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 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 your estimate.
5. Provide specific examples to illustrate your concerns.
6. Offer alternatives.
7. Make sure to submit your comments by the comment period deadline identified.
8. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your response. It would also be helpful if you provided the name, date, and **Federal Register** citation related to your comments.

II. Background

A. What Action is the Agency Taking?

Under section 4 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), EPA is reevaluating existing pesticides to ensure that they meet current scientific and regulatory standards. Using a modified, streamlined version of its public participation process, EPA has completed a RED for the low risk pesticide, nitrogen under section 4(g)(2)(A) of FIFRA. EPA has determined that the data base to support reregistration is substantially complete and that products containing nitrogen will be eligible for reregistration, provided the risks are mitigated either in the manner described in the RED or by another means that achieves equivalent risk reduction. Upon submission of any required product specific data under section 4(g)(2)(B) and any necessary changes to the registration and labeling (either to address any concerns identified in the RED or as a result of product specific data), EPA will make a final reregistration decision under section 4(g)(2)(C) for products containing nitrogen.

Nitrogen is used commercially to generate an inert atmosphere usually for product packaging. In the food industry, it is used to preserve packaged foods, such as ground coffee, by displacing oxygen. As a pesticide active ingredient, nitrogen may be used as a fumigant to control insects in structures and on stored food commodities. Currently there is only one registered end-use product containing nitrogen as the active ingredient. As a pesticide inert it is used as an aerosol propellant.

EPA must review tolerances and tolerance exemptions that were in effect when the Food Quality Protection Act (FQPA) was enacted in August 1996, to ensure that these existing pesticide residue limits for food and feed commodities meet the safety standard established by the new law. Tolerances are considered reassessed once the safety finding has been made or a revocation occurs. EPA has reviewed and made the requisite safety finding for the nitrogen tolerances included in this notice.

EPA is applying the principles of public participation to all pesticides undergoing reregistration and tolerance reassessment. The Agency's Pesticide Tolerance Reassessment and Reregistration; Public Participation Process, published in the Federal Register on May 14, 2004, explains that in conducting these programs, the Agency is tailoring its public participation process to be commensurate with the level of risk, extent of use, complexity of issues, and degree of public concern associated with each pesticide. EPA can expeditiously reach decisions for pesticides like nitrogen, which pose no risk concerns, have low use, affect few if any stakeholders, and require little/no risk mitigation. Once EPA assesses uses and risks for such pesticides, the Agency may go directly to a decision and prepare a document summarizing its findings. The Agency therefore is issuing the low risk nitrogen RED, risk assessments, and related documents simultaneously for public comment.

The reregistration program is being conducted under Congressionally mandated time frames, and EPA recognizes the need both to make timely decisions and to involve the public in finding ways to effectively mitigate pesticide risks. Nitrogen, however, poses no risks that require mitigation. The Agency therefore is issuing the nitrogen RED, its risk assessments, and related support materials simultaneously for public comment. The comment period is intended to provide an opportunity for public input and a mechanism for initiating any necessary amendments to the RED. All comments should be submitted using the methods in Unit I. of the **SUPPLEMENTARY INFORMATION**, and must be received by EPA on or before the closing date. These comments will become part of the Agency Docket for nitrogen. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments.

EPA will carefully consider all comments received by the closing date and will provide a Response to Comments Memorandum in the Docket and electronic Edocket. If any comment significantly affects the document, EPA also will publish an amendment to the RED in the **Federal Register**. In the absence of substantive comments requiring changes, the nitrogen RED will be implemented as it is now presented.

B. What is the Agency's Authority for Taking this Action?

Section 4(g)(2) of FIFRA as amended directs that, after submission of all data concerning a pesticide active ingredient, ''the Administrator shall determine whether pesticides containing such active ingredient are eligible for reregistration,'' before calling in product specific data on individual end-use products and either reregistering products or taking other ''appropriate regulatory action.''

