comments and input to the Agency on the preliminary risk assessments for the pesticides specified in this notice. Such comments and input could address, for example, the availability of additional data to further refine the risk assessments, such as percent crop treated information or submission of residue data from food processing studies, or could address the Agency's risk assessment methodologies and assumptions as applied to these specific chemicals. Comments should be limited to issues raised within the preliminary risk assessments and associated documents. EPA will provide other opportunities for public comment on other science issues associated with the organophosphate pesticide tolerance reassessment program. Failure to comment on any such issues as part of this opportunity will in no way prejudice or limit a commenter's opportunity to participate fully in later notice and comment processes. All comments should be submitted by March 13, 2000 at the address given under Unit I. Comments will become part of the Agency record for each individual organophosphate pesticide to which they pertain.

List of Subjects

Environmental protection, Chemicals, Pesticides and pests.

Dated: January 5, 2000.

Lois Rossi,

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

[FR Doc. 00–627 Filed 1–11–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[OPP–30000/51A; FRL–6380–6]

1,3-Dichloropropene; Proposed Determination to Terminate Special Review

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed Determination to Terminate Special Review.

SUMMARY: This Notice sets forth EPA's proposal to terminate the Special Review of 1,3-Dichloropropene (1,3-D). This proposal is based on Dow AgroSciences' changes to their product labels and EPA's determination that, with these label revisions, the benefits of 1,3-D use outweigh the risks. In making this determination, EPA considered several factors, including the risk reduction provided by numerous mitigation measures that have been added to 1,3-D labels, the benefits of 1,3-D use and the risks and benefits of alternative soil fumigants, in particular the phase-out of methyl bromide production and imports by 2005. In December, 1998, EPA issued the Reregistration Eligibility Decision (RED) document for 1,3-D and has determined that all uses of 1,3-D are eligible for reregistration.

DATES: Comments, data and information relevant to the Agency's proposed decision, identified by the docket control number OPP–30000/51A, must be received on or before March 13, 2000.

ADDRESSES: Comments may be submitted by mail, electronically or in person. Please follow the detailed instructions for each method provided in the ''SUPPLEMENTARY INFORMATION'' section.

FOR FURTHER INFORMATION CONTACT: Phil Budig, Special Review and Reregistration Division (7508C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Telephone (703) 308–8029. E-mail address: budig.philip@epa.gov.

SUPPLEMENTARY INFORMATION

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are a pesticide registrant with registered products which contain 1,3-D as an active ingredient, or if you are an agricultural producer using products containing 1,3-D as an active ingredient.

B. How Can I Get Additional Information, Including Copies of Support Documents?

1. *By mail.* You may request copies of this document and supporting documents by writing to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460 or calling 703–305–5805 between 8:30 a.m. and 4 p.m., Monday through Friday, excluding legal holidays. Be sure to include the docket control number [OPP–30000/51A] in your request.

2. *In person.* The Agency has established an official record for this action under docket control number [OPP–30000/51A]. The official records consist of the documents specifically referred to in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). The official record includes documents that are physically located in the docket, as well as documents that are referred to in those documents. The public version of the official record does not include any information claimed as CBI. The public version of this record, including printed, paper versions of any electronic comments, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is 703–305–5805.

3. *Electronically.* You may obtain electronic copies of this document and various support documents from the EPA Home page at the **Federal Register** - Environmental Documents entry for this document under ''Laws and Regulations'' (www.epa.gov/fedrgstr/).

C. How and to Whom do I Submit Comments?

You may submit comments through the mail, in person, or electronically:

1. *By mail.* Submit comments to Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

2. *In person.* Deliver comments to Public Information and Records Integrity Branch in Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

3. *Electronically.* Submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk by mail as described above in Unit I.C.1. Electronic submission on disks will be accepted in Wordperfect 5.1/6.1 or ASCII file format. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. All comments in electronic form must be identified by the docket control number [OPP–30000/51A]. Electronic comments may also be filed online at many federal Depository Libraries.

The record for the Special Review is kept in paper form. Accordingly, EPA will transfer all comments received electronically into printed paper form as they are received and will place the paper copies in the official record, which will also include all comments submitted directly in writing. The official record is the paper record maintained at the address for the Public Information and Records Integrity Branch listed above.

D. How Should I Handle Information that I Believe is Confidential?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit in response to this document as confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice.

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

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

- Explain your views as clearly as possible.
- Describe any assumptions you used.
- Provide copies of technical information or data that support your views.
- If you estimate potential burden or costs, explain how you arrived at the estimate you provide.
- Provide specific examples to illustrate your concerns.
- Offer alternative ways to improve the Agency's proposed action.
- Make sure to submit your comments by the deadline in this notice.
- To ensure proper receipt by EPA, be sure to identify the docket control 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. Introduction

1,3-Dichloropropene (1,3-D) is a soil fumigant used mainly to control plant-parasitic nematodes. A second formulation containing chloropicrin also controls soil fungi. The primary registrant of 1,3-D products is Dow AgroSciences. Dow AgroSciences' main products are Telone II, which is used to treat soils to be planted to any crop, including vegetables, orchard trees, and ornamentals, and Telone C-17, which contains chloropicrin to enhance fungicidal properties. Two other registrants also reformulate Telone II into eight end-use products. Dow AgroSciences also holds a Special Local Needs (FIFRA section 24(c)) registration for a pre-plant underground drip product, Telone EC.

1,3-D is injected as a liquid into the soil by shanks, or knives, that are inserted 12 to 18 inches beneath the soil surface. The volatile chemical then diffuses through the air spaces in the soil inhabited by nematodes and other soil-borne pests. The rate of diffusion is affected by the size of the soil particles, the amount of soil moisture present, the amount of organic material, and pH. 1,3-D can move up and into the atmosphere or down to ground water under certain conditions. The half-life of 1,3-D in soil depends on several factors; in field studies the dissipation half-life ranged from 1 to 7 days and in laboratory studies up to 54 days. For more information on 1,3-D use, see Unit VI of this document.

1,3-D is classified as a B_2, or probable human, carcinogen by both the oral and inhalation routes of exposure. Studies show that 1,3-D residues do not occur in foods planted to treated soils when 1,3-D is used as a pre-plant soil fumigant. Oral exposures can occur through consumption of contaminated ground water. Workers and residents in the vicinity of treated fields can be exposed to 1,3-D vapors during application and for approximately a 2–week period as some of the applied material offgasses following application. 1,3-D is classified as Toxicity Category II (moderately toxic) for oral toxicity and primary eye irritation and Toxicity Category III (low toxicity) for dermal irritation. There are two degradates of toxicological concern, 3-chloroallyl alcohol and 3-chloroacrylic acid.

A. Legal Background

In order to obtain a registration for a pesticide under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA, 7 U.S.C. 136 et seq., as amended by the Food Quality Protection Act of 1996, Public Law 104–170), an applicant must demonstrate that the pesticide will not cause ''unreasonable adverse affects on the environment'' when used according to label directions [FIFRA section 3(c)(5)]. The term unreasonable adverse effects on the environment means (1) ''any unreasonable risk to humans or the environment, taking into account the economic, social and environmental costs and benefits of the use of any pesticide'' [FIFRA section 2(bb)] or (2) ''a human dietary risk from residues that results from use of a pesticide in or on any food inconsistent with the standard under section 408 of the Federal Food, Drug and Cosmetic Act'' (21 U.S.C. 346a).

Tolerances, or the establishment of maximum permissible levels of pesticides in foods, are required when a pesticide or its identifiable degradates or metabolites are expected to be present in food. The Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 301 et seq., as amended by the Food Quality Protection Act (FQPA) of 1996, (Public Law 104–170), authorizes EPA to establish such tolerances (21 U.S.C. 346(a)). Without such a tolerance or an exemption from a tolerance, a food containing a pesticide residue is ''adulterated'' under section 402 of the FFDCA and may not be legally moved in interstate commerce (21 U.S.C. 342).

In determining a pesticide's safety for establishing a tolerance or an exemption from the requirement of a tolerance, the FFDCA also requires that EPA examine aggregate exposures from all sources of pesticide residues, whether infants and children have heightened susceptibility to pesticide residues, and whether there are cumulative effects of pesticides and other compounds with a common mechanism of toxicity (21 U.S.C. 346a).

Certain pesticides are classified as non-food use when no residues are expected to occur in crops from pesticide treatment. This class of pesticides includes several soil fumigants which degrade in the soil to compounds of non-toxicological concern, and thus are not available for uptake by plants. Non-food use pesticides do not require a tolerance or an exemption from a tolerance.

Under the registration requirements of FIFRA, the burden of proving that a pesticide satisfies the standard for registration is on the proponent(s) of registration and continues as long as the registration remains in effect. Under FIFRA section 6, the Administrator may cancel the registration of a pesticide or require modification of the terms and conditions of a registration if the Administrator determines that the pesticide product causes unreasonable adverse effects to man or the environment. EPA created the Special Review process to provide a public procedure to gather and evaluate information about the risks and benefits of uses that exceed EPA's risk criteria.

The Act also provides that all pesticides registered prior to November 1, 1984 must be reregistered. Congress amended FIFRA to include reregistration for older pesticides because of advances in scientific knowledge and testing capabilities not available when many pesticides were first registered.

The Special Review risk criteria are set out in the regulations at 40 CFR part 154. When EPA believes that a pesticide has met such criteria, a notice announcing the initiation of the Special Review is published in the **Federal Register**. After the Notice of Special Review is issued, registrants and other interested persons are invited to review

the data and risk assessments upon which EPA's determination is based and to submit data and information to rebut EPA's conclusions. In addition to submitting rebuttal evidence, commenters may submit relevant information to support EPA's initial conclusions or to aid in the determination of whether the economic, social and environmental benefits of the use of the pesticide outweigh the risks. After reviewing the comments, EPA makes a preliminary decision of the future status on the pesticide's registration.

Typically, a Special Review is concluded in one of three ways. If information is submitted which successfully rebuts EPA's risk case, the Agency may propose no changes to the terms and conditions of a pesticide's registrations. Secondly, EPA may propose changes to the terms and conditions of registration such that the proposed measures reduce risk(s) to a point where the benefits of the pesticide's use(s) outweigh the risk concerns. Such changes might include additional protective clothing, lower application rates or engineering controls.

However, EPA may determine that no changes in the terms and conditions of a registration will adequately assure that use of the pesticide will not cause any unreasonable adverse effects. If EPA makes such a determination, it may seek cancellation, suspension, or change in classification of the pesticide's registration. Any final decision on a pesticide's registration through the Special Review process is set forth in a Notice of Final Determination issued in accordance with 40 CFR 154.33.

B. Regulatory Background

1,3-D was placed into Special Review in 1986 (51 FR 36160, October 8, 1986) based on carcinogenicity concerns. At that time, EPA focused on inhalation exposure to workers who load and apply 1,3-D, as well as to workers who enter fields shortly after 1,3-D application. EPA also noted risk concerns for potential dietary exposures through food crops and ground water contamination with 1,3-D or its contaminant 1,2-dichloropropane (1,2-D). The focus of the Special Review was to gather data to better define 1,3-D's toxicity, environmental fate and factors which most influence exposures and to seek ways to reduce those exposures.

In 1986, EPA also issued the Registration Standard for 1,3-D (Guidance for the Reregistration of Pesticide Products Containing 1,3-Dichloropropene, USEPA, September 18, 1986). This standard outlined studies required to fill data gaps and maintain the 1,3-D registration. Many of the data gaps involved residue chemistry and environmental fate, which were needed to investigate the Special Review concerns for worker, dietary and ground water risks. Most studies in the 1986 Registration Standard were scheduled for completion within 2 years.

