TABLE 2.—SECTION 3 REGISTRATIONS CANCELED FOR NON-PAYMENT OF MAINTENANCE FEE—Continued

Registration no.	Product Name
072992–00006	T430 Vase Solution
072992–00009	T426 Hydrating Solution
073049–00021	Gibberellic Acid, 10%
073049–00022	Release Plus
073049–00044	Gibrel 4%
073049–00053	Gibrel Plus 2x Plant Growth Regulator Soluble Powder
073062–00001	VP Paraformaldehyde
073134–00001	Bugaway! TSP Formula 1
073368–00002	LRS Gas Liquid Chlorine #140
073368–20007	LRS Liquid Sodium Hypochlorite #10
073465–00001	Shellshock Insecticide
073637–00001	Tillam 6-E Selective Herbicide
073637–00002	Tillam Technical Selective Herbicide
073727–00013	Verox -7.5
073727–00017	Verox-37
073727–00018	Verox-15
073727–00020	Verox-2
074210–00004	Sanital II
074246–00001	Zydox 25
074292–00001	Southwest Select Diatomaceous Earth
074424–00001	Zenkill 1 Flying Insect
074530–00002	Pendimethalin Tech.
074655–00017	Daracide 2302
074812–00001	The Graden Guy Diatomaceous Earth
074812–00002	Garden-Ville Diatomaceous Earth
075341–00007	Osmoplastic SD Wood Preserving Compound

IV. Public Docket

Complete lists of registrations canceled for non-payment of the maintenance fee will also be available for reference during normal business hours in the OPP Public Docket, Room 119, Crystal Mall #2, 1921 Jefferson Davis Highway South, Arlington VA, and at each EPA Regional Office. Product-specific status inquiries may be made by telephone by calling toll-free 1–800–444–7255.

List of Subjects

Environmental protection, pesticides and pest.

Dated: October 23, 2003.

James Jones,

Director, Office of Pesticide Programs.

[FR Doc. 03–27954 Filed 11–5–03; 8:45 a.m.]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0208; FRL–7321–1]

Boscalid; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket ID number OPP–2003–0208, must be received on or before December 8, 2003.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Cynthia Giles-Parker, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–7740; e-mail address: *giles-parker.cynthia@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

- Crop Production (NAICS Code 111)
- Animal Production (NAICS Code 112)
- Food Manufacturing (NAICS Code 311)
- Pesticide Manufacturing (NAICS Code 32532)

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 this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any 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 Copies of this Document and Other Related Information?

1. *Docket.* EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0208. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis

Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access.* You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically.* If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets.* Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2003–0208. The system is an''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail.* Comments may be sent by e-mail to *opp-docket@epa.gov*, Attention: Docket ID Number OPP–2003–0208. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM.* You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail.* Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID Number OPP–2003–0208.

3. *By hand delivery or courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, Attention: Docket ID Number OPP–2003–0208. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI To the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then

identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

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

List of Subjects

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

Dated:October 23, 2003.

Debra Edwards,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

BASF Corporation

PP 2F6434 and 3F6580

EPA has received pesticide petitions (PP 2F6434 and 3F6580) from BASF Corporation, Research Triangle Park, NC, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of Boscalid (3-pyridinecarboxamide, 2-chloro-*N*-(4′-chloro(1,1′-biphenyl)-2-yl) in or on the following raw agricultural and processed commodities: pome fruit at 3.0 ppm; apple pomace at 20.0 ppm and hops at 35.0 ppm, and soybean aspirated grain fraction at 2.5 ppm. EPA has determined that the petitions contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

These individual summaries are printed below as they were received from the petitioner.

PP 2F6434

A. Residue Chemistry

1. *Plant metabolism*. Nature of the residue studies (OPPTS Harmonized Guidline 860.1300) were conducted in grapes, lettuce and beans as representative crops in order to characterize the fate of BAS 510 F in all crop matrices. In all three crops the BAS 510 F Residues of Concern (ROC) were characterized as parent (BAS 510 F). A confined rotational crop study also determined that parent was the residue of concern in the representative crops of radish, lettuce and wheat.

2. *Analytical method*. In plants the parent residue is extracted using an aqueous organic solvent mixture followed by liquid/liquid partitioning and a column clean up. Quantitation is by gas chromatography using mass spectrometry (GC/MS). In livestock the residues are extracted with methanol. The extract is treated with enzymes in order to release the conjugated glucuronic acid metabolite. The residues are then isolated by liquid/liquid partition followed by column chromatography. The hydroxylated metabolite is acetylated followed by a column clean-up. The parent and acetylated metabolite are quantitated by gas chromatography with electron capture detection.

3. *Magnitude of the residues*. Field trials were carried out in order to determine the magnitude of the residue in the apples, pears and hops. Field trials were conducted in the United States in the required regions. Field trials were carried out using the maximum label rate, the maximum number of applications, and the minimum preharvest interval for each crop or crop group. In addition, a processing study was conducted on apples to determine concentration factors during normal processing of the raw agricultural commodity into the processed commodities.

B. Toxicological Profile

1. *Acute toxicity*. Based on available acute toxicity data BAS 510 F and its formulated products do not pose acute toxicity risks. The acute toxicity studies place technical BAS 510 F in toxicity category IV for acute oral; category III for acute dermal and category IV for acute inhalation. BAS 510 F is category IV for both eye and skin irritation, and it is not a dermal sensitizer. Two formulated end use products are proposed, a wettable granule (WG) termed BAS 510 02 F containing 70% BAS 510 F and a wettable granule (WG) termed BAS 516 02 F containing a 2:1 mixture of BAS 510 F and BAS 500 F. BAS 510 02 F has an acute oral toxicity category of III, acute dermal of category III, acute inhalation of category IV, eye irritation of category III, skin irritation of category IV, and is not a dermal sensitizer. BAS 516 02 F has an acute oral toxicity category of III, acute dermal of category III, acute inhalation of category IV, eye irritation of category III, skin irritation of category IV, and is not a dermal sensitizer.