Section 408(q) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(q), requires EPA to review tolerances and exemptions for pesticide residues in effect as of August 2, 1996, to determine whether the tolerance or exemption meets the requirements of section 408(b)(2) or (c)(2) of FFDCA. This review is to be completed by August 3, 2006.

List of Subjects

Environmental protection, Pesticides and pests.

Dated: December 17, 2004.

Debra Edwards,

Director, Special Review and Reregistration Division, Office of Pesticide Programs.

[FR Doc. 05–1025 Filed 1–18–05; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0297; FRL–7690–5]

[S,S]-Ethylene diamine disuccinic acid; 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 identification (ID) number OPP–2004–0297, must be received on or before February 18, 2005.

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: Bipin Gandhi, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–8380; e-mail address: *gandhi.bipin@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 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 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 ID number OPP–2004–0297. 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, 1801 S. Bell St., 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–2004–0297. 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–2004–0297. 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–2004–0297.

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, 1801 S. Bell St., Arlington, VA, Attention: Docket ID Number OPP–2004–0297. 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: December 29, 2004.

Betty Shackleford,

Acting 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 summary may have been edited by EPA if the terminology used was unclear, the summary contained extraneous material, or the summary unintentionally made the reader conclude that the findings reflected EPA's position and not the position 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.

The Associated Octel Company, Limited

PP 4E6818

EPA has received a pesticide petition (4E6818) from The Associated Octel Company, Limited, P.O. Box 17, Oil Sites Road, Ellesmere Port, South Wirral L65 4HF, United Kingdom proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish an exemption from the requirement of a tolerance for [S,S]-ethylene diamine disuccinic acid, CAS Reg. No. 20846–91–7. EPA has determined that the petition contains 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.

A. Residue Chemistry

1. *Plant metabolism.* [S,S]-Ethylene diamine disuccinic acid is a chelating agent that is used as a vehicle to deliver micronutrients essential for healthy and rapid growth, such as iron and cobalt, to plants. It is unknown whether or not plants would uptake [S,S]-ethylene diamine disuccinic acid that might leach into the soil when applied as a minor component of pesticide formulations. However, organic chelating agents are not absorbed normally by growing plants. It appears that the primary role the chelate plays is to hold the metallic cations near the root surface until direct absorption of the free cation can take place. Once the micronutrient cations are inside the plant, other organic chelates (such as citrates) may be carriers of these cations to different parts of the plant (Ref. 1). Therefore, it is unlikely that [S,S]-ethylene diamine disuccinic acid would accumulate within plant tissue through its application to the soil as a minor component of pesticide formulations.

2. *Analytical method.* An analytical method has not been proposed because [S,S]-ethylene diamine disuccinic acid residues harmful to plants and animals are highly unlikely to occur when it is applied as part of the proposed pesticide formulation and according to that formulation's label directions for use.

3. *Magnitude of residues.* A waiver of the residue data has been requested because [S,S]-ethylene diamine disuccinic acid is produced by actinomycetes, *Amycolatopis japonica sp. nov.* (Ref. 2) and *Amycolatopsis orientalis* (Ref. 3), which are naturally occurring bacteria, degrades rapidly and is completely mineralized in the soil, will have limited accessibility to plants in the proposed use pattern, and exhibits low mammalian toxicity. [S,S]-Ethylene diamine disuccinic acid is a siderophore produced by actinomycetes, and it functions symbiotically with plants to assist in the transport of soil metals to plant rootlets. The use of [S,S]-ethylene diamine disuccinic acid, therefore, does not constitute the addition of a foreign material to the soil; rather, it is a compound that soil microorganisms and plants already encounter. Natural mechanisms already exist for the degradation and/or utilization of [S,S]-ethylene diamine disuccinic acid in the soil/plant microsystem. Moreover, organic chelates are not absorbed normally by growing plants, and residues are not expected in plants.