In 1990, EPA notified Dow AgroSciences (then DowElanco) of its concerns regarding the many delays in obtaining the studies required in the 1986 Registration Standard, namely for the residue chemistry and several of the retrospective ground water studies. Dow AgroSciences stated that the delays were due to difficulties in obtaining radiolabeled 1,3-D and the unexpected collapse of testing systems in one of the ground water studies. EPA established a new 2–year schedule for these data. Also in 1990, California suspended 1,3-D use permits because unexpectedly high levels of the fumigant were found during air monitoring required under California law. California regulates the use of certain pesticides by permits, which are issued annually and which specify use conditions such as the application rates, location and crops [Ref. 1]. Since 1,3-D use patterns in California were unique to the state, EPA initiated a review of use and exposure scenarios throughout the United States. EPA issued a Data Call-In (DCI) in 1991 for information on exposure, usage and product performance by state and by crop.

In 1990, Title VI of the Clean Air Act was amended to include regulation of chemicals which deplete stratospheric ozone. Under the amendments, EPA's Office of Air and Radiation originally proposed to phase-out use of methyl bromide by 2001 due to its potential to deplete stratospheric ozone (56 FR 49548, September 30, 1991). Because the 1,3-D Special Review considered methyl bromide to be a major alternative to 1,3-D, EPA looked more closely at the risks and benefits of all the remaining soil fumigants and contact nematicides. Specifically, EPA looked at the potential increase in benefits and risks associated with 1,3-D use in light of the scheduled phase-out of methyl bromide. The phase out was extended to 2005 under legislation passed in 1999. For more information on the methyl bromide phase out, refer to http://www.epa.gov/docs/ozone/mbr/mbrqa.html.

EPA contacted Dow AgroSciences in 1992 when the additional residue chemistry and ground water studies were not submitted according to the revised schedule. EPA also sought measures to reduce inhalation exposures, since EPA's assessments based on the incomplete data sets yielded risk estimates for workers and residents who live near treated fields that exceeded those EPA generally considers to be acceptable. In order to maintain 1,3-D registrations, the registrant agreed to set a strict timetable for completing data submissions, to develop new exposure data, and to add engineering controls and additional personal protective gear for workers to all 1,3-D labels [Ref. 2].

EPA also raised concerns about the results of the retrospective ground water studies. While results from North Carolina and California were acceptable, unexpectedly high levels from the Nebraska site, and the lack of results from Florida required attention. Since Dow AgroSciences had already approached Florida with plans to expand use as a methyl bromide alternative, EPA and Florida developed a joint schedule to oversee the study. EPA believed that the high levels in Nebraska were linked to cold temperatures, and required a prospective ground water study in Wisconsin to determine whether 1,3-D can be safely used in cold climates.

In 1995, Dow AgroSciences and EPA met a second time to review the data that had been collected, as well as California's decision to allow limited re-introduction of 1,3-D use [Ref. 3]. On January 19, 1996, Dow AgroSciences requested changes to their Telone labels to incorporate mitigation requirements and also included a time table for submitting interim and final studies for ground water monitoring taking place in Florida and Wisconsin [Ref. 4].

In 1997 and 1998, the results of the ground water studies showed levels of 1,3-D in ground water which were high enough to warrant additional mitigation measures. On September 30, 1998, Dow AgroSciences requested a third modification of their Telone labels to include measures to mitigate potential exposures through contaminated ground water (see Table 1 below) (Ref. 5). This label modification was included as part of the reregistration eligibility determination for 1,3-D. Dow AgroSciences also has agreed to conduct additional studies on the alcohol and acid degradates of toxicological concern and additional environmental fate studies. In addition, Dow AgroSciences agreed to conduct a tap water monitoring study to assess 1,3-D and degradate levels in water used for drinking. Should residues of 1,3-D and/or the alcohol or acid degradates be detected at levels exceeding the Office of Water health advisory of 0.2 parts per billion (ppb), Dow AgroSciences has

agreed to implement label use restrictions on further applications in the vulnerable use areas before the next use season commences. Label changes may include restrictions based on depth to ground water or soil type characteristics. Table 1 outlines all of the requirements which now appear on the new 1,3-D labels (effective August 1, 1999) as well as measures adopted earlier.

TABLE 1.— SUMMARY OF REQUIREMENTS ON 1,3-D LABELS

Regulatory Action (date when measures took effect)	Label Requirements
Registration Standard (1986) (effective 1987).	Precautionary statements; Cancer hazard warning; Classification change to ''Restricted Use'' pesticide; Reentry increased to 72 hours; Clothing for applicators and handlers (coveralls, chemical-resistant gloves and boots, liquid-proof hat).
1992 Interim Risk Mitigation (effective 1992/1993).	Ground water advisory; Lowered maximum rates; Deletion of selected use sites; Revised respirator requirements; Closed loading requirements; Technology to minimize 1,3-D spillage during application.
Worker Protection Standard (August 1992, see 57 FR 38102).	Coveralls over short-sleeved shirt and short pants; Chemical-resistant gloves and footwear; Chemical-resistant apron (for direct handlers).
1995 Risk Mitigation (effective August 1996).	A respirator requirement for all 1,3-D handlers; Restricted entry increased to 5 days; Prohibition of use within 300 feet of occupied structures; Soil moisture and soil sealing requirements; Modified application techniques; Lower maximum use rates.
1998 Risk Mitigation (effective August 1999).	100' buffer between drinking water wells and treated fields; prohibition in areas overlying karst geology; prohibition of use in ND, SD, MN, NY, ME, NH, VT, MA, UT, MT, WI where ground water is less than 50 feet from the surface and soils are classified as hydrologic type ''A.''

Based on the submission of label changes and a completed data base showing that 1,3-D can be used without unreasonable adverse effects to humans or the environment, EPA has found all uses of 1,3-D eligible for reregistration. The Reregistration Eligibility Decision (RED) document is contained in the 1,3-D docket (the location is listed under ''ADDRESSES'' in this Notice), or can also be accessed from the Internet at http://www.epa.gov/REDs for case 0328. Please refer to the 1,3-D RED for a more detailed discussion of the data summarized in this Notice.

C. Summary of EPA's Proposed Action

EPA has determined that the benefits associated with the continued use of 1,3-D under the recently revised terms and conditions of 1,3-D's registration outweigh the risks. Thus, EPA is proposing to terminate the Special Review of 1,3-D.

III. Summary of Hazard Assessment

A. Short and Intermediate Term Toxicity

The acute toxicity values and categories for 1,3-D are summarized in Table 2 below:

TABLE 2.— ACUTE TOXICITY STUDY RESULTS FOR 1,3-D

Study Type	Results	Toxicity Category
Acute Oral ...	LD_{50} = 300 mg/kg (M), 224 mg/kg (F)	II
Acute Dermal - Rabbit.	LD_{50} = 333 mg/kg	II
Acute Inhalation.	LC_{50} = 3.88 mg/L (M), 4.1 mg/L(F)	IV
Primary Eye Irritation.	Intermediate irritant	II
Primary Skin Irritation.	Slight irritant	III
Dermal Sensitization.	Sensitizer	.....
Acute Neurotoxicity.	None required	.....

EPA has placed 1,3-D in Toxicity Category II (moderately toxic, the second highest toxicity classification out of four levels). EPA has reviewed the available toxicological data for 1,3-D and concluded that the data do not indicate any evidence of significant oral or inhalation toxicity from a single exposure event that may occur with labeled uses.

EPA has established an intermediate-term endpoint based on results from a 2–year combined chronic/carcinogenic inhalation study in rats. Fischer 344 rats (50/sex/group plus 10/sex/group to 6– and 12–month exposure groups) were exposed by whole-body inhalation to Telone II (92.1% active ingredient (a.i.)) at aerosol concentrations of 0, 5, 20 or 60 parts per million (ppm) (equivalent to approximately 0, 0.023, 0.091 or 0.272 mg/L), 6 hours/day, 5 days/week for a total of 509 days over a 2–year period. There was no effect of exposure to 1,3-D on the survival of males or females. Slight (approximately 5% in 60 ppm males and females, as well as 3% in 20 ppm males) decreases in body weight gains were observed (statistically significant, $p<0.05$) but generally only during the first year of the study. The olfactory region of the nasal cavity appeared to be the target tissue as determined by histopathological examination. Males and females having been exposed to 60 ppm (no evidence reported at lower concentrations of 20 or 5 ppm) showed decreased thickness and erosions of the epithelium as well as minimal submucosal fibrosis. For chronic toxicity, the No-Observed Adverse Effect Level (NOAEL) was 20 ppm (0.091 mg/L) and the Lowest Observed Adverse Effect Level (LOAEL) was 60 ppm (0.272 mg/L) based on histopathological changes in nasal tissue as well as the suggestion of decrease in body weight gain compared with controls during the first year of the study.

B. Carcinogenicity

EPA initiated the Special Review of 1,3-D based on evidence that 1,3-D induced cancer in rats and mice exposed to 1,3-D. The potential for human carcinogenicity is based on inhalation exposures for workers handling the fumigant and for area residents who may be exposed to air borne levels of 1,3-D and oral exposures to levels in contaminated ground water.

1. *Oral studies*. In 1985, the National Toxicology Program (NTP) tested the chronic toxicity and carcinogenic potential of 1,3-D (Telone II - 89% 1,3-D, 6% inert ingredients, 1% epichlorohydrin) in F344 rats and B6C3F1 mice [Ref. 6].

a. *Rat Feeding Study by Gavage*. Male and female F344 rats received oral administration by gavage (feeding tube) of 1,3-D in corn oil at 0, 25, or 50 mg/kg/day, 3 days per week, for 104 weeks. A total of 77 rats per sex were used for each dose group, including those sacrificed for examination during the course of testing. Statistically significant increases in the incidence of the following tumors were observed at the highest dose tested (HDT) by pairwise comparison with controls:

i. Forestomach squamous cell papillomas in males and females.

ii. Combined forestomach squamous cell papillomas and carcinomas combined in males.

iii. Liver neoplastic nodules in males and combined neoplastic nodules and hepatocellular carcinomas in males.

The increased incidence of forestomach tumors was accompanied

by a statistically significant positive trend for forestomach basal cell hyperplasia in male and female rats of both treated groups (25 and 50 mg/kg). There were also positive trends for other tumors in rats (i.e. in females, mammary gland adenomas or fibromas and thyroid gland follicular cell adenomas or carcinomas; in males, adrenal gland pheochromocytomas). The highest dose tested in rats (50 mg/kg) appeared to be adequate for carcinogenicity testing.

b. *Mouse Feeding Study by Gavage*. In the mouse study, groups of 50 mice/sex were fed Telone II in corn oil (with 1% epichlorohydrin as a stabilizer) through a gavage feeding tube at 0, 50, or 100 mg/kg, 3 days per week for a total of 104 weeks. The results of the study were confounded by an excessive mortality in control males (those not receiving 1,3-D) from myocarditis. The survival of female mice was lower in the high dose group than in the other dose level groups (46/50, 45/50, 36/50 for control, low dose and high dose respectively). Significantly elevated incidence of the following tumors were observed either at the HDT or at both dose levels:

i. Forestomach squamous cell papillomas or papillomas and carcinomas combined in males and females, and squamous cell carcinomas in females.

ii. Urinary bladder transitional cell carcinomas in males and females.

iii. Lung adenomas or adenomas and carcinomas combined in males and females.

Several deficiencies were noted in the mouse study, including excessive mortality in control males and inadequate randomization procedures at the study initiation. The highest dose tested appears to have been excessive for testing. While this study was not used for quantitatively estimating 1,3-D's carcinogenic potential, the Agency has included the stomach, bladder and lung effects in its weight-of-the-evidence findings (see Unit III.D of this document.).

c. *Rat study by microencapsulation*. In 1992, the registrant conducted a second feeding study using time-released (microencapsulated) doses of 1,3-D in food since the stomach tumors seen in the NTP study occurred in the area where the feeding tube was inserted. In addition, the NTP study results may have been confounded by the presence of a stabilizer, epichlorohydrin, which is a known carcinogen.

