Register notice describing the electronic docket at 67 FR 38102 (May 31, 2002), or go to *http://www.epa.gov./edocket.*

Title: NSPS for Lime Manufacturing (40 CFR part 60, subpart HH) (OMB Control No. 2060–0063, EPA ICR Number 1167.07). This is a request to renew an existing approved collection that is scheduled to expire on January 31, 2003. Under the OMB regulations, the Agency may continue to conduct or sponsor the collection of information while this submission is pending at OMB.

Abstract: The New Source Performance Standards (NSPS) for Lime Manufacturing Plants were proposed on May 3, 1977 and promulgated on April 26, 1984. These standards apply to each rotary lime kiln used in lime manufacturing, which commenced construction, modification or reconstruction after May 3, 1977. The standards do not apply to facilities used in the manufacture of lime at kraft pulp mills. The purpose of this NSPS is to control the emissions of particulate matter (PM) from lime manufacturing plants, specifically from the operation of the rotary lime kilns. The standards limit particulate emissions to 0.30 kilogram per megagram (0.60 lb/ton) of stone feed, and limit opacity to 15% when exiting from a dry emission control device. This information is being collected to assure compliance with 40 CFR part 60, subpart HH.

There are three types of reporting requirements for owners or operators of facilities under this NSPS: (1) Notifications (*e.g.*, notice for new construction or reconstruction, anticipated and actual startup dates, initial performance test, and demonstration of the CMS); (2) a report on the results of the performance test; and (3) semiannual reports of instances of occurrence and duration of any startup, shutdown, or malfunctions. The purpose of the notifications are to inform the Agency or delegated authority when a source becomes subject to this standard. Performance tests are conducted to ensure that the new plants operate within the boundaries outlined in the standard. The semiannual reports are used for problem identification, as a check on source operation and maintenance, and for compliance determinations. Under this standard the data collected by the affected industry is retained at the facility for a minimum of two years and made available for inspection by the Administrator.

The Administrator has judged that PM emissions from lime manufacturing plants cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare. Owners/operators of lime manufacturing plants must notify EPA of construction, modification, startups, shutdowns, malfunctions and performance test dates, as well as provide reports on the initial performance test and annual excess emissions. The industry costs associated with the information collection activity in the standards are capital costs and O&M costs associated with continuous emissions monitoring and labor costs associated with recordkeeping and reporting. In order to ensure compliance with the standards promulgated to protect public health, adequate reporting and recordkeeping is necessary. In the absence of such information, enforcement personnel would be unable to determine whether the standards are being met on a continuous basis, as required by the Clean Air Act.

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR part 9 and 48 CFR chapter 15, and are identified on the form and/or instrument, if applicable.

Burden Statement: The annual public reporting and recordkeeping burden for this collection of information is estimated to average 42 hours per response. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

Respondents/Affected Entities: Lime Manufacturing Plants.

Estimated Number of Respondents: 53.

Frequency of Response: On occasion, initial, and semiannual.

Estimated Total Annual Hour Burden: 4,434 hours.

Estimated Total Annual Cost: $91,500.

Changes in the Estimates: There is an increase of 244 hours in the total estimated burden currently identified in the OMB Inventory of Approved ICR Burdens. This increase is due to an increase in the number of existing facilities subject to this standard resulting from the availability of more accurate data.

Dated: December 10, 2002.

Oscar Morales,

Director, Collection Strategies Division.

[FR Doc. 02–32399 Filed 12–23–02; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2002–0283; FRL–7277–5]

Bronopol; 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–2002–0283, must be received on or before January 23, 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: 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, pesticide manufacturer, or antimicrobial pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Industry (NAICS 111), e.g., Crop production.
- Industry (NAICS 112), e.g., Animal production.
- Industry (NAICS 311), e.g., Food manufacturing.

• Industry (NAICS 32532), e.g., Pesticide manufacturing.

• Industry (NAICS 32561), e.g., Antimicrobial pesticide.

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–2002–0283. 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–2002–0283. 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–2002–0283. 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–2002–0283.

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–2002–0283. 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 10, 2002.

Peter Caulkins,

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 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.

PP 2E6475

EPA has received a pesticide petition (PP 2E6475) from BASF Corporation: 3000 Continental Drive - North, Mount Olive, NJ 07828–1234; 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 2-bromo-2-nitro-1,3-propanediol (Bronopol) (CAS Reg. No. 52–51–7) in or on all raw agricultural commodities when used as an in-can preservative in pesticide formulations applied to growing crops, raw agricultural commodities after harvest, and animals. 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* Residue chemistry data are not generally required by EPA regarding tolerance exemption petitions. Consequently no plant metabolism data have been generated.

2. *Analytical method.* Since this petition is for an exemption from the requirement of a tolerance, an enforcement analytical method for 2-bromo-2-nitro-1, 3-propanediol is not needed.

3. *Magnitude of residues.* Based on the proposed amount of 2-bromo-2-nitro-1,3-propanediol to be used in the final products (0.04% or less by weight of the total formulation) and the recommended frequency and rates of application to growing crops, raw agricultural commodities after harvest, and animals, the residues are expected to be essentially undetectable and not toxicologically significant.

B. Toxicological Profile

1. *Acute toxicity.* Bronopol was given as single oral doses of 200, 280, 390, 550, or 770 mg/kg, as a solution in distilled water, to groups of ten male and ten female rats. The rats were observed for a seven-day period. Overt signs of toxicity were seen immediately after dosing with 280 mg/kg or more, and within 1 hour in males given 200 mg/kg. The signs included sedation, wheezing, gasping, nasal exudate, cyanosis, increased salivation and ataxia. Animals given 550 or 770 mg/kg

also had slow or labored respiration, and two females became prostrate. Most deaths occurred within 19 hours after dosing, but some occurred up to 72 hours. There were no gross abnormalities at autopsy of the decedents or in animals killed at the end of the study. The LD_{50} in male rats was 307 mg/kg and in female rats was 342 mg/kg.