B. Toxicological Profile

1. *Acute toxicity.* The acute toxicity of [S,S]-ethylene diamine disuccinic acid was studied in several studies using male and female rats via the oral, dermal, and inhalation routes. In two acute oral toxicity studies, the lethal dose $(LD)_{50}$ for both males and females was established at >2,700 milligrams/kilogram body weight (mg/kg bwt) and >2,000 mg/kg bwt, respectively, which were the highest dose levels tested. For the two acute dermal toxicity studies, the LD_{50} for both males and females was established at >2,640 mg/kg bwt and > 2,000 mg/kg bwt, respectively, which were the highest dose levels tested. For the acute inhalation study, the lethal concentration $(LC)_{50}$ was established at >1.49 milligrams/liter (mg/L), which was the highest concentration that could be produced using the procedures prescribed. [S,S]-Ethylene diamine disuccinic acid also was studied in several primary eye irritation, primary skin irritation, and dermal sensitization studies. In two primary eye irritation studies, two primary skin irritation studies and a 24–hour repeat application patch test, the substance was considered a non-irritant. In a dermal sensitization study and a human repeat insult patch test, the substance was found not to be a dermal sensitizer.

2. *Genotoxicty.* [S,S]-Ethylene diamine disuccinic acid was shown not to be genotoxic in a battery of standard short-term studies. In a bacterial mutation assay, it was concluded that, when tested at dose levels up to 5,000 µg/plate of histidine dependent auxotrophic mutants of *Salmonella typhimurium* in water, [S,S]-ethylene diamine disuccinic acid was not mutagenic. In a Salmonella/mammalian (Ames test) and *Escherichia coli* WP2 mutagenesis assay, [S,S]-ethylene diamine disuccinic acid was tested using tester strains TA98, TA100, TA1535, TA1537, TA1538, WP2 uvrA (pHM101), and WP2 (pHM101) in the presence and absence of Aroclor-induced rat liver microsomal enzymes at a maximum dose of 5,000 µg per plate and was found not to cause a positive response. Further, [S,S]-ethylene diamine disuccinic acid was tested in a L5178Y TK+/- mouse lymphoma mutagenesis assay in the absence and presence of aroclor induced rat liver S-9, using doses of 4,028 to 2,765 µg/mL in the initial assay and 5,028 to 2,765 µg/mL in the confirmatory assay, and was found to be negative in both the absence and presence of exogenous metabolic activation. In an *in vitro* cytogenetics assay with Chinese hamster ovary (CHO) cells, in both definitive and confirmatory assays, the test system was exposed to dose levels of 79, 157, 313, 625, 1,250, 2,500, and 5,000 µg/mL for 6 hours with a 12–hour recovery period in the absence and presence of an S-9 reaction mixture. In addition, the test system was exposed to 5, 10, 20, 40, 79, 157, 313, 625, and 1,250 µg/mL continuously for 42 hours in the absence of a S-9 reaction mixture. In the definitive assay, survival at the highest dose level was scored 82% in the non-activated 6–hour treatment study, 70% in the non-activated 18–hour treatment study, 38% in the non-activated 42–hour study, and 84% in the S-9 activated study. The three highest doses with 200 scorable metaphase cells, i.e., 313, 625, and 1,250 µg/mL in the 6–hour non-activated study, 157, 313, and 625 µg/mL in the 6–hour activated study, and 5, 10, and 20 µg/mL in the 42–hour non-activated study, were selected for microscopic analysis. The test article did not induce a significant increase in structural chromosome aberrations in either the absence or presence of S-9 activation, regardless of the treatment condition or harvest time ($p \geq 0.025$, Fisher's exact test). However, in the non-activated 18–hour treatment study, there were no scorable metaphase cells in any of the test article dose groups. In addition, there was a statistically significant increase in numerical aberrations in the non-activated 42–hour study at 20 µg/mL ($p < 0.025$, Fisher's exact test). There was also a statistically significant dose response in numerical aberrations in the non-activated 42–hour study ($p < 0.05$, Cochran-Armitage test). In the confirmatory assay, survival at the highest dose level scored was 78% in the non-activated 6–hour treatment study, 77% in the non-activated 18–hour study, 29% in the non-activated 42–hour treatment study, and 109% in the S-9 activated study. The three highest doses with 200 scorable metaphase cells, i.e., 157, 313, and 625 µg/mL in the 6–hour treatment study, 313, 625, and 1,250 µg/mL in the 18–hour non-activated study, and 10, 20, and 40 µg/mL in the non-activated 42–hour study, were selected for microscopic analysis. The test article did not induce a significant increase in structural or numerical chromosome aberrations in either the absence or presence of S-9 activation in the 6–hour or 18–hour treatment studies ($p \geq 0.025$, Fisher's exact test). There was a statistically significant increase in structural chromosome aberrations at the 40 µg/mL dose level in the non-activated 42–hour study ($p < 0.025$, Fisher's exact test) and a statistically