Charles River Fischer 344 (''Fischer 344'') rats (60/sex/dose) were fed doses of 0, 2.5, 12.5, and 25 mg/kg/day for 2 years, with an examination of one group made after 1 year. Body weight gains were decreased for males and females at the middle and high doses compared to controls. There was an increase in liver masses/nodules in males only at the 12.5 and 25 mg/kg doses. The NOAEL was 2.5 mg/kg. There was an increased incidence of basal cell hyperplasia of the nonglandular mucosa of the stomach of both sexes at the 12 and 24 month sacrifice at the middle and high doses. The incidence of primary hepatocellular adenomas in male rats exceeded that in the control group at the middle and high doses tested. The incidence of hepatocellular ademonas in female rats showed an increase over the control only at the high dose. The highest dose tested appeared adequate for carcinogenicity testing [Ref. 7]. EPA used the test results of this study to confirm the carcinogenicity finding of the earlier study in rats. The results of this study were also used to develop the chronic non-cancer Reference Dose.

d. *Mouse study by microencapsulation*. Male and female $B_6C_3F_1$ mice (50/sex/dose) were fed microencapsulated 1,3-D at levels of 0, 2.5, 25 or 50 mg/kg/day for 2 years, with an examination of 10 mice/sex/dose made after 1 year. As seen in the rat study, body weight gains were lower in both sexes at the middle and high doses compared to controls. In addition, hepatocytes of the high dose males were decreased in size at the 12 and 24 month sacrifice. While liver effects were seen, there was no treatment-related incidence of tumors observed in mice ingesting microencapsulated 1,3-D [Ref. 8]. EPA notes that the negative cancer findings do not affect the Agency's position on the carcinogenicity of 1,3-D due to the results of the rat study.

2. *Inhalation studies*. Because 1,3-D is a volatile compound which can move up and into the atmosphere after application, EPA also required studies on the potential carcinogenicity of 1,3-D via the inhalation route of exposure.

a. *Rat study*. In the rat study, 50/sex/group were exposed to 0, 5, 20 or 60 ppm 1,3-D for 6 hours/day, 5 days/week, for approximately 2 years. Ancillary groups of rats (10/sex/group) were similarly exposed for 6 or 12 months. Clinical signs of toxicity were not observed and no significant differences in survival were found in any of the test groups. No significant increase in treatment-related incidence of tumors in rats was observed [Ref. 9].

b. *Mouse study*. The mouse study followed the same study design as the rat study (50 mice/sex/group dosed at 0, 5, 20, or 60 ppm, 6 hours/day, 5 days/week for approximately 2 years; 2 groups of mice to be sacrificed and studied at the 6 month and 1 year mark of the study). In male mice at the 2–year sacrifice, a statistically significant increase in the incidence of bronchioloalveolar adenoma (a benign lung tumor) was found at the highest dose tested (HTD) (60 ppm) by pairwise comparison with controls (9/50, 6/50, 13/50, and 22/50 for 0, 5, 20, and 60 ppm respectively). For controls (0 ppm) the historical incidence for bronchioloalveolar adenoma is in the 7-32% range; this includes a 20% control incidence from another 2–year inhalation study. Additionally, male mice had a significant difference in lacrimal gland cystadenomas in the pair-wise comparison of control and the 20 ppm dose group. No tumors were seen in treated female mice. Although a hyperplastic response was seen in the urinary bladders of both male and female mice, no tumorigenic response was found [Ref. 10].

3. *Dermal studies*. EPA also has studies that tested the potential carcinogenicity of 1,3-D through short-term dermal exposure. Van Duuren et al., (1979) administered subcutaneous injections of 1,3-D weekly to 30 female HA:ICR mice at a dose of 3 mg/mouse. The author noted a positive finding of fibrosarcomas in 6 of the 30 mice after 538 days. No tumors developed in untreated or vehicle-treated animals (i.e. treated with the serum minus the compound being tested).

The same study also investigated the tumor-initiating potential of 1,3-D when applied to the skin of female HA:ICR mice (30 animals). Mice received 1,3-D in 0.2 mL acetone as the initiator at a single dermal dose of 122 mg, followed by promotion with phorbal myristate acetate (5 μg) in acetone 3 times/week for 440–594 days. No significant differences in tumor incidence were found between the treated and control animals. Additionally, when 1,3-D was tested for carcinogenic potential following repeated dermal administration with 122 mg. 1,3-D in 0.2 mL in 0.2 acetone, 3 times/week for 440–594 days, only 1/30 treated animals had papilloma and carcinoma of the skin; the authors noted statistical significance was not attained. None of the control animals developed any skin tumors [Ref. 11]. EPA did not consider this study in its consideration of 1,3-D's carcinogenicity since the authors' conclusions and statistical tests used could not be confirmed.

4. *Structure-Activity Relationships*. 1,3-D bears a structural resemblance to several short chain halogenated hydrocarbon compounds that are known human and/or animal carcinogens, namely vinyl chloride and epichlorohydrin. There is no

information, however, that establishes a common mode of carcinogenicity between these chemicals and 1,3-D.

C. Mutagenicity

A series of mutagenicity studies has been performed which show that 1,3-D has some mutagenic activity. This activity would also provide support for a carcinogenicity concern. 1,3-D produced gene mutations in bacterial and mammalian test systems *in vitro* but did not produce structural chromosomal aberrations in mammalian test systems. 1,3-D is also a germ cell mutagen in *Drosophila*. The *Drosophila* result suggests an interaction with germ cells in an eukaryotic organism. There are studies in the open literature that show the *in vivo* mouse liver conversion of 1,3-D to mutagenic cis and trans epoxides, the *in vitro* formation of four DNA adducts when 1,3-D epoxides are reacted with 2′-deoxygenase and the *in vivo* formation of DNA lesions in the stomach, colon, liver, kidneys, bladder, lungs, brain and bone marrow.

For the 1,3-D reregistration and Special Review, Dow AgroSciences submitted information to support regulation of 1,3-D as a non-linear carcinogen (i.e., that there is no risk associated with exposure below a certain dose) because 1.3-D is not mutagenic. EPA has reviewed the information and determined that the weight-of-the-evidence shows 1,3-D is mutagenic. [Ref. 12].

In addition, Dow AgroSciences is performing the Ames assay, mouse lymphoma and mouse mocronucleus study on the alcohol and acid degradates to test EPA's assumption that the degradates exhibit the same mutagenicity as the parent.

D. Human Incidents Data

The Agency is aware of several reports in the open literature describing adverse effects related to accidental 1,3-D exposure. In 1973, nine firemen were exposed during a clean-up operation in California after a 1,3-D transport tank overturned [Ref. 13]. Reports show two of the nine men exposed were treated for neck pain, nausea and breathing difficulty following exposure. Follow-up revealed that both men died from hematological malignancies within 7 years of exposure. In a separate case in the same report, a farmer was repeatedly sprayed in the face with 1,3-D through a leaky hose. The man first went to the doctor in 1975, when he was found to have mucosal lesions in his ear and pharynx, as well as symptoms of fatigue. He also required transfusions to correct low red and white blood cell counts. He returned to field work in 1976, where he was again sprayed with 1,3-D. The next year, fatigue became more severe and his gums began bleeding. Red and white cell counts were diminished and the patient was diagnosed with acute myelomonocytic leukemia. The patient died within 5 weeks of admission.

In another report of 1,3-D exposure [Ref. 14], a worker drank a clear fluid which he thought was water from a container. The first signs of injury were acute gastrointestinal distress, sweating, tachycardia, tachypnoea and lividity in the lower legs. His condition worsened within 9 hours; blood abnormalities did not respond to numerous treatments. The patient died 38 hours after admission; the autopsy revealed multiple organ failure and extensive damage to the respiratory tract and liver. While this case involves an acute poisoning, rather than a chronic effect, EPA has concluded that this report supports concern for 1,3-D toxicity to the human hematologic system as was seen in the other cases cited above. It should be noted that these accidental exposures to 1,3-D are less likely under the current labels because of strict requirements for closed loading, check valves, and protective equipment.

While these reports alone do not provide an adequate basis for making a determination of human carcinogenicity (i.e. that 1,3-D is a Group A, human, carcinogen), they provide evidence to support EPA's concerns regarding the target organs of 1,3-D's effects in humans (hematopoietic system, lungs, liver) and its potential to induce cancer.

E. Weight-of-the-Evidence and Carcinogenicity Summary

The EPA Cancer Peer Review Committee (CPRC) met in 1989 to consider all the data relevant to developing a position on 1,3-D's carcinogenicity. The Committee based its determination on the following:

1. The CPRC looked at the original NTP oral carcinogenicity studies to determine whether the epichlorohydrin stabilizer was the carcinogenic agent. The CPRC concluded that the tumors could not solely be attributed to epichlorohydrin because tumors were seen at sites other than the forestomach (i.e. liver, mammary gland and thyroid) and the dose of epichlorohydrin was far below that associated with forestomach tumors in gavage and drinking water carcinogenicity studies. A comparison between the mutagenic activities of 1,3-D and epichlorohydrin showed that even if epichlorohydrin did contribute some activity to the 1,3-D preparation, its relative contribution would be very small because epichlorohydrin constituted a small percent of the total test material. Epichlorohydrin by itself did not appear to induce as large a mutagenic response as 1,3-D on an equimolar basis based on studies administering epichlorohydrin alone.

2. 1,3-D, when administered by oral gavage to Fischer 344 rats, was associated with an increase in (i) forestomach tumors in both sexes; (ii) liver tumors in males; and (iii) positive trends for other tumor types in mammary and thyroid glands.

3. 1,3-D, when administered by oral gavage to $B_6C_3F_1$ mice, was associated with an increase in (i) forestomach tumors; (ii) urinary bladder tumors and cell changes; and (iii) lung adenomas (benign lung tumors) in both sexes at both dose levels and lung adenomas and carcinomas combined in males at both dose levels.

4. No compound-related increase in tumors was observed in inhalation studies in Fischer 344 rats. However, the dose levels used were not considered to be high enough to fully assess the carcinogenic potential of 1,3-D.

5. 1,3-D, when administered by inhalation to $B_6C_3F_1$ mice, was associated with an increase in bronchioloalveolar adenomas in males at the highest dose tested. Cellular changes in the urinary bladder, nasal passages and non-glandular stomach were noted. Based on toxicity parameters, the data suggest that higher dosing could have been utilized in this study.

6. The CPRC concluded that the benign lung tumors observed in mice after inhalation were biologically significant, because tumor induction was dose-dependent, tumor incidence was outside the range of historical controls, and the tumor type was also seen in the mouse oral study.

7. EPA has concluded that, based on available evidence in bacterial, *Drosophila* and mammalian cell mutagenicity studies, 1,3-D has mutagenic capability.

8. 1,3-D bears a structural resemblance to several short chain halogenated hydrocarbons that are known carcinogens.

9. Confidence in the compound-related induction of tumors was strengthened by the observation of site concordance for neoplastic and non-neoplastic effects seen for the two routes (oral and dermal) of 1,3-D administration [Ref. 15].

Based on the above data (evidence of carcinogenicity in two rodent species via two different routes of exposure), EPA has classified 1,3-D as a Group B_2, or probable human, carcinogen.

For the 1,3-D reregistration and Special Review, Dow AgroSciences submitted information to support regulation of 1,3-D as a non-linear carcinogen (i.e., that there is not risk associated with exposure below a certain dose). The Office of Pesticide Programs has reviewed the information and determined that the evidence on 1,3-D's mutagenicity does not support Dow AgroScience's claim that 1,3-D is a candidate for regulation as a non-linear carcinogen [Ref. 12]. Thus, EPA will continue to regulate 1,3-D as a B_2 carcinogen under a linear approach.

