; and with a parental NOAEL of 4.9 mg/kg/day based on general toxicity consistent with previous rat studies at levels >29 mg/kg/day. Study A was performed to clarify an equivocal finding of decreased absolute and relative weight of the epididymides without any morphological correlation in the male FY and FZ generations in Study B. However, EPA stated ''the effects at the 4.9 mg/kg/day dose level was minimal and considered sufficiently close to a NOAEL. The study is acceptable and 4.9 mg/kg/day dose level was considered to be the No Observed-Effect Level (NOEL).''

6. *Mutagenicity*

A Modified Ames Test (3 studies; point mutation): Negative; Host-Mediated Assay (point mutation): Negative; Mouse Lymphoma Test (point mutation): Negative; *In Vitro* CHO Cells (point mutation): Negative; *In Vitro* Cytogentics - CHO Cells (Chromosome Aberrations): Negative; *In Vivo* Dominant Lethal Test - Male NMRI Mouse (Chromosome Aberrations):Negative; Rec Assay (2 test; DNA damage and repair): Negative; *In Vitro* UDS Test Using Hepatocyte (DNA damage and repair): Negative; *In Vivo* SCE Using Chinese Hamster (DNA damage and repair): Negative

Based on the data present and weight of evidence, BASF concludes that vinclozolin does not pose a mutagenic hazard to humans.

7. *Other Relevant Testing*

a. Mechanistic Studies/Mode of Action - Anti-androgenicity Activity

A series of mechanistic studies were performed to elucidate and define the anti-androgenic properties of vinclozolin. The following conclusions can be drawn from the *in vivo* data:

The anti-androgenic effects observed are not related to an inhibition of androgen-steroid hormone synthesis.

The anti-androgenic effects are not related to an inhibition of 5 alpha-reductase activity.

The anti-androgenic effects are a result of a competitive binding to the androgen receptor resulting in an inactivation of this receptor.

The anti-androgenic effects are mediated by the hydrolysis metabolite M1 and probably not by vinclozolin or the main metabolite, R8.

The anti-androgenic effects are related to a second hydrolysis metabolite M2 which is a slightly more potent anti-androgen

than M1. However M2 concentrations are very low and the compound may not contribute much to the in vivo effects.

b. Metabolism - Rat

i. Oral studies. BASF has submitted results from a number of metabolism studies using wistar rats. The results of these studies can be summarized as follows: vinclozolin is well absorbed (@85 percent) and intensively metabolized, the liver playing an important role (@65 percent of the radioactivity administered was found in the bile and no unchanged active ingredient was excreted in the urine). The determination of radioactivity in the plasma over a period of seven days showed that slight accumulation took place.

ii. Dermal study. In an *in vivo* dermal absorption study, male Wistar rats were dosed with, ^{14}C vinclozolin. Dose levels of 0.002, 0.02, 0.2, and 2.0 mg/cm^2 were administered to 24 rats per dose level, applied to a shaved area of approx. 13 cm^2 on the back of the rat. Groups of 4 rats were sacrificed at 0.5, 1, 2, 4, 10, or 72 hours following application of the dose. Urine and feces were collected during this period. At the end of the exposure period (10 hours in the case of the 72 hour treatment group), the skin site was washed with cotton swabs moistened with water. A blood sample was taken prior to sacrifice. The treated skin along with the GI tract, liver, kidneys, adrenals, testes, eyes, brain and carcass were subjected to radioactive mass balance analysis. Urine from the bladder was added to the voided samples. Results of this analysis showed recoveries of between 81.6–104 percent. The lowest dose of 0.002 mg/cm^2 from the 10-hour exposure period is considered to be the most appropriate dose for use in the occupational risk assessment, as this dose most closely approximates the dermal deposition results obtained in the worker exposure studies. After the 10–hour exposure the total percent absorbed at this dose level was 29.l percent.

Percutaneous absorption of [^{14}C]-vinclozolin was also assessed in vitro using rat and human epidermis in flow-through diffusion cells. The test substance was applied at two dose levels, 200 ug/cm^2 (high) and 2 ug/cm^2 (low), and assessed over 24 hours. A total of 32 samples (16 rat and 16 human) were used at the high dose level, and 34 (17 rat and 17 human) at the low dose level. Samples of human skin were obtained at postmortem. Human epidermis was prepared from full thickness skin by immersion in water at 60 degrees Celsius for one minute. Rat epidermis was prepared by soaking the skin in 2M sodium bromide for approximately 24 hours. With respect to the worker exposure relevant time of eight hours, penetration through human skin was 16.7 times less at the high dose tested and 4.2 times less at the low dose tested than through rat skin.