In a further oral study groups of ten male rats were given single doses of Bronopol at 36, 54, 80, 120, 270, 400, or 600 mg/kg, as a suspension in 0.4% aqueous Cellosize solution. The rats were observed for up to ten days after treatment. Overt signs of toxicity were seen within 30 minutes after dosing with 80 mg/kg or more, and included wheezing, gasping or labored respiration and nasal exudate. Animals in the higher dose groups were inactive and adopted a low or hunched body position. Deaths occurred in these groups up to five days after treatment; macroscopic findings in the decedents included evidence of gastrointestinal irritation at 120 mg/kg or more, enlarged and dark red adrenals in some animals given 400 or 600 mg/kg, small spleens in a few rats given 80 or 120 mg/kg, and pale areas on the livers at 600 mg/kg. At terminal autopsy, one animal given 400 mg/kg also had a small spleen. Statistical analysis of the mortality data indicated that the LD_{50} was 254 mg/kg.

In an acute inhalation study a group of six rats and two groups of eight rats were exposed for 6-hour periods to Bronopol dust at nominal concentrations of 5, 0.5, or 0.05 mg per liter air respectively. The animals were then kept under observation for up to 14 days. Exposure of rats to 5 mg dust per liter air caused severe eye irritation, dyspnea and loss of bodyweight. Exposure to 0.5 mg dust per liter air caused only slight eye irritation and mild dyspnea, while no definite signs of irritation were observed in animals exposed to 0.05 mg dust per air.

In a second inhalation study four groups of 10 rats (5 males and 5 females) were exposed to Bronopol at 0 (filtered air negative control), 0.038, 0.089 or 0.588 mg/ by inhalation (nose-only) over a period of 4 hours. Exposure was followed by an observation period of 14 days. In the high dose group one animal died overnight after exposure, and 2 more animals were killed during the following day because of severe eye inflammation. Signs of marked irritancy were recorded in high dose animals but disappeared by the third observation day. Minor treatment-related signs (piloerection and hunched posture) were observed on the day of treatment in some intermediate dose rats. There was no effect in the low dose group. There were no treatment-related effects on body weight or treatment-related pathological findings except for local dermatitis and ulceration in 2 high dose animals possibly attributable to dermal exposure to the test article.

Several studies as summarized below determined 2-bromo-2-nitro-1,3-propanediol to be irritant to the eye. Bronopol in polyethylene glycol 300– 0.1 ml volumes of 0.5 or 2% Bronopol in polyethylene glycol 300 were instilled into one eye of each of six rabbits, three rabbits per concentration. The other eye in each case was treated with solvent only. The 2% solution was instilled only once, whereas the 0.5% solution was instilled on four successive days. The 2% solution of Bronopol in polyethylene glycol 300, instilled once, caused moderate inflammation and slight conjunctival edema which subsided after 5 hours. The 0.5% solution, instilled on four successive days, had effects similar to those produced by the solvent alone.

Bronopol in saline - Two drops of a solution containing 0.5% w/v Bronopol in normal saline were applied to one eye of three New Zealand White rabbits once daily on four successive days. The other eye (control) of each rabbit was treated with normal saline. The eyes were examined for irritation at 15 and 30 minutes, and at 1, 2, 3, 4, and 24 hours after treatment each day. One rabbit developed moderate inflammation and very slight edema of the conjunctiva between two and four hours after the first application, but this subsided within 24 hours. No other reactions were observed.

Bronopol in polyethylene glycol 400 - One drop of Bronopol at 0 (vehicle control), 0.5, 2, or 5% in polyethylene glycol 400 was added to one eye of 12 rabbits, 3 animals per test concentration. The other eye of each rabbit was left untreated. After 24 hours the eyes were irrigated with 300 ml of lukewarm water. Ocular reactions were assessed according to the FDA method at 1, 24, 48, and 72 hours, and then 7, 14, and 21 days after treatment.Immediately after treatment, with all the solutions, most rabbits exhibited head shaking and blinking and/or rubbing the treated eye. After 1 hour all the animals developed conjunctival reactions which had largely subsided by 24 hours, except in the most severely affected cases. One rabbit treated with 5% Bronopol had conjunctival reactions that persisted for 72 hours. The lower concentrations produced less severe and less persistent conjunctival reactions, and none of the concentrations elicited reactions in the cornea or iris. It was concluded that Bronopol in polyethylene glycol 400 was irritant at 5% but not at 2 or 0.5%, when instilled once only into the eye of the New Zealand White rabbit.

Bronopol is also irritant to the skin. In a cumulative irritancy study dilutions of Bronopol at 0 (vehicle control), 0.1, 0.5, 1, 2.5, and 5% in petrolatum was applied daily for 21 days to the same site on the back of 8 men. The treatment sites were occluded. Readings were made daily on a scale of 0 to 4. The skin irritancy threshold concentration of Bronopol was approximately 0.5 to 1.0%. To determine if the subjects had been sensitized, they were further elicited after a 10-day rest period. Two subjects reacted at 0.5 and 1% Bronopol. One reacted at 0.1%. These men received a product use test consisting of applications (without patching) to the cubital fossa twice daily for 7 days. These were negative.

