unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4). Nor does it require any prior consultation as specified by Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998); special considerations as required by Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994); or require OMB review or any Agency action under Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). Since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 et seq.) do not apply. In addition, the Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999). Executive Order 13132 requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.'' This final rule directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4).

VIII. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this rule in the **Federal Register**. This rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

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

Dated: June 29, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180—[AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 321(q), (346a), and 371.

2. Section 180.555 is amended by alphabetically adding the following entries to the table in paragraph (a) to read as follows:

§ 180.555 Trifloxystrobin; tolerances for residues.

(a) * * *

Commodity	Parts per million
Almond, hulls	3.0
Almond, nutmeat	0.04
* * * * *	
Aspirated grain fractions	5.0
* * * * *	
Fruiting vegetables	0.5
* * * * *	
Hops, dried cones	11.0
* * * * *	
Potato, tubers	0.04
* * * * *	
Sugar beet, dried pulp	0.4
Sugar beet, molasses	0.2
Sugar beet, roots	0.1
Sugar beet, tops	4.0
Wheat, bran	0.15
Wheat, forage	0.3
Wheat, grain	0.05
Wheat, hay	0.2
Wheat, straw	5.0

* * * * *

[FR Doc. 00–18100 Filed 7–17–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–301015; FRL–6594–8]

RIN 2070–AB78

Vinclozolin; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for combined residues of vinclozolin, 3-(3,5-dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione and its metabolites containing the 3,5-dichloroaniline moiety in or on the raw agricultural commodities: succulent beans at 2.0 parts per million (ppm); canola at 1.0 ppm; eggs, milk, and the meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep at 0.05 ppm; and in the meat, fat, and meat byproducts of poultry at 0.1 ppm. These tolerances will expire and are revoked on September 30, 2003. BASF Corporation requested these tolerances under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996.

DATES: This regulation is effective July 18, 2000. Objections and requests for hearings, identified by docket control number OPP–301015, must be received by EPA on or before September 18, 2000.

ADDRESSES: Written objections and hearing requests may be submitted by mail, in person, or by courier. Please follow the detailed instructions for each method as provided in Unit VII. of the **SUPPLEMENTARY INFORMATION.** To ensure proper receipt by EPA, your objections and hearing requests must identify docket control number OPP–301015 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Mary L. Waller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection

Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–9354; and e-mail address: waller.mary@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

Categories	NAICS	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

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

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number OPP–301015. The official record consists of the documents specifically referenced in this action, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

II. Background and Statutory Findings

In the **Federal Register** of April 21, 2000 (65 FR 78) (FRL–6555–6), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Public Law 104–170) announcing the filing of a pesticide petition (PP 0F6079) for tolerances by BASF Corporation, Agricultural Products, P.O. Box 13528, Research Triangle Park, NC 27709. This notice included a summary of the petition prepared by BASF Corporation, the registrant. In addition, on June 2, 2000, the Agency added a supplemental notice of filing to the docket which summarized the toxicity and risk associated with the proposed tolerances. The Agency received comments from the Natural Resources Defense Council (NRDC), Earthjustice Legal Defense Fund (EJLDF), and BASF Corporation. The comments from outside parties are summarized in Unit IV below, followed by the Agency's response.

The petition requested that 40 CFR 180.380 be amended by establishing tolerances for combined residues of the fungicide vinclozolin, 3-(3,5-dichlorophenyl)-5-ethenyl-5-methyl-2,4-oxazolidinedione and its metabolites containing the 3,5-dichloroaniline moiety, in or on succulent beans at 2.0 ppm and canola at 1.0 ppm. The petition was later amended to request tolerances on eggs, milk, and the meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep at 0.05 ppm and in the meat, fat, and meat byproducts of poultry at 0.1 ppm.

Section 408(b)(2)(A)(i) of the FFDCA allows EPA to establish a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance is ''safe.'' Section 408(b)(2)(A)(ii) defines ''safe'' to mean that ''there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.'' This includes exposure through drinking water and in residential settings, but does not include occupational exposure. Section 408(b)(2)(C) requires EPA to give special consideration to exposure of infants and children to the pesticide chemical residue in establishing a tolerance and to ''ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue....''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. For further discussion of the regulatory requirements of section 408 and a complete description of the risk assessment process, see the final rule on Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997) (FRL–5754–7).

III. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances for combined residues of vinclozolin in or on succulent beans at 2.0 ppm; canola at 1.0 ppm; eggs, milk, and the meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep at 0.05 ppm; and the meat, fat, and meat byproducts of poultry at 0.1 ppm. EPA's assessment of the exposures and risks associated with establishing the tolerance follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children. The nature of the toxic effects caused by vinclozolin are discussed in this unit.

1. *Acute toxicity.* A battery of acute toxicity studies placed technical vinclozolin in toxicity category IV for acute oral toxicity (LD_{50} of > 10,000 mg/kg), and acute inhalation toxicity (LC_{50} of 29.1 mg/l); and toxicity category III for acute dermal toxicity (LD_{50} of > 5,000 mg/kg). Technical vinclozolin caused minimal eye and dermal

irritation and the technical material is positive for skin sensitization.

2. *Chronic toxicity.* i. A 1–year chronic 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 in 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), adrenal, liver, spleen, and thyroids in the 4.9 or 48.7 mg/kg/day dose groups; (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.

ii. A combination of two chronic feeding studies and one carcinogenicity study 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 male and female rats at dose levels ≥ 23 mg/kg/day with a progression of severity to the upper levels; (2) decreased food consumption in both male and female rats at dose levels ≥ 71 mg/kg/day with a progression of severity to the upper dose levels; (3) cataracts with associated 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 with increase of severity at the higher doses tested; (5) increased absolute and/or relative weights for adrenal 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) at 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, adrenal, pituitary, prostate (males), uterus (females), and ovaries (females) at dose levels ≥ 143 mg/kg/day. In the testes (males), Leydig cell adenomas were seen at the MTD for dose levels ≥ 23.0 mg/kg/day due to the anti-androgenic nature of vinclozolin.

3. *Carcinogenicity.* A carcinogenicity 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:

i. Increased mortality in the HDT as compared to controls;

ii. Decreased body weights and significant signs of clinical toxicity were observed in both male and female mice at the upper two dose levels with a progression of severity;

iii. Hematological and clinical chemistry value changes were observed at the highest dose tested;

iv. Increased absolute and/or relative weights for adrenal 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;

v. Microscopic findings were observed in the liver, adrenal, testes (males), ovaries and uterus (females), and related sexual organs in the upper two dose levels;

vi 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.* i. In four developmental toxicity studies, vinclozolin was given orally from gestational day (gd) 6 through 19 as follows: Study 4—dose levels of 0, 15, 50, or 150 mg/kg/day; study 5—dose levels of 0, 50, 100, 200 mg/kg/day, study 6—dose levels of 0, 200, 400 mg/kg/day and study 8—dose levels of 0, 600, and 1,000 mg/kg/day. At the gd 20, the fetuses were evaluated.

Maternal toxicity was demonstrated at 600 and 1,000 mg/kg/day by the statistically significant increase in absolute and relative adrenal and liver weight in study 8. This was the only study where organ weights were determined. A maternal NOAEL could not be established and therefore, the study was not considered to demonstrate any extra sensitivity. No histology was conducted on the organs, but other studies have demonstrated lipid accumulation in the adrenals, and centrilobular cloudiness of the liver. In addition, a dermal developmental study has indicated adrenal and liver weight increases occurred at 180 mg/kg/day and higher. Statistically significant increases and decreases occurred in the body weight gain and in food consumption with no apparent dose relatedness in any of the studies. The relative efficiency of food utilization was too variable to be definitive.

Statistically significant male and female fetal body weight decrement occurred at 1,000 mg/kg/day. These weight decrements were considered test material related. A statistically significant decrease occurred in anogenital distance among male fetuses. The term pseudohermaphroditism was used to describe the effect because these males exhibited decreased anogenital distances, but exhibited superficially normal internal testes. The anogenital distance in male fetuses was statistically decreased at 50 mg/kg/day and higher in studies 4, 6, and 8. (The anogenital index was statistically significantly depressed at 150 mg/kg/day and higher). The anogenital distance and index were not determined in study 5. The response was dose related. Although the anogenital index was not statistically significantly depressed at 50 mg/kg/day, it was nominally depressed. Considering the significantly depressed anogenital distance at 50 mg/kg/day and higher and the nominally depressed anogenital index at 50 mg/kg/day, the NOAEL for this study was considered to be 15 mg/kg/day, the lowest dose tested (LDT). These results are consistent with hormonal or anti-hormonal effects from the test material.

Soft tissue examination of fetuses indicated that increased incidence occurred in dilated renal pelvis and hydro-ureter at 400 mg/kg/day in study 6. At higher dose levels in study 8, the incidence of dilated renal pelvis and hydro-ureter was nominally increased. The failure of the dilated renal pelvis, and hydro-ureter to be significantly increased in study 8 was attributed to the fewer litters used (7, 5, and 8 in controls, 600, and 1,000 mg/kg/day). The NOAEL for these renal effects is considered to be 200 mg/kg/day.

Skeletal examination of fetuses indicated increased incidence of accessory 14th rib at 400 mg/kg/day and in fetuses and litters at 600, and 1,000 mg/kg/day. These effects on the 14th rib may be related to dose administration. Evaluation of the Preliminary Study suggested a dose related increase in 14th ribs at these high dose levels. No other dose related effects were reported.

The developmental toxicity NOAEL was set at 15 mg/kg/day and the developmental LOAEL was 50 mg/kg/day based on decreased anogenital distance in males. Increased incidence of dilated renal pelvis, hydro-ureter, and accessary 14th rib may have occurred at 400 mg/kg/day and higher. The maternal toxicity LOAEL was < 600 mg/kg/day based on increases in absolute

and relative adrenal and liver weight. Organ weights were not determined at lower dose levels.

ii. A developmental study in rats via dermal exposure for 6 hours/day on intact skin with dosages of 0, 60, 180, and 360 mg/kg/day HDT had a developmental NOAEL of 60 mg/kg/day and a maternal 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.

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

iv. 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 and high number of abortions at the HDT; and (2) increased absolute and/or relative weights for adrenal in the mid and high dose groups.

v. A two-generation rat 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 a reproductive NOAEL of 4.9 mg/kg/day based on decreased epididymal weight and male's inability 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, the Agency concluded that the effects at the 4.9 mg/kg/day dose level were minimal and considered sufficiently close to the NOAEL. The study is acceptable and the 4.9 mg/kg/day dose level was considered to be the NOAEL.