2. *Genotoxicity*. Ames Test (1 Study; point mutation): Negative; *In Vitro* CHO/HGPRT Locus Mammalian Cell Mutation Assay (1 Study; point mutation): Negative; *In Vitro* V79 Cell Cytogenetic Assay (1 Study; Chromosome Damage): Negative; *In Vivo* Mouse Micronucleus (1 Study; Chromosome Damage): Negative; *In Vitro* Rat Hepatocyte (1 Study; DNA damage and repair): Negative. BAS 510 F has been tested in a total of 5 genetic toxicology assays consisting of *in vitro* and *in vivo* studies. It can be stated that BAS 510 F did not show any mutagenic, clastogenic or other genotoxic activity when tested under the conditions of the studies mentioned above. Therefore, BAS 510 F does not pose a genotoxic hazard to humans.

3. *Reproductive and developmental toxicity*. The reproductive and developmental toxicity of BAS 510 F was investigated in a two-generation rat reproduction study as well as in rat and rabbit teratology studies.

There were no adverse effects on reproduction in the two-generation study at any dose tested. Pup effects were observed, with parental toxicity, at the highest dose tested only. In both parental generations, reduced food consumption and reduced bodyweight gain were observed at 10,000 ppm. Both absolute and relative liver weights were increased 21% in F_1 generation parental females at the high dose of 10,000 ppm only. Hepatocellular centrilobular hypertrophy (usually slight) was observed in many animals of both sexes in both the F_0 and F_1 generations at 1,000 ppm, and in all animals of both sexes at 10,000 ppm. Additionally, some of the parental male rats at 10,000 ppm, in both generations, displayed centrilobular liver cell degeneration. Developmental toxicity was seen at 1,000 ppm in the form of decreased pup weights in the F_2 males, and at 10,000 ppm in the form of decreased pup weight for both males and females of both the F_1 and F_2 generations. The parental systemic and developmental toxicity NOAEL's are both 100 ppm (12 mg/kg/day).

No teratogenic effects were noted in either the rat or rabbit developmental studies. In the rat study, evidence of maternal or developmental toxicity were not observed at any dose (highest dose tested of 1,000 mg/kg/day). Neither a maternal nor developmental LOAEL were found since the highest dose tested was the NOAEL in both studies. In the rabbit teratology study, maternal toxicity observed at the mid dose of 300 milligrams/kilogram of body weight (mg/kg bw) consisted of discolored/reduced feces in one dam and an abortion in one dam. This finding is not necessarily indicative of a definitive test substance related adverse effect. The dam which displayed the fecal alterations and abortion also displayed decreased body weight and body weight gain - compared to the group mean - during gestation. These decreases occurred even prior to compound administration. Food consumption was also dramatically decreased in this dam compared to the other animals in the group. Every day from gestation day (GD) 1–12, this dam had food consumption values which were less than half the mean for the group (compound administration began on GD 7). From GD 13 to 26 (when the animal aborted and was sacrificed) this dam ate essentially nothing (food consumption during this time period was less than or equal to 1.5 grams/day). These decreases in body weight, body weight gain, and food consumption, prior to compound administration, all indicate an animal in poor health and this poor state of health, rather than compound exposure, was likely the reason for the fecal alterations and abortion.

At the high dose of 1,000 mg/kg bw a maternal body weight gain decrease compared to controls of 81% was observed during the treatment period. Reduced food consumption, reduced body weight and abortions in three dams, were also seen at 1,000 mg/kg/day. Evidence of developmental toxicity was not seen at any dose tested.

Developmental neurotoxicity was not observed at any dose in the developmental neurotoxicity study. No maternal toxic effects were noted at any dose in this study. No developmental toxicity was seen at the low dose of 12 mg/kg/day (100 ppm). Reduced body weights and body weight gains were seen at 118 mg/kg/day (1,000 ppm) during post natal day (PND) 1–4. Reduced body weights and body weight gains were seen at 1,183 mg/kg/day (10,000 ppm) as well as decreased absolute pup brain weight at day 11 post partum (p.p.) (both sexes) and decreased brain length (males only) at day 11 p.p. The reduced pup brain weights and decreased brain length go hand-in-hand and both are due to the decreased pup weights seen at this dose. In this respect, it should be noted that pup brain weights relative to body weight at p.p. 11 were not significantly different from controls at this dose. Though no maternal toxicity was seen in this study, other studies using similar doses of BAS 510 resulted in maternal toxicity. A dose of 118 mg/kg/day in female rats of the same strain in the multigeneration study, resulted in an increased incidence of hepatic centrilobular hypertrophy - a parameter which could not have been detected in the developmental neurotoxicity (DNT) study as liver histopathology on parental animals was not performed in the DNT study.

4. *Subchronic toxicity*. The subchronic toxicity of BAS 510 F was investigated in 90–day feeding studies with rats, mice and dogs, and in a 28–day dermal administration study in rats. A 90–day neurotoxicity study in rats was also performed. Generally, mild toxicity was observed. At high dose levels (doses above the LOAELs) in feeding studies, all three species displayed alterations in various clinical chemistry parameters. These clinical chemistry alterations were likely secondary to general toxicity. Statistically significant increased absolute and relative thyroid weights were observed in male rats only at doses at and above the LOAEL. Increased absolute and relative liver weights were observed in both sexes at doses above the LOAEL in rats and dogs. Increased absolute and relative liver weights were seen in both sexes of the mouse at lower doses. However, the increases in liver weights at these lower doses in the mouse were not deemed to be compound related due to the unusually low concurrent control liver weight values. At doses above the LOAELs, liver weight increases were supported by histopathology alterations in the rat and mouse, but not in the dog. Overall, only mild toxicity was observed in oral subchronic testing.

In the 28–day repeat dose dermal study, no systemic effects were noted up to the highest dose tested of 1,000 mg/kg/day.