In a single, 4 hour, semi-occluded dermal application of undiluted Bronopol to the skin of six rabbits produced severe dermal reactions, including eschar formation, necrosis and severe edema. Other adverse dermal reactions noted were slight hemorrhage of the dermal capillaries, blanching or brown discoloration of the skin, desquamation and scar tissue. The absence of fur growth was also occasionally noted on day fourteen with further effects indicative of corrosion. A primary irritation index of 6.2 was produced and evidence of corrosive effects were noted fourteen days after treatment. Undiluted Bronopol was found to be a severe irritant/corrosive to rabbit skin.

An acute rabbit dermal toxicity study gave a dermal LD_{50} of > 2,000 mg/kg body weight. The study was based on the EEC, OECD and EPA/OPPTS guidelines. A single oral dose of 2,000 mg/kg body weight of the test material preparation in 0.5% Tylose was applied in a group of ten rats (five males and five females) to the clipped epidermis (dorsal and dorsolateral parts of the trunk) and covered by a semi occlusive dressing for 24 hours. No mortality occurred. Signs of toxicity noted in the 2,000 mg/kg groups comprised poor general state, dyspnea and apathy. Findings were observed until including study day 1. The following skin effects were observed at the application site: white discoloration, erythema, edema, eczematoid skin change, scaling, and crust formation. Findings were observed until termination of the study. The animals did not gain weight during the first post exposure observation week but restarted to gain weight thereafter. No abnormalities were noted in the animals necropsied at the end of the study,

except in the skin of the application site, where incrustation and full thickness necrosis (9/10 animals) was observed. Under the conditions of this study, the acute dermal median lethal dose (LD_{50}) of the test substance was found to be greater than 2000 mg/kg body weight for male and female animals.

2-bromo-2-nitro-1,3-propanediol is classed as a weak skin sensitizer as indicated in four Magnusson and Kligman guinea pig skin sensitization studies as summarized below.

Study 1 - The test method was the Magnusson and Kligman guinea pig maximization test, but using 10 test animals, 4 treated controls and 4 untreated controls. Induction in the test animals was by intradermal injections of 0.03% w/v Bronopol in saline and Complete Freunds Adjuvant in the shoulder region. The induction process was supplemented 7 days later by 1.5% w/v Bronopol in distilled water applied under occlusion to the injection sites. Fourteen days later the animals were challenged on the shaved flank by occluded patch with 0.4% w/v Bronopol in distilled water. Twenty-four hours after the challenge the patch was removed and the reaction site examined 24 and 48 hours after removal. A further 3 challenges were made at either 1 or 2 week intervals. The treated controls were 4 guinea pigs treated the same as the test animals except that the test substance was omitted from the intradermal injection and the covered patch induction procedures. At each challenge 4 previously untreated animals were challenged as per the test animals. This group formed the untreated control. In the Magnusson and Kligman Maximization test, sensitization is normally assessed after one challenge. At this stage in this test there was no sensitization. One animal was sensitized after 2 challenges and a further animal after 3 challenges. In this test 2/10 animals sensitized after one challenge is classified as a mild sensitizer (Grade II), but since 3 challenges were necessary before 2/10 animals were sensitized, the sensitization potential must be regarded as less than mild, hence Bronopol was found to be a weak sensitizer by this method.

Study 2 - Induction was carried out as in Study 1 except that 9 guinea pigs were used; induction was 0.02% Bronopol in saline and induction supplementation was 6–7 days later with 5% Bronopol in saline. Fourteen days later the animals were challenged (24 hour occluded patch) with 1% Bronopol in saline. One week later the animals were subjected to a cross-reaction challenge with 2% formalin. Further challenges were made with Bronopol and formalin after 2 and 3 weeks. Any challenge reactions were recorded after 24 and 48 hours. 2/9 animals showed sensitization reactions to Bronopol at challenge 1. Animals were not challenged with Bronopol at challenge 2. No sensitization reactions were seen at challenge 3 and 1/9 animals showed an equivocal reaction at challenge 4. 1/9 animals showed an equivocal reaction to formalin at challenge 2, but there was no evidence of cross-reaction at challenges 3 and 4. It was concluded that Bronopol was a weak sensitizer under the conditions of this test. There was no significant evidence of cross-reaction to challenge with formalin.

Study 3 - Induction was carried out as in Study 1 except that 9 guinea pigs were used; induction was 0.02% Bronopol in saline and induction supplementation was 6–7 days later with 2.5% Bronopol in saline. Fourteen days later the animals were challenged (24 hour occluded patch) with 0.25% Bronopol in saline; a second challenge was made after a further 7 days. Any challenge reactions were recorded after 24 and 48 hours. There was no evidence of sensitization in the 9 animals tested at either challenge, and it was concluded that Bronopol was not a sensitizer under the conditions of this test.

Study 4 - Induction was carried out as in Study 1 except that induction was 0.02% Myacide BT (a minimum of 98% Bronopol) in saline and induction supplementation was 6–7 days later with 2.5% Myacide BT in saline. Fourteen days later the animals were challenged (24 hour occluded patch) with 0.25% Myacide BT in saline; a second challenge was made after a further 7 days. Any challenge reactions were recorded after 24 and 48 hours. There was no evidence of sensitization in the 10 animals tested at either challenge, and it was concluded that Myacide BT was not a sensitizer under the conditions of this test. The overall conclusion was that Bronopol has a very low, and variable, sensitization potential in the stringent Magnusson and Kligman guinea pig maximization test and is at most a weak sensitizer in this species. There was no evidence that the animals had become sensitized to formalin.