5. *Mutagenicity.* The following test/assays showed no evidence of mutagenic activity: Modified Ames Test (3 studies, point mutation); Host-Mediated Assay (point mutation); Mouse Lymphoma Test (point mutation); *In Vitro* CHO Cells (point mutation); *In Vitro* Cytogenetics—CHO Cells (Chromosome Aberrations); *In Vivo* Dominant Lethal Test—Male NMRI Mouse (Chromosome Aberrations); Rec Assay (2 test, DNA damage and repair); *In Vitro* UDS Test Using Hepatocyte (DNA damage and repair); and *In Vivo* SCE Using Chinese Hamster (DNA damage and repair).

6. *Mechanistic studies-anti-androgenicity activity.* A series of mechanistic studies (*In Vivo* and *In Vitro*) were conducted to define the anti-androgenic properties of vinclozolin. The results of these studies showed that vinclozolin elicits the anti-androgenic effects by binding to androgen sensitive organs.

B. Toxicological Endpoints

1. *Acute toxicity.* EPA selected the NOAEL of 6 mg/kg/day (adjusted for a single dose) from a developmental toxicity study in rats based on decreased ventral prostate weight in male offspring observed at the adjusted LOAEL of 11.5 mg/kg/day. The endpoint is the most sensitive indicator of acute anti-androgenic developmental toxicity. The population subgroup of concern is females (13+) because the endpoint is an *in utero* effect applicable only to females of childbearing age. An uncertainty factor of 100 was used to account for interspecies extrapolation and intraspecies variation. On this basis, the acute reference dose (aRfD) is 0.06 mg/kg/day. EPA determined that a 10X FQPA safety factor is applicable, and the margin of exposure (MOE) for the population subgroup of concern, females (13+) is 1,000X. The acute population adjusted dose (aPAD) is 0.006 mg/kg/day. An acute dose and endpoint were not identified for other population subgroups.

2. *Chronic toxicity.* EPA has established the Reference Dose (RfD) for vinclozolin at 0.012 mg/kg/day. This RfD is based on a NOAEL of 1.2 mg/kg/day from the combined chronic toxicity/carcinogenicity study in rats in which histopathological lesions occurred in the lungs and livers of male rats, in ovaries of females, and in the eyes of both sexes at the LOAEL of 2.3 mg/kg/day. An uncertainty factor of 100 was used to account for interspecies extrapolation and intraspecies variation. A 10X FQPA safety factor was added resulting in a cPAD of 0.0012 mg/kg/day.

3. *Short- and intermediate-term toxicity.* For short- and intermediate-term dermal and inhalation toxicity, the NOAEL of 3 mg/kg/day from a rat developmental toxicity study was selected for the population subgroup of concern, females (13+). The LOAEL of 6 mg/kg/day was based on decreased ventral prostate weights. For short- and intermediate-term dermal and inhalation toxicity, the NOAEL of 5 mg/kg/day from a rat developmental toxicity study was selected for the population subgroup of concern, infants and children. The LOAEL of 15 mg/kg/day was based on delayed puberty. A dermal absorption factor of 25% was used to correct for route-to-route extrapolation (oral to dermal exposure) and a default inhalation absorption factor of 100% was assumed for oral to inhalation exposure. The MOE for females (13+), infants and children is 1,000X.

4. *Long-term dermal and inhalation toxicity (cancer and non- cancer).* For chronic non-cancer and cancer dermal and inhalation toxicity, EPA selected the chronic NOAEL of 1.2 mg/kg/day from the combined rat chronic toxicity/carcinogenicity study in which histopathological lesions occurred in the lungs and livers of male rats, in ovaries of females, and in the eyes of both sexes at the LOAEL of 2.3 mg/kg/day. The $Q_1{}^*$ calculated in a low-dose linear extrapolation is 2.9×10^{-1} (mg/kg/day)$^{-1}$. A dermal absorption factor of 25% was used to correct for route-to-route extrapolation (oral to dermal exposure) and a default inhalation absorption factor of 100% was assumed for oral to inhalation exposure. The cancer assessment includes not only the adult U.S. population but also infants and children as well.

5. *Carcinogenicity.* Vinclozolin is classified as a Group C carcinogen based on Leydig (interstitial testicular) cell tumors in a perinatal rat developmental toxicity study. A non-linear (MOE) approach was determined to be appropriate based on a weight-of-the-evidence conclusion that tumor induction is via an anti-androgenic mechanism. Prostate weight decreases occurred at the LOAEL of 6 mg/kg/day; the point of departure for use in the non-linear risk assessment is 3 mg/kg/day (NOAEL). EPA believes that use of the population adjusted dose (PAD) for overall anti-androgenic effects (0.0012 mg/kg/day) is also protective of cancer effects because it is protective of the anti-androgenic effects that are, in effect, precursors to tumor formation.

6. *Overall anti-androgenic effects.* The Agency has determined that use of the most sensitive regulatory toxicity endpoint and the highest uncertainty factor (UF) would be protective of the anti-androgenic effects on all population subgroups caused by vinclozolin including developmental/reproductive effects as well as carcinogenic effects. In the case of vinclozolin, the most sensitive toxicity endpoint/dose and UF

are derived from the rat oral chronic/carcinogenicity study, i.e., the NOAEL of 1.2 mg/kg/day and an UF of 1,000. The PAD of 0.0012 mg/kg/day was used in assessment of risks resulting from the anti-androgenic activity of vinclozolin.

C. Exposures and Risks

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.380) for the combined residues of vinclozolin and its metabolites containing the 3,5-dichloroaniline moiety, in or on the following raw agricultural commodities: Belgian endive tops, cucumbers, wine grapes, kiwi fruit, head and leaf lettuce, dry bulb onions, bell peppers, raspberries, stone fruit (except plums/fresh prunes), and strawberries. There are no U.S. registered vinclozolin products for use on wine grapes, cucumbers, and peppers, and the current tolerances for these commodities are for imported commodities only. In addition, as a risk mitigation measure, BASF requested deletion of the strawberry and stone fruit uses from their vinclozolin label on June 30, 1998. The Agency published a **Federal Register** notice announcing the use deletion on July 30, 1998, (63 FR 40710) (FRL–6020–9) and under the existing stock plan, vinclozolin could be used on strawberries and stone fruit until January 30, 2000. Revocation of the stone fruit and strawberry tolerances are expected in the near future.

To further mitigate risk associated with the use of vinclozolin, the Agency is considering a proposal submitted by the registrant which includes the following items to occur over the next 5 years: A phase out of all domestic food uses of vinclozolin except for the use on canola, and the reinstatement of the snap bean tolerance for a period of 5 years; revocation of all import tolerances except for wine grapes to cover residues in wine; future phase out of use on sod farms resulting in the remaining turf use limited to golf courses; and voluntary cancellation of use on ornamental plants. In addition as a short-term risk reduction measure, label amendments were approved on June 14, 2000 to add a 24–day pre-harvest interval for sod harvested for residential uses.

The Agency has been petitioned by BASF Corporation to establish tolerances on the following commodities: Succulent beans; canola; eggs, milk, meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep; and fat, meat, and meat byproducts of poultry. Risk assessments were conducted by EPA to assess dietary exposure from vinclozolin as a result of all current tolerances (excluding stone fruit and strawberries) and all proposed tolerances. Strawberries and stone fruit were excluded because the use of vinclozolin on these crops was deleted and significant residues are not expected to occur in these crops as the latest possible use of vinclozolin under the existing stocks plan was January 30, 2000.

Section 408(b)(2)(E) authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide chemicals that have been measured in food. If EPA relies on such information, EPA must require that data be provided 5 years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the levels anticipated. Following the initial data submission, EPA is authorized to require similar data on a time frame it deems appropriate. As required by section 408(b)(2)(E), EPA will issue a data call-in for information relating to anticipated residues to be submitted no later than 5 years from the date of issuance of this tolerance.

Section 408(b)(2)(F) states that the Agency may use data on the actual percent of food treated for assessing chronic dietary risk only if the Agency can make the following findings: Condition 1, that the data used are reliable and provide a valid basis to show what percentage of the food derived from such crop is likely to contain such pesticide residue; Condition 2, that the exposure estimate does not underestimate exposure for any significant subpopulation group; and Condition 3, if data are available on pesticide use and food consumption in a particular area, the exposure estimate does not understate exposure for the population in such area. In addition, the Agency must provide for periodic evaluation of any estimates used. To provide for the periodic evaluation of the estimate of percent of crop treated (PCT) as required by section 408(b)(2)(F), EPA may require registrants to submit data on PCT.

The Agency used PCT data for domestic crops and percent of imported crop treated (PICT) data for all imported crops. Data on stone fruits and strawberries were not included as the uses have been deleted from labels. For the acute analysis, the estimated maximum PCT was used and for the chronic analyses, the weighted average PCT was incorporated.

The Agency believes that the three conditions listed above have been met. With respect to Condition 1, PCT estimates are derived from Federal and private market survey data, which are reliable and have a valid basis. EPA uses a weighted average PCT for chronic dietary exposure estimates. This weighted average PCT figure is derived by averaging State-level data for a period of up to 10 years, and weighting for the more robust and recent data. A weighted average of the PCT reasonably represents a person's dietary exposure over a lifetime, and is unlikely to underestimate exposure to an individual because of the fact that pesticide use patterns (both regionally and nationally) tend to change continuously over time, such that an individual is unlikely to be exposed to more than the average PCT over a lifetime. For acute dietary exposure estimates, EPA uses an estimated maximum PCT. The exposure estimates resulting from this approach reasonably represent the highest levels to which an individual could be exposed, and are unlikely to underestimate an individual's acute dietary exposure. The Agency is reasonably certain that the percentage of the food treated is not likely to be an underestimate. As to Conditions 2 and 3, regional consumption information and consumption information for significant subpopulations is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Use of this consumption information in EPA's risk assessment process ensures that EPA's exposure estimate does not understate exposure for any significant subpopulation group and allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency. Other than the data available through national food consumption surveys, EPA does not have available information on the regional consumption of food to which the pesticide may be applied in a particular area.