In a 90–day rat neurotoxicity study, there was no mortality, signs of clinical toxicity, or adverse effects on food consumption or body weight at any dose level in either sex. No signs of neurotoxicity were observed during clinical observations, functional observation batteries, motor activity measurements of neuropathology. Therefore, there were no selective neurotoxic effects. Adverse effects were not seen even at the highest dose level tested. A LOAEL was not found and the NOAEL is the highest tested of 15,000 ppm (1,050 mg/kg/day in males; 1,272 mg/kg/day in females).

5. *Chronic toxicity*. Based on review of the available data, the Reference Dose (RfD) for BAS 510 F will be based on a 24–month feeding study in rats with a threshold no observed effect level (NOEL) of 5 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.05 mg/kg/day. The

following are summaries of chronic toxicity studies submitted to EPA.

The chronic toxicity/oncogenicity studies with BAS 510 F include a 12–month feeding study with Beagle dogs, an 18–month B63CF1 mouse feeding study, a 24–month Wistar rat chronic feeding study and a 24–month Wistar rat oncogenicity study.

At the highest dose tested in dogs, effects observed consisted primarily of increased liver and thyroid weights and some serum clinical chemistry changes. The NOAEL was 800 ppm (21.8 mg/kg bw males; 22.1 mg/kg bw females).

Decreased body weights were seen in males in the mouse chronic study at doses of 400 ppm and above. Decreased female body weight was seen at doses of 2,000 ppm and above. The target organ in this study was the liver. In both the rat chronic and oncogenicity studies, the highest dose tested of 15,000 ppm exceeded a maximum tolerated dose (MTD) and was discontinued after 17 months. Effects observed at the next highest dose of 2,500 ppm primarily centered around the thyroid and liver.

Overall, mild toxicity was observed with chronic exposure to BAS 510 F. No evidence of treatment-induced oncogenicity was observed in the mouse or dog studies. A slight increase in thyroid follicular cell adenomas was seen in both sexes at the high dose when the data from both rat bioassays are combined.

A mode of action (MOA) for the thyroid follicular cell adenomas has been proposed. This MOA is based on the EPA publication ''Assessment of Thyroid Follicular Cell Tumors,'' March 1998, EPA/630/R–97/002. This document describes the criteria which must be met in order for a compound to be considered under the MOA described in that publication. BASF Corporation believes that BAS 510 F has met the cited criteria.

6. *Threshold effects*. Based on a review of the available chronic toxicity data, BASF believes EPA will establish the RfD for BAS 510 F at 0.05 mg/kg/day. This RfD for BAS 510 F is based on the 2–year chronic and 2–year oncogenicity studies in rats with a threshold average NOEL of 5 mg/kg/day for males and females. Using an uncertainty factor of 100, the RfD is calculated to be 0.05 mg/kg/day. Based on the acute toxicity data, BASF believes that 510 F does not pose any acute dietary risks.

BAS 510 F was shown to be non-carcinogenic in mice and dogs. There was a slight increase in thyroid follicular cell ademonas at the high dose in both sexes in the rat. A threshold-based MOA for these tumors based on the EPA publication ''Assessment of Thyroid Follicular Cell Tumors'' (EPA/630/R–97/002, March, 1998), has been proposed. BASF believes the data to support this proposed mode of action are strong, and that the thyroid tumors seen in the rat following BAS 510 exposure have a threshold. In addition, a battery of genotoxicity studies demonstrated that BAS 510 F has no genotoxic or clastogenic potential. Therefore, BASF believes that the threshold approach to regulating BAS 510 F is appropriate. Also, it should be noted that, while the Agency has in the past considered tumors of this type to be potential human carcinogens, the European Union has published a policy which considers these tumor types, when they occur at low incidence rates in the rat, to not be relevant to man. (The publication: European Commission, European Chemicals Bureau, ECBI/49/99 – Add. 1 Rev. 2; ''Draft Summary Record, Commission Group of Specialized Experts in the fields of Carcinogenicity, Mutagenicity and Reprotoxicity'' Meeting at Arona, 1 – 2 September 1999), Therefore, BASF believes that these tumors are not likely relevant to humans and, if these tumors are to be considered relevant to humans, the threshold approach to cancer risk assessment is appropriate.

7. *Animal metabolism*. In the rat, the predominat route of excretion of BAS 510 F is fecal with urinary excretion being minor. The half life of BAS 510 F is less than 24 hours. Saturation of absorption appears to be occurring at the high dose level. BAS 510 F is rapidly and intensively metabolized to a large number of biotransformation products. The hydroxylation of the diphenyl moiety was the quantitatively most important pathway. Second most important was the substitution of the Cl of the 2-chloropyridine part against SH by conjugation with glutathione. No major differences were observed. In hens and goats the residues of concern were determined to be parent, the hydroxylated metabolite M510 F01 (2-chloro-*N*-(4′chloro-5-hydroxy-biphenyl-2-yl)nicotinamide), and the glucuronic acid of the metabolite M510 F02.

8. *Metabolite toxicology*. No additional studies were required for metabolite toxicology.

Endocrine disruption. No specific tests have been conducted with BAS 510 F to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity studies (i.e., subchronic and chronic toxicity, teratology and multi-generation reproductive studies) which would suggest that BAS 510 F produces endocrine related effects.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. A chronic dietary exposure analysis was conducted for BAS 510 F to include the proposed uses of apples and hops. The dietary exposure included prior tolerances for beet root, root vegetables, tuberous and corm vegetables, bulb vegetables, leafy vegetables, head and stem brassica, leafy brassica greens, legume vegetables, fruiting vegetables, cucurbit vegetables, stonefruit, berries, tree nuts, pistachios, cereal grains, mint, grapes, raisins, strawberries, peanut, peanut meal, peanut oil, cotton seed, soybean seed, canola, flax seed and sunflower seed in addition to the new tolerances for apples and hops. The analysis assumed 100% of the crops were treated, default processing factors (even though much lower experimentally-derived processing factors are available), and used the tolerance value for residues. The one exception to the use of defaults was for the apple processing, where an average calculated processing factor of 0.09 was used for apple juice. For apple juice concentrate, the juice factor of 0.09 was adjusted by the ratio of the default concentrate (3.9) and default juice (1.3) processing factors, which led to an estimated processing factor of 0.27 for apple juice concentrate. Even with these worst-case assumptions, it was determined that the Theoretical Maximum Residue Contribution (TMRC) was only 34.0% of the reference dose for the U.S. population and 77.1% for children 1–6 years (the highest exposed age-related subpopulation).