significant dose response ($p<0.05$, Cochran-Armitage test). This increase in the percentage of structural chromosome aberrations in this dose was within the acceptable range of the historical control values, and therefore this increase was not viewed as being biologically relevant. Last, in an *in vivo* cytogenetic assay in rats, male and female Sprague-Dawley rats were treated with [S,S]-ethylene diamine disuccinic acid by single-dose gavage administration of 200, 670, or 2,000 mg/kg bwt. The percentage of structurally damaged first division metaphase cells was not significantly increased in the test-article-treated groups, regardless of sex, dose, or sacrifice time ($p\leq0.025$, Fisher's exact test). The percentage of numerically changed second division metaphase cells was not significantly increased in the test-article-treated groups, regardless of sex, dose, or sacrifice time ($p>0.025$, Fisher's exact test). It was concluded that [S,S]-ethylene diamine disuccinic acid was negative in the *in vivo* cryogenic assay in rats.

3. *Reproductive and developmental toxicity*. Two range-finding developmental toxicity studies, two developmental toxicity studies and one plasma mineral level study were conducted with rats. In the first range-finding study, mated Charles River CRl:CD VAF/Plus female rats were administered 2,000, 8,000, 16,000, 24,000, and 40,000 parts per million of the test substance in the diet on gestation days 6 through 15. Maternal toxicity resulted at the 16,000 ppm level and higher, as evidenced by two test article-related deaths at the highest dose level, test article-related emaciation, soft stool, decreased defacation and no stool, and inhibited bodyweight gain, body weight loss, and dose-related decreases in food consumption when compared with the control group. Developmental toxicity was evidenced at 16,000 ppm by reduced gravid uterine weight and at doses of 24,000 ppm and above by increases in post-implantation loss when compared with the controls, and a concomitant decrease in the numbers of live fetuses. Developmental toxicity also was evidenced from the fetuses found to be severely malformed in the 24,000 ppm group. Based on the results of this study, dosage levels of 0, 2,000, 8,000, and 16,000 ppm were selected by the sponsors for the definitive developmental toxicity study. In the second range-finding study, mated Charles River Crl:CD VAF/Plus female rats were administered dosage levels of the test article of 0, 50, 200, 400, 600, and 1,000 mg/kg/day by gavage on gestation days 6 through 15. There were no significant observations of maternal toxicity at any dosage level. No indication of developmental toxicity was observed at the dose levels tested. The study's conclusion was the dose levels evaluated produced no apparent maternal or developmental toxicity that was test article related. In the first developmental toxicity study, mated Charles River Crl:CD VAF/Plus female rats were administered dosage levels of 2,000, 8,000, and 16,000 ppm of the test substance in their diet on gestation days 6 through 15. Maternal toxicity was evidenced at the high-dose level by body weight and food consumption inhibition as compared with the control group. Blood zinc levels were decreased in all treated groups, and iron and copper levels were reduced in the high-dose treated dams. Developmental toxicity was indicated by a statistically significant increase in post-implantation losses at the high-dose level. Post-implantation losses at the high-dose appeared to selectively affect the sex ratio and, as a consequence, the percentage of live male fetuses was reduced while the percentage of live female fetuses was