E. Threshold Effects

The established Reference Dose (RfD) for vinclozolin is based on a 2-year feeding study in rats with a threshold NOAEL of 1.2 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.012 mg/kg/day.

F. Non-Threshold Effects

Vinclozolin is known to be an anti-androgenic agent, thus the consequence of hormonal imbalance are two-fold; the primary anti-androgenic effect is a suppression in androgen target organs such as epidymides, prostate or seminal vesicle, whereas stimulation is seen in organs involved in steroid hormone synthesis (testes, adrenals, ovaries). Target organs for hormones must be able to respond to changes in physiological levels of hormones, which can fluctuate significantly as evidenced by the hormone changes during the female estrus cycle. It was indeed demonstrated that changes induced in these organs were be reversible when hormone levels return to normal concentrations. It is only when hormone imbalance continues over a long time that irreversible changes occur.

In the case of suppression the affected organ is forced into a hypofunctional state. Progressively, the organ becomes hypotrophic and hypoplastic. With stimulation on the other hand the initial changes can be described as hyperfunction, hypertrophy and hyperplasia. As mentioned before, it is only when the hormonal imbalance continues over a long time that the ultimate reversible adaptation of the affected organ (hypoplasia or hyperplasia) is still not sufficient to handle the situation and only then an irreversible transition takes place. In the case of hormonal suppression atrophy is the ultimate consequence, in the case of stimulation, the ultimate consequence are tumors in the affected organs.

It is thus plausible that at dose levels which do not result in hypertrophy/hyperplasia or hypotrophy/hypoplasia the ultimate consequence of these adaptive changes, i.e. tumors or-atrophy, respectively, cannot occur. For risk assessment purposes this mode of action offers the possibility to determine a threshold for both tumor formation and atrophy by histopathological examination of the hyper- or hypo-functional organ. Thus, at dose levels which do not affect these organs, a mechanistic NOAEL can be defined and risk assessment can be carried out using assessment or safety factors.

The increase in neoplasia observed in the adrenals, ovaries and uterus were only seen in female rats at the high dose levels which was ≥143 mg/kg/day of the chronic toxicity study and/or carcinogenicity study. As determined by BASF and EPA, the 71 mg/kg/day dose level of the rat chronic/oncogenicity toxicity study exceeded the criteria for a MTD. Therefore, based the physiological status of the animals may be deteriorated in such a way that low dose extrapolation of results obtained at this dose level is not possible. Similarly, the liver tumors arising in the mouse oncogenicity at the 1,411 mg/kg/day dose level in which severe body weight losses and significant mortality were observed, clearly exceeding the MTD (as determined by BASF and EPA - **Cancer Peer Review Document**, September, 1996) and is not relevant for risk assessment purposes.

Additionally, vinclozolin is not a genotoxic agent and mechanistic studies have shown the increased incidence of liver tumors in male rat and female mice is a result of liver tumor promoting properties of the test substance. Vinclozolin is not an initiator of the carcinogenic event. Based on the available data, the mechanism of promotion is the induction of liver cell proliferation of the test substance. The data available also indicate that dose levels which do not induce liver toxicity also do not induce cell proliferation nor enhance the carcinogenic process. Therefore, BASF concludes that a threshold for liver carcinogenicity can be defined to be at least 143 mg/kg/day in the rat and at least 557 mg/kg/day in the mouse.

Concerning the testicular tumors (Leydig cell tumors), results of the long-term studies with vinclozolin demonstrate that hormone-related carcinogenesis was only observed in rats, and with the exception of Leydig cell tumors only at dose levels which exceeded the MTD criteria. The relevance of Leydig cell tumors to men should be seen in the light that this is a very rare human tumor and that the precursor change (i.e. Leydig cell hyperplasia) has not been observed in patients treated with flutamide. In addition, the toxicology of cimitidine, a H2-receptor antagonist with anti-androgenic properties resulting in a size reduction and atrophy of the prostate and seminal vesicles in chronic rat studies. Moreover, an increase in benign Leydig cell tumors, and a decrease in

pituitary and mammary tumor incidence were noted; hence a toxicity potential not unlike that of vinclozolin is evident. Despite the fact that over 30 million patients have been treated with cimitidine, this therapeutic agent has been demonstrated to be extremely safe, clearly indicating that the rat Leydig cell tumors have very little relevance for humans. A similar conclusion is drawn by other investigators ''Leydig cell tumors of the rat have limited significance because of the fundamental differences in testicular control mechanisms.'' It is therefore concluded that the observed neoplastic changes do not pose a relevant hazard to humans. EPA in the September, 1996, **Cancer Peer Review Document**, came to the same basic conclusion that the Leydig cell tumors are a very uncommon tumor type in humans which implies the threshold dose for humans would be greater than for rats. EPA based this conclusion on the work performed by Dr. C. Capen (Professor Charles C. Capen, Leydig Cell Tumors: Pathology, Physiology, and Mechanistic Considerations in Rats, The Toxicology Forum, 1994 Annual Summer Meeting, p. 110).