F. Dose-Response Assessment for 1,3-D

By using data from carcinogenicity studies, EPA quantifies the carcinogenic potential of chemicals based on a dose-response relationship. This measure is known as the carcinogenic potency factor, or the Q^1*. For 1,3-D, EPA has calculated two carcinogenic potency factors: one for the oral route and the other for inhalation. The Q_1* for the oral route was presented in the 1986 Notice of Special Review as 1.75×10^{-1} (mg/kg/day)$^{-1}$, based on the combined tumors (either (i) adrenal and thyroid, (ii) forestomach or (iii) liver tumors) in the oral gavage rat study using the Multistage model. In 1994, Office of Pesticide Programs revised the Q_1* for the oral route to 1.22×10^{-1} based on a scaling factor of 3/4 instead of 2/3 to extrapolate data from humans to animals. The Q_1* for the inhalation route using the 3/4 scaling factor is 5.33×10^{-2} (mg/kg/day)$^{-1}$, based on the lung bronchioalveolar tumor rates in male mice [Ref. 16].

G. Toxicity and Carcinogenicity of 1,2-Dichloropropane

The 1986 Notice initiating the Special Review for 1,3-D mentioned concerns for the contaminant 1,2-dichloropropane (1,2-D). In the mid 1980's, 1,2-D was registered as an active ingredient and was present in 1,3-D formulations at levels up to 5%. All 1,2-D pesticide registrations were canceled as of 1987 and 1,2-D levels in the Telone II formulation (which is also used by reformulators) have been reduced to less than 0.1% for products sold after August 1, 1999. Nonetheless, EPA has been tracking 1,2-D levels in ground water studies and reviews due to 1,2-D's persistence.

EPA has not conducted a formal evaluation of the toxicology database for 1,2-D at this time because 1,2-D is no longer registered as a pesticide. However, 1,2-D has been evaluated by the Office of Research and Development (ORD) to support development of the Drinking Water Criteria Document by the Office of Water (USEPA 1987). ORD evaluated the limited available database for 1,2-D and concluded that the liver was the principal target organ of toxicity. ORD also found effects from acute exposures; the effects were seen in the lungs, liver, kidneys, central nervous system and eyes. A more detailed description is on EPA's IRIS data base at http://www.epa.gov/ordntrnt/ORD/dbases/iris/index.html.

1,2-D has been classified as a Group B_2, probable human carcinogen, with a Q_1* of 3.69×10^{-2}(mg/kg/day)$^{-1}$ based on the statistically significant increased incidence of hepatocellular adenomas and carcinomas in male and female $B_6C_3F_1$ mice. In addition, a dose-related trend in mammary adenocarcinomas was noted in female Fischer 344 rats. This is considered significant because Fischer 344 rats have a relatively low background incidence of these tumors (56 FR 3540, January 30, 1991). In addition, 1,2-D was mutagenic in the *Salmonella* and in *Aspergillus nidulans*. 1,2-D also induced sister chromatid exchange and chromosome aberrations in Chinese hamster ovary cells.

The Agency has not cumulated 1,3-D risks with the impurity 1,2-D or other chemicals since no determination has been made that these chemicals share a common mechanism of toxicity.

IV. Summary of Exposure

A. Dietary Exposure

1. *Food sources.* The 1986 Registration Standard concluded that the characteristics of 1,3-D were not well enough understood to ascertain whether residues might be expected in raw agricultural commodities, and therefore metabolism data were required for reregistration.

In 1992, Dow AgroSciences submitted metabolism studies demonstrating that 1,3-D is extensively metabolized and incorporated into natural components such as sugars, amino acids and fatty acids. EPA determined that residues of 1,3-D and its degradates of toxicological concern are not expected in foods from pre-plant fumigant uses of 1,3-D. Thus, EPA has determined that the pre-plant fumigation uses of 1,3-D are non-food uses and no tolerances or exemptions from the requirement for a tolerance are required. [Ref. 17].

2. *Drinking water sources.* Although EPA believes there are no residues of 1,3-D in foods grown on 1,3-D treated soils, studies show that 1,3-D can contaminate ground water, including that which is used for drinking water. While 1,3-D was not specifically placed into Special Review because of ground water concerns, EPA noted that 1,3-D could reach ground water since monitoring had yielded detections of 1,3-D and 1,2-D. EPA's Office of Water (OW) has not established a Maximum Contaminant Level (MCL) set for 1,3-D. For carcinogens, OW typically sets a Maximum Contaminant Level Goal (MCLG) at zero. In 1987, OW set the Health Advisory level of 0.2 ppb, which is the daily level of consumption over a lifetime associated with a 1×10^{-6} cancer risk. Health Advisories are not enforceable standards, but rather are advisory in nature.

The MCL for 1,2-D is 0.005 mg/L (5 µg/L or 5 ppb). For 1,2-D, EPA's Office of Water has a children's 10-day Health Advisory of 0.09 mg/L (90 µg/L or 90 ppb).

1,3-D is considered highly mobile and is more persistent when 1,3-D enters ground water in colder climates. 1,3-D has been detected and its presence confirmed in ground water in New York, Florida, Nebraska, Washington state and the Netherlands under normal field use. In 1991, the General Accounting Office (GAO) issued a report which listed detections of 1,3-D in seven states. This list also included detections of the impurity 1,2-D [Ref. 18].

The 1986 Registration Standard required that retrospective ground water monitoring studies be conducted at five sites. From the study results, no 1,3-D was found at the California, North Carolina or Washington state sites. Retrospective ground water monitoring studies require sampling in known use areas for a pesticide, but do not require extensive information on past use, well integrity or other historical information to help characterize any detections. A sinkhole collapsed and interfered with obtaining results at the Florida site. 1,3-D residues were found at the Nebraska site, leading EPA to suspect that the increased persistence of 1,3-D under colder conditions had contributed to 1,3-D's presence in ground water there.

In 1995 and 1996, Dow AgroSciences initiated prospective ground water studies in Wisconsin and Florida. Prospective studies are conducted under predetermined conditions in areas of no known prior use, thereby reducing the chance that prior use or changes in use practices could interfere with study results. The Wisconsin site was chosen to better define 1,3-D's fate in a cold climate. Dow AgroSciences initiated the Florida study to determine if 1,3-D products could be used without adverse effects to ground water.

At the Wisconsin study site, 1,3-D, its degradates and 1,2-D were found in both on-site wells and in one off-site monitoring well at concentrations well

above levels considered acceptable. These levels were detected for more than a year after the 1,3-D application occurred (see Unit V.B.1.c. for more information on concentrations associated with unacceptable risks). Cancer risks associated with prolonged exposures to the detected levels were unacceptably high for all age groups, as were chronic non-cancer risks for infants and children. In the Wisconsin study, on-site wells yielded concentrations of 1,3-D as high as 579 ppb. Concentrations of 1,3-D in off-site wells were as high as 84 ppb [Ref. 19].

In the Florida study, 1,3-D, its degradates and 1,2-D were also found, though at lower levels than those seen in the Wisconsin study. In Florida, residents tap both surficial aquifers and deeper ground water for drinking water and thus the study was designed to look at levels 10 feet and 70 feet below the surface. There were also a limited number of off-site wells to look at downgradient concentrations from a single application. Time-weighted average (TWA) concentrations of 1,3-D plus its degradates in the on-site wells were 1.15 ppb in 10 feet wells and 0.17 ppb in the 70 feet wells (note that time-weighted averages are used to describe the exposures to pesticides which pose chronic risks, while peak levels are used to describe exposures to pesticides which pose acute risks). TWA concentrations of 1,3-D plus degradates measured in wells located 100 feet down-gradient from the treated field were 0.074 ppb. Levels of 1,3-D plus its degradates did not persist beyond a year after application [Ref. 20].

EPA also reviewed the U.S. Geological Survey's (USGS) National Water Quality Assessment (NAWQA) reports. The assessment, which is on-going, monitors both surface and ground water for pesticides, nitrates and other contaminants in the United States. Some USGS-monitored sites were located in counties that have reported the highest use rates of 1,3-D, although there was no information in the reports to directly link 1,3-D treatments with sampled wells. Moreover, the assessment did not test for 1,3-D's alcohol and acid degradates. None of the NAWQA reports released to date have shown detections of 1,3-D in ground or surface water. 1,2-D detections were widespread and thought to be related to past use of 1,2-D as a soil fumigant. Although no information in the reports directly links 1,3-D use to the monitored wells, the absence of detections suggests that 1,3-D use probably does not result in widespread aquifer contamination. For more details on the NAWQA program and 1,3-D and 1,2-D sampling, please refer to http://water.usgs.gov/lookup/get?nawqa/.

EPA used the results of the prospective ground water studies to assess exposure to 1,3-D and its degradates in drinking water because of the Agency's confidence in the high quality of the data. EPA has estimated dietary exposure to 1,3-D via drinking water using these study results and a daily water consumption value of 2 L/day for adult males and females with bodyweights of 70 kg and 60 kg, respectively, and 1 L/day consumption for infants and children with a 10 kg bodyweight. The following equation used to estimate exposure to 1,3-D through drinking water for adult males is provided as an example of how EPA calculated exposure to 1,3-D and its degradates in drinking water:

Exposure (mg/kg/day)(Adult male) = (conc'n, µg/L)(2 L/day)(0.001 mg/µg) ÷ 70 kg adult body weight

The following table 3 presents the exposure estimates for 1,3-D, its degradates and 1,2-D.

TABLE 3.— CHRONIC EXPOSURE ESTIMATES FOR 1,3-D, 1,3-D+ DEGRADATES, AND 1,2-D

(Based on Time-Weighted Average (TWA) concentrations from the Florida and Wisconsin Prospective Ground Water Studies)

Populations	Compound	Florida Prospective Study (365 days)						Wisconsin Prospective Study (after 337 days, on-site wells)	
		10-ft wells		70-ft wells		10-ft wells, 100 ft off-site		shallow aquifer (15-22 ft)	
		TWA µg/L	Exposure (mg/kg/day)	TWA µg/L	Exposure (mg/kg/day)	TWA µg/L	Exposure[1] (mg/kg/day)	TWA µg/L	Exposure (mg/kg/day)
Adult males	1,3-D	0.30	8.6×10^{-6}	0.04	1.1×10^{-6}	0.026		134	3.8×10^{-3}
Adult females			1×10^{-5}		1.3×10^{-6}				4.5×10^{-3}
Infants & Children			3×10^{-5}		4×10^{-6}				1.3×10^{-2}
Adult males	1,3-D + Degradates	1.15	3.3×10^{-5}	0.17	4.9×10^{-6}	0.074		357	1×10^{-2}
Adult females			3.8×10^{-5}		5.6×10^{-6}				1.2×10^{-2}
Infants & children			1.2×10^{-4}		1.7×10^{-5}				3.6×10^{-2}
Adult males	1,2-D	0.22	6.3×10^{-6}	0.06	1.7×10^{-6}	NA		1.69	4.9×10^{-5}
Adult females			7.3×10^{-6}		2×10^{-6}				5.6×10^{-5}
Infants & children			2.2×10^{-5}		6×10^{-6}				1.7×10^{-4}

[1] Note these wells were not used for risk assessment purposes, therefore, TWA concentration values are only presented to compare to levels found in other wells.

In summary, the prospective studies show that 1,3-D can move to ground water under use conditions allowed on 1,3-D labels. EPA believes that the conditions most likely to result in 1,3-D treatment-related ground water contamination are shallow water tables, cold temperatures and high soil permeability. 1,3-D labels have a ground

water advisory and, as of August 1, 1999, will require a 100 feet setback from drinking water wells. The labels will also prohibit use in ND, SD, MN, NY, ME, NH, VT, MA, UT, MT, WI where ground water is less than 50 feet from the surface and soils are classified as hydrologic type ''A,'' and in areas overlying karst geology.

B. Non-Dietary Exposure and Mitigation

Dow AgroSciences conducted several studies to assess both worker and residential exposures to air borne concentrations of 1,3-D. The Agency and Dow AgroSciences designed special studies not only to measure air levels following fumigation, but also to determine which measures are best suited to mitigate exposures. This section describes those studies, their limitations, and how EPA reached regulatory decisions based on the study results [Ref. 21].