2. *Genotoxicty*. Mutagenicity studies including *in vitro/in vivo* in mouse erythrocytes (micronucleus assay), chromosomal aberration test in human lymphocytes, Salmonella typhimurium plate (Ames) tests with and without activation were negative. Bronopol did not induce mutations in the *in vitro* bacterial mutagenicity assay (TX 86004) or the V79 cell mutation assay (TX 86043), neither was there evidence of activity in assays for host-mediated bacterial mutagenicity or dominant lethality conducted in mice TX 74034). Furthermore, there was no increase in the incidence of micronuclei in polychromatic erythrocytes of bone marrow from male and female mice, 24, 48, or 72 hours after administration of single oral doses up to a maximum tolerated level of 160 mg/kg (TX 86001). However, weak *in vitro* clastogenic activity was detected in cultured human lymphocytes exposed for 24 hours, in the absence of S–9, to Bronopol at 30 µg/ml (TX 86049). Bronopol is normally self-stabilizing at about pH 4 in aqueous media, but decomposes at elevated temperature and more alkaline pH to release formaldehyde as a breakdown product. Under the conditions of the human lymphocyte chromosome assay, only about 10% of an initial 30 µg/ml concentration of Bronopol in the culture medium (pH 6.9) could be detected by analysis after 2 hours incubation at 370 C (DT 86029), and a formaldehyde concentration of 4.2 µg/ml was found at this time (DT 86030); the calculated value for formaldehyde released from complete breakdown of the 30 µg/ml concentration of Bronopol is 4.5 µg/ml. Formaldehyde shows clastogenic properties *in vitro* that include the induction of chromosome aberrations in human lymphocytes. Furthermore, in a lymphocyte assay conducted in-house (TX 86050), formaldehyde, in the absence of S–9 activation, elicited chromosome damage that was qualitatively and quantitatively similar to that seen in the assay of Bronopol. These findings, supported by the analytical data, indicate that the *in vitro* clastogenicity seen with Bronopol is due to its breakdown to formaldehyde. Although formaldehyde is a clastogen *in vitro,* its reactivity precludes distribution *in vivo,* so it is inactive in bone marrow and germ cells. The relative instability of Bronopol, like that of other non-carcinogenic formaldehyde-releasing agents, does not allow it to transport formaldehyde to these sites. In contrast, the carcinogen, hexamethylphosphoramide (HMPA), is more stable and requires metabolic activation to release formaldehyde; as a result, HMPA is clastogenic in bone marrow and has adverse effects in germ cells. In conclusion, the testing of Bronopol over a wide range of genetic endpoints has revealed only a single adverse finding, namely weak *in vitro* clastogenicity, and this result is clearly

attributable to the release of formaldehyde from Bronopol under the conditions of the lymphocyte assay. The consensus of negative findings in short-term *in vitro* tests, together with the negative finding in an *in vivo* test for chromosome damage and the absence of oncogenicity in the life span studies in rats and mice (see below), indicates that Bronopol does not present a genotoxic hazard.

In a 2-year rat (drinking water) chronic toxicity and tumorgenicity, Bronopol dissolved in tap water was dosed to 28 day old rats in 4 groups (45 male and 45 female in the main groups and 15 male and 15 female in the satellite groups) via the drinking water for 104 weeks at 0 (untreated control), 10, 40, and 160 mg/kg/day. The main groups were reserved for evaluation of tumorigenic potential and were not used for blood and urine samples during the study; the satellite groups were used for blood and urine samples during the study and were not included in the tumorigenicity assessment. The results at the various dose levels may be summarized as follows:

160 mg/kg/day

- Reduced grooming activity during the final year of treatment.
- Significantly increased mortality.
- Reduced weight gain from week 3 onwards among males and from week 7 onwards among females.
- Lower food intake among males from week 13 onwards.
- Marked reduction in water intake throughout the dosing period and an associated reduction in urine volume noted at weeks 25, 52, and 103.
- Increase incidence of progressive glomerulonephrosis in males and females.
- At week 52, urine repeatedly positive for hemoglobin in 4/10 males and 1/10 females, at week 77 in 4/10 males and 3/10 females, and at week 103 in 10/10 males and 1/10 females.
- Stomach lesions in 20 males and 15 females and the gastric lymph nodes showed dilation of the sinusoids in 4 males and 5 females.
- Squamous metaplasia, inflammation or atrophic acini in the salivary glands of 12 males and 11 females.

40 mg/kg/day

- Reduced weight gain from weeks 27 to 78 among males.
- Lower food intake from weeks 53 to 78 among males.
- Moderate reduction in water intake throughout the dosing period.
- At week 77, urine repeatedly positive for hemoglobin in 6/10 males and at week 103 in 3/10 males.
- Stomach lesion in 1 male.
- Squamous metaplasia, inflammation or atrophic acini in the salivary glands of 12 males and 2 females.

10 mg/kg/day

- Small but definite reduction in water intake throughout the dosing period.
- At week 77, urine repeatedly positive for hemoglobin in 2/10 males and at week 103 in 2/9 males.
- Stomach lesions in 1 male and 1 female.
- Squamous metaplasia and/or inflammation or atrophic acini in the salivary glands of 5 males and 1 female.

Control

- At week 52, urine repeatedly positive for hemoglobin in 1/10 males and 0/10 females, at week 77 in 2/10 males and 0/10 females, and at week 103 in 3/10 males and 1/10 females.
- Stomach lesions in 1 male and 2 females.
- Squamous metaplasia and/or inflammation or atrophic acini in the salivary glands of 3 males and 2 females.