Based on the toxicology results, an acute dietary risk assessment for BAS 510 F is most likely not required, but if so, only for non-nursing infants <1 year old. For dietary exposure estimation, 100% crop treated and tolerance values for residues were used. The resulting acute exposure prediction for non-nursing infants (the highest exposed age-related subpopulation) resulted in an acceptable 10.6% of the acute reference dose at the 95th percentile. If a more realistic scenario were used assuming percent crop treated and the range of residues, a much lower exposure would be obtained.

ii. *Drinking water*. Estimates of ground and surface water levels were determined using SCIGROW and FIRST models, respectively. The drinking water level of concerns (DWLOCs) for chronic exposure are obtained by subtracting the chronic dietary food. This is outlined in the following table.

PERCENTAGES OF REFERENCE DOSE FOR CHRONIC EXPOSURE TO BAS 510 F

	U.S. Population (% of RfD)	Children 1–6 (% of RfD)
Chronic dietary exposure	34.0	77.1
Remainder of RfD available for water (%) (Drinking Water Level of Concern)	66.0	22.9
SCIGROW ground water estimation[1]	0.015	0.044
FIRST surface water estimation1	0.08	0.24
Total of RfD used by diet and water	34.1	77.4

[1] Used highest values predicted from the model for all agricultural uses; assumes 2L/day and 60 kg for adult; 1L/day and 10 kg for child

Overall, using worst-case parameters the predicted aggregate exposure by all potential routes for both adults and children is less than the chronic reference dose.

2. *Non-dietary exposure*. BAS 510 F is not currently planned for residential uses. Thus, residential exposure is not aggregated into the risk assessment.

D. Cumulative Effects

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.'' BAS 510 F is a foliar fungicide chemically belonging to the carboxin class of fungicides. BAS 510 F acts in the fungal cell by inhibiting mitochondrial respiration through inhibition of the succinate-ubiquinone oxidase reductase system in Complex II of the mitochondrial electron transport chain. BAS 510 F shares this mode of action with only one other currently registered U.S. pesticide - carboxin.

The EPA is currently developing methodology to perform cumulative risk assessments. At this time, there is no available data to determine whether BAS 510 F has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, BAS 510 F does not appear to produce a toxic metabolite produced by other substances.

E. Safety Determination

1.*U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and the reliability of the toxicity data, BASF has estimated that aggregate exposure to BAS 510 F will utilize 34.1% of the RfD for the U.S. population. For the highest exposed age-related subpopulation (children 1–6 years), the maximum aggregate exposure is predicted to be 77.4% of the reference dose. BASF concludes that there is a reasonable certainty that no harm will result from the aggregate exposure to residues of BAS 510 F, including anticipated dietary and drinking water exposures and non-occupational exposures.

2. *Infants and children*—i. *Developmental toxicity in the Rat*. A developmental study was conducted via oral gavage in rats with dosages of 0, 100, 300 and 1,000 mg/kg bw/day with a maternal and developmental No-Adverse-Effect Level (NOAEL) of 1,000 mg/kg. No evidence of developmental toxicity was observed up to the highest dose tested.

ii. *Developmental toxicity in the rabbit*. A developmental study was conducted via oral gavage in rabbits with dosages of 0, 100, 300 and 1,000 mg/kg bw/day. The NOAEL for maternal toxicity was 100 mg/kg bw/day and was 1,000 mg/kg/day for developmental toxicity. As noted above in section B.3. (Reproductive and developmental toxicity), this NOAEL is based on fecal alterations and an abortion in a single dam at the next highest dose of 300 mg/kg/day. The dam which displayed the fecal alterations and abortion also displayed decreased body weight, body weight gain and food consumption, compared to the group mean, during gestation. These decreases occurred even prior to compound administration. These decreases in body weight, body weight gain, and food consumption, prior to compound administration, all indicate an animal in poor health and this poor state of health, rather than compound exposure, was likely the reason for the fecal alterations and abortion. No teratogenic effects were observed at any dose level.

iii. *Reproductive toxicity*. A two-generation reproduction study in rats was conducted with dosages of 0, 12, 118, and 1,183 mg/kg bw/day. No impairment of reproductive function was noted at any dose. The parental and developmental NOAEL are both 12 mg/kg/day. Mild effects in both the parents and pups were noted at 118 mg/kg/day and consisted of an increased incidence of hepatic centrilobular hypertrophy in parents and, in the pups, slightly decreased body weight and body weight gain (7%) in F_2 generation only, and only in males. At 1,183 mg/kg/day paternal effects included decreased body weights and food consumption, increased liver weights and increased incidence of hepatic centrilobular hypertrophy and degeneration. Pup effects at this dose were an increase in pup mortality in the F_2 only and a decreased body weight in F_1 and F_2.

iv. *Reference dose*. In all reproductive studies, the NOAEL's for developmental effects were either equal to or higher than those for the parents. Therefore, BAS 510 F shows no selective toxicity for the young. In addition, there were no direct neurotoxicity effects noted in either the acute or subchronic neurotoxicity studies.

Based on these results, no additional safety factors to protect children are warranted. Since the reproductive studies NOAEL's are higher than the RfD calculated from the chronic rat study, BASF believes the Reference Dose of 0.05 mg/kg/day is also appropriate to measure safety for infants and children. Therefore, the chronic Population Adjusted Dose (cPAD) is also 0.05 mg/kg bw/day.