increased. Developmental toxicity also was indicated for the high-dose group by reduced fetal body weights. Administration of the test article resulted in teratogenicity in the majority of fetuses and litters at a concentration of 16,000 ppm. Fetuses from this group were observed with singular or multiple external, visceral and/or skeletal malformations and developmental variations. All major organ systems and skeletal structures were affected. The developmental period affected covers the entire dose administration period; therefore, the results of the study indicate the test article is a non-selective teratogen capable of producing a variety of malformations and developmental changes. A depletion of one or more metals in the blood, most likely zinc, may be correlated with these changes. In conclusion, the no observed adverse effect level (NOAEL) for the test substance when administered orally via the diet to the mated rats was 8,000 ppm with regard to maternal toxicity and developmental toxicity. In the second developmental toxicity study, the test substance was administered to mated Charles River Crl:CD VAF/Plus female rats by oral gavage at dose levels of 0, 50, 400, and 1,000 mg/kg/day on gestation days 6 through 15. Maternal toxicity was indicated at the 1,000 mg/kg/day dose level by a significant reduction in mean carcass weights, a significant reduction in food consumption, and an increased incidence of clinical observations; therefore, the NOAEL was considered to be 400 mg/kg/day. Developmental toxicity was not indicated at any dose level evaluated, and the NOAEL with respect to developmental toxicity was considered greater than 1,000 mg/kg/day. The plasma mineral levels in pregnant rats were evaluated. In this study, mated Charles River Crd:CD VAF/Plus female rats were used to determine the effect of the test substance on plasma levels of zinc, iron and copper in pregnant rats. Dose levels of 50, 400, and 1,000 mg/kg/day were administered by gavage as a single daily dose on gestation days 6 through 15 at a volume of 10.0 mL/kg. This resulted in maternal toxicity at the 1,000 mg/kg/day dose level, as indicated by soft stool and reduced (non-statistically significant) weight gain during the treatment period. Treatment also resulted in a dose-dependant, statistically significant reduction in zinc plasma levels for all dose groups at both the 2 and 4 hour-time points, as compared with the control group, and a statistically significant dose-dependant reduction in plasma copper levels in all treated groups at 4 hours and at the two highest dose levels at 2 hours. Plasma levels of iron fluctuated in all treated groups at both the 2 and 4 hour-time points, as compared with the control group, and these changes were not considered due to treatment with the test article. Oral administration of the test article at dosages of 50, 400, and 1,000 mg/kg/day during gestation days 6 to 15 resulted in a dose-dependant reduction in plasma zinc and copper in samples obtained 2 and 4 hours after the last dose on gestation day 15. Plasma iron levels were reduced in the 50 and 400 mg/kg/day groups in a dose-dependant fashion, as compared with the control group, from samples obtained at 2 and 4 hours following the last dose on gestation day 15. This trend was not observed at the 1,000 mg/kg/day dosage, and there was no treatment-related effect on plasma iron levels at this dose level. Administration of the test article during the period of gestation days 6 to 15 effectively lowered the plasma levels of zinc and copper in a dose-related fashion. There was no dose-related effect in plasma iron levels attributable to administration of the test article.