The evidence of toxic effects related to the administration of Bronopol was a reduction in food intake, impaired food utilization efficiency associated with reduced bodyweight gain, and increased mortality. Changes in the stomach and gastric lymph nodes were attributed to the irritant effect of Bronopol. Unpalatability reduced the water intake and was associated with a reduced output of urine, an increased incidence of hemoglobinuria and an exacerbation of the spontaneous incidence of progressive glomerulonephrosis. Treatment with Bronopol exacerbated a spontaneous change in the salivary glands. These effects were dose related and apart from a small effect on water intake that was related to palatability, there was no evidence of toxicity at 10 mg/kg/day. There was no evidence to suggest that the administration of Bronopol affected the tumor incidence. In summary, the study gave a systemic no observed adverse effect level (NOAEL) of 10 mg/kg/day, a lowest effect level (LEL) of 40 mg/kg/day and found 2-bromo-2-nitro-1,3-propanediol (Bronopol) to be not carcinogenic.

3. *Reproductive and developmental toxicity*. In a two-generation reproduction study in rats Bronopol was administered to rats in the drinking water at concentrations of 25, 70, or 200 mg/kg/day. Thirteen males and 26 females were treated for a minimum of 80 days prior to mating. They were mated on two separate occasions to produce the F1a and F1b litters. Weanlings from the F1b litters were randomly selected (13 males and 26 females) to become parents of the next generation. The F1 parents were treated for a minimum of 87 days prior to mating, and were mated on two separate occasions to produce the F2a and F2b litters. In the F0 generation, one female from each of the control and low-dose groups, and one male and five females from the high-dose group died or were sacrificed in extremis during the study; in the F1 generation, one female from each of the low-, mid- and high-dose groups died before the end of the study. There were no treatment-related aspects, so these deaths were considered to have been incidental to Bronopol. Food consumption for the high-dose group was consistently lower than controls for the F0 males, for F0 females during the initial two weeks of treatment and the lactation periods for both mates, and for F1 females during the lactation period of the F2a mate. Water consumption was reduced in all treated groups, in a dose-related manner, throughout most of the study; this contributed to the lower achieved dosages of Bronopol that animals received, namely 22.55, 55.2, or 147 mg/kg. The female fertility index for the high-dose group was slightly lower than control at the F1 mate only. Mean body weights of the offspring of the F0 and F1 high-dose parents (F1a and F1b, and F2a and F2b, respectively) were lower than the control throughout the lactation periods. Mean body weights of the F1b pups from the low- and mid-dose groups were slightly lower than control on day 21 of the lactation period. There were no other test article-related macroscopic or microscopic changes. There was a dose-related increase in the kidney weights of treated F0 females, though the difference between the low dose group and controls was minimal. In the high-dose group animals there was a decrease in the absolute weights of the livers, and possibly also the hearts, of F1 males, and in the absolute liver weights of F2b males and females; these females also had lower absolute kidney weights. In conclusion, ingestion of Bronopol elicited signs of toxicity at all dosages, though the only reproductive or litter parameter affected at the 25 and 70 mg/kg/day dosages was body weight of F1b pups at weaning, where a minimal decrease was seen.

An early rat dermal developmental toxicity study gave a maternal NOAEL > 40 mg/kg/day (HDT) considering 2-bromo-2-nitro-1,3-propanediol as a severe dermal irritant in rats. Further development toxicity studies have been carried out for both the rat and the rabbit. In the rat study three groups of 24 timed-mated female rats were dosed once daily, orally by gavage, with

solutions of Bronopol at dose levels of 10, 28, or 80 mg/kg/day from days 6 to 15 of pregnancy, inclusive. A similar group of females were dosed with the vehicle (purified water acidified to pH 4) by the same route and over the same period, and served as controls. Maternal clinical signs, bodyweights and food consumption were recorded. On day 20 of pregnancy, the females were killed and a necropsy was performed. Numbers of corpora lutea and live and dead implantations were recorded. Live fetuses were weighed, sexed and examined for external and visceral abnormalities. Two thirds of the fetuses were also examined for skeletal abnormalities. There was evidence of maternal toxicity following oral gavage administration of Bronopol at 80 mg/kg/day, characterized by retarded bodyweight gain over days 6 to 7 of pregnancy. There was no evidence of maternal toxicity at either 10 or 28 mg/kg/day. There was no evidence of developmental toxicity at any of the dose levels investigated. There may be an association of treatment at 80 mg/kg/day with advanced ossification of sacral arches and at 28 and 80 mg/kg/day with advanced ossification of the forelimb phalanges. However, neither of these findings in these groups was unusually advanced when compared to historical background data.

In a second study using rabbit groups of 18, 19, or 20 timed-mated female animals were dosed daily between 7 and 19 days of pregnancy, inclusive, by the oral route with aqueous solutions of Bronopol at dose levels of 0 (control), 5, 20, 40, and 80 mg/kg/day. Day 0 of pregnancy was the day of mating. 80 mg/kg/day was selected as a level which should elicit maternal effects. However, in the event that the effects may have been too severe, 40 mg/kg/day was selected as the next highest level known to be tolerated by the pregnant rabbit. The lower dose level of 5 mg/kg/day and the intermediate dose level of 20 mg/kg/day were expected to be 'no effect' levels. Maternal clinical condition, bodyweight, and food consumption were recorded. The females were killed on day 28 of pregnancy and a necropsy was performed. They were weighed, sexed and examined for external, visceral, and skeletal abnormalities. At 80 mg/kg/day, Bronopol elicited severe maternal toxicity at the onset of dosing. The animals recovered after dosing ceased, but the outcome of pregnancy was affected. There was embryotoxicity characterized by growth retardation and a slightly higher than expected incidence of fetal abnormalities. This embryotoxicity was considered likely to be related to the maternal toxicity. At 40 mg/kg/day, which was considered to be the highest level likely to be tolerated by the pregnant rabbit without eliciting severe maternal toxicity, there was no evidence of adverse effects of treatment on the pregnant rabbit or developing embryos. This dose level was therefore considered to be the 'no effect' level of Bronopol with regard to developmental toxicity.