1. *Worker and area resident exposure studies—* a. *Exposure studies in the Notice of Special Review.* In the 1986 Notice of Special Review, the non-dietary worker exposure assessment was based on nine studies conducted in California and Florida. The excess lifetime cancer risk estimates based on these exposure studies ranged from 10^{-5} (one excess cancer death in 10,000 exposed workers over a lifetime) to 10^{-2} (one excess cancer death in 100 exposed workers over a lifetime). In the 1986 Registration Standard, EPA noted the variability in the data and risk estimates, but ascribed this to 1,3-D's high volatility and variations in crop practices. During the reregistration process, the registrant submitted environmental fate studies which showed that in controlled laboratory studies, 1,3-D behaves differently according to soil type, temperature, the amount of organic matter in the soil and other variables [Ref. 22]. There were, however, only limited data describing how 1,3-D moves in the field under actual use conditions. EPA determined that, in order to make regulatory determinations for the Special Review, study designs would have to take into account some of the environmental conditions that appeared to influence air borne concentrations under actual field conditions.

b. *Exposure studies for the PD2.* When EPA and Dow AgroSciences met in 1992 to assess the potential effectiveness of risk reduction measures, the discussions focused on the environmental factors and work practices which would likely lead to the highest exposures and how best to control exposures. The registrant agreed to take certain steps, including reducing maximum application rates, reducing high exposures to loaders during fumigant transfers, using closed systems and discontinuing the practice of continuously pumping 1,3-D when the application rig was lifted out of the ground at row turns. These exposure reduction measures were placed on 1,3-D labels in 1992 and 1993.

In addition to label changes, the meetings defined exposure study designs which would take into account the different use conditions in the United States and the effectiveness of mitigation measures (e.g., enclosed cabs, respirators, loading from 1,000 gallon bulk containers instead of 55 gallon drums). The 1,000 gallon bulk containers, also called mini-bulk or traveler systems, reduce exposures because the frequency of loading events is reduced. AgroSciences conducted air monitoring studies in three locations to measure exposures to fumigant loaders, applicators, re-entry workers and area residents.

For the three study sites, two types of sampling for worker tasks took place: 4 hour sampling to estimate full-day exposure and short term sampling. The three representative sites chosen each had different soil types, moisture conditions, organic soil content and cropping patterns.

For residential exposure estimates, data were pooled to account for random shifts in prevailing wind direction. For residents, EPA also assumed 16 hours/day spent in and around the house. EPA also assumed 1,3-D air concentrations to be the same indoors and outdoors since 1,3-D is a small, highly volatile chemical and since there are no data demonstrating any indoor/outdoor difference. Exposure estimates for residents are presented in Table 5 in Unit IV of this document, at fixed distances from a treated field.

Moses Lake, Washington. This study was conducted in October and November of 1992. 1,3-D was applied at 25 gallons per acre in loamy sand soil. The delivery system used was bulk loading with dry disconnects. Application was by the broadcast method. This type of application is crucial to root crops because the economically important part of the plant is entirely underground and is susceptible to direct nematode damage. For residential air monitoring, there were 20 monitoring locations surrounding the 20-acre treatment test site.

Buckeye, Arizona. This study was conducted in March of 1993. 1,3-D (Telone II) was applied by the row method at a rate of 12 gallons per acre. In the row method less material is used, since the fumigant is being applied to discrete rows of soil, generally for vegetable crops, cotton and tobacco. The soil was sandy loam, and bulk loading was used both with and without dry disconnects. A second study performed in Buckeye, AZ was similar to the first, except that drum loading was used. For residential air monitoring, there were 28 locations surrounding the 20–acre plot.

Hookerton, North Carolina. This study was conducted in December of 1992. Telone C-17 was used at a rate of 20 gallons per acre to a field that was sandy loam. Drum loading was used and applied by the broadcast method. For residential air monitoring, there were 20 monitoring locations surrounding the 12–acre test plot.

Ainger, North Carolina. In April of 1995, after the 1992 negotiations and data call-in, Dow AgroSciences conducted an additional worker exposure monitoring study using a new mini-bulk packaging and delivery system for 1,3-D (the ''traveler'' study). 1,3-D was applied using the row method at a rate of 10 gallons/acre to a tobacco field. The soil type was not specified.

Lifetime exposures were estimated by using information Dow AgroSciences collected on use and usage of 1,3-D. In 1991, Dow AgroSciences surveyed the 17 states where 1,3-D was used (the survey did not include California) to obtain information on use patterns around the country. Information included the crops planted on 1,3-D treated soil, the amount of 1,3-D (and its alternatives) handled, and the amount of time spent handling 1,3-D.

Exposure estimates for workers are presented in Table 4, while estimates for exposure to residents around treated fields are presented in Table 5.

TABLE 4.—1,3-D AIR CONCENTRATION MONITORING DATA FOR AGRICULTURAL WORKERS

Activity	Sample Duration	Study sites	Total reps.	Air Concentration (µg/m3)	
				Range	Mean
Loading [a]	4 hr	WA, AZ	10	177-5932	1,631
Loading [a]	task only	WA, AZ	10	526-32490	10,833
Loading [a]	task only	NC	12	52-1180	464
Application [b]	4 hr & task	WA, AZ, NC	28	43-6581	1,359

[a] With use of dry disconnects
[b] With use of end-row spill control

TABLE 5.— OFFSITE AIR MONITORING DATA USING AVERAGE CONCENTRATIONS FROM THREE STUDY SITES (AZ, NC, WA)

Distance from treated field (m)	Mean Conc. 7 day (µg/m3)	Mean conc. 15 day (µg/m3)
1600 (AZ)	3	2
1,200 (AZ)	6	4
800	11	7
500	19	10
125 Edge of buffer zone[1]	92	56
25	196	63
5	185	67
onsite	181	171

[1] Edge of buffer zone - EPA uses this distance to approximate risks at 300 feet buffer.

V. Worker and Area Resident Risk Assessment

Cancer risk is the product of exposure and cancer potency. EPA used the results of the air monitoring studies to assess inhalation exposure. EPA used the air levels at the 125 meter distance, which is used to represent the 300 foot buffer, to approximate an upper-bound worst case scenario for inhalation risk. EPA used the levels detected in the 10-foot wells from the Florida prospective ground water monitoring study as an upper-bound worst case scenario for drinking water risk. Because the new 1,3-D labels will prohibit 1,3-D use in areas similar to the Wisconsin site, those levels were not used to develop risk estimates for the general population.

A. The Cancer Potency Estimate

EPA calculates lifetime cancer risks as the product of exposure and the cancer potency estimate (Q_1*). EPA has classified 1,3-D as a Group B_2 (probable human) carcinogen based on tumor induction in rats and mice by the oral and inhalation routes of exposure. The inhalation Q_1* is 5.33×10^{-2} (mg/kg/day)$^{-1}$. For oral (water) exposures, the Q_1* is 1.22×10^{-1} (mg/kg/day)$^{-1}$.

B. The Risk Assessment

1. *Dietary risk assessment.* The dietary risk assessment for 1,3-D is based solely on drinking water exposures through contaminated ground water. Studies show that 1,3-D and its degradates of toxicological concern do not appear in foods grown on treated soils as long as 1,3-D is applied as a pre-plant soil fumigant. The assessment does not include any exposure through surface water. While models used to estimate movement of pesticides to surface water show the potential for 1,3-D movement to surface water, these models are not designed to track volatile, soil applied pesticides. EPA will review the results of a run-off study Dow AgroSciences is conducting in order to assess whether run-off to surface water is a significant source of dietary exposure.

The dietary (drinking water) risk assessment consists of exposures to 1,3-D and its two degradates of toxicological concern, 3-chloroallyl alcohol and 3-chloroacrylic acid. EPA does not have toxicity data on the degradates, and thus assumed that the degradates are of equal toxicity and carcinogenicity to 1,3-D. A separate assessment is presented based on 1,2-D levels found in the prospective studies.

a. *Acute- and intermediate-term drinking water risks.* No acute or intermediate endpoints were identified for 1,3-D exposure, and thus no acute or intermediate risk assessment was conducted.

b. *Chronic drinking water risk.* For chronic non-cancer risks, EPA determined that an oral Reference Dose (RfD) should be 0.025 mg/kg/day based on a NOAEL of 2.5 mg/kg/day from a 2-year chronic/carcinogenicity study in rats and an uncertainty factor of 100. The RfD is a level at or below which daily aggregate exposure over a lifetime is not expected to pose appreciable non-cancer chronic risk to human health; EPA generally considers exposures which occupy less than 100% of the RfD to be acceptable.

The chronic drinking water risk is calculated as a percent of the RfD taken up by drinking water. For 1,3-D, groundwater is considered to be the only source for chronic drinking water exposure to 1,3-D, and exposure includes the acid and alcohol degradates.

The following calculation was used:

% RfD = (Drinking Water Exposure, mg/kg/day) ÷ RfD of 0.025 mg/kg/day × 100%

Drinking water exposures for the U.S. population were developed using concentrations from the Florida prospective ground water monitoring study. For all population sub-groups (adult males, adult females, infants/children), the % RfD was less than 1, and therefore is considered acceptable [Ref. 23].

c. *Cancer risk estimates - drinking water.* For 1,3-D, EPA looked at aggregate risks from multiple routes of exposures (i.e., food, water, air, dermal). In order to aggregate exposures from multiple routes of exposure, EPA developed Drinking Water Levels of Comparison (DWLOC's). A DWLOC, which is not an enforceable standard, is the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from all other exposure routes. The DWLOC for 1,3-D is based on ground water levels as EPA did not have information to determine whether surface water should also be a component of the DWLOC.

For 1,3-D, EPA has calculated two DWLOC's. For residents who live near treated fields, as defined at the 300 feet buffer, the DWLOC for cancer is zero because the inhalation risk estimates were calculated to be greater than 1×10^{-6} for this population. While the cancer risk estimates at distances between 300 feet up to 800 meters are presented as greater than 1×10^{-6}, EPA believes these risks are overstated because the value of all mitigation measures has not been factored into the assessment. Thus, EPA believes the DWLOC of zero is overly conservative.

For the general population, defined as residents who live at distances greater than 300 feet from 1,3-D treated fields,

the DWLOC for cancer has been calculated to be 0.3 ppb, which is the level of daily consumption of a pesticide over a lifetime associated with a 10^{-6} risk. The DWLOC for cancer differs from OW's Health Advisory (HA) of 0.2 ppb, in part because of differing assumptions on exposure, but also because the DWLOC is based on more reliable cancer data developed after the 1987 HA had been established.

EPA compared the ground water levels of 1,3-D found in the Wisconsin and Florida study sites to the DWLOC for cancer of 0.3 ppb. In the Wisconsin study, time-weighted average levels were 357 ppb, far greater than the 0.3 ppb level considered to be acceptable. In the Florida study, time-weighted average levels from on-site wells were 1.15 ppb, which is associated with lifetime cancer risks of 4×10^{-6} [Ref. 24]. As of August 1, 1999, 1,3-D labels will require applicators to leave a 100 foot set-back from any drinking water well. Therefore the levels from on-site wells in the studies would overestimate risks at an application site. EPA did not have accurate information to develop risk estimates with the 100 foot buffer because the registrant requested the setback from drinking water wells after ground water studies were well underway. Although the information from the off-site wells is limited, EPA views these levels (27 ppb in WI, 0.074 ppb in FL) as indicative of an expected decline in residues with the well setback from a one-time application.