4. *Subchronic toxicity.* Several short-term studies were conducted using male and female rats. In a 14–day oral feeding study, one control and four dose groups of male and female Wistar rats were administered 0, 50, 500, 2,500, and

5,000 mg/kg/bwt/day of the test substance. In Group 5, the highest dose group, one male was found dead on day 9 of treatment. In groups 1, 2, 3, and 4, no deaths occurred. Test article related clinical signs of reaction to treatment with the test substance were noted in Group 5 before death or sacrifice; ruffled fur, diarrhea, emaciation, hunched posture, and sedation were noted. In Group 4, ruffled fur, diarrhea, emaciation and hunched posture were noted in both male and female animals at the end of the first week and during the second week. No clinical signs or symptoms of ill health were noted in the animals of Groups 1, 2, or 3. In a second 14–day oral feeding study with SFR-bred male Wistar rats administered dose levels of 0, 750, 1,000, and 1,250 mg/kg/bwt/day, all animals survived until scheduled necropsy, and no test article related clinical signs were evident in any animal. The mean food consumption, body weight development and relative food consumption were unaffected by the test article. Based on the results of this study, the no observed effects level (NOEL) was considered to be above 1,250 mg/kg/bwt/day. In a sub chronic 13–week oral (feeding) toxicity study, male and female SPF-bred Wistar rats were fed nominal dose levels of the test substance of 0, 50, 300, 700, and 1,000 mg/kg/bwt/day. Based on the results, the NOEL of the test substance was considered to be 300 mg/kg/bwt/day. A mineral balance 28–day oral toxicity (feeding) study using male rats fed dose levels of the test substance of 0, 50, 150, 300, and 400 mg/kg/bwt/day was conducted. Up to and including the highest dose level, there were no test article-related death or sign of reaction to treatment. Food and water consumption were not affected by treatment with the test article. The clinical laboratory data, opthalmoscopic examination as well as the recording of organ weights gave no indication of test article related effects. At macroscopic and microscopic examinations, no treatment-related histopathologic alterations in any of the organs or tissues examined were noted. There were no statistically significant changes in body weight or body weight gain. However, there was a trend towards a decreased body weight and body weight gain as the dose increased. The increased urinary output of minerals (Cu, Zn, Mg) was considered to be test article-related. This increase in urinary output was compensated by a decrease in fecal elimination of the respective minerals. There was no effect on total mineral output relative to control values. Tissue mineral (Cu, Zn, Mg) levels were not affected in the sternum, femur or liver. In the kidneys there was a statistically significant decrease in tissue Zn levels for two test groups. The lack of a dose-response effect did not allow for a definitive statement, but in consideration of the effects of treatment on Zn elimination, a test article-related effect was not ruled out.

5. *Chronic toxicity*. [S,S]-ethylene diamine disuccinic acid and its metabolites are not structurally related to a recognized carcinogen, and the weight-of-the-evidence from the reported genotoxicity and subchronic toxicity studies indicates that [S,S]-ethylene diamine disuccinic acid is not mutagenic and does not produce a morphologic effect in any organ that could lead to neoplastic change.