The dietary (food only) risk assessments used anticipated residues from field trial data which EPA believes are very conservative for the following qualitative reasons: (1) Field trial data assumes that all crops are treated at the maximum application rate and harvested at the minimum pre-harvest interval (PHI). In practice, crops are sometimes treated at lower application rates and harvested at longer PHI's leading to lower residues in the crops; (2) Field trial data assumes no decline between harvest and consumption of the crop. However, residues of vinclozolin will decline between harvest and consumption. Data are not available to

quantify the extent of this decline; (3) Home ''processing'' was not accounted for in the risk assessment. Practices such as washing, peeling, and cooking could lead to significantly lower residues than those from field trial data; and (4) For the acute dietary risk assessment, the vinclozolin metabolites of greatest concern are those closely related to the parent compound. Use of field trial data in the acute dietary assessment assumes that all residues have structures closely related to the parent compound and that they all elicit the developmental effects of concern. In reality, many metabolites convertible to 3,5-DCA may have structures different from the parent such that they are not of acute concern.

Although EPA cannot quantify for vinclozolin the combined residue reduction from the factors identified above, for many pesticides the difference in residues between field trial and monitoring data can be an order of magnitude 10X or more. The registrant is submitting processing (washing/cooking) studies which could allow for further future refinement of the dietary risk assessment.

The Dietary Exposure Evaluation Model (DEEM®), which incorporates consumption data generated in USDA's Continuing Surveys of Food Intakes by Individuals (CSFII), 1989–1992 was used to conduct the dietary risk assessments. For refined acute dietary risk assessments, the entire distribution of consumption events for individuals is multiplied by the distribution of residues to obtain a distribution of exposures in mg/kg/day. This is a probabilistic analysis, referred to as a ''Monte Carlo'' analysis and the risk is reported at various percentiles of exposure. For chronic dietary risk assessments, the 3–day average of consumption for each population subgroup is combined with residues in commodities to determine average exposure in mg/kg/day.

i. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1–day or single exposure. The acute dietary exposure estimates for the only population subgroup of concern (taking into account the toxicological studies on vinclozolin), females (13+), utilized the following percentage of the aPAD (0.006 mg/kg/day) at the various percentiles of exposure as indicated: 120% of the aPAD at the 99.9th percentile; 98% of the aPAD at 99.85th percentile; 83% of the aPAD at the 99.8th percentile; 73% of the aPAD at the 99.75th percentile; 60% of the aPAD at the 99.6th percentile; and 49% of the aPAD at the 99.5th percentile. Because the anticipated residues are based on field trial data and are conservative estimates (i.e. they overestimate residue levels), the Agency believes that basing its exposure estimate on the very upper ranges of potential exposure (the 99.5th and above) will unreasonably overestimate exposure. Considering this factor in choosing a population percentile of exposure that is adequately protective was explicitly discussed in EPA's policy on the use of population percentiles of exposure in acute risk assessments. U.S. EPA (Office of Pesticide Programs), ''Choosing A Percentile of Acute Dietary Exposure as a Threshold of Regulatory Concern'' (March 2000). In addition, as part of the reregistration process for vinclozolin, the registrant is proposing to further reduce the dietary exposure to vinclozolin, and the Agency may request future tolerance revocations for certain commodities as well. The very conservatively estimated acute dietary risk (food only) does not exceed the Agency's level of concern.

ii. *Chronic exposure and risk*. The chronic dietary exposure estimates expressed as a percentage of the cPAD (0.0012 mg/kg/day) were 4% for the U.S. population and 7% for the most highly exposed population subgroup, children (1–6 years old). EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Therefore, the chronic dietary risk (food only) does not exceed the Agency's level of concern.

iii. *For cancer and anti-androgenic risk assessment.* EPA believes that vinclozolin should be classified as a Group C carcinogen. The cancer risk assessment included both the U.S. population and infants and children. EPA believes the key concern for infants and children exposed to vinclozolin is the potential for developmental/reproductive effects related to the anti-androgenic properties of vinclozolin. In addition, the possibility of increased incidence of testicular Leydig cell tumors in adults as a result of exposure to vinclozolin as infants or children cannot be ruled out. However, due to the relationship between vinclozolin's anti-androgenic properties and its carcinogenic effects, the Agency believes protecting against the anti-androgenic effects would also be protective against potential carcinogenic effects to all population subgroups (including infants and children).

Accordingly, the cPAD will be protective against potential carcinogenic effects as well as the developmental/reproductive effects. The cPAD already incorporates the full, additional 10x safety factor for the protection of infants and children (i.e., it is derived from the NOAEL of 1.2 mg/kg/day with an MOE of 1,000 – 10x for intraspecies extrapolation; 10x for interspecies variation; and 10x for FQPA). Since this approach (using the cPAD) would be more protective than the proposed POD for cancer risk assessment of 3 mg/kg/day, and includes an additional 10x factor for the protection of infants and children, a separate non-linear risk assessment for cancer is not necessary.

Exposure estimates expressed as a percentage of the anti-androgenic PAD (0.0012 mg/kg/day) were 4% for the general U.S. population and 7% for the most highly exposed population subgroup, children (1–6 years old). In addition, as a point of comparison, the MOE was calculated to be 75,000 for the general U.S. population and 38,000 for children (1–6 years old).

2. *From drinking water*. In general, available monitoring data are of limited use because metabolite concentration measurements were not performed. For both surface water and groundwater, the sum of vinclozolin and its principal metabolites, assumed to degrade completely to 3,5-dichloroaniline (hereafter referred to as 3,5-DCA), have been used to assess the cancer risk associated with 3,5-DCA whereas vinclozolin per se has been used for the vinclozolin risk assessments.

In the absence of reliable, available monitoring data, EPA uses models to calculate the estimated environmental concentrations (EECs) of pesticides in ground and surface water. However, EPA does not use these model estimates to quantify risk. Currently, EPA uses DWLOCs as a surrogate to capture risk associated with exposure to pesticides in drinking water. A DWLOC represents the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses (if any). A DWLOC will vary depending on the residue level in foods, the toxicity endpoint and the drinking water consumption patterns and body weights for specific population subgroups. The calculated DWLOC is compared to the model estimate (EEC), and if the model estimates are below the DWLOC, the risks are not considered to be of concern.

For estimating groundwater concentrations of vinclozolin and 3,5-DCA, EPA used the Screening

Concentration in Ground Water (SCI-GROW) model. The SCI-GROW model is based on scaled groundwater concentration from groundwater monitoring studies, and environmental fate properties (aerobic soil half-lives and organic carbon partitioning coefficients-Koc's). SCI-GROW provides a screening concentration which is an estimate of likely groundwater concentrations if the pesticide were used at the maximum allowed label rate in areas with groundwater vulnerable to contamination. In most cases, a majority of the pesticide use area will have groundwater that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimate. Using SCI-GROW, the acute and chronic ground water EEC of vinclozolin per se is 0.53 parts per billion (ppb), and the acute and chronic ground water EEC of 3,5-DCA is 2.65 ppb.

For estimating surface water concentrations of vinclozolin and 3,5-DCA, EPA used tier II models, Pesticide Root Zone Model (PRZM) 3.12 and Exposuer Analysis Modeling System (EXAMS) 2.975, which assumed decline of parent vinclozolin and formation and decline of metabolites in a sequential degradation pattern in both field and pond such that degradation proceeds completely to 3,5-DCA. Vinclozolin per se is a major residue near application, but eventually the metabolites are the principal residues in both surface and drinking water. The metabolites are the only residues that are likely to be found in the environment except fairly soon after application. The scenario used in the model (application to onions in California) is the worst-case scenario for water modeling. A tier II EEC for a particular crop or use is based on a single site that represents a high exposure scenario for the crop or use. Weather and agricultural practices are simulated at the site for 36 years to estimate the probability of exceeding a given concentration (maximum concentration or average concentration) in a single year. Maximum EECs are calculated so that there is a 10% probability that the maximum concentration in a given year will exceed the EEC at the site; peak and chronic EECs were calculated so that there is a 10% probability the maximum average concentration for a given duration (4–day, 21–day, etc.) will equal or exceed the EEC at the site. This can also be expressed as an expectation that water concentrations will exceed EECs once every 10 years. The acute (peak) surface water EEC for vinclozolin is 5.68 ppb and for 3,5-DCA is 26 ppb. The chronic (annual mean) surface water EEC for vinclozolin is 0.165 ppb and for 3,5-DCA is 3.12 ppb.

i. *Acute exposure and risk*. For the population subgroup of concern, females (13+), the DWLOCs for vinclozolin per se at the various percentiles of exposure are as follows: 0 ppb at the 99.9th percentile; 4 ppb at the 99.85th percentile; 30 ppb at the 99.8th percentile; 47 ppb at the 99.75th percentile; 80 ppb at the 99.6th percentile; and 92 at the 99.5th percentile. At all but the very highest percentiles of exposure (99.85th and above), the DWLOC for vinclozolin per se is higher than the EEC of 5.68 ppb in surface water and 0.53 ppb in ground water. As explained above, given the level of refinement in the vinclozolin exposure estimate, EPA believes using the highest percentiles of exposure in estimating risk would unreasonably overstate risk. Therefore, EPA is reasonably certain that exposure to vinclozolin per se in drinking water will result in no harm.

ii. *Chronic exposure and risk*. The following chronic DWLOCs were calculated for vinclozolin per se: general U.S. population, 41 ppb; females (13+) 35 ppb; and children (1–6 years old), 11 ppb. The lowest DWLOC of 11 ppb for children 1–6 years old is higher than the EEC of 0.165 ppb in surface water and 0.53 ppb in ground water. Therefore, EPA is reasonably certain that exposure to vinclozolin in drinking water will result in no harm.

3. *From non-dietary exposure*. There are no vinclozolin pesticide products registered for use by homeowners. Therefore, there is no potential for homeowner handler exposure to vinclozolin pesticide products. Vinclozolin can, however, be occupationally used in a manner that may lead to post-application exposures to the general population, in particular, golfers playing on treated golf courses and homeowners and their families coming into contact with or playing on sod which was previously treated on a sod farm. A chemical-specific turf exposure study was used to measure human exposure as well as residue dissipation over time.

All residential exposures are considered to be short-/intermediate-term duration (i.e., 1 day to 1 week and 1 week to several months, respectively), and the same endpoint applies to both durations of exposure. As the endpoints selected are from oral toxicity studies (NOAEL of 3 mg/kg/day for females (13+)) and NOAEL of 5 mg/kg/day for infants and children, route-to-route exposure was corrected by applying a 25% dermal absorption factor and a 100% default inhalation absorption factor was assumed. A 100% safety factor was used and a 10X FQPA safety factor was added raising the Agency's level of concern to 1,000.