In the EPA **Carcinogenicity Peer Review Document** of September 1996, the Agency stated ''[T]his classification of Group B2 was based on statistically significant increases in multiple tumor types in male Wistar rats and ovarian tumors in female Wistar rats at a dose which was excessive. The MOE approach was chosen because the tumors were benign at dose levels which were considered to be excessive, and there was little concern for mutagencity of vinclozolin. Mechanistic data for the Leydig cell tumors also provided further support for the use of the MOE approach.'' It is BASF's understanding that EPA has performed another **Cancer Peer Review** in January, 1997. This rereview is based the pathology peer review performed by Dr. Charles Capen (Professor of Pathology at The Ohio State University) concerning the criteria used in diagnosing the ovarian and prostate tumors seem in the chronic/oncogenicity rat study for vinclozolin and the report of the FIFRA Scientific Advisory Panel (SAP) concerning vinclozolin. The data generated by Dr. Capen were also presented to the SAP by BASF and are a bases for the conclusions drawn by the Panel. BASF is awaiting the result of this second **Cancer Peer Review**.

To further support the conclusion stated above, the most recent meeting of the FIFRA SAP concluded the following: (Proceeding of the October, 1996, meeting were issued in December, 1996)

''[B]ased on these data (as presented by BASF Corporation and EPA), it is far from established that vinclozolin is carcinogenic to the rat. It is not ruled out, however. In addition, there is little concern for mutagenicity as expressed by the Agency reviews...

''[B]ased on (1), we would consider the possibility that vinclozolin is a carcinogen in rats or mice, but the evidence for this is not compelling. The Panel believes that the classification of vinclozolin using the new guidelines would be ''not likely to be a carcinogenic hazard to humans...''

''[T]he most appropriate method of risk quantification is on a non-linear model, MOE approach based on a NOEL for non-neoplastic effects...''

Therefore both the Agency and the SAP have agreed that vinclozolin should be regulated as a threshold chemical using the standard margin of exposure approach, BASF concurs with those opinions.

G. Aggregate Exposure

1. *Dietary exposure.* For the purpose of assessing the potential chronic dietary exposure, BASF has estimated aggregate exposure based on Theoretical Maximum Residue Contribution (TMRC) for all exisiting tolerances and registered uses of vinclozolin including the proposed tolerance of vinclozolin on succulent beans at 5.0 ppm. In this analysis tolerance levels were used for all crops except stonefruit were used. In the case of stonefruit, anticipated residues based on the available residue data. Where reliable data were available and acceptable to the Agency percent crop treated was also used. This analysis revealed that for the general U.S. population and children - ages 1-6 (the most sensitive sub-population), vinclozolin treated crops utilized 25 percent and 45 percent, respectively, of the RfD (0.012 /kg/day). BASF estimates that withdrawal of the tolerances in prunes, plums, tomatoes, grapes (except those grown for wine production) and raisins will reduce the percent RfD consumed by at least one-third; reducing the percent RfD consumed in children - ages 1-6 from 45 percent to 30 percent and for the general population from 25 percent to 16 percent. BASF is currently analyzing the available monitoring data for vinclozolin residues to determine the actual exposure to vinclozolin residues. Preliminary analysis indicates that no sub-population in the United States is exposed to over 1 percent of the RfD.

EPA has expressed concern for acute dietary risk in the draft RED for the subgroup population -women of childbearing age (13 years and older) due to the hormonal effects of vinclozolin.

In response to this concern, BASF requested that Technical Assessment Systems, Inc. (TAS), conduct an acute dietary analysis for vinclozolin that used the current consumption data and exposure models capable of calculating a real world estimates of potential exposure to residues in food.