6. *Animal metabolism*. The absorption, distribution and elimination of [S,S]-ethylene diamine disuccinic acid were evaluated in three studies. In the first study, succinate-14C(U)-[S,S]-ethylene diamine disuccinic acid sodium salt at 2,106 mg/kg was administered to male Wistar rats by oral (gavage) dosing. This resulted in increased levels of radioactivity in bone marrow over the first 24 hours followed by biphasic elimination. The identity of the radioactivity in tissues was not determined. The mean peak bone marrow radioactivity level was 37 µg [S,S]-ethylene diamine disuccinic acid sodium salt equivalents/g (ppm) at the 24–hour time point. Bone marrow radioactivity levels declined thereafter to 10 ppm at the end of the 72–hour study period. Results of this study demonstrate that bone marrow is exposed to [S,S]-ethylene diamine disuccinic acid and/or its metabolites following oral (gavage) dosing under conditions similar to those employed in *in vivo* cytogenics studies. In the second study, female Wistar rats were dosed orally (gavage) with succinate-14C-(U)-S,S-[S,S]-ethylene diamine disuccinic acid sodium salt at 2053 mg/kg. This resulted in elevated levels of radioactivity in bone marrow during the 72–hour study period. The identity of the radioactivity in tissues was not determined. The highest mean bone marrow radioactivity level was 14 µg [S,S]-ethylene diamine disuccinic acid sodium salt equivalents/g (ppm) at the 24–hour time point. Bone marrow radioactivity declined slowly thereafter to 5 ppm at the end of the 72–hour period. Results of this study demonstrate that bone marrow is exposed to [S,S]-ethylene diamine disuccinic acid and/or its metabolites following oral (gavage) dosing under conditions similar to those employed in *in vivo* cytogenics studies. In the third study, groups of male and female Wistar rats were administered 14C-[S,S]-[S,S]-ethylene diamine disuccinic acid sodium salt by oral gavage and dermal application. Target dosing for the groups varied between 10.0 ± 0.3 uCi/rat and 18.6 ± 0.5 uCi/rat. After oral administration of 14C-[S,S]-[S,S]-ethylene diamine disuccinic acid sodium salt, radioactivity was rapidly eliminated, mainly via the feces. Based on the recovery of radioactivity in the urine, expired air and tissues, the oral absorption was less than approximately 5% of the dose in both gender groups. Based on the radioactivity recoveries in the excreta and the residue tissue content, approximately 11.1% of the applied dermal dose of 14C-[S,S]-[S,S]-ethylene diamine disuccinic acid sodium salt was absorbed by males and 5.18% was absorbed by females. During dermal exposure of 14C-[S,S]-[S,S]-ethylene diamine disuccinic acid sodium salt, the amount of radioactivity eliminated in the excreta of both gender groups was less than 9% of the dose. There was an apparent gender effect in the amount of absorbed radioactivity eliminated in the excreta for urine only. There was no statistically significant gender effect in the oral or dermal absorption of radioactivity on the basis of the radioactivity recoveries in the excreta and tissue. The overall recovery of radioactivity after oral administration of 14C-[S,S]-[S,S]-ethylene diamine disuccinic acid sodium salt was 84.4 ± 1.52% (males) and 89.5 ± (females) and after dermal application was 59.1 ± 8.03% (males) and 62.8 ± 18.6% (females) of the dose. There was no statistically-significant difference in the radioactivity recoveries between the male and female animals after both routes of administration.

7. *Metabolite toxicology*. [S,S]-Ethylene diamine disuccinic acid occurs in nature and is a siderophore produced by the Actinomycetes, *Amycolatopis japonica* sp. nov. (Ref. 2) and *Amycolatopsis orientalis* (Ref 3). [S,S]-Ethylene diamine disuccinic acid is rapidly and completely mineralized (Ref. 4). The degradation pathway of [S,S]-ethylene diamine disuccinic acid is not fully understood. However, the catabolism of [S,S]-ethylene diamine disuccinic acid was initiated by carbon-nitrogen lyase catalysing the non-hydrolytic cleavage of the C-N bond between the ethlenediamine part of the molecule and one of the succinyl residues without any collectors being required. The reaction led to the formation of fumarate and AEAA [N-(2-aminoethyl) aspartic acid]. The further degradation of AEAA remains still to be

unraveled. To date, one can merely speculate that, catalysed by DH (dehydrogenase) or a MO (monooxygenase), the C-N bond between the succinyl residue and the ethylene diamine part of the molecule is split, or that an aspartyl residue is removed by the cleavage of a C-N bond within the ethylenediamine part of AEAA. (Ref. 5). [S,S]-ethylene diamine disuccinic acid and related [S,S] homologues comply with internationally accepted criteria for ready biodegradability of chemicals ''ostensibly because the metabolic products of the biodegradation are naturally occurring biochemicals such as succinic acid'' (Ref. 6).

8. *Endocrine disruption.* [S,S]-Ethylene diamine disuccinic acid does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that [S,S]-ethylene diamine disuccinic acid had any effect on endocrine function in the developmental or reproduction studies.