Post-application risks to the general population were considered for golfers following treatment of greens, tees, and fairways. Adult golfer exposures, women (13+), were less than the Agency's level of concern even on the day of application (MOE = 1,700). Given the magnitude of the MOE for adult women golfers, the Agency does not believe that the risks to child golfers would exceed the Agency level of concern either because the skin surface area/body weight ratio of the typical child golfer is similar to that of adults (within 15%). Therefore, the MOE for a child golfer is only slightly less than the MOE for adult golfers.

The exposure scenario used for toddlers playing on treated sod was the worst case scenario. The exposure scenario assumed that toddlers were playing on sod which had been treated with vinclozolin on a sod farm that same day, cut and laid in a residential setting. The MOE for toddlers is 33. This MOE represents an upper-bound exposure which includes dermal and non-dietary ingestion pathways (dermal exposure and hand-to-mouth oral exposure to grass and dirt). EPA has calculated that foliar dislodgeable residues on the sod decline such that risks fall beneath the Agency's level of concern 26 days after application (MOE = 1,100). To mitigate the unacceptable risk resulting from exposure before the 26–day period has elapsed; the registrant has proposed deletion of use on sod farms; amended the label to add a 24–day pre-harvest interval; and initiated the immediate restickering of all product in the channels of trade to require a 24–day period before sod can be harvested. It is assumed that, at a minimum, sod harvesting and replanting in a residential setting would take an additional 2 days; thereby, providing a total of 26 days for residues of vinclozolin to decline to an acceptable level. Although the Agency's level of concern is exceeded, EPA believes that these risk reduction measures when taken into consideration with the extremely conservative exposure scenario and exposure assumptions will immediately reduce the exposure such that it is below the Agency's level of concern.

4. *Cumulative exposure to substances with a common mechanism of toxicity.* Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative

effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.''

Vinclozolin, procymidone, and iprodione are members of the imide group of the dicarboximide class of fungicides. Each of these three pesticides can metabolize to 3,5-DCA. FQPA requires EPA to estimate cumulative risk from consumption of food and water containing 3,5-DCA derived from vinclozolin, iprodione, and procymidone.

i. *Acute exposure and risk.* EPA has certain evidence that these compounds induce similar toxic effects but has not yet determined whether or not these compounds modulate androgens by a common mechanism of toxicity. In fact, there is evidence that iprodione does not share a common mechanism of toxicity as it disrupts the endocrine system by inhibiting androgen synthesis rather than competing for the androgen receptor as vinclozolin does. In addition, these three chemicals do not have any known metabolites/degradates in common with the possible exception of 3,5-DCA which is structurally and toxicologically different from the parent compounds and unlikely to be an androgen receptor antagonist.

EPA has, at this time, some data which suggests that vinclozolin and procymidone have a common mechanism of toxicity. An article published in *Toxicology & Industrial Health* (Vol. 15, ISS 1–2, 1999, page 80–93) which reports the findings by Dr. Earl Gray, National Health and Environmental Effects Research Laboratory, U.S. EPA, Research Triangle Park, NC, suggests that procymidone alters sexual differentiation in the male rat by acting as an androgen-receptor antagonist *in vivo* and *in vitro.* The Agency has yet to make a conclusion as to whether these data are sufficient to evaluate whether vinclozolin and procymidone have a common mechanism of toxicity. Within the next year, the Agency expects to reach a conclusion as to whether these data are sufficient to determine that vinclozolin and procymidone have a common mechanism of toxicity.

Even if it is assumed that vinclozolin and procymidone share a common mechanism of toxicity, EPA believes that it can still make the finding of reasonable certainty of no harm for vinclozolin because any cumulative risk resulting from adding procymidone residues in wine to vinclozolin exposure is unlikely to differ significantly from the risk of vinclozolin alone. This conclusion is based on a number of factors. The exposure assessment for vinclozolin estimates that vinclozolin exposure through wine grapes contributes < 2% of the total vinclozolin exposure. The percent of imported wine grapes that are treated with procymidone is similar to that of vinclozolin (estimated 10% of wine grapes treated with vinclozolin and 9.4% of wine grapes treated with procymidone), and therefore, the exposure pattern for these chemicals is similar. In addition, the exposure estimates conservatively assume that all wine bearing vinclozolin residues also contain procymidone residues. In all likelihood, wine grapes would be treated with either vinclozolin or procymidone but not both chemicals. Therefore, EPA believes that vinclozolin exposure and procymidone exposure through wine grapes would each add < 2% to the ''cumulative exposure''. As noted above, the acute food-only risk of vinclozolin is 83% of the aPAD at the 99.8th percentile of exposure, and the acute ground water EEC of 0.53 ppb and the acute surface water EEC of 5.68 ppb are lower than the drinking water DWLOC which is 30 ppb at the 99.8th percentile of exposure. EPA believes there is ultimately enough room in the risk cup to accommodate vinclozolin and procymidone risk, even, if in the future, EPA does determine that procymidone and vinclozolin share a common mechanism of toxicity.

ii. *Carcinogenic exposure and risk.* Since 3,5-DCA is not a registered pesticide, there is no FIFRA toxicology data base for this compound. EPA has used the Q_1* for *p*-chloroaniline (PCA) to assess the carcinogenicity (only toxicological endpoint identified for 3,5-DCA) for other structurally related chloroanilines. EPA's approach on chloroanilines is to consider chloroaniline metabolites to be toxicologically equivalent to PCA unless there is sufficient evidence that the metabolite is not carcinogenic. A Q_1* of 6.38×10^{-2} (mg/kg/day)$^{-1}$ has been calculated for *p*-chloroaniline based on the spleen sarcoma rate in male rats from a National Toxicology Program bioassay.

Exposure to 3,5-DCA was evaluated from the following sources: residues of vinclozolin- and iprodione-derived 3,5-DCA in food and wine, residues of procymidone-derived 3,5-DCA in imported wine, and 3,5-DCA residues in water from domestic agricultural uses of iprodione and vinclozolin. There are no U.S. registrations for procymidone. Therefore, an evaluation of exposure to procymidone-derived 3,5-DCA in water is not appropriate.

a. *Food risk*— (1) *From vinclozolin-derived 3,5-DCA residues.* Cancer risks were 5.1×10^{-7} for all crops, including strawberries and stone fruits. Cancer risks were 2.6×10^{-7} for all crops, excluding strawberries and stone fruits. Neither of these risks exceed the Agency's level of concern.

(2) From iprodione-derived 3,5-DCA residues. As stated in the July 1998 Iprodione RED, the cancer risk associated with 3,5-DCA derived from iprodione was 6×10^{-9}. This risk does not exceed the Agency's level of concern.

(3) *From procymidone-derived 3,5-DCA residues.* The cancer risk associated with 3,5-DCA in imported wine produced from grapes treated with procymidone was estimated to be 3.7×10^{-7}. This risk does not exceed the Agency's level of concern

b. *Drinking water risk*—(1) *From vinclozolin derived 3,5- DCA.* As stated previously, Tier II surface water EECs were generated using PRZM/EXAMS for 3,5-DCA. Onions grown in California were considered to be the worst-case scenario for water modeling. The highest chronic EEC is 3.12 ppb in surface water while the carcinogenic DWLOC for 3,5-DCA has been calculated to range from 0.47 ppb to 1.6 ppb. Therefore, the EEC exceeds the DWLOC indicating a potential for concern. The onion scenario was selected because this use site represents the highest maximum seasonal rate currently allowed on vinclozolin labels. However, the registrant has requested deletion of onions after this growing season (July 15, 2000). If the Agency accepts this request, this scenario is not appropriate for use in a carcinogenic risk assessment which represents life-time exposure.

Assuming acceptance of BASF's use deletion request, the carcinogenic DWLOC for 3,5-DCA (based on the commodities available for consumption after this use season) has been calculated to range from 0.46 ppb to 1.6 ppb. Using Tier II PRZM/EXAMS, the modeled EECs are 0.64 ppb for lettuce and 0.34 ppb for canola. The use site which represents the highest modeled exposure in drinking water is golf courses. Application to golf course turf is currently permitted on grass mowed at 1 inch or less. Using the Tier I generic expected environmental concentration (GENEEC) model, the Agency has calculated a chronic EEC of 0.29 ppb based on application to tees and greens and a chronic EEC of 2.33 ppb assuming application to tees, greens, and fairways. These EECs were the result of refinements to the GENEEC model. These refinements included the incorporation of an 87 percent crop area (PCA) factor as well as the percentage of the golf course that actually receives

pesticide treatment, bringing the resulting PCA factor down to 17%. It was assumed that tees and greens comprise 2.8% of the acreage of a golf course. When fairways are included, an additional 16.7% of the golf course is treated. The EEC of 2.33 ppb exceeds the DWLOC. In evaluating whether this EEC indicated a risk of concern EPA considered the following factors:

(i) The drinking water assessment on turf is based on GENEEC, a screening-level Tier I model. At present, PRZM-EXAMS, the Tier II model, does not have the appropriate parameters to accurately model turf runoff. Although GENEEC is not an ideal tool for use in drinking water risk assessments, it can provide high-end estimates of the concentrations that might be found in a confined pond of one hectare. Drinking water from surface water sources does not typically come from this type of scenario, but rather from bodies of water that are substantially larger than such ponds and from diverse watersheds. Unlike a confined pond, there is always some flow (in a river) or turn over (in a lake or reservoir) resulting in an over-estimation of the persistence of the chemicals near the drinking water utility intakes. Although a PCA of 17% was used to refine the model, the Agency recognizes that there are still uncertainties in the accuracy of the model to represent drinking water concentrations.

(ii) The GENEEC model uses the 56–day average of pesticide concentrations immediately after an event (application of pesticide). This short time-period may not adequately characterize a person's average daily exposure over a year, even more so, over a life time of 70 years.

(iii) The GENEEC model assumes that once in every 10 years the EEC will be exceeded. For the other 9 out of 10 years the level of residue in drinking water is likely to be below the EEC with at least one half of the years falling significantly below by a factor of 5 to 10. Therefore, a person may be exposed to the EEC once in every 10 years or a total of seven times during a lifetime of 70 years. The Agency believes the potential for such a lifetime exposure is minimal.

The first of these factors raises some concern because there is a possibility that GENEEC may underpredict residue levels although such underprediction would not be expected to be great. The second and third factors, on the other hand, could lead to a significant overstatement of drinking water exposure values. In light of all of these factors, EPA believes that it is likely there is no risk of concern from exposure to vinclozolin-derived 3,5-DCA.