4. *Subchronic toxicity.* A 13-week rat gavage study showed a NOAEL of 20 mg/kg/day and a lowest observed adverse effect level (LOAEL) of 80 mg/kg/day. Bronopol as a solution in distilled water was dosed to CD rats (4 groups of 20 males and 20 females) by oral gavage once per day, seven days per week for 13 weeks at 0 (untreated control), 20, 80, and 160 mg/kg/day. Reaction to treatment was as follows:

160 mg/kg/day - Severe respiratory distress and abdominal distension; reduced bodyweight gain and food consumption; death of 22 males and 14 females (includes 4 male and 3 female rats which replaced rats dying after one dose); all surviving rats were killed on day 9; autopsy showed gaseous and fluid distension of the gastro-intestinal tract in the majority of decedents; ulceration, epithelial hyperplasia and hyperkeratosis or congested vessels in the stomachs of 2 males and 4 females.

80 mg/kg/day - Severe respiratory distress and abdominal distension, the latter sign confined to 6 males and 6 females which subsequently died. At week 6, only 4 males and 2 females showed slight respiratory difficulty. Seven males and 9 females died with autopsy showing gaseous and fluid distension of the gastro-intestinal tract; reduced bodyweight gain and food consumption for the first week of treatment only; renal changes in 2 males.

20 mg/kg/day - In one male, respiratory distress, which subsequently regressed; renal changes in 2 males.

A 13-week dog gavage study showed a NOAEL of 8 mg/kg/day and LOAEL of 20 mg/kg/day. Bronopol dissolved in water was dosed to Beagle dogs (4 groups of 3 males and 3 females) by oral gavage once per day, seven days per week for 3 months (13 weeks) at 0 (untreated control), 4, 8, and 20 mg/kg/day. One pair of dogs was dosed at levels of 20– 40 mg/kg/day, over a period of 2 weeks in order to determine the vomiting threshold of Bronopol. This was found to be at a dosage of approximately 20 mg/kg/day. During the study vomiting occurred within 30 minutes of dosing and no other clinical signs were observed. Macroscopic post mortem examination revealed no abnormalities. In the main study there were no deaths. Vomiting, mainly at 20 mg/kg/day, within 0.5 hour of dosing was observed with occasional passage of liquid feces and red-stained mucus in isolated animals, both dosed and control. There were no adverse effects on food or water consumption, or on bodyweight. There were no abnormalities of the eye; no macroscopic post mortem abnormalities; or morphological changes or variations from normal in histological tissue examination which could be related to dosage of the test compound. After dosing for 6 weeks, one animal receiving 8 mg/kg/day had a serum alkaline phosphatase value approximating to the upper limit of normality of 35 King Armstrong units; after 12 weeks, however, the value was well within normal limits. After dosing for 12 weeks the group mean total white cell count, although within normal limits, was significantly lower in dogs receiving 8 and 20 mg/kg/day than in the controls. One animal receiving 4 mg/kg/day had a serum glutamic-pyruvic transaminase value after 12 weeks which exceeded the upper limit of normality of 50 mU/ml. Apart from the liver of one dog receiving 20 mg/kg/day which was heavier than would normally be expected, all organ weights were within normal limits. However, when expressed as a percentage of bodyweight the mean liver and spleen weights for dogs receiving 20 mg/kg/day were significantly heavier than the control values.

5. *Chronic toxicity.* A 2-year toxicity/carcinogenicity Bronopol study (administration via drinking water) in rats showed a NOAEL of $\geq$ 7 mg/kg/day and a LEL of < 32 mg/kg/day. For more detail see the carcinogenicity summary in Unit B.2.

In a study on potential local and tumorigenic effects from repeated dermal application to mice Bronopol dissolved in 90% acetone/water was applied to the shaved dorsum of 3 groups of mice (52 male and 52 female per group) at 0 (vehicle control), 0.2%, and 0.5%. Application was at the rate of 0.3 ml per mouse on three days (Monday, Wednesday, and Friday) in each week for 80 weeks. The results are summarized as follows:

- Among some mice treated with 0.5% Bronopol, there was minimal hair loss at the periphery of the shaved area during the first three weeks of treatment.
- A marginally inferior survival rate was recorded among male mice, although the prime cause of death

among decedents showed no relation to treatment.

• Between weeks 26 and 52, an inferior bodyweight gain was recorded among male mice treated with 0.5% Bronopol, although bodyweight gain over the 80 week treatment period was comparable with that of the controls. Bodyweight gain among other treated mice was not disturbed by treatment.

• Food intake and efficiency of food utilization showed no disturbance by treatment.

• Macroscopic examination of decedents and mice killed after 80 weeks of treatment, revealed pathology which was common to some animals from control and treated groups.

• Microscopic examination of decedents and mice killed at termination revealed changes consistent with the age and strain of mouse employed.

• Treatment with Bronopol did not alter the spontaneous tumor profile of the mice.