C. Aggregate Exposure

1. *Dietary exposure.* As a minor formulation component, and given its rapid and complete mineralization, there is no reasonable expectation that [S,S]-ethylene diamine disuccinic acid will appear in the diet.

i. *Food.* As a minor formulation component, and given its rapid and complete mineralization, there is no reasonable expectation that [S,S]-ethylene diamine disuccinic acid will appear in the diet.

ii. *Drinking water.* As a minor formulation component, and given its rapid and complete mineralization, there is no reasonable expectation that [S,S]-ethylene diamine disuccinic acid will appear in water.

2. *Non-dietary exposure.* Non-dietary exposures to [S,S]-ethylene diamine disuccinic acid will be both occupational and residential. Occupational exposures include those to applicators and handlers of pesticides containing this substance. However, precautionary measures prescribed by the labels of pesticide products containing this substance will minimize these exposures. Also, [S,S]-ethylene diamine disuccinic acid is used in the U.S. in the metal treatment industry as a chelating agent. However, the precautionary measures prescribed by the product's material safety data sheet will minimize exposure to workers in this industry. [S,S]-Ethylene diamine disuccinic acid also is used in the U.S. in hair dye products as a chelating agent to stabilize the peroxide bleach portion. Exposure to [S,S]-ethylene diamine disuccinic acid in these residential products should be minimal because the products are used for limited periods and [S,S]-ethylene diamine disuccinic acid is used in minor amounts in the products.

D. Cumulative Effects

The potential for [S,S]-ethylene diamine disuccinic acid and other substances that have a common mechanism of toxicity has been considered. [S,S]-Ethylene diamine disuccinic acid is a naturally occurring substance produced by certain common bacteria, and it is rapidly and completely mineralized in the environment. There is no reliable information to indicate that toxic effects produced by [S,S]-ethylene diamine disuccinic acid would be cumulative with those of any other chemicals, including another pesticide. Therefore, the Associated Octel Corporation, Limited believes that it is appropriate to consider only the potential risks of [S,S]-ethylene diamine disuccinic acid in an aggregate risk assessment.

E. Safety Determination

1. *U.S. population.* As presented previously, the exposures of the U.S. general population to [S,S]-ethylene diamine disuccinic acid are low, few hazards are presented by [S,S]-ethylene diamine disuccinic acid, and the risks are minimal. Use of [S,S]-ethylene diamine disuccinic acid as a minor component of pesticide formulations applied to growing crops would not contribute significantly to the level of [S,S]-ethylene diamine disuccinic acid found naturally in the environment and to which man is exposed. Further, there is adequate information to show that any toxicological concern raised by the potential contribution of [S,S]-ethylene diamine disuccinic acid to growing crops is minimal. Occupational exposure to [S,S]-ethylene diamine disuccinic acid is expected to be well controlled and limited if worker-safety procedures are routinely practiced. Residential exposure also should be minimal, because of the low levels of [S,S]-ethylene diamine disuccinic acid contained in hair dyes and the infrequent, intermittent use of these products.

2. *Infants and children.* The complete toxicological data base, including the developmental toxicity studies, was considered in assessing the potential for additional sensitivity of infants and children to residues of [S,S]-ethylene diamine disuccinic acid. The developmental toxicity studies did indicate an increased sensitivity of rats to *in-utero* exposure to [S,S]-ethylene diamine disuccinic acid. However, this increased sensitivity appeared at very high dose levels which also caused maternal toxicity, and these levels are not expected to appear in or on growing crops, because [S,S]-ethylene diamine disuccinic acid is a minor component of pesticide formulations and it will rapidly and completely mineralize after application.

F. International Tolerances

There are no known international tolerances for residues of [S,S]-ethylene diamine disuccinic acid in food or animal feed.

G. References

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ENVIRONMENTAL PROTECTION AGENCY

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Carolina Steel Drum Superfund Site; Notice of Proposed Settlement

AGENCY: Environmental Protection Agency.