Nonetheless, the exceedance of the DWLOC, based on a screening level model, does indicate a need to take steps to insure that exposures do not raise a risk of concern. Therefore, the Agency is considering requiring the registrants of vinclozolin and iprodione to submit targeted surface water monitoring studies. The studies would be used to compare the existing modeled results to the more accurate data. The Agency will also consider requiring BASF to develop a survey of golf course superintendents to obtain current information on actual vinclozolin use patterns. Estimates for turf use will be examined further pending receipt of better usage characterization data. Lastly, the Agency is considering requiring additional toxicological information on 3,5-DCA from the registrants of vinclozolin, iprodione and procymidone, including data for use in evaluating the carcinogenic potential of this metabolite.

(2) *Iprodione 3,5-DCA.* As stated in the RED, the DWLOC for 3,5-DCA derived from domestic uses of iprodione was estimated to be 0.55 ppb. The 3,5-DCA EEC in surface water associated with the use of iprodione alone was estimated to be 0.45 ppb. Thus, the iprodione derived 3,5-DCA carcinogenic DWLOC is not exceeded.

(3) *From procymidone 3,5-DCA.* There is no drinking water exposure because procymidone is not registered for use in the United States.

c. *Cumulative risk.* The cumulative, food-only cancer risk associated with 3,5-DCA derived from all three of these imide fungicides is 9.2×10^{-7} (includes stone fruit and strawberries) and the cumulative food-only cancer risk is 6.3×10^{-7} when stone fruit and strawberries are excluded. There is uncertainty in the above risk estimates in that a surrogate Q_1* is being used for 3,5-DCA. However, due to the structural similarities of 3,5-DCA and *p*-chloroaniline (PCA), EPA believes that for 3,5-DCA, the use of the PCA Q_1* represents an upper-bound estimate. The Agency is considering requiring registrants of vinclozolin, iprodione, and procymidone to provide additional toxicological information on 3,5-DCA including data for use in evaluating the carcinogenic potential. The cumulative, food-only cancer risk estimates are conservative and are considered to be a negligible cancer risk.

The 3,5-DCA DWLOC from all three imide fungicides (including canola, snap beans and those currently registered vinclozolin uses which are not being supported after this use season) ranges from 0.26 ppb to 1.4 ppb. The estimated concentration of 3,5-DCA in water from applications of iprodione (1998 iprodione RED) is 0.45 ppb and falls within the range of the aggregated DWLOC cited above. The estimated concentration of 3,5-DCA in water from applications of vinclozolin after this use season is estimated to range from 0.29 ppb to 2.33 ppb. As already stated, this range could potentially present a risk of concern based on the model, however, based on how the model estimates residue concentrations for cancer assessment, EPA believes that it is unlikely that a cancer risk of concern is present.

D. Aggregate Risks and Determination of Safety for U.S. Population

1. *Acute risk.* The acute dietary (food only) risk does not exceed the Agency's level of concern at the percentiles of exposure up to the 99.8th percentile. Using anticipated residues, PCT data, and PICT data, the population subgroup of concern, females (13+) utilized 83% of the dietary (food only) aPAD at the 99.8th percentile of exposure. For drinking water, the EEC of 5.68 ppb in surface water and the EEC of 0.53 in groundwater did not exceed the DWLOC of 30 ppb at the 99.8th percentile of exposure.

2. *Chronic risk.* Using the exposure assumptions described above, EPA believes that aggregate dietary exposure to the U.S. population will use 4% of the cPAD and exposure to the most highly exposed population subgroup, children (1–6 year old) will use 7% of the cPAD. The chronic DWLOCs for vinclozolin were 41 ppb for the general U.S. population and 35 ppb for the most highly exposed population subgroup, women (13+). The chronic DWLOCs were higher than the chronic EEC of 0.53 ppb in ground water and 0.165 ppb in surface water. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

3. *Short- and intermediate-term risk.* Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. All residential exposures are considered to be short- and intermediate-term duration and since the same endpoint applies to both durations of exposures, the dermal and inhalation exposures must be aggregated together with the food and water exposures for each population subgroup

of concern, females (13+) and infants and children. The risks currently exceed the Agency's level of concern. However, when considering the conservative method of exposure estimations previously discussed, and the following risk mitigation measures (stone fruit and strawberry use deletion, and the immediate restickering of all vinclozolin products for sod farm use to include a 24–day period before harvesting), the MOE is ≥1,010 for aggregate risks to the population subgroups of concern, females (13+) and infants and children as well as the general U.S. population resulting from vinclozolin uses are not of concern. Therefore, the risks do not exceed the Agency's level of concern.

4. *Aggregate cancer risk for U.S. population*. Because the overall anti-androgenic effects are a prerequisite for hyperplasia and tumor formation, and are considered to be protective of the potential carcinogenic outcome of exposure to the anti-androgenic vinclozolin and its metabolites, the overall anti-androgenic aggregate risk which are identical to the chronic aggregate risk. The chronic aggregate risks are presented. The chronic (non-cancer) aggregate risk was below the Agency's level of concern for food and drinking water sources of exposure. Chronic food-source risks were ≤7% of the cPAD when stone fruit and strawberries are excluded (uses have been canceled). Estimated environmental concentrations were compared to the chronic DWLOCs. The chronic EEC for residues of vinclozolin per se in ground water (0.53 ppb) was below the chronic DWLOCs for water consumption by adults (41 ppb for the general U.S. population and 35 ppb for females (13+)) and by children (11 ppb).

Cancer risks from vinclozolin derived 3,5-DCA were 2.6×10^{-7} for all crops, excluding strawberries and stone fruits. This risk does not exceed the Agency's level of concern. The 3,5-DCA DWLOC from all three Imide fungicides (including canola, snap beans and those currently registered vinclozolin uses which are not being supported after this use season) ranges from 0.26 ppb to 1.4 ppb. The 3,5-DCA EEC resulting from iprodione use is 0.45 ppb and falls with the range of the aggregated DWLOC cited above. The 3,5-DCA EEC resulting from vinclozolin use after this use season is estimated to range from 0.29 ppb to 2.33 ppb. As already stated, this range could potentially present a risk of concern based on the model, however, based on how the model estimates residue concentrations for cancer assessment, EPA believes that it is unlikely that a cancer risk of concern is present.

5. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to vinclozolin residues.

E. Aggregate Risks and Determination of Safety for Infants and Children

1. *Safety factor for infants and children*—i. *In general*. In assessing the potential for additional sensitivity of infants and children to residues of vinclozolin, EPA considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children.

ii. *Prenatal and postnatal sensitivity*. The rationale for retaining the 10X FQPA safety factor is explained below:

a. There is evidence of increased susceptibility of offspring following *in utero* exposure to vinclozolin in the prenatal developmental toxicity study in rats.

b. A developmental neurotoxicity study in rats with an expanded protocol is required for vinclozolin as a result of concern for the anti-androgenic properties of vinclozolin and its metabolites.

iii. *Conclusion*. Based on the developmental and reproductive data for vinclozolin, EPA determined that an additional 10X safety factor for the protection of infants and children (as required by FQPA) should be retained.

2. *Acute risk*. No study with vinclozolin indicated that acute exposure to vinclozolin is likely to cause an adverse effect of concern on infants or children or the general public with the exception of the *in utero* effects on the developing fetus. Risks to the fetus are estimated by examining exposure to women of child-bearing age.

3. *Chronic risk*. Using the exposure assumptions described in this unit, EPA has concluded that aggregate exposure to vinclozolin from food will utilize 7% of the cPAD for infants and children. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Since the EEC's for residues of vinclozolin per se are lower than the chronic DWLOC's, EPA does not expect the aggregate exposure to exceed 100% of the cPAD.

4. *Short- or intermediate-term risk*. The short- and intermediate-term risks currently exceed the Agency's level of concern (MOE = 1,000). However, the Agency believes the exposure estimates are conservative, as previously discussed, and therefore, overestimate risk. When the following risk mitigation measures (stone fruit and strawberry use deletion, and the immediate restickering of all vinclozolin products for sod farm use to include a 24–day period before harvesting) are taken into consideration, the MOE is ≥ 1,010 for aggregate risks to infants and children resulting from use of vinclozolin. Therefore, the risks do not exceed the Agency's level of concern.

5. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to vinclozolin residues.

IV. Response to Public Comments

A. Natural Resources Defense Council Comments

1. *Comment number 1*. NRDC argues that EPA is not authorized to use percent crop treated information in acute risk assessments. NRDC bases this argument on the fact that the provision explicitly addressing percent crop treated information, section 408(b)(2)(F), only mentions use of such information in chronic assessments.

Agency response. EPA disagrees with this interpretation of the FFDCA. FFDCA Section 408(b)(2)(D)(vi) directs EPA, in making tolerance decisions, to consider ''available information concerning the aggregate exposure levels of consumers to the pesticide chemical residue.'' 21 U.S.C. 346a(b)(2)(D)(vi). This is a broad mandate that includes all manner of information bearing on exposure, not the least of which would be percent crop treated information. Thus, EPA believes that subsection (b)(2)(D)(vi) authorizes use of percent crop treated information in both acute and chronic risk assessments.

Congress explicitly addressed use of percent crop treated information in section 408(b)(2)(F) where it imposed certain conditions on EPA's use of

percent crop treated information in chronic risk assessments. Section 408(b)(2)(F) states:

> In establishing, modifying, leaving in effect, or revoking a tolerance for a pesticide chemical residue, the Administrator may, when assessing chronic dietary risk, consider available data and information on the percent of food actually treated with the pesticide chemical (including aggregate pesticide use data collected by the Department of Agriculture) only if the Administrator—
> (i) finds that the data are reliable and provide a valid basis to show what percentage of the food derived from such crop is likely to contain such pesticide chemical residue;
> (ii) finds that the exposure estimate does not understate exposure for any significant subpopulation group;
> (iii) finds that, if data are available on pesticide use and consumption of food in a particular area, the population in such area is not dietarily exposed to residues above those estimated by the Administrator; and
> (iv) provides for the periodic reevaluation of the estimate of anticipated dietary exposure.

21 U.S.C. 346a(b)(2)(F) (emphasis added). Although this paragraph affirms the ability of EPA to use percent crop treated information for chronic dietary risk assessments, the clear thrust of this paragraph is to impose four limitations on the use of such information in chronic risk assessments (i.e. the limitations set forth in clauses (i)—(iv) following the ''only if''). Because the limitations expressly apply only ''when assessing chronic dietary risk'', Congress did not impose any limitation on the authority in subsection (b)(2)(D)(vi) to consider percent crop treated for risk assessments that consider risks other than chronic ones (i.e. acute risks).