6. *Animal metabolism*. Rat and dogs were used in a metabolic study with both oral and cutaneous dosing as follows: Oral Dosing in Rats was by stomach tube with aqueous solutions of [14C]-Bronopol (1 mg/kg). Oral Dosing Dogs - Beagle dogs were dosed with [14C]-Bronopol (2 mg) mixed with unlabelled Bronopol (6–8 mg) as an aqueous solution in gelatin capsules. Cutaneous Dosing Rats and Rabbits - Initially solutions of [14C]-Bronopol (4 mg/kg) in water, acetone and acetone/water (9:1, v/v) were applied to the clipped backs of rats to determine the influence of the vehicle on percutaneous absorption. Acetone was determined to be the preferred application vehicle. In the main tests an acetone solution of [14C]-Bronopol (4.8 mg/ml) was applied to shaved/depilated areas of the backs of rats and rabbits at the rates of 0.05 ml per rat and 0.2–0.4 ml per rabbit, the treated areas being occluded with secured polythene. After an oral dose of [14C]-Bronopol (1 mg/kg) to rats or dogs, the radioactivity was completely absorbed, evenly distributed and rapidly excreted. Excretion was almost complete in 24 hours. During 5 days, rats excreted 83.3% in the urine, 5.8% in the feces (via the bile) and 8.4% in the expired air; 1.6% was still retained probably by incorporation into pathways of intermediary metabolism of [14C]-glycerol produced by biotransformation of [14C]-Bronopol. During 5 days, dogs excreted 81.8% in the urine and 3.1% in the feces. After an oral dose of [14C]-Bronopol (1 mg/kg), peak blood levels of radioactivity were reached in rats and dogs within 2 hours, and declined with an initial half-life of 4 ± 1 hour. After an oral dose of [14C]-Bronopol (1 mg/kg) to the rat and the dog, Bronopol and its metabolites were evenly distributed. Only in tissues concerned with excretion did levels of radioactivity exceed those in the blood. When applied to the skin of rats, [14C]-Bronopol was absorbed to a greater extent from an acetone solvent vehicle than from water:acetone (1:9, v/v) or water alone. In rats, at least 7 and 15% of an applied dose was percutaneously absorbed during 24 and 96 hours respectively. In rabbits, at least 9% of an applied dose was percutaneously absorbed during 24 hours. Pretreatment of rabbit skin with a depilatory enhanced absorption. Microhistoautoradiographs of rabbit skin showed that [14C]-Bronopol was mainly localized on the epidermis around the hair follicles. The limited percutaneous absorption of Bronopol may occur through the hair follicles. Five metabolites, which were more polar than Bronopol, were detected in the urine of rats and dogs given an oral dose of [14C]-Bronopol. One metabolite, shown by comparison of infra-red and mass spectra with synthetic material to be 2-nitropropane-1,3-diol, accounted for more than 40% of the administered dose. Unchanged Bronopol, which is unstable in plasma, was not detected. A similar pattern of urinary metabolites of [14C]-Bronopol was found after cutaneous application as after oral administration of the compound.

Further metabolic studies were carried out in male and female rats following single oral doses of [14C]-Bronopol at 10 and 50 mg/kg and repeated dosing at 10 mg/kg/day with Bronopol for 14 days followed by a single oral dose, 10 mg/kg of [14C]-Bronopol. The compound was well absorbed and rapidly excreted mainly via urine. Radioactivity found in the carcass and tissues at 168 hours after dosing accounted for less than 3% of dose. There were no major consistent differences between male and female rats. Bronopol was highly metabolized and intact compound was not detected in the urine. The urinary metabolite chromatographic patterns contained numerous polar metabolites and similar patterns were found for each group. The major metabolite observed was equivalent to desbromo-bronopol (2-nitro-propane-1,3-diol). Extensive metabolism led to radiolabeled one-carbon units excreted as carbon dioxide in expired air.

7. *Metabolite toxicology*. As determined in the animal metabolism studies in Unit B.6. numerous polar metabolites were identified in urine from rat and dog. Unchanged 2-bromo-2-nitro-1,3-propanediol was not detected. The major peak in most samples corresponded to desbromobronopol (debrominated bronopol), i.e. 2-nitropropane-1, 3-diol. This metabolite is not considered of toxicological concern.

8. *Endocrine disruption*. No specific tests have been conducted with 2-bromo-2-nitro-1,3-propanediol 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 tests, i.e., teratology and multi-generation reproduction studies, which would suggest that 2-bromo-2-nitro-1,3-propanediol produces effects characteristic of the disruption of endocrine functions.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. The proposed use of 2-bromo-2-nitro-1, 3-propanediol as a preservative in end-use pesticide formulations applied to growing crops, raw agricultural commodities after harvest, and animals is not expected to result in any significant additional, dietary exposure, due to the low concentration of 2-bromo-2-nitro-1, 3-propanediol employed in the formulation and the extremely low probability of significant contact by the general public following treatment.

2-bromo-2-nitro-1, 3-propanediol has FDA approval for indirect food contact use as a preservative in adhesives that are components of food packaging or storage materials (21 CFR 175.105); as a slimicide for use in pulp and papermaking at a maximum level of 0.6 lb/ton of dry weight fiber (21 CFR 176.300); and paper components in contact with aqueous and fatty foods at a level not to exceed 0.01% by weight of those components (21 CFR 176.170). These uses are not expected to result in quantifiable residues of 2-bromo-2-nitro-1, 3-propanediol in the diet. Uses as a preservative in concentrates of agricultural pesticide products also is not expected to be a source of quantifiable residues in food.

There are no acute or chronic toxicological concerns associated with the proposed use of 2-bromo-2-nitro-1,3-propanediol as an inert ingredient in concentrates of agricultural pesticide products. An acute dietary risk assessment, therefore, is not required. Chronic exposure to 2-bromo-2-nitropropane-1, 3-diol through food is essentially insignificant.

ii. *Drinking water*. Contamination of drinking water would not be expected to

occur under the proposed use conditions of 2-bromo-2-nitro-1, 3-propanediol as a preservative at very low concentrations in pesticide products intended for applications, principally to growing crops, raw agricultural commodities after harvest, and animals; as either a direct pour-on application or as a spray. Neither method of application is expected to contaminate water supplies intended for human consumption. Bronopol is not applied to water and is not used for the disinfection of human or animal drinking water.