Although EPA is not performing a cumulative risk assessment for 1,3-D and 1,2-D, EPA developed a DWLOC for 1,2-D to compare with the levels found in the ground water studies. The oral Q_1* for 1,2-D was used to calculate a DWLOC for cancer effects, which is 1 ppb. This 1,2-D DWLOC of 1 ppb compares to 0.22 ppb found in 10' Florida wells, 0.06 ppb found in 70' Florida wells and 1.7 ppb found in the WI study. It should be noted that the new labels prohibit use of 1,3-D products in areas with conditions similar to Wisconsin. The inhalation exposure studies did not monitor for levels of 1,2-D in air. Therefore, the DWLOC only estimates oral exposures.

2. *Inhalation risk assessment*— a. *Factors that influence exposures.* Occupational and residential/bystander inhalation exposure occurs as a result of 1,3-D volatilization. 1,3-D is a volatile chemical which is applied at least 12 inches below the soil surface. The liquid 1,3-D then diffuses through the soil spaces and as much as 25% can volatilize into the atmosphere.

Volatilization can also occur during product loading; several measures have been added to 1,3-D labels to minimize leaks. 1,3-D products do not require mixing and are loaded into tanks which are attached to tractors or application rigs directly from a bulk or mini-bulk container through closed loading systems. Bulk loading from tanker trucks is the predominant practice where custom applicators are the primary 1,3-D users (e.g., the Pacific Northwest). Mini-bulk systems are portable 1,000-gallon ''traveler'' cylinders with dry disconnects to prevent 1,3-D leaks.

Variations in use patterns and application methods can affect exposures. The rate and amount of 1,3-D volatilization is affected by application method, soil sealing method, soil composition (e.g., amount of clay and organic matter), soil moisture, and a variety of other local environmental factors. Meteorological conditions, such as temperature, precipitation, wind, and atmospheric stability vary greatly from day to day and also have an effect on exposure. Studies showed that average exposures are inversely related to distance from the treated field; 1,3-D air concentrations measured 125 meters from treated fields were 45 to 72 percent lower than air concentrations measured 5 meters from treated fields [Ref. 25].

b. *Exposure estimates used for risk assessment.* EPA based its risk assessment on 1,3-D air concentrations measured from the monitoring sites in Washington, Arizona and the two sites in North Carolina (one using drum loading for residential exposure and another using mini-bulk for worker exposure). Only inhalation exposure was estimated; dermal exposure is expected to be negligible because of 1,3-D's volatility and the protective measures required on 1,3-D product labels.

Because the number of monitored replicates at each site was small (5 to 13), EPA pooled the results from different sites to obtain the largest possible sample sizes for each exposure scenario.

For intermediate-term worker exposure, 4–hour samples were used over the first 7–day period to calculate the mean air concentrations over all pooled replicates. All worker air concentration estimates were adjusted using a protection factor of 0.10 for respirators. For intermediate term risks, EPA calculates a Margin of Exposure, or MOE. The MOE is a quotient of the NOAEL divided by estimated human exposures. EPA generally regards MOE's of less than 100 to be unacceptable. For 1,3-D, the Agency chose an intermediate term NOAEL of 0.091 mg/L, derived from the 2-year combined chronic/carcinogenicity inhalation study in rats.

For intermediate-term residential/bystander exposure, a time-weighted average (TWA) air concentration was calculated for the first 8 days of exposure only (day of application and the first 7 days of a 14-day study). These are the mean 7–day air concentrations in Table 5 in Unit IV of this document, which were used to calculate intermediate term MOE's, also using the NOAEL of 0.091 mg/L.

For lifetime worker and residential/bystander exposure, the TWA air concentration was calculated for the entire sampling period for each monitoring station. This time-weighted average was the arithmetic mean of the mean daily air concentrations. For all but the on-site samples, this calculation included the air concentrations measured during the application process. This value was normalized over a 24 hour period, and incorporated into an overall 15 day TWA (the day of application plus the 14 days following). The exposure period of 15 days is used based on study results showing almost complete volatilization during the 2–week period following application.

For each distance from a treated field, the mean TWA over all four directions (N, S, E, W) was calculated for the entire monitoring period. Data for all three sites were then pooled, and an overall average for each distance was calculated for the entire data set. These values appear in Table 5 under the heading of ''Mean conc. 15 day'' air concentrations.

Exposures to agricultural handlers entering treated fields after the 5 day Re-entry Interval (REI) were calculated using the on-site air monitoring data from the residential/bystander studies. For each of the three monitored sites, the TWA 1,3-D air concentration was calculated for the period consisting of days 6-14 post-application and was adjusted by 0.10 for a respirator.

Chronic, lifetime exposures to workers and area residents were expressed as lifetime average daily dose (LADD). The LADD of 1,3-D was calculated according to the following formula:

LADD (mg/kg/day) = [(air concentration, $\mu g/m^3$)(mg/1,000 μg)(ventilation rate, m^3/hr)(hr/day) (days/yr)(1 yr/365 days)(yrs exposed/70 yrs)] ÷ 70 kg body wt

using the following values for workers and residents/bystanders:

TABLE 6.—ASSUMPTIONS USED IN ASSESSING WORKER AND RESIDENTIAL/BYSTANDER RISK

	Workers	Residents/ Bystanders
Ventilation rate.	1.74 m^3/h (light work)	0.81 m^3/h
Lifetime Exposure.	30 years, grower, 20 years, commercial	30 years
Average Lifetime.	70 years	70 years
Exposure Duration.	crop specific	16 h/day
Exposure Frequency.	crop specific	15 days/event, 1 event/yr

LADDs for commercial ''for-hire'' handlers were calculated by first estimating average daily doses (ADDs) in mg/kg/day, from the air concentrations. Information on days per year and hours per day were obtained for each crop, state by state, from Dow AgroSciences' Use, and Usage Summary Report (1991). However, for loaders, the report lists only the total hours per day spent actively engaged in loading (0.5 to 1.25 hour/day), not total hours spent on site. To estimate ADDs, the Agency therefore assumed loaders to be on site for the same number of hours per day as the applicators (5 to 10 hours/day, depending on state and crop).

LADDs for growers assumed that the majority of the work day is spent applying 1,3-D, and only as much time as is required to load the tank is spent engaged in loading. Therefore, the 4–hour samples were used in the calculation of the portion of the exposure resulting from application, and the task-specific samples were used to calculate the exposure incurred while loading (because four-hour samples were not collected for the mini-bulk study, the Agency made the assumption that, for the use of mini-bulk cylinders, the task-specific loader air concentrations are experienced for the duration of a work cycle). The loading and application exposures were then added to estimate the total exposure for these individuals. For growers, the Agency assumed that the same person conducts both loading and application of 1,3-D. Tables 7 through 9 present worker and residential/bystander risk.

TABLE 7.— 1,3-D CUSTOM HANDLER INTERMEDIATE-TERM NON-CANCER RISKS AND CANCER RISKS

Delivery Method	Example Crop	Task	Conc. µg/m^3 from Table 4	hr/d	day/yr	LADD	Cancer Risk	Int.-Term MOE[a]
Bulk	Cotton, AZ	Loader	1,631	10	36	1.1×10^{-3}	6.1×10^{-5}	560
		Applicator	1,359	10	20	5.3×10^{-4}	2.8 x 10-5	670
Bulk	Potatoes, WA	Loader	1,631	8	24	6.1×10^{-4}	3.2×10^{-5}	560
		Applicator	1,359	8	24	5.1×10^{-4}	2.7×10^{-5}	670
Mini-bulk	Tobacco, NC	Loader	464	5	10	4.5×10^{-5}	2.4×10^{-6}	1960
		Applicator	1,359	5	10	1.3×10^{-4}	7.0×10^{-6}	670

[a] Adjusted for wearing of respirator or use of enclosed tractor cab (PF = 0.1). MOEs greater than 100 are generally considered to be acceptable.

TABLE 8.— 1,3-D GROWER INTERMEDIATE-TERM NON-CANCER RISKS AND CANCER RISKS

Delivery Method	Example Crop	Loading Conc. µg/m^3	hr/d	Application:			LADD	Cancer Risk	Int.-Term MOE[a]
				Conc. µg/m^3 from Table 4	hr/d	d/yr			
Bulk	Cucurbits, TX	10833	0.25	1,359	6	15	6.3×10^{-4}	3.4×10^{-5}	670
Bulk	Pineapples, HI	1,0833	1.25	1,359	6	11	9.3×10^{-4}	5.0×10^{-5}	670
Mini-bulk	Tobacco, NC	464	0.5	1,359	5	3.5	9.6×10^{-5}	5.1×10^{-6}	670
Mini-bulk	Peanuts, GA	464	1	1,359	3	5	8.8×10^{-5}	4.7×10^{-6}	670

[a] Adjusted for wearing of respirator or use of enclosed tractor cab (PF = 0.1)

TABLE 9.—RESIDENTIAL/BYSTANDER EXPOSURE

Distance from treated field(m)	Study Site(s)	Doses (mg/kg/day)		Cancer Risk	Int.-Term MOE
		ADD	LADD		
1,600	AZ	7.6×10^{-7}	3.3×10^{-7}	1.7×10^{-8}	2,800
1,200	AZ	2.9×10^{-5}	1.2×10^{-5}	6.6×10^{-7}	1,600
800	overall	5.7×10^{-5}	2.4×10^{-5}	1.3×10^{-6}	8,500
500	overall	7.7×10^{-5}	3.3×10^{-5}	1.8×10^{-6}	6,100
[1]125	overall	2.6×10^{-4}	1.1×10^{-4}	5.9×10^{-6}	1,700
25	overall	4.8×10^{-4}	2.1×10^{-4}	1.1×10^{-5}	920
5	overall	5.1×10^{-4}	2.2×10^{-4}	1.2×10^{-5}	870
Onsite	overall	8.3×10^{-4}	3.6×10^{-4}	1.9×10^{-5}	500

[1]Labels require buffer zone of 300 ft (approximately 125 meters) from an occupied structure.

C. Aggregate and Cumulative Risk

Aggregate risk, which considers the various routes of exposure for a pesticide, and cumulative risk, which looks at the risks posed from all pesticides with a common mechanism of action are factors that EPA must consider when it evaluates risks from a pesticide chemical residue under the Federal Food, Drug and Cosmetic Act, as amended by the Food Quality Protection Act. These requirements apply specifically to tolerance actions. As mentioned in the Introduction, EPA classifies 1,3-D as a non-food use chemical. Thus, tolerances are not required. Therefore, EPA regulates 1,3-D under FIFRA's risk/benefit standard. However, these risk assessment factors reflect advances in risk assessment methodology which EPA believes are appropriate when assessing 1,3-D's risk, even though no tolerance action is involved.

EPA has aggregated inhalation and oral exposures to 1,3-D. The aggregate risk estimate is calculated as follows:

$$\text{cancer risk}_{\text{inhalation}} + \text{cancer risk}_{\text{water}} = \text{aggregate lifetime cancer risk}$$

In calculating aggregate risk, EPA has determined that a reasonable worst-case exposure scenario would be comprised of the inhalation risk at the 300 foot buffer, derived from the average of three air monitoring studies, and water exposure risk from the on-site concentrations from the Florida study. EPA did not use the Wisconsin study values because, as of August 1, 1999, use in areas similar to this site is prohibited. Thus, the aggregate risk is estimated as follows:

$$6 \times 10^{-6}{}_{\text{inhalation}} + 4 \times 10^{-6}{}_{\text{water}} = 1 \times 10^{-5}$$

This aggregate cancer risk estimate, however, is based on assessments which contain numerous uncertainties from both the inhalation and water routes of exposure. Those uncertainties are detailed in Unit V.D. below.

For cumulative risk, EPA has made a determination not to cumulate the risks posed by exposures to 1,3-D and any other chemical. This determination could change in the future based on policy changes or new mechanistic data on 1,3-D or other chemicals.