NRDC contends that subparagraph (F) impliedly bars EPA from relying on percent crop treated information for acute risk assessments under subparagraph (D)(vi) because subparagraph (F) only mentions chronic risk assessments. EPA, however, does not believe that the statutory silence on acute risk assessments in subparagraph (F) compels such an interpretation. In fact, the statutory structure suggests the converse conclusion. Subparagraph (F) clearly sets forth that percent crop treated information may be used in chronic risk assessments ''only if'' four conditions can be met. If Congress had intended that this provision limit EPA's general authority to consider percent crop treated information other than as applied to chronic risk assessments, the reference to chronic risk assessments should not have been included as part of the introductory clause but as one of the ''only if'' conditions. Failure to include it as one of the ''only if'' conditions suggests that Congress was merely setting out rules for chronic risk assessments and not making a broader statement about use of percent crop treated information generally.

Moreover, it is not surprising that Congress chose only to address use of percent crop treated information in the context of chronic risk assessment given EPA's historical practice regarding use of percent crop treated data. Although EPA has considered percent crop treated information in chronic risk assessment for decades, use of such information in acute risk assessments is a relatively recent phenomenon, and Congress, in 1996, may have either not been aware of the rapidly evolving risk assessment techniques for acute hazards or believed that it was premature to enact statutory requirements as to such assessments.

There were two key events in 1995 that triggered the use of percent crop treated information in acute risk assessments: (1) A new focus on acute hazards; and (2) a new risk methodology for assessing acute risks. In 1995, EPA began for the first time consistently identifying acute endpoints and performing acute risk assessments for each pesticide. EPA was initially reluctant to use percent crop treated information in such assessments due to the difference between acute and chronic risks. With chronic risk, EPA is concerned with hazards that occur from exposure over an extended time period. Thus, in assessing chronic risk, EPA generally combines percent crop treated information with data on residue levels to produce an estimate of the residue level a person is exposed to over an extended time-frame assuming the person gets a mixture of treated and untreated commodities. With acute hazards, EPA is concerned with the risk from a single exposure and thus is interested in the exposure that can come from a single commodity. Accordingly, for acute risk assessments it is inappropriate to produce a single estimate of the residue level in commodities if such estimate does not reflect high end values that are likely to occur. Use of percent crop treated data in the manner used in chronic assessment, however, reduces high-end values in proportion to percent of crop treated. To overcome this problem, a new risk assessment methodology was developed that used a complex, probabilistic model that incorporated all residue values, including the high end values, and percent crop treated information. EPA first accepted these probabilistic acute risk assessments late in 1995, and use of this new risk assessment technique in regulatory actions was still relatively infrequent prior to the drafting and passage of the FQPA in the summer of 1996.

In sum, NRDC can demonstrate, at best, that the statute is silent regarding use of percent crop treated information in acute risk assessments. Given the general language in section 408 directing EPA to consider ''available information'' on aggregate exposure levels, EPA's interpretation of section 408 as permitting use of percent crop treated data in acute risk assessments is certainly reasonable. NRDC's interpretation, on the other hand, would erect an absolute bar to the use of the most advanced scientific techniques for reliably and accurately estimating anticipated exposure to pesticide residues.

2.*Comment number 2*. EPA fails to identify the correct NOAEL for vinclozolin's endocrine disrupting effects. Dr. Gray has reported an apparent lack of a NOAEL for vinclozolin's developmental effects. Therefore, use of a NOAEL of 6 mg/kg/day for the acute analysis and use of 3 mg/kg/day as the NOAEL for short-term, intermediate-term, and carcinogenic risk assessments is not scientifically supportable. NRDC feels that a LOAEL of 3 mg/kg/day, adjusted for the lack of a true NOAEL, should be used as the hazard component in risk assessments.

Agency response. First, the Agency stresses that it conducted a statistical analysis of anogenital distance in response to dose in the Gray developmental rat study, and it was determined that the NOAEL for acute effects was 6 mg/kg/day and the LOAEL was 12 mg/kg/day. In a 12/8/99 memorandum, the Agency determined that decreased ventral prostate weight, observed at 6 mg/kg/day, was an even more sensitive indicator of the anti-androgenic activity of vinclozolin; the next lower dose (3 mg/kg/day) was thus selected as the study NOAEL.

Second, the Agency must stress that the NOAEL of 6 mg/kg/day for the acute dietary analysis represents the 3 mg/kg/day treatment level (study NOAEL) in the [multidose] perinatal oral developmental rat study noted above that has been adjusted by a plasma equilibrium factor to derive an adjusted NOAEL that reflects a single dose; the adjusted LOAEL causing decreased ventral prostate weight has been calculated to be 11.5 mg/kg/day.

The perinatal oral developmental rat study mentioned above was also used as the source of the NOAEL for short-term and intermediate-term dermal and inhalation risk assessments for women (13–50); note that the study NOAEL of 3 mg/kg/day was not adjusted for the

plasma equilibrium factor because the applicable short-term and intermediate-term routes of exposure are not oral and because they typically reflect multiple exposure events more closely approximated by the multidose oral developmental rat study.

The Agency disagrees with NRDC's suggestion that the 3 mg/kg dose from the Gray, et al. oral developmental rat study is a LOAEL. As noted above, EPA's statistical analysis shows that the anogenital distance effect has a NOAEL of 6 mg/kg/day in the Gray study. NRDC has not offered any explanation of why it does not agree with that statistical analysis. Second, as to the decreased ventral prostate weight effect, EPA's review of the data shows that this adverse affect was not present at 3 mg/kg; however, this adverse effect was a dose-related effect in male offspring at 6 mg/kg and above. No adverse effects were observed at the 3 mg/kg/day dose level. Thus, EPA cannot agree with NRDC that the 3 mg/kg/day dose should be treated as a LOAEL in conducting the risk assessment for vinclozolin.

The perinatal rat developmental toxicity study was also used to derive the point of departure (POD = NOAEL of 3 mg/kg/day) to be used in the non-linear carcinogenicity risk assessments; the effect seen at the LOAEL of 6 mg/kg/day was prostate weight decrease, seen as an early manifestation of the anti-androgenic action of vinclozolin ultimately resulting in Leydig (testicular interstitial) cell tumors in the chronic/cancer studies. However, note that the NOAEL of 1.2 mg/kg/day from the rat chronic/cancer studies is considered to be protective of cancer effects because it is protective of the anti-androgenic effects that are the likely precursors to tumor formation. The chronic Population Adjusted Dose (cPAD), used to calculate risk, is derived by dividing the NOAEL of 1.2 mg/kg/day by the safety factor of 1,000 (10X for intraspecies extrapolation, 10X for interspecies variation, and 10X for FQPA). Because this approach (using the cPAD) would be more protective than the proposed POD for cancer risk assessment of 3 mg/kg/day, and includes an additional 10X factor for the protection of infants and children, a separate non-linear risk assessment for cancer is not necessary.

3. *Comment number 3*. Vinclozolin and iprodione do share a common mechanism of toxicity. NRDC disagrees with EPA's judgement that vinclozolin and iprodione do not share a common mechanism because they are both known anti-androgens, both have the metabolite 3,5-dichloroaniline in common, and both cause the same effect even if the exact manner of androgen interference is different. In fact, they may act additively or synergistically as a result of affecting the androgen pathway at different sites as opposed to the potential competition for the same binding site if both act at the exact same point in the process.

Agency response. FQPA requires EPA to consider available information concerning the cumulative effects of compounds that have a common mechanism of toxicity. It should be stressed, however, that EPA is moving in a stepwise fashion to evaluating the cumulative assessment of anti-androgenic pesticides.

Vinclozolin, procymidone, and iprodione are members of the imide group of the dicarboximide class of fungicides. There is some evidence that these compounds induce similar toxic effects. Further, all of these fungicides appear to be anti-androgenic. The mechanistic basis for their anti-androgenic properties have been studied to different degrees. There are studies underway at EPA's National Health and Environmental Effects Laboratory to better elucidate the mechanism of toxicity for these anti-androgenic fungicides as well as mixture studies on how they interact. Although all three of these fungicides effectively reduce the level of testosterone, they do so by different pathways. Vinclozolin and procymidone bind and compete for the androgen receptor. Iprodione disrupts the endocrine system by inhibiting androgen synthesis rather than competing for the androgen receptor. It should be noted that these three chemicals do not have any known metabolites/degradates in common with the possible exception of 3,5-dichloroaniline which is structurally and toxicologically different from the parent compounds and unlikely to be anti-androgenic.

The androgen system may be modulated in different ways including competitive binding to androgen receptors, interference with gene control over the synthesis of several enzymes or other factors associated with synthesis of androgen and testosterone. All of these variables relate to the potency, specificity, and site of action of the anti-androgen and determine the expression of the anti-androgenicity induced by various compounds. Because of the complexity of the androgen system, a careful evaluation of all the available data is needed as well as peer review by the FIFRA Science Advisory Panel before a formal decision is made regarding whether or not these compounds modulate androgens by a common mechanism of toxicity. The evaluation of a common mechanism would follow the 1999 EPA Guidance for Identifying Pesticide Chemicals and Other Substances That Have A Common Mechanism of Toxicity (64 FR 5796, February 5, 1999) (FRL–6060–7). Furthermore, procymidone has yet to be subjected to the Reregistration Eligibility Decision (RED) process and, as part of this process, its toxicology database must meet current standards of acceptability. Although there are data suggesting that these dicarboximide fungicides induce some of the same anti-androgenic effects, the mechanism by which they cause these toxic effects have not been adequately evaluated.

Even after an evaluation of all the data and a decision is made regarding a common mechanism of toxicity, other analyses are important to conduct regarding the integration of exposure and hazard data to determine the likelihood that such groupings might result in a cumulative risk as described in the Agency's Proposed Guidance on Cumulative Risk Assessment of Pesticide Chemicals That Have a Common Mechanism of Toxicity (http://www.epa.gov/scipoly/sap/1999/september/cumdoc.pdf). Only then can it be determined whether there is a need to conduct a cumulative risk assessment on these dicarboximide fungicides.

Nonetheless, because of the apparent similarity of mechanism of toxicity between vinclozolin and procymidone EPA has considered, as discussed above, whether the cumulative effects from vinclozolin and procymidone (assuming these pesticides' effects are cumulative) would raise a risk of concern. EPA is unwilling, at this time, to make the same assumption concerning iprodione and vinclozolin. NRDC hypothesizes that, because iprodione and vinclozolin operate in a different manner on the androgen system, they are likely to have an additive anti-androgenic effect. A conclusion that chemicals that operate at different stages in the androgen pathway are acting through a common mechanism of toxicity or otherwise merit a cumulative assessment is beyond any cumulative effects determination EPA has made either pre- or post-FQPA. That does not mean that further evaluation of the science of cumulative effects concerning anti-androgenic effects will not lead to a conclusion that iprodione and vinclozolin have a common mechanism of toxicity. At this time, however, given the scientific understanding of the mechanisms of these two pesticides, EPA is unwilling to presume that such common mechanism exists or that there is some other justification for treating

these pesticides as having cumulative effects.