The acute exposure analysis, utilized the principles of Tier 1 and Tier 3 analyzes presented to the FIFRA Science Advisory Panel in September, 1995, and subsequently implemented by OPP/EPA. Using appropriate methodology, available residue distribution data, and percent crop treated information it was determined the margin of exposure to the most sensitive sub-population exceeded 100 (the value generally accepted by the Agency as sufficient) at the very conservative 99.9th percentile of the population; when all crops having tolerances; plus succulent beans and cranberries were included in the analysis. When prunes, plums tomatoes, grapes (excluding those grown for wine production) and raisins were removed from the analysis, the margin of exposure at the 99.9th percentile was determined to be 425 for women of childbearing age.

2. *''Other'' Exposure.* Other potential sources of potential exposure to vinclozolin for the general population to residues of vinclozolin are residues in drinking water and exposure from non-occupational sources. For drinking water, based on the available environmental fate data, BASF does not anticipate routine exposure to residues of vinclozolin in drinking water. There is no established Maximum Concentration Level (MCL) or Health Advisory Level (HAL) for vinclozolin under the Safe Drinking Water Act (SDWA).

For non-occupational exposure, vinclozolin is included in a number of formulations used for professional treatment of golf-courses and turf. Posting and notification procedures ensure that there is no exposure to the general public either during or following treatment.

BASF has a flowable formulation containing vinclozolin which is available to the homeowner for use on residential lawns. Treatment rates (1.0 oz a.i./1,000 sq. ft.) and the number of treatments allowed per year are low. BASF believes that this minor use will not impact significantly on the aggregate exposure to vinclozolin since this use represents less than 0.5 percent of total vinclozolin use.

Additionally, for the homeowner handler and homeowner postapplication, EPA has estimated these non-occupational exposure for vinclozolin in the draft Registration Eligibility Document issued to BASF on February 2, 1996. EPA stated the following:

a. ''The MOE's for homeowners handlers were greater than 100 for both the low-pressure hand-held sprayer and backpack sprayer assuming long-sleeve shirt, long pants, shoes, and socks are worn. All homeowner handlers were assumed not be exposed seven days or more in a 90-day period and, the short-term endpoint is used in determining the MOE's. The Application rate for homeowners was assumed to be five gallons of dilute spray per day.''

b. In a simmilar analysis for postapplication exposure for homeowners a margin of exposure greater than 100 was calculated based on a worst case analysis using strawberry data as a surrogate.

Therefore, based on the completeness and reliability of the toxicity data, and the assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of vinclozolin, including all anticipated dietary exposure.

H. Cumulative Exposure

BASF has considered the potential for cumulative effects of vinclozolin and other substances that have a common mechanism of toxicity. BASF is aware of two other substance active ingredients which are structurally similar, iprodione and procymidone. However, BASF believes that consideration of a common mechanism of toxicity is not appropriate at this time. This conclusion was similarly drawn by Rhone-Poulenc the manufacturer of iprodione in a recent Notice of Filing for that compound.

The Agency has previously noted both structural and toxicological similarities between iprodione, procymidone, and vinclozolin. BASF believes that there are clear differences in both the type and magnitude of effects observed after exposure to vinclozolin when contrasted with iprodione. BASF believes that there is no reliable data to indicate cumulative effects should be considered in reference to iprodione. As to procymidone, BASF is unaware of any conclusive data that would indicate a common mode of action with procymidone. It should also be noted that procymidone's tolerances are limited to grapes grown for wine production outside the United States.

I. Determination of Safety for U.S. Population

Reference Dose (RfD): Using the exposure assumptions described above and the completeness and the reliability of the toxicity data, BASF has estimated that aggregate exposure to vinclozolin will utilize less than 16 percent of the RfD for the US population. EPA generally has no concern for exposure below 100 percent of the RfD. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of vinclozolin, including all anticipated dietary exposure and all other non-occupational exposures.

J. Determination of Safety for Infants and Children

Reference Dose: Based on the completeness of vinclozolin's toxicological database and the risk assessment information cited above BASF believes the RfD used to assess safety to children should be the same as that for the general population, 0.012 mg/kg/day. BASF concluded that the most sensitive child population group is that of children ages 1 to 6. BASF has calculated that the exposure to this group to be <30 percent of the RfD for all uses including that proposed in this document. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to residues of vinclozolin, including all anticipated dietary exposure and all other non-occupational exposures.

K. Other Considerations

The qualitative nature of the residues in plants is adequately understood. Residues of the parent molecule, and metabolites containing the 3,5-dichloroaniline moiety are the only residues of concern. There is a practical analytical method for detecting and measuring levels of vinclozolin in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. There has been no need to establish meat, milk, poultry or egg tolerances as the crops including succulent beans on which vinclozolin is used do not represent animal feed items.