D. Strengths, Weaknesses and Uncertainties of the Risk Assessment

The evidence for the inhalation carcinogenicity endpoint is strong. Carcinogenicity was confirmed at multiple sites in two species of test animals. Further, the lung tumors used for quantitative risk assessment were seen in both the mouse oral and inhalation studies. Positive results in bacterial, *Drosophila* and mammalian mutagenicity studies also contribute to the weight-of-the-evidence for carcinogenicity. EPA acknowledges that there are uncertainties in extrapolating from rodent studies to possible human effects. While there are human incidents suggesting a link between 1,3-D exposure and hematological malignancies, they are too few to support a change to the cancer classification.

The main difficulty in assessing exposure is trying to measure air concentrations of a volatile chemical under highly variable conditions. Although there is an extensive exposure monitoring data base for 1,3-D, many factors influence exposure. Many of these factors are specific to the application method and local environmental conditions. Soil conditions (moisture, organic content, temperature), soil sealing methods, injection depth and meteorological conditions all affect 1,3-D air concentrations to various degrees. Since these factors are uncontrollable under field conditions, additional studies are not likely to yield information which would substantially improve the accuracy of the current risk assessment.

In addition, based on available data, EPA extrapolated to estimate levels of use on crops and in states for which there was no actual data. The assessment also assumes that treatment patterns are the same every year; however, the 1992 Use, Usage and Product Performance DCI noted that treatment typically varies from year to year, depending on anticipated pest pressures, crop rotations, weather conditions, and economic factors.

There is also no information available to assess whether there are current 1,3-D handlers whose exposure would increase due to the methyl bromide phase out. A cursory review of usage over the past five years shows that there has been an overall increase in 1,3-D use. EPA believes this increase is due, in part, to growers making the transition away from methyl bromide. EPA believes that the phase out will increase the numbers of people exposed, but not any one 1,3-D user's exposure, because growers typically use either 1,3-D or methyl bromide.

EPA believes residential risks may be overstated because most individuals are not likely to spend 16 hours a day at a fixed distance from a treatment site for the 2–week period following fumigation over 30 years.

Drinking water risks were based on levels found in on-site wells. Because the new labels will require a 100 foot setback, these levels are likely overestimates, and thus add to the uncertainty in the risk estimates presented in this document.

Most importantly, the protective value of only some of the mitigation measures required on 1,3-D labels can be quantified. Given that many of the measures have not been factored into the assessment, risks are likely to be lower than those presented.

E. Comments on Risk from the Notice of Special Review and EPA's Response

Several comments on the health concerns were submitted in response to EPA's 1986 decision to initiate a Special Review. Many of these comments are no longer applicable as changes have been made to the formulation of 1,3-D products, use patterns and 1,3-D labels. For completeness of the record, EPA will present and respond to these comments.

Comment. The Natural Resources Defense Council (NRDC) submitted extensive comments on 1,3-D. First, NRDC criticized the exposure assessment for not taking into account dermal exposure. Secondly, they mentioned that bioaccumulation in aquatic animals should be addressed. In addition, NRDC asserted that tolerances or an exemption from a tolerance should be established to cover residues of 1,3-D in commodities grown in treated soil.

EPA's Response. At the time of the Notice of Special Review, EPA's position was that, due to 1,3-D's volatility, the dermal contribution to risk was minimal compared to the inhalation risk. Because of closed loading and other personal protective equipment requirements, dermal exposure to workers should be minor, if any. Dermal exposure to bystanders and those living 300 feet from treated fields is not expected.

As to bioaccumulation in aquatic animals, the Registration Standard noted that laboratory studies show the parent compound, 1,3-D, is low to moderately toxic to waterfowl and upland game birds, moderately toxic to fish and highly toxic to freshwater invertebrates. In water, 1,3-D rapidly

dissolves by photolysis and hydrolysis, reducing the potential for exposure to non-target organisms and thus the potential for bioaccumulation [Ref. 26].

Regarding tolerances, EPA has determined that residues of concern are not likely to appear in foods from pre-plant fumigant uses of 1,3-D and has classified such 1,3-D uses as non-food uses which do not require tolerances.

Comment. NRDC asserted that ground water should have been included as a trigger for the Special Review, and that 1,2-D and 3-chloroallyl-alcohol should have been examined in greater detail.

EPA's Response. At the time EPA issued the Notice of Special Review, a main force driving the ground water concern was the higher percentage of 1,2-dichloropropane in Telone products. Since that time, the amount of 1,2-D has been reduced, although EPA is still tracking how 1,2-D moves in the environment.

EPA agrees with NRDC's comment that the acid and alcohol degradates should be included in the 1,3-D risk assessment. In the dietary assessments, EPA required that Dow AgroSciences track the residue chemistry of the alcohol and acid degradates. There were no residues of either 1,3-D or its degradates in crops planted to 1,3-D treated soils. For water monitoring and subsequent risk assessments, EPA included the degradate levels and assigned the same toxicity and carcinogenicity as the parent. Dow AgroSciences is conducting several toxicity and environmental fate studies to test this assumption. EPA did not include ecological risk as a trigger for the Special Review; the 1998 reregistration review of ecological data supports that 1,3-D use does not pose unacceptable ecological risks.

Comment. The state of Massachusetts commented that residues of 1,3-D had never been detected in ground water there, but that an on-going monitoring system was in place.

EPA's Response. EPA is aware that 1,3-D has not been detected to date in Massachusetts. However EPA's review of 1,3-D monitoring is on-going and the Agency would like to receive any available information about 1,2-D and 1,3-D monitoring (including degradates) from the states.

Comment. The U.S. Department of the Interior commented that the Notice of Special Review did not take into account the effects of 1,3-D on wildlife.

Response. In the Registration Standard, EPA noted that there were no known effects on wildlife or endangered species. Studies submitted for reregistration show that 1,3-D is moderately toxic to waterfowl and upland game birds. In ecotoxicity tests, 1,3-D is moderately toxic to coldwater fish, moderately toxic to warm water fish and highly toxic to freshwater invertebrates. EPA believes that, since 1,3-D is injected into the soil and dissipates relatively soon thereafter, there should be low exposure to wildlife through plants or insects. While ecological effects were not included in the Notice of Special Review, EPA has reviewed data applicable to wildlife effects for reregistration and found that 1,3-D is not likely to pose unreasonable risks to wildlife. Because use of 1,3-D is expected to expand to coastal areas, Dow AgroSciences is conducting estuarine ecotoxicity and environmental fate data on 1,3-D and the alcohol and acid degradates. EPA will take appropriate regulatory action if the study results show that the increased 1,3-D use poses unreasonable risks.

VI. Benefits Assessment

1,3-D is a pre-plant soil fumigant labeled for the control of all plant-parasitic nematodes and some plant diseases, insects and weeds. Nematodes are the principle target pests for most use sites. 1,3-D, methyl bromide, metam-sodium and chloropicrin are broad-spectrum soil fumigants registered for use on all food and non-food sites. Dazomet is a nematicide registered for selected sites. Non-fumigant alternatives are aldicarb, ethoprop, fenamiphos, oxamyl and terbufos. Non-chemical alternatives (e.g., fallowing, non-host crop rotations, resistant varieties, soil solarization, deep plowing of crop residue) are often classified as supplemental control measures because they are used in conjunction with the pesticide alternatives. The amount of 1,3-D used is variable from year to year. EPA estimates that 20 to 40 million pounds of the active ingredient 1,3-D are applied yearly to approximately 400,000 to 500,000 acres.

A. Scope and Methodology

Individual site analyses were completed for 1,3-D use on 15 sites. Most of the usage data in the benefits analyses were obtained from the 1991 Use, Usage and Product Performance DCI; other information was gathered from USDA published statistics, state extension officials and crop specialists, literature searches and comments on the Notice of Special Review. The 15 sites comprised about 95% of the 1,3-D usage between 1988 and 1990.

EPA has conducted three reviews of benefits information: (1) the 1986 Initiation of Special Review; (2) a 1994 analysis based mainly on information from the 1991 DCI [Ref. 27]; and (3) a 1997 update of the 1994 analysis [Ref. 28]. The 1994 review estimated economic impacts if 1,3-D were restricted or canceled. The 1997 review was not as comprehensive as the DCI and 1994 analysis, and thus the more recent analysis may not have captured the full extent of use between 1994 and 1997.

The basic economic approach used was a partial budgeting method and simple supply-demand analysis using possible cost changes and yield effects. If 1,3-D use were canceled for a given site, EPA made projections on the alternatives that growers would use to control the target pests on acreage currently treated with 1,3-D. The assessment does not project economic impacts if both 1,3-D and methyl bromide are unavailable.

California 1,3-D usage was not included in the benefits assessment because of California's suspension of use permits between 1990 and 1994 and the limited re-introduction of 1,3-D since then.

B. Impacts if 1,3-D were not Available

Based on the 1994 review, short-term grower economic impacts for all sites are estimated to range from $37 million to $89 million annually. EPA considers these impacts to be substantial. These impacts are the result of increased costs for alternative treatments and reduced yields with the use of alternatives and are presented in Table 11. EPA estimates project that growers would shift an average of 50% of their use to the fumigant alternatives and 44% of the use to non-fumigant alternatives. The remaining 6% represents a shift to non-chemical and unknown alternatives. Metam-sodium is the fumigant alternative with the largest quantity of additional acres treated, followed by methyl bromide and chloropicrin. Aldicarb is the non-fumigant alternative with the largest shift in additional acres treated, followed by ethoprop and fenamiphos.

Crops with the greatest total value of impacts if 1,3-D were canceled would be Irish potatoes, tobacco, sugar beets, cucurbits (e.g., cucumbers, pumpkins, squashes), onions, strawberries and peppers. Geographically, the regions most affected would be the Pacific Northwest (Washington, Oregon and Idaho) and the southeastern states (Georgia, Alabama, Florida, Virginia and North and South Carolina). Impacts on users growing fruit and nut trees and grapevines, crucifers, pineapples and strawberries would occur when methyl bromide is no longer available as an alternative. The following table 10 presents estimated usage of 1,3-D and reflects a recent update.

TABLE 10.— MAJOR 1,3-D USAGE SITES - 1997 REVIEW[1]

Crop	Acres Treated (000)	% Crop Treated	lbs a.i. applied (000)	States where most usage occurs
	weighted average	weighted average	weighted average	
Crucifers	10	4	2000	AZ,TX,GA, SC, NC,CA
Peppers	5	4	400	NM,NC,CA
Cucurbits	13	2	600	TX,AZ,SC, NC,GA,CA
Sugar Beets	45	3	4000	NE,WY,CO, ID
Cotton	85	1	2000	AZ,NC,GA, FL,CA
Tobacco	80	11	7200	NC,SC,GA
Irish Potato	80	6	13,500	WA,ID,OR, CO,ND,MI
Sweet Potato	N/A[2]	N/A[2]	N/A[2]	NC, GA, SC
Peanut	12	1	700	AL,GA,TX
Fruit/Nut Trees and Grape Vines	27	6	2400	CA,SC,NC, AZ,GA,NJ
Onions	5	5	1000	OR,WA,ID
Tomato	2	0	200	GA,FL,AL
Carrots	2	2	150	CA,WA,TX
Pineapple	5	14	1300	HI
Strawberries	1	1	80	CA,FL,NJ
Total	382		35530	

[1] Usage data covers 1990-1995 for most sites and as early as 1987 for other sites, primarily using data from the 1991 Use, Usage and Product Performance DCI. California data is only available for 1994 and 1995, due to the 1991-1993 use permit suspension and limited re-entry program. ''Weighted average'' weights the more recent years' estimates because they tend to be more reliable estimates than for possibly outdated earlier estimates.

[2]N/A - not available for sweet potatoes during the 1997 review.

The following table 11 presents the 1994 summary of short term (annual) economic assessment.