4. *Comment number 4*. EPA should not approve tolerances that exceed safe levels. The elevated risk numbers in the case of short-term and intermediate-term risk and the exceedance of the drinking water level of concern (DWLOC) are especially of concern and there is doubt that the proposed mitigation measures will alleviate the estimated risks. There is concern that EPA's assessments are not sufficiently conservative to protect public health and the Agency should not ignore or explain-away its own elevated risk estimates.

Agency response. EPA has high confidence in the short-term and intermediate-term risk assessments (these involve treated sod) because a chemical-specific turf exposure study was used and because foliar residue dissipation over time was determined. We, therefore, have confidence that the mitigation measure to require a 24-day interval between final treatment and harvest of sod before it is shipped for placement in a residential setting will be protective. Only in the case of acute aggregate risk from vinclozolin and carcinogenic risk from 3,5-DCA was there an indication of a potential drinking water concern. The exposure estimates (EECs) were based on conservative modeling. Also, the food exposures (subtracted from the aPAD to determine the DWLOC) are very conservative because they are based on field trial residue data. DWLOCs cannot be used in a quantitative risk assessment as representative monitoring data may. Rather, they are used to determine the magnitude of potential concern by comparison to the EEC's. As the 99.9th percentile of food exposure to vinclozolin is considered to be overly conservative given the use in this exposure assessment and the overly conservative drinking water assessment, EPA has little concern for an apparent elevated risk particularly in light of the registrant's mitigation proposals. Finally, discussion of the strengths and weaknesses of our assessments, the assumptions made, and our level of confidence are all part of the risk characterization component of risk assessment. We must provide qualitative descriptors to facilitate the risk management process.

B. Earthjustice Legal Defense Fund comment

Comment. EPA is asked to consider Earthjustice's prior comments and objections to the previous vinclozolin tolerance.

Agency response. EPA has addressed Earthjustice's prior comments and objections in the Agency letter of May 11, 2000 to the Earthjustice Legal Defense Fund, and therefore, the Agency believes that further detailed discussion is not necessary. In brief, Earthjustice's prior comments focused on two issues: the use of the additional safety factor for the protection of infants and children; and the cumulative effects of vinclozolin, iprodione, and procymidone. In considering Earthjustice's comments in the course of assessing vinclozolin, EPA has acceded to Earthjustice's request to retain the additional safety factor for the protection of infants and children and has assumed, for this tolerance rulemaking, that vinclozolin and procymidone have a common mechanism of action that will lead to cumulative effects. EPA decided against reaching that conclusion as to vinclozolin and iprodione for the reasons explained above. EPA's full response to Earthjustice has been included in the docket for this action.

C. BASF Corporation Comments

1. *Comment number 1*. BASF has supplied information which would allow the Agency to further refine the acute dietary risk by using monitoring data provided in response to the Agency's preliminary risk assessment. Use of this information would significantly reduce the calculated acute dietary risk.

Agency response. BASF did submit grape and lettuce metabolism studies and a proposal that monitoring data be used as a source of refined dietary exposure estimates, i.e., anticipated residues. FDA and USDA/Pesticide Data Program monitoring data are available for most foods expected to bear vinclozolin residues. However, these monitoring data are not useful for risk assessment purposes because these programs do not analyze all 3,5-DCA containing metabolites, which are the residues of concern. Agency review of the plant metabolism studies reveals that a significant portion of the vinclozolin residue may exist as 3,5-DCA per se or conjugates, all of which tend to increase with time as they are the terminal, more stable residues. Conjugates and 3,5-DCA per se are not analyzed by either FDA or PDP. These residues are, however, analyzed by the data collection method used to generate the field trial data because the method converts all of these residues to a common moiety (derivatized 3,5-DCA). Also, there was significant variability in the ratios of vinclozolin per se to total residues with time, between crops, and between studies on the same crop. Therefore, at this time the Agency does not believe that the plant metabolism studies provide sufficient additional information supporting use of monitoring data to generate anticipated residues (ARs) and that field trial data should be used to calculate AR values for dietary exposure in food.

2. *Comment number 2*. BASF has submitted or cited information it feels supports their contention that 3,5-DCA should not be assumed to be toxicologically equivalent to *p*-chloroaniline, i.e., that 3,5-DCA should not be considered to be a carcinogen like *p*-chloroaniline for risk assessment purposes. Based on physicochemical and stereochemical differences from *p*-chloroaniline, BASF thinks that 3,5-DCA would not be mutagenic. Calculations indicate that the amino group of *p*-chloroaniline is 1,300 times more reactive than the amino group of 3,5-DCA in a peroxidation reaction, a step necessary to generate the corresponding hydroxylamine which is a prerequisite for mutagenicity. Side-by-side Ames Bioassays demonstrated that *p*-chloroaniline is clearly mutagenic whereas 3,5-DCA is nonmutagenic in the presence of metabolic activation and a cocarcinogen. This indicates that the two chloroanilines behave different biologically.

Agency response. While the submitted information provides some support for the claim that 3,5-DCA may be less potent than *p*-chloroaniline, there is insufficient evidence to show that 3,5-DCA is not mutagenic or carcinogenic. The available mutagenicity data are insufficient because 3,5-DCA was tested using only one of the four or five *Salmonella typhimurium* strains usually tested in the Ames bioassay; also, 3,5-DCA was not the subject of any other *in vitro* mutagenicity study required for pesticide registration.

Only long-term studies in which two mammalian species are exposed to a potential carcinogen can provide concrete evidence of carcinogenicity. Therefore, until sufficient data are submitted, DCA will continue to be regulated based on a Q_1* calculation for *p*-chloroaniline.

3. *Comment number 3*. BASF claims that recently submitted details of calculations of turf foliar dislodgeable residues provides evidence that a 9-day preharvest interval (PHI), rather than the Agency-calculated 24-day PHI, is sufficient to bring the children's MOE to a level below the Agency's level of concern. Regardless of the outcome of the Agency review, BASF is willing to impose the 24-day PHI suggested in the supplemental notice.

Agency response. These data are currently under review, and no comment can be provided at this time.

V. Other Considerations

A. Metabolism in Plants and Animals

1. *Plant metabolism*. The qualitative nature of the residue in plants is adequately understood based on metabolism studies on strawberries, lettuce, peaches, and grapes. The plant metabolism studies indicate that metabolism in plants results from the hydrolytic cleavage of the oxazolidinedione ring and/or loss of the ethenyl moiety. Formation of conjugates and hydrolysis to 3,5-DCA occur and these may increase with time. The residues of concern are vinclozolin per se and its metabolites containing the 3,5-DCA moiety.

2. *Animal metabolism*. The qualitative nature of the residue in livestock is adequately understood based on adequate ruminant and poultry metabolism studies submitted in conjunction with pesticide petitions PP#7H5531 and PP#9F3750. The residues of concern are vinclozolin, a mixture of the diastereomers of *N*-(3,5-dichlorophenyl)-2-methyl-2,3,4-trihydroxybutyramide (BF 352–25), and a mixture of diastereomers derived by dihydroxylation of the vinclozolin vinyl group (BF 352–37). These metabolites are covered by the present tolerance expression, i.e., they contain the 3,5-DCA moiety.

B. Analytical Enforcement Methodology

1. *Plants*. Adequate analytical methodology is available for data collection and enforcing tolerances of vinclozolin per se and its metabolites containing the 3,5-DCA moiety in/on plant commodities. Method I in PAM, Vol. II, which underwent a successful EPA method validation on strawberries, involves base hydrolysis of residues to convert vinclozolin and its metabolites to 3,5-DCA. After steam distillation and organic solvent extraction, the isolated DCA is derivatized to *N*-(3,5-dichlorophenyl)chloroacetamide using chloroacetyl chloride prior to quantitation by gas chromatography/electron capture detection (GC/ECD). The limit of quantitation is 0.05 ppm.

2. *Livestock*. EPA has concluded that the following methods are available for the enforcement of tolerances for livestock tissues: method A9004A, a GC/ECD method, and method A9207, a High Performance Liquid Chromatography method. Method A9004A is based on conversion of vinclozolin and its metabolites to 3,5-DCA. However, it does not distinguish between residues of vinclozolin and other compounds convertible to 3,5-DCA. The LOQ is generally 0.05 ppm (0.1 ppm for poultry commodities). To confirm that the 3,5-DCA detected by method A9004A is derived from vinclozolin, method A9207 is used to measure 2,3,4-trihydroxy-*w*-methylbutanoic acid-(3,5-dichloroanilide) (BF 352–25), the major metabolite of vinclozolin in livestock commodities. The LOQ and the limit of detection are estimated to be 0.05 and 0.025 ppm, respectively. Both methods have been successfully validated.

3. The FDA PESTDATA database dated 1/94 (PAM, Vol. I, Appendix II) indicates that vinclozolin is completely recovered (> 80%) using FDA Multiresidue Protocols D and E (oily and non-oily matrices). Vinclozolin metabolite B is completely recovered using Protocols D and E (for oily matrices), and only partially recovered (50–80%) using Protocol E for non-oily matrices. Metabolite E is completely recovered using Protocol D. Metabolite F is recovered using Protocol D but no quantitative information is available. Metabolite S is partially recovered using Protocol E (non-oily matrices). The FDA multiresidue methodology differentiates between vinclozolin and iprodione, a pesticide that also contains the DCA moiety.

C. Magnitude of Residues

1. *Snap beans*. Sixteen (16) residue trials were conducted in a total of 7 states. Each trial consisted of a single residue sample. The residue trials were conducted using the Ronilin WP formulation. Eight of the trials involved application to lima beans and eight to snap beans. Ground applications were made in approximately 50 gallons of finish spray per acre and air applications in 5 to 15 gallons per acre. Samples of beans, cannery waste, green forage, and dry forage were analyzed. Residues in snap beans were as follows: 0.38, 0.53, 0.62, 0.64, 0.73, 0.76, 0.95, and 2.40 ppm.