F. International Tolerances

A maximum residue level (MRL) has not been established for BAS 510 F in any crop by the Codex Alimentarius Commission.

PP 3F6580

A. Residue Chemistry

1. *Plant metabolism*. Nature of the residue studies (OPPTS Harmonized

Guideline 860.1300) were conducted in grapes, lettuce and beans as representative crops in order to characterize the fate of Boscalid (BAS 510 F) in all crop matrices. In all three crops the BAS 510 F Residues of Concern (ROC) were characterized as parent BAS 510 F. A confined rotational crop study also determined that parent was the residue of concern in the representative crops of radish, lettuce and wheat.

2. *Analytical method*. In plants the parent residue is extracted using an aqueous organic solvent mixture followed by liquid/liquid partitioning and a column clean up. Quantitation is by GC/MS. The extract is treated with enzymes in order to release the conjugated glucuronic acid metabolite. The residues are then isolated by liquid/liquid partition followed by column chromatography. The hydroxylated metabolite is acetylated followed by a column clean-up. The parent and acetylated metabolite are quantitated by GC/ECD.

3. *Magnitude of the residues*. Field trials were carried out in order to determine the magnitude of the residue in soybean and soybean aspirated grain fraction. Field trials were conducted in the United States and Canada in the required regions. Field trials were carried out using the maximum label rate, the maximum number of applications, and the minimum preharvest interval. In addition, a processing study was conducted on the soybean to determine concentration factors. Tier III field rotational crop studies were conducted to support rotational crop tolerances for soybean.

B. Toxicological Profile

1. *Acute toxicity*. Based on available acute toxicity data BAS 510 F and its formulated products do not pose acute toxicity risks. The acute toxicity studies place technical BAS 510 F in toxicity category IV for acute oral; category III for acute dermal and category IV for acute inhalation. BAS 510 F is category IV for both eye and skin irritation, and it is not a dermal sensitizer. Two formulated end use products are proposed, a Water Dispersible Granule (WG) termed BAS 510 02F containing 70% BAS 510 F and a Water Dispersible Granule (WG) termed BAS 516 02F containing a 2:1 mixture of BAS 510 F and BAS 500F. BAS 510 02F has an acute oral toxicity category of III, acute dermal of III, acute inhalation of IV, eye irritation of III, skin irritation of IV, and is not a dermal sensitizer. BAS 516 02F has an acute oral toxicity category of III, acute dermal of III, acute inhalation of IV, eye irritation of III, skin irritation of IV, and is not a dermal sensitizer.

2. *Genotoxicity*. Ames Test (1 Study; point mutation): Negative; *In Vitro* CHO/HGPRT Locus Mammalian Cell Mutation Assay (1 Study; point mutation): Negative; *In Vitro* V79 Cell Cytogenetic Assay (1 Study; Chromosome Damage): Negative; *In Vivo* Mouse Micronucleus (1 Study; Chromosome Damage): Negative; *In Vitro* Rat Hepatocyte (1 Study; DNA damage and repair): Negative. BAS 510 F has been tested in a total of 5 genetic toxicology assays consisting of *in vitro* and *in vivo* studies. It can be stated that BAS 510 F did not show any mutagenic, clastogenic or other genotoxic activity when tested under the conditions of the studies mentioned above. Therefore, BAS 510 F does not pose a genotoxic hazard to humans.

3. *Reproductive and developmental toxicity*. The reproductive and developmental toxicity of BAS 510 F was investigated in a two-generation rat reproduction study as well as in rat and rabbit teratology studies.

There were no adverse effects on reproduction in the two-generation study at any dose tested. Pup effects were observed, with parental toxicity, at the highest dose tested only. In both parental generations, reduced food consumption and reduced bodyweight gain were observed at 10,000 ppm. Both absolute and relative liver weights were increased 21% in F_1 generation parental females at the high dose of 10,000 ppm only. Hepatocellular centrilobular hypertrophy (usually slight) was observed in many animals of both sexes in both the F_0 and F_1 generations at 1,000 ppm, and in all animals of both sexes at 10,000 ppm. Additionally, some of the parental male rats at 10,000 ppm, in both generations, displayed centrilobular liver cell degeneration. Developmental toxicity was seen at 1,000 ppm in the form of decreased pup weights in the F_2 males, and at 10,000 ppm in the form of decreased pup weight for both males and females of both the F_1 and F_2 generations. The parental systemic and developmental toxicity NOAEL's are both 100 ppm (12 mg/kg/day).

No teratogenic effects were noted in either the rat or rabbit developmental studies. In the rat study, evidence of maternal or developmental toxicity was not observed at any dose (highest dose tested of 1,000 mg/kg/day). Neither a maternal nor developmental LOAEL were found since the highest dose tested was the NOAEL in both studies.

In the rabbit teratology study, maternal toxicity observed at the mid dose of 300 mg/kg bw consisted of discolored/reduced feces in one dam and an abortion in one dam. This finding is not necessarily indicative of a definitive test substance related adverse effect. The dam which displayed the fecal alterations and abortion also displayed decreased body weight and body weight gain - compared to the group mean - during gestation. These decreases occurred even prior to compound administration. Food consumption was also dramatically decreased in this dam compared to the other animals in the group. Every day from gestation day 1 to 12, this dam had food consumption values, which were less than half the mean for the group (compound administration began on day GD 7) From gestation day 13 to 26 (when the animal aborted and was sacrificed) this dam ate essentially nothing (food consumption during this time period was less than or equal to 1.5 grams/day). These decreases in body weight, body weight gain, and food consumption, prior to compound administration, all indicate an animal in poor health and this poor state of health, rather than compound exposure, was likely the reason for the fecal alterations and abortion.

At the high dose of 1,000 mg/kg bw a maternal body weight gain decrease compared to controls of 81% was observed during the treatment period. Reduced food consumption, reduced body weight and abortions in three dams, were also seen at 1,000 mg/kg/day. Evidence of developmental toxicity was not seen at any dose tested.