L. International Tolerances

A maximum residue level for succulent beans has not been established for vinclozolin by the Codex Alimentarius Commission.

M. Conclusions

BASF Corporation believes that the proposed use of vinclozolin on snap beans would not pose a significant risk to human health, including that of infants and children, and is in compliance with the requirements of the Food Quality Protection Act of 1996. Moreover, BASF believes that the proposed tolerance for vinclozolin on snap beans should be established.

II. Public Record

Interested persons are invited to submit comments on this notice of filing. Comments must bear a notation indicating the docket control number, [PF–720]. All written comments filed in response to this petition will be available in the Public Response and Program Resources Branch, at the address given above from 8:30 a.m. to 4 p.m., Monday through Friday, except legal holidays.

A record has been established for this notice under docket control number [PF–720] 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:

opp-docket@epamail.epa.gov

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 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, Pesticides and pests, Reporting and recordkeeping.

Dated: March 10, 1997.

Stephen L. Johnson,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 97–6909 Filed 3–18–97; 8:45 am]

BILLING CODE 6560–50–F

[OPP–181037; FRL 5593–8]

Dimethomorph; Receipt of Application for Emergency Exemption, Solicitation of Public Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has received a specific exemption request from the Florida Department of Agriculture and Consumer Services (hereafter referred to as the ''Applicant'') to use the pesticide dimethomorph (CAS 110488–70–5) to treat up to 7,250 acres of tobacco to control metalaxyl-resistant blue mold.The Applicant proposes the use of a new (unregistered) chemical; therefore, in accordance with 40 CFR 166.24, EPA is soliciting public comment before making the decision whether or not to grant the exemption.

DATES: Comments must be received on or before April 3, 1997.

ADDRESSES: Three copies of written comments, bearing the identification notation ''OPP–181037,'' should be submitted by mail to: Public Response and Program Resource 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, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

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 [OPP–181037]. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic comments on this proposed rule may be filed online at many Federal Depository Libraries. Additional information on electronic submissions can be found below in this document.

Information submitted in any comment concerning this notice may be claimed confidential by marking any part or all of that information as CBI. Information so marked 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 provided by the submitter for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments filed pursuant to this notice will be available for public inspection in Rm. 1132, Crystal Mall No. 2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, except legal holidays.

FOR FURTHER INFORMATION CONTACT: By mail: Libby Pemberton, Registration Division (7505W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number and e-mail: Floor 6, Crystal Station #1, 2800 Jefferson Davis Highway, Arlington, VA, (703) 308–8326; e-mail: pemberton.libby@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Pursuant to section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (7 U.S.C. 136p), the Administrator may, at her discretion, exempt a state agency from any registration provision of FIFRA if she determines that emergency conditions exist which require such exemption. The Applicant has requested the Administrator to issue a specific exemption for the use of dimethomorph on tobacco to control blue mold. Information in accordance with 40 CFR part 166 was submitted as part of this request.

In 1995 and 1996 a national epidemic of tobacco blue mold, caused by metalaxyl-resistant strains of the pathogen, occurred. Resistant strains are becoming more widely disseminated, a situation which is exacerbated with a protracted wet weather pattern. Previously, blue mold was controlled primarily by treatment with metalaxyl, with significant assistance from ferbam and mancozeb. A very high level of control was possible with these materials until metalaxyl-resistant strains appeared. Labeled pesticides made under ideal spray conditions but high disease pressure do not provide acceptable economic levels of control.

The Applicant states that presently, there are no fungicides registered in the U.S. that will provide adequate control of the metalaxyl-resistant strains of blue mold. The Applicant states that dimethomorph has been shown to be effective against these strains of blue mold. Dimethomorph holds current registrations throughout many European countries. The Applicant estimates that losses in 1997 could be greater than $7 million without use of dimethomorph. Under appropriate conditions, it is possible that this disease could develop to epidemic proportions, causing major changes and losses to the U.S. tobacco industry.

The Applicant proposes to apply dimethomorph at a maximum rate of 0.225 lbs. a.i. (2.5 lbs. of product) per acre, by ground with a maximum of 5 applications per crop, to a maximum of 7,250 acres of tobacco.

This notice does not constitute a decision by EPA on the application. The regulations governing section 18 require publication of a notice of receipt of an application for a specific exemption proposing use of a new chemical (i.e., an active ingredient not contained in any currently registered pesticide). Such notice provides for opportunity for public comment on the application. Accordingly, interested persons may submit written views on this subject to the Field Operations Division at the address above.

A record has been established for this notice under docket number [OPP–181037] (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.

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