2. *Non-dietary exposure*. 2-bromo-2-nitro-1, 3-propanediol is used as an industrial biocide for the prevention of biofouling in areas such as recirculating water in cooling towers and evaporative condensers, air conditioners, air washers and humidifier systems, oil, gas and industrial process water, metal working fluids and paper mill pulp and process water; and for the preservation of surfactants, adhesives, starch, pigment and extender slurries, paints, latex and antifoam emulsions, absorbent clays, water based printing inks and print solutions, water based pesticides and chemical toilet solutions. The margins of exposure (MOEs) calculated for direct applicators occupationally exposed by either the dermal or inhalation route, based on worst-case estimates, revealed there is no level for concern. Estimated exposures to professional painters using paint preserved with 2-bromo-2-nitro-1, 3-propanediol were used as the worst-case for estimating secondary occupational exposure risk. MOEs were not exceeded and EPA has concluded that risk associated with secondary exposure are not of concern.

2-bromo-2-nitro-1, 3-propanediol is also used in the preservation of consumer, household and institutional products. Based on the worst-case estimate for professional painters chronically exposed to 2-bromo-2-nitro-1, 3-propanediol, EPA has concluded that risk associated with these uses are not of concern.

2-bromo-2-nitro-1, 3-propanediol also is used to preserve pharmaceuticals, cosmetics, and toiletries, which are regulated by FDA. The Cosmetic, Toiletries and Fragrance Association's (CTFA's) Cosmetic Ingredient Review (1980) states that 2-bromo-2-nitro-1,3-propanediol is safe as a cosmetic ingredient at concentrations up to 0.1% except where there is a risk of nitrosamine or nitrosamide formation. Similarly, 2-bromo-2-nitro-1,3-propanediol is listed in Annex VI of the EC Cosmetics directive as an approved preservative for use up to 0.1% except where there is a risk of nitrosamine formation.

Based on toxicity data, an aggregate risk or likelihood of the occurrence of an adverse health effect resulting from all routes of exposure to 2-bromo-2-nitro-1, 3-propanediol is not expected.

D. Cumulative Effects

There is no reliable information that would indicate or suggest that 2-bromo-2-nitro-1, 3-propanediol has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population*. The reference dose (RfD) for 2-bromo-2-nitro-1, 3-propanediol based on the 2-year chronic study (drinking water) in rats with a NOAEL of 10 mg/kg/day and using an uncertainty factor of 100 is calculated to be 0.1 mg/kg of body weight (bwt)/day. The estimated worst-case theoretical maximum residue contribution (TMRC) resulting from this action will be 0.000024 mg/kg/bwt/day for the overall U.S. population and represents 0.024 percent of the RfD. Based upon this information and review of its use, EPA has found that, when used in accordance with good agricultural practice, this ingredient is useful and a tolerance is not necessary to protect the public health.

2. *Infants and children*. Nothing in the available literature would suggest that infants and children are more sensitive to the effects of 2-bromo-2-nitro-1, 3-propanediol than adults. Exposure of infants to 2-bromo-2-nitro-1, 3-propanediol resulting from its proposed use as an inert ingredient in certain pesticide formulations is expected to be negligible and will not put infants and children at increased risk.

F. International Tolerances

BASF Corporation is not aware of the existence of any international tolerances for 2-bromo-2-nitro-1, 3-propanediol.

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FEDERAL RESERVE SYSTEM

Agency Information Collection Activities: Proposed Collection; Comment Request

AGENCY: Board of Governors of the Federal Reserve System

SUMMARY: Background. On June 15, 1984, the Office of Management and Budget (OMB) delegated to the Board of Governors of the Federal Reserve System (Board) its approval authority under the Paperwork Reduction Act, as per 5 CFR 1320.16, to approve of and assign OMB control numbers to collection of information requests and requirements conducted or sponsored by the Board under conditions set forth in 5 CFR 1320 Appendix A.1. Board–approved collections of information are incorporated into the official OMB inventory of currently approved collections of information. Copies of the OMB 83–I's and supporting statements and approved collection of information instruments are placed into OMB's public docket files. The Federal Reserve may not conduct or sponsor, and the respondent is not required to respond to, an information collection that has been extended, revised, or implemented on or after October 1, 1995, unless it displays a currently valid OMB control number.

Request for Ccomment on Information Collection Proposal.

The following information collection, which is being handled under this delegated authority, has received initial Board approval and is hereby published for comment. At the end of the comment period, the proposed information collection, along with an analysis of comments and recommendations received, will be submitted to the Board for final approval under OMB delegated authority. Comments are invited on the following:

a. whether the proposed collection of information is necessary for the proper performance of the Federal Reserve's functions; including whether the information has practical utility;

b. the accuracy of the Federal Reserve's estimate of the burden of the proposed information collection, including the validity of the methodology and assumptions used;

c. ways to enhance the quality, utility, and clarity of the information to be collected; and

d. ways to minimize the burden of information collection on respondents, including through the use of automated collection techniques or other forms of information technology.

DATES: Comments must be submitted on or before February 24, 2003.

ADDRESSES: Comments may be mailed to Ms. Jennifer J. Johnson, Secretary, Board of Governors of the Federal Reserve System, 20th Street and Constitution Avenue, N.W., Washington, DC 20551. However, because paper mail in the Washington area and at the Board of Governors is subject to delay, please consider submitting your comments by e-mail to *regs.comments@federalreserve.gov*, or