Developmental neurotoxicity was not observed at any dose in the developmental neurotoxicity study. No maternal toxic effects were noted at any dose in this study. No developmental toxicity was seen at the low dose of 12 mg/kg/day (100 ppm). Reduced body weights and body weight gains were seen at 118 mg/kg/day (1,000 ppm) during PND 1–4. Reduced body weights and body weight gains were seen at 1,183 mg/kg/day (10,000 ppm) as well as decreased absolute pup brain weight at day 11 p.p. (both sexes) and decreased brain length (males only) at day 11 p.p. The reduced pup brain weights and decreased brain length go hand-in-hand and both are due to the decreased pup weights seen at this dose. In this respect, it should be noted that pup brain weights relative to body weight at p.p. 11 were not significantly different from controls at this dose.

Though no maternal toxicity was seen in this study, other studies using similar doses of BAS 510 F resulted in maternal toxicity. A dose of 118 mg/kg/day in female rats of the same strain in the

multigeneration study, resulted in an increased incidence of hepatic centrilobular hypertrophy — a parameter which could not have been detected in the DNT study as liver histopathology on parental animals was not performed in the DNT study.

4. *Subchronic toxicity*. The subchronic toxicity of BAS 510 F was investigated in 90–day feeding studies with rats, mice and dogs, and in a 28–day dermal administration study in rats. A 90–day neurotoxicity study in rats was also performed. Generally, mild toxicity was observed. At high dose levels (doses above the LOAELs) in feeding studies, all three species displayed alterations in various clinical chemistry parameters. These clinical chemistry alterations were likely secondary to general toxicity. Statistically significant increased absolute and relative thyroid weights were observed in male rats only at doses at and above the LOAEL. Increased absolute and relative liver weights were observed in both sexes at doses above the LOAEL in rats and dogs. Increased absolute and relative liver weights were seen in both sexes of the mouse at lower doses. However, the increases in liver weights at these lower doses in the mouse were not deemed to be compound related due to the unusually low concurrent control liver weight values. At doses above the LOAELs, liver weight increases were supported by histopathology alterations in the rat and mouse, but not in the dog. Overall, only mild toxicity was observed in oral subchronic testing.

In the 28–day repeat dose dermal study, no systemic effects were noted up to the highest dose tested of 1,000 mg/kg/day.

In a 90–day rat neurotoxicity study, there was no mortality, signs of clinical toxicity, or adverse effects on food consumption or body weight at any dose level in either sex. No signs of neurotoxicity were observed during clinical observations, functional observation batteries, or motor activity measurements of neuropathology. Therefore, there were no selective neurotoxic effects. Adverse effects were not seen even at the highest dose level tested. A LOAEL was not found and the NOAEL is the highest tested of 15,000 ppm (1,050 mg/kg/day in males; 1,272 mg/kg/day in females).

5. *Chronic toxicity*. Based on review of the available data, the Reference Dose (RfD) for BAS 510 F will be based on a 24–month feeding study in rats with a threshold No-Effect Level (NOEL) of 5 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.05 mg/kg/day. The following are summaries of chronic toxicity studies submitted to EPA.

The chronic toxicity/oncogenicity studies with BAS 510 F include a 12–month feeding study with Beagle dogs, an 18–month B63CF1 mouse feeding study, a 24–month Wistar rat chronic feeding study and a 24–month Wistar rat oncogenicity study.

At the highest dose tested in dogs, effects observed consisted primarily of increased liver and thyroid weights and some serum clinical chemistry changes. The NOAEL was 800 ppm (21.8 mg/kg bw males; 22.1 mg/kg bw females).

Decreased body weights were seen in males in the mouse chronic study at doses of 400 ppm and above. Decreased female body weight was seen at doses of 2000 ppm and above. The target organ in this study was the liver. In both the rat chronic and oncogenicity studies, the highest dose tested of 15,000 ppm exceeded a maximum tolerated dose (MTD) and was discontinued after 17 months. Effects observed at the next highest dose of 2,500 ppm primarily centered around the thyroid and liver.

Overall, mild toxicity was observed with chronic exposure to BAS 510 F. No evidence of treatment-induced oncogenicity was observed in the mouse or dog studies. A slight increase in thyroid follicular cell adenomas was seen in both sexes at the high dose when the data from both rat bioassays are combined.

A mode of action (MOA) for the thyroid follicular cell adenomas has been proposed. This MOA is based on the EPA publication ''Assessment of Thyroid Follicular Cell Tumors,'' March 1998, EPA/630/R–97/002. This document describes the criteria, which must be met in order for a compound to be considered under the MOA described in that publication. BASF Corporation believes that BAS 510 F has met the cited criteria.

6. *Threshold effects*. Based on a review of the available chronic toxicity data, BASF believes EPA will establish the Reference Dose (RfD) for BAS 510 F at 0.05 mg/kg/day. This RfD for BAS 510 F is based on the 2–year chronic and 2–year oncogenicity studies in rats with a threshold average NOEL of 5 mg/kg/day for males and females. Using an uncertainty factor of 100, the RfD is calculated to be 0.05 mg/kg/day. Based on the acute toxicity data, BASF believes that 510 F does not pose any acute dietary risks.

BAS 510 F was shown to be non-carcinogenic in mice and dogs. There was a slight increase in thyroid follicular cell adenomas at the high dose in both sexes in the rat. A threshold-based mode of action for these tumors based on the EPA publication ''Assessment of Thyroid Follicular Cell Tumors'' (EPA/630/R–97/002, March, 1998) has been proposed. BASF believes the data to support this proposed mode of action are strong, and that the thyroid tumors seen in the rat following BAS 510 exposure have a threshold. In addition, a battery of genotoxicity studies demonstrated that BAS 510 F has no genotoxic or clastogenic potential. Therefore, BASF believes that the threshold approach to regulating BAS 510 F is appropriate. Also, it should be noted that, while the Agency has in the past considered tumors of this type to be potential human carcinogens, the European Union has published a policy which considers these tumor types, when they occur at low incidence rates in the rat, to not be relevant to man. (The publication: ''European Commission, European Chemicals Bureau, ECBI/49/99 — Add. 1 Rev. 2; Draft Summary Record, Commission Group of Specialized Experts in the fields of Carcinogenicity, Mutagenicity and Reprotoxicity, Meeting at Arona, 1 – 2 September 1999).'' Therefore, BASF believes that these tumors are not likely relevant to humans and, if these tumors are to be considered relevant to humans, the threshold approach to cancer risk assessment is appropriate.