2. *Canola*. Four field trials were conducted in Canada (two in Alberta and one each in Manitoba and Saskatchewan). These sites represent Regions 5, 7, and 14. A single treatment was applied at 0.22, 0.33, or 0.45 lb active ingredient per acre (ai/A) (0.44X, 0.66X, and 0.89X the maximum rate of 0.5 lb ai/A proposed on the U.S. label) in 40 gallons of water per acre using ground equipment. Two major canola varieties were treated at 20–35% bloom; the treatment-to-harvest intervals were 37–57 days. The canola seed were stored frozen for 330 days. The preponderance of data support the storage stability of the 3,5-DCA moiety for this length of time in canola seed. At the 0.44X application rate, canola seed contained 3,5-DCA-containing residues of 0.038–0.20 ppm. At the 0.33X rate, residues were detected at 0.065–0.28 ppm. At the 0.88X rate, residues were found at 0.068–0.42 ppm. An additional six field trials were conducted in Canada between 1982 and 1996 to support Section 18 requests. A single application was made at 0.22–0.67 lb ai/A (0.44X – 1.34X) during the early bloom to the mid-bloom stage using aerial and ground equipment. The treatment-to-harvest intervals were 36–69 days. Residues containing the 3,5-DCA moiety in canola seed were ≤ 0.93 ppm. The highest residue value resulted from an application of 0.44 lb ai/A (0.88X). Although some of the available trials do not reflect the maximum rate, others represent exaggerated rates. The earlier-submitted data, combined with the four Canadian field trials submitted with this petition, provide sufficient magnitude of the residue data upon which to base a canola seed tolerance.

A canola seed processing study was conducted on seed harvested from a Saskatchewan field trial. A single treatment at 0.45 lb ai/A (0.89X) occurred at 40% bloom. At maturity, 49 days later, seeds were subjected to typical processing into oil and meal. The seed, crude oil, refined oil, and meal byproduct were analyzed in Germany by BASF using method P–14.003.02. Residues containing the 3,5-DCA moiety were detected at 0.62 – 0.89 ppm in four replicates of seed (mean = 0.76 ppm). Residues in crude oil were 0.85 = 0.94 ppm (mean = 0.88 ppm) indicating very slight concentration in this intermediate component of the process that is not used for food or feed. Upon purifying, refined oil (the product for commerce) did not contain detectable residues (< 0.05 ppm) indicating residue reduction. In addition, the byproduct canola meal contained residue levels identical to those in the seed (0.68 – 0.89 ppm) demonstrating a lack of concentration of vinclozolin residues in this livestock feed.

3. *Meat, milk, poultry, and eggs*. There are no feed items associated with the currently registered use sites or succulent beans. However, canola meal may be fed to beef and dairy cows, swine, and poultry at up to 15% of the diet. The canola seed tolerance level of 1 ppm was used for canola meal to calculate livestock diets because the processing study indicated that vinclozolin concentrations in seed remains the same in the meal. The meal dry matter content of 88% (corrected for

cattle only) was also used to calculate livestock diets for tolerance-setting purposes. The dietary burdens are thus: 0.17 ppm for beef and dairy cattle and 0.15 ppm for swine and poultry.

Based on livestock feeding studies, the theoretical residues in tissues were calculated using tissue residues at the lowest feeding level (100 ppm) extrapolated to the dietary burdens provided above. Livestock commodity residues resulting from the three feeding levels (100, 300, and 1,000 ppm) were fairly linear lending some support to the assumed linearity down to the dietary burden levels. Theoretical residues ranged from 0.004 ppm to 0.015 ppm in cattle tissues and milk, 0.001 ppm to 0.004 ppm in poultry tissues and eggs, and 0.003 ppm to 0.014 ppm in swine tissues. In accordance with 40 CFR 180.6(a)(2), EPA believes that the available data indicate that there is a reasonable expectation of finite residues of vinclozolin transferring from treated canola to livestock commodities via canola meal in the diet. Accordingly, EPA recommends that tolerances at the LOQ of the method be proposed as follows: 0.05 ppm in eggs, milk, and the meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep; and 0.1 ppm in the meat, fat, and meat byproducts of poultry.

D. International Residue Limits

CODEX maximum residue limits (MRLs) for residues of vinclozolin and its metabolites containing the 3,5-DCA moiety have been established in common bean at 2 ppm, rape seed at 1 ppm (no limit for canola), cattle meat and milk at 0.5 ppm, and chicken meat and eggs at 0.05 ppm. No Canadian or Mexican tolerances have been established for vinclozolin residues in succulent beans, rape, canola, meat, milk, poultry, or eggs.

The CODEX MRLs for canola (rape seed), cattle meat, cattle milk, and poultry eggs are in harmony with the proposed tolerances associated with this petition. The chicken meat MRL (0.05 ppm) is not in harmony with the proposed tolerance in poultry meat (0.1 ppm) due to recovery discrepancies with the analytical method.

E. Rotational Crop Restrictions

Based on a limited field rotational crop study which was adequate to satisfy the data requirement, vinclozolin residues were all < 0.05 ppm (LOQ of method) in all plant commodities (wheat, cabbage, and potatoes) at the minimum plant-back interval of 30 days. Therefore, EPA has concluded that it is permissible to rotate to small grains, leafy vegetables and root crops after a 30–day interval.

VI. Conclusion

Therefore, tolerances are established for combined residues of vinclozolin, 3-(3,5-dichlorophenyl)-5-ethynyl-5-methyl-2,4-oxazolidinedione and its metabolites containing the 3,5-dichloroaniline moiety, in or on succulent beans at 2.0 ppm; canola at 1.0 ppm; eggs, milk, and the meat, fat, and meat byproducts of cattle, goats, hogs, horses, and sheep at 0.05 ppm; and the meat, fat, and meat byproducts of poultry at 0.1 ppm.

VII. Objections and Hearing Requests

Under section 408(g) of the FFDCA, as amended by the FQPA, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. The EPA procedural regulations which govern the submission of objections and requests for hearings appear in 40 CFR part 178. Although the procedures in those regulations require some modification to reflect the amendments made to the FFDCA by the FQPA of 1996, EPA will continue to use those procedures, with appropriate adjustments, until the necessary modifications can be made. The new section 408(g) provides essentially the same process for persons to ''object'' to a regulation for an exemption from the requirement of a tolerance issued by EPA under new section 408(d), as was provided in the old FFDCA sections 408 and 409. However, the period for filing objections is now 60 days, rather than 30 days.

A. What Do I Need to Do to File an Objection or Request a Hearing?

You must file your objection or request a hearing on this regulation in accordance with the instructions provided in this unit and in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket control number OPP–301015 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk on or before September 18, 2000.

1. *Filing the request.* Your objection must specify the specific provisions in the regulation that you object to, and the grounds for the objections (40 CFR 178.25). If a hearing is requested, the objections must include a statement of the factual issues(s) on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the objector (40 CFR 178.27). Information submitted in connection with an objection or hearing request 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 information 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.

Mail your written request to: Office of the Hearing Clerk (1900), Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460. You may also deliver your request to the Office of the Hearing Clerk in Rm. C400, Waterside Mall, 401 M St., SW., Washington, DC 20460. The Office of the Hearing Clerk is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Office of the Hearing Clerk is (202) 260–4865.

2. *Tolerance fee payment.* If you file an objection or request a hearing, you must also pay the fee prescribed by 40 CFR 180.33(i) or request a waiver of that fee pursuant to 40 CFR 180.33(m). You must mail the fee to: EPA Headquarters Accounting Operations Branch, Office of Pesticide Programs, P.O. Box 360277M, Pittsburgh, PA 15251. Please identify the fee submission by labeling it ''Tolerance Petition Fees.''

EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding the waiver of these fees, you may contact James Tompkins by phone at (703) 305–5697, by e-mail at tompkins.jim@epa.gov, or by mailing a request for information to Mr. Tompkins at Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460.

If you would like to request a waiver of the tolerance objection fees, you must mail your request for such a waiver to: James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460.

3. *Copies for the Docket.* In addition to filing an objection or hearing request with the Hearing Clerk as described in Unit VI.A., you should also send a copy of your request to the PIRIB for its inclusion in the official record that is described in Unit I.B.2. Mail your copies, identified by docket control number OPP–301015, to: Public

Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460. In person or by courier, bring a copy to the location of the PIRIB described in Unit I.B.2. You may also send an electronic copy of your request via e-mail to: opp-docket@epa.gov. Please use an ASCII file format and avoid the use of special characters and any form of encryption. Copies of electronic objections and hearing requests will also be accepted on disks in WordPerfect 6.1/8.0 file format or ASCII file format. Do not include any CBI in your electronic copy. You may also submit an electronic copy of your request at many Federal Depository Libraries.

B. When Will the Agency Grant a Request for a Hearing?

A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is a genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues(s) in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32).

VIII. Regulatory Assessment Requirements

This final rule establishes a tolerance under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4). Nor does it require any prior consultation as specified by Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998); special considerations as required by Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994); or require OMB review or any Agency action under Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). Since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. In addition, the Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999). Executive Order 13132 requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.'' This final rule directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4).

IX. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 et seq., as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this final rule in the **Federal Register**. This final rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

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

Dated: June 21, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180—[AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 321(q), (346a) and 371.

2. In § 180.380, the table to paragraph (a) is amended by revising the entry for ''beans, succulent'', and by adding new entries to read as follows:

§ 180.380 Vinclozolin; tolerances for residues.

(a) * * *

Commodity	Parts per million	Expiration/ Revocation Date
Beans, succulent ..	2.0	9/30/03
* * *	*	*
Canola	1.0	9/30/03
Cattle, fat	0.05	9/30/03
Cattle, mbyp	0.05	9/30/03
Cattle, meat	0.05	9/30/03
* * *	*	*
Eggs	0.05	9/30/03
Goats, fat	0.05	9/30/03
Goats, mbyp	0.05	9/30/03
Goats, meat	0.05	9/30/03
Hogs, fat	0.05	9/30/03
Hogs, mbyp	0.05	9/30/03
Hogs, meat	0.05	9/30/03
Horses, fat	0.05	9/30/03
Horses, mbyp	0.05	9/30/03
Horses, meat	0.05	9/30/03
* * *	*	*
Milk	0.05	9/30/03
* * *	*	*
Poultry, fat, ...	0.1	9/30/03
Poultry, meat	0.1	9/30/03
Poultry mbyp	0.1	9/30/03
* * *	*	*
Sheep, fat	0.05	9/30/03
Sheep, mbyp	0.05	9/30/03
Sheep, meat	0.05	9/30/03
* * *	*	*

* * * * *

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