7. *Animal metabolism*. In the rat, the predominant route of excretion of BAS 510 F is fecal with urinary excretion being minor. The half-life of BAS 510 F is less than 24 hours. Saturation of absorption appears to be occurring at the high dose level. BAS 510 F is rapidly and intensively metabolized to a large number of biotransformation products. The hydroxylation of the diphenyl moiety was the quantitatively most important pathway. Second most important was the substitution of the Cl of the 2-chloropyridine part against SH by conjugation with glutathione. No major differences were observed with regard to label, sex, and dose level.

In hens and goats the residues of concern were determined to be parent, the hydroxylated metabolite M510 F01 (2-chloro-*N*-(4′chloro-5-hydroxy-biphenyl-2-yl)nicotinamide), and the glucuronic acid of the metabolite M510 F02.

8. *Metabolite toxicology*. No additional studies were required for metabolite toxicology.

9. *Endocrine disruption*. No specific tests have been conducted with BAS 510 F to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant

toxicity studies (i.e., subchronic and chronic toxicity, teratology and multi-generation reproductive studies) which would suggest that BAS 510 F produces endocrine related effects.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food.* A chronic dietary exposure analysis was conducted for BAS 510 F including crops which are target uses as well as inadvertent residues in rotational crops. The analysis assumed 100% of the crops were treated, default processing factors (even though much lower experimentally-derived processing factors are available), and used the tolerance value for residues. Even with these worst-case assumptions, it was determined that the Theoretical Maximum Residue Contribution (TMRC) was only 30.1% of the reference dose for the U.S. population and 62.5% for children 1–6 years (the highest exposed age-related subpopulation).

Based on the toxicology results, an acute dietary risk assessment for BAS 510 F is most likely not required, but if so only for children 1–6 years. For dietary exposure estimation, 100% crop treated and tolerance values for residues were used. The resulting acute exposure prediction for children 1–6 years (the highest exposed age-related subpopulation) resulted in an acceptable 8.8% of the acute reference dose at the 95th percentile. If a more realistic scenario were used assuming percent crop treated and the range of residues, a much lower exposure would be obtained.

ii. *Drinking water.* Estimates of ground and surface water levels were determined using SCIGROW and FIRST models, respectively. The drinking water level of concerns (DWLOCs) for chronic exposure is obtained by subtracting the chronic dietary food. This is outlined in the following table.

PERCENTAGES OF REFERENCE DOSE FOR CHRONIC EXPOSURE TO BAS 510 F

K	U.S. Population (% of RfD)	Children 1–6 (% of RfD)
Chronic dietary exposure	30.1	62.5
Remainder of RfD available for water (%) (Drinking Water Level of Concern)	69.9	37.5
SCIGROW ground water estimation[1]	0.015%	0.044%
FIRST surface water estimation[1]	0.08%	0.24%
Total of RfD used by diet and water	30.2%	62.8%

[1] Used highest values predicted from the model for all agricultural uses; assumes 2L/day and 60 kg for adult; 1L/day and 10 kg for child

Overall, using worst-case parameters the predicted aggregate exposure by all potential routes for both adults and children is less than the chronic reference dose.

2. *Non-dietary exposure.* BAS 510 F is not currently planned for residential uses. Thus, residential exposure is not aggregated into the risk assessment.

D. Cumulative Effects

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.'' BAS 510 F is a foliar fungicide chemically belonging to the carboxin class of fungicides. BAS 510 F acts in the fungal cell by inhibiting mitochondrial respiration through inhibition of the succinate-ubiquinone oxidase reductase system in Complex II of the mitochondrial electron transport chain. BAS 510 F shares this mode of action with only one other currently registered U.S. pesticide — carboxin.

The EPA is currently developing methodology to perform cumulative risk assessments. At this time, there is no available data to determine whether BAS 510 F has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, BAS 510 F does not appear to produce a toxic metabolite produced by other substances.

E. Safety Determination.

1. *U.S. population.* Using the conservative exposure assumptions described above and based on the completeness and the reliability of the toxicity data, BASF has estimated that aggregate exposure to BAS 510 F will utilize 30.2% of the RfD for the U.S. population. For the highest exposed age-related subpopulation (children 1–6 years), the maximum aggregate exposure is predicted to be 62.8% of the reference dose. BASF concludes that there is a reasonable certainty that no harm will result from the aggregate exposure to residues of BAS 510 F, including anticipated dietary and drinking water exposures and non-occupational exposures.

2. *Infants and children*—i. *developmental toxicity in the Rat.* A developmental study was conducted via oral gavage in rats with dosages of 0, 100, 300 and 1,000 mg/kg bw/day with a maternal and developmental No-Adverse-Effect Level (NOAEL) of 1,000 mg/kg. No evidence of developmental toxicity was observed up to the highest dose tested.

3. *Developmental toxicity in the rabbit.* A developmental study was conducted via oral gavage in rabbits with dosages of 0, 100, 300 and 1,000 mg/kg bw/day. The NOAEL for maternal toxicity was 100 mg/kg bw/day and was 1,000 mg/kg/day for developmental toxicity. As noted above in section 3.0, this NOAEL is based on fecal alterations and an abortion in a single dam at the next highest dose of 300 mg/kg/day. The dam which displayed the fecal alterations and abortion also displayed decreased body weight, body weight gain and food consumption, compared to the group mean, during gestation. These decreases occurred even prior to compound administration. These decreases in body weight, body weight gain, and food consumption, prior to compound administration, all indicate an animal in poor health and this poor state of health, rather than compound exposure, was likely the reason for the fecal alterations and abortion. No teratogenic effects were observed at any dose level.

i. *Reproductive toxicity.* A two-generation reproduction study in rats was conducted with dosages of 0, 12, 118, and 1,183 mg/kg bw/day. No

impairment of reproductive function was noted at any dose. The parental and developmental NOAEL are both 12 mg/kg/day. Mild effects in both the parents and pups were noted at 118 mg/kg/day and consisted of an increased incidence of hepatic centrilobular hypertrophy in parents and, in the pups, slightly decreased body weight and body weight gain (7%) in F_2 generation only, and only in males. At 1,183 mg/kg/day paternal effects included decreased body weights and food consumption, increased liver weights and increased incidence of hepatic centrilobular hypertrophy and degeneration. Pup effects at this dose were an increase in pup mortality in the F_2 only and decreased body weight in F_1 and F_2.

ii. *Reference dose*. In all reproductive studies, the NOAEL's for developmental effects were either equal to or higher than those for the parents. Therefore, BAS 510 F shows no selective toxicity for the young. In addition, there were no direct neurotoxicity effects noted in either the acute or subchronic neurotoxicity studies.

Based on these results, no additional safety factors to protect children are warranted. Since the reproductive studies NOAEL's are higher than the RfD calculated from the chronic rat study, BASF believes the Reference Dose of 0.05 mg/kg/day is also appropriate to measure safety for infants and children. Therefore, the chronic Population Adjusted Dose (cPAD) is also 0.05 mg/kg bw/day.

F. International Tolerances

A maximum residue level (MRL) has not been established for BAS 510 F in any crop by the Codex Alimentarius Commission.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–7583–8]

Regulatory Innovation Pilot Projects (Project XL)

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of availability of the final project agreement modifications to Buncombe County Leachate Recirculation/Gas Recovery (Bioreactor) Project XL pilot.

SUMMARY: EPA is requesting comments on modifications to the Project XL Final Project Agreement (FPA) for Buncombe County. The FPA is a voluntary agreement that was developed collaboratively by Buncombe County, the North Carolina Department of Environment and Natural Resources (NCDENR), and EPA. The original FPA was agreed upon and signed by each participant on September 18, 2001. Since that time, Buncombe County has utilized the expertise of a couple of widely-recognized experts in the bioreactor field—Dr. Morton Barlaz (North Carolina State University), and Dr. Debra Reinhart (University of Central Florida). These technical experts have made a few professional recommendations to Buncombe County regarding the Buncombe County bioreactor landfill project. These recommendations have been documented in a Preliminary Design Report (PDR) submitted to EPA and the State in September 2002. The Preliminary Design Report contains a table that lays out seven specific proposed FPA modifications. For each of the proposed modifications, the table identifies: the FPA agreed-upon original criteria, proposed modification to FPA language, and reason for the modification. The recommendations are based upon the best professional judgement of the technical experts being utilized by Buncombe County. The FPA modifications will help to further clarify the existing FPA. The FPA modifications also identify what parameters the recognized experts perceive to be necessary (*e.g.*, where the original FPA language may have been silent), or unnecessary and not very useful. The proposed FPA modifications contain suggestions for specific parameters that are directly applicable to the decomposition of wastes, thereby steering the State of North Carolina, EPA, and Buncombe County towards more useful and consistent measuring of critical data. EPA has determined that these FPA modifications would not warrant a change to the rule; however, EPA is providing notice to the public and stakeholders regarding these modifications to the FPA for Buncombe County.

The Project XL program, announced in the **Federal Register** on May 23, 1995 (60 FR 27282), gives regulated entities the flexibility to develop alternative strategies that will replace or modify specific regulatory or procedural requirements on the condition that they produce greater environmental benefits. In 1995, EPA had set a goal of implementing fifty XL projects undertaken in full partnership with the States. The Agency had achieved the goal of implementing 50 innovative pilot projects, and as of January, 2003 EPA is no longer accepting proposals for new Project XL pilot projects. The implementation of several of these innovative pilots is on-going. Buncombe County is one of the many innovative pilots that is currently in the implementation phase.

In the Final Project Agreement, Buncombe County proposes to use certain bioreactor techniques (*e.g.*, leachate recirculation) at its municipal solid waste landfill (MSWLF), to accelerate the biodegradation of landfill waste and decrease the time it takes for the waste to stabilize in the landfill. The principal objectives of this bioreactor XL project are to evaluate performance of an alternative landfill liner and to assess waste decomposition when recirculated leachate is added to the landfill. To achieve the objectives of the project, Buncombe County proposes to recirculate leachate in MSWLF cells to be constructed with a liner that differs in certain respects from the liner design specified in the Subtitle D regulations. In order to carry out this project, Buncombe County sought relief from current Resource Conservation and Recovery Act (RCRA) Subtitle D regulations (40 CFR part 258), which set forth design and operating criteria. Buncombe County desires to construct the remainder of its landfill cells with an approved alternative liner while implementing this leachate recirculation/gas recovery project. Buncombe County also sought regulatory flexibility from the prohibition in 40 CFR 258.28, Liquid Restrictions, which precludes the addition of useful bulk or non-containerized liquid amendments. During periods of low leachate generation, Buncombe County wanted to be able to supplement the leachate flow with water from the adjoining French Broad River to maintain moisture levels in the landfill. Some of the superior environmental benefits that Buncombe County expects to achieve with this project include: Improved leachate quality; reduction in the potential for uncontrolled releases of leachate to contaminate the groundwater, or gas to contaminate the air during the post-closure phase (should a containment system failure occur); increased gas yield and capture; rapid waste biodegradation and stabilization; increased lifespan of the landfill resulting in less need for construction of additional landfills; reduced post-closure costs; and faster reclamation of land for future use. The Buncombe County proposal is one of several bioreactor XL project proposals that are currently being implemented through the Project XL program. This