TABLE 11.— SUMMARY OF SHORT-TERM, ANNUAL IMPACTS IF 1,3-D WERE CANCELED (1991 ESTIMATES)

Crop	Average Pounds a.i. applied (000)	Average acres treated (000)	Average Percent crop-treated	Total Short term Annual Impact from Use of Next-Best Alternative(s)(in $000)	
				Increase in Treatment Costs	Yield Losses Cost
Carrots	450	4	1	500–1,000	400
Cotton	1550	31	8	insignificant	300–3,300
Crucifers	950	26	4	unknown[1]	unknown[1]
Cucurbits	1500	19	5	6,000–6,500	unknown
Fruit/Nut Trees & Grapevines	2,500	9	2	0–500	none in short run[2]
Onions	1,750	10	2	1,500–8,000	unknown
Peanuts	750	12	3	insignificant	insignificant
Peppers	3,650	18	4	5,600–6,700	none in short run[2]
Pineapples	1,950	6	2	400–500	(2,100–2,700)
Potatoes (Irish)	16,500	95	24	4,000	9,000–22,000
Strawberries	75	<1	<1	100	none in short run[2]
Sugar Beets	4,500	51	13	insignificant	1,000–13,000
Sweet Potatoes	1,900	29	7	insignificant	unknown
Tobacco	8,150	91	23	2,000–3,000	8,000–13,000
Tomatoes	300	2	1	insignificant	none in short run[2]
Total	46,475	403		20,000–40,000	[3]17,000–49,000

[1] The information from the 1991 DCI did not provide enough comparative information for alternatives and thus no estimates could be derived.

[2] Methyl bromide is the main alternative; absent development of a suitable alternative, losses would occur without 1,3-D after the 2005 phase-out.

[3] With next best alternative (methyl bromide), yield increases would be expected.

C. Strengths and Limitations in the Benefits Assessment

The data used to conduct the benefits assessment for 1,3-D are relatively comprehensive. The results of the Use, Usage and Product Performance DCI allowed EPA to identify specific use states, amount of 1,3-D used, acreage treated and use of alternatives for many use sites. EPA was able to quantify potential economic impacts where yield

data for 1,3-D and its alternatives was available.

However, there are weaknesses associated with this assessment, as the information is now as much as 10 years old. Changes in the regulatory status of alternatives, agricultural markets and the laws governing agriculture are likely to have influenced some 1,3-D users' practices. Although the 1997 review shows a decrease in use from the 1994 analysis, a cursory review of 1,3-D trends indicates that 1,3-D use has been increasing, and likely will continue to do so. This is mainly due to increased usage in California as the state's permitting program has increased the amount of 1,3-D used there. In addition, 1,3-D use has increased (mainly in Florida and California) as growers seek alternatives to methyl bromide. Overall, the figures presented in Tables 10 and 11 likely understate to some degree the benefits associated with current 1,3-D use. EPA is interested in obtaining comments (preferably data) from areas or for crops which have experienced substantial fluctuations in 1,3-D use over the past 5 to 7 years.

There are also limitations in how the assessment was conducted. Some of the data EPA collected on product performance came from crop specialists' opinions where studies were not available. Also, usage data for a few vegetable crops were aggregated under different groupings for some states. For example, one state listed tomatoes as an individual crop, while another listed tomatoes under the grouping ''vegetables.''

For crops where methyl bromide is the fumigant of choice, EPA attempted to predict whether 1,3-D would be used when methyl bromide is no longer available, and the resulting increase in 1,3-D usage. Crop specialists and growers are not sure what major pest(s) are currently being controlled by methyl bromide since it is a broad spectrum biocide. Accordingly, it is not clear to what extent 1,3-D would serve as a suitable alternative for all of the methyl bromide uses. In addition, the pending phase-out of methyl bromide has spurred a great deal of research on alternative nematode controls; development of less costly or more effective alternatives could also have an effect on future use of 1,3-D. Because of the uncertainties related to the methyl bromide phase-out, EPA decided to present its benefits assessment on a short-term, annual basis. Despite the uncertainties associated with the pending phase-out, EPA believes the information accurately depicts the high benefits associated with 1,3-D use.

The 1,3-D benefits assessment provides valuable information defining use and usage patterns. The benefits analyses present biological and economic information on the use and usage of 1,3-D. Biological assessments provided information on pests controlled and their damage, use rates, methods of application and the comparative performance of alternatives. Economic analyses estimated the total usage, the cost of market shifts to alternatives and the relative impacts on users and the industry.

VII. Risks Associated with 1,3-D Alternatives

In developing a regulatory proposal, EPA considered whether canceling 1,3-D use could actually increase risk based on shifts to the next best alternative. The main limitation in developing a comparative risk assessment is that the main alternatives pose acute rather than chronic risks, making these different endpoints difficult to compare. As such, this Unit provides only a summary of the risks of alternative nematicides.

For the two fumigant alternatives, methyl bromide and metam sodium, short-term animal studies were used to determine at what level of exposure adverse effects are observed. The NOAEL is the lowest tested level where no observable adverse effects are seen. A quotient of the NOAEL over human exposures is used to calculate an MOE. EPA generally regards MOEs of less than 100 to be unacceptable.

A. Methyl Bromide

Like 1,3-D, methyl bromide is a liquid soil fumigant that is injected into the soil. Since methyl bromide is more volatile than 1,3-D, tarping generally follows application in order to improve methyl bromide retention in the treated volume of soil.

Inhalation of 1,600 ppm for 10–20 hours, or 7,900 ppm for 1.5 hours is lethal to humans [Ref. 29]. The lowest inhalation level found to cause toxicity in humans is 35 ppm in air. At lower levels, there can be neurological effects and low-level chronic exposures are associated with dizziness, vision and hearing disturbances, and personality changes. Most human exposures are through inhalation. OSHA has established a Permissible Exposure Level of 20 ppm time-weighted average over an 8–hour period [Ref. 30].

For methyl bromide, EPA did not have a complete data base on usage. Therefore, the risk assessment was conducted on the crop where the total amount of methyl bromide used is highest - strawberries. The study used was conducted by the Alliance of the Methyl Bromide Industry in June 1993 to measure worker exposure only; there was no monitoring to assess residential exposure [Ref. 31]. No mitigation is factored into the assessment, even though a self-contained breathing apparatus (SCBA) is required when methyl bromide levels exceed the Threshold Limit Value of 5 ppm. The NOAEL is 20 ppm based on a rabbit study. MOEs for workers range from 5 to 7,600. The workers most at risk are those who remove the tarps several days after application. MOEs for this group of handlers range from 5 to 19.

Ground water testing for methyl bromide has been conducted in California, Florida and Hawaii. Of 20,429 wells tested, 2 wells in California contained methyl bromide residues at 2.5 and 6.4 ppb. There is no Maximum Contaminant Level (MCL) established for methyl bromide.

As mentioned in Unit II.B. of this document, methyl bromide production and importation is scheduled for phase-out in 2005 because of its potential to deplete stratospheric ozone.

B. Metam Sodium

Metam sodium is also a liquid soil fumigant typically applied by injection or chemigation methods. Chemigation application is preferred because water is required for transporting the chemical through the soil. The type of irrigation system used depends on the crop grown and farm size. Metam sodium rapidly breaks down to methyl isothiocyanate (MITC) and carbon disulfide (CS_2), which are both developmental toxicants based on animal studies. California now requires buffer zones for fields near residential areas based on the odor nuisance associated with CS_2.

The MOEs, based on MITC and CS_2, for mixer/loaders and applicators for several types of application systems range from 23 (shank injection similar to 1,3-D applications) to 261 (center pivot irrigation). MOEs for residents are estimated to be 135 at the 500 meter buffer. The Agency does not have information on ground water monitoring for metam sodium or MITC [Ref. 32].

C. Aldicarb

Aldicarb is a granular carbamate pesticide. Aldicarb controls insects, mites and nematodes and is used on certain crops where 1,3-D is also used: cotton, citrus, peanuts, sugar beets, sweet potatoes and tobacco. Use on Irish potatoes is restricted to the Pacific Northwest, Florida and certain counties in Utah and Nevada. EPA has classified aldicarb in ''Toxicity Class I,'' meaning it is highly toxic by the oral, dermal and

inhalation routes of exposure. In 1993, EPA identified aldicarb as one of the five most acutely toxic pesticides to handlers and field workers. Since then, both EPA and Rhone-Poulenc, the main producer of aldicarb, have pursued risk mitigation proposals to reduce the risk to handlers and applicators of aldicarb.

Residues of aldicarb have been detected in foods, and in some cases, the higher levels exceeded levels of concern for acute toxicity. EPA has taken steps to reduce the possibility of high residues in foods, especially potatoes.

Aldicarb has been detected in ground and drinking water supplies. EPA is in the process of establishing an MCL for aldicarb and for the sulfoxide and sulfone degradates.

Since the detection of aldicarb residues in wells on Long Island, New York in 1979, an extensive amount of ground water monitoring has been conducted by the registrants and state and local authorities. Aldicarb residues have been detected in ground water in 26 states. EPA has identified a positive correlation between aldicarb detections in ground water and vulnerable soils (i.e., soil conditions that are more likely to lead to ground water contamination), usage, and climatic data. Geologic and hydrologic factors, such as the lateral movement of water along an impermeable layer, are viewed as significant in controlling the movement of aldicarb to ground water. Other controls, such as well set-backs, have not been completely effective in preventing ground water contamination. Because of this, EPA has been looking at a variety of controls to augment set-backs such as regulating based on local soil and water conditions, and lower rates to control the potential for ground water contamination [Ref. 33].

D. Fenamiphos

Fenamiphos is an organophosphate, contact nematicide which is sold as either a granular or an emulsifiable concentrate. Fenamiphos is used primarily on tobacco, orchard crops, cotton, peanuts, citrus, grapevines, and pineapples as an alternative to 1,3-D or as a supplemental nematicide once crop growth is underway. Fenamiphos has a low soil/water partition coefficient, resistance to hydrolysis, and low Health Advisory level (2 ppb). The risk concerns with fenamiphos and its degradates are high acute toxicity (Classified in EPA's Toxicity Category I), residues in food, ground water contamination and surface water contamination. The parent compound, fenamiphos, has been detected in ground water in Florida at over 10 times the adult health advisory of 2 ppb. High levels of the two major degradates of toxicological concern have also been found in ground water in Florida. Unlike 1,3-D and methyl bromide, fenamiphos does not volatilize rapidly. Bird and fish kills have been associated with fenamiphos use, and label restrictions (setbacks from waterways) have been placed on fenamiphos labels. EPA is also looking into ecological concerns for terrestrial, fresh water and marine/estuarine animals.

In conjunction with the overall review of organophosphates, EPA is posting risk and use information for fenamiphos on the internet. The most current risk assessment for fenamiphos is available on www.epa.gov/oppsrrd1/op/status.htm.

E. Summary of the Risks Associated with Alternatives to 1,3-D

EPA reviewed the risks associated with the alternatives to 1,3-D to determine whether cancellation of 1,3-D registrations would actually reduce risks or shift risks due to exposure to alternatives. The Agency found that considerable risks are associated with the most likely alternative nematicides. Like 1,3-D, the four major alternatives pose risks to workers. Aldicarb and fenamiphos residues also present dietary concerns. There are ground water contamination concerns associated with the use of fenamiphos and aldicarb. Fenamiphos also is a surface water contaminant and has caused fish kills. While there is no way to compare chronic and acute risks directly, EPA believes the potential acute risks of 1,3-D's alternatives raise concerns about the desirability of shifting use from 1,3-D to the next-best alternatives.

VIII. Risk/Benefit Analysis

A. Introduction to the 1,3-D Risk/Benefit Analysis

FIFRA directs EPA to consider both the risks and benefits of a pesticide's use when developing and choosing among regulatory options. In looking at the benefits, EPA considers the availability and effectiveness of alternative treatments and the risks posed by the alternatives. In addition, EPA takes into account uncertainties in both the risk and benefits assessments.

In 1996, FQPA amended the requirements for what EPA must consider in taking any action on pesticide tolerances, including aggregate and cumulative risks, and whether infants and children have heightened susceptibility to a pesticide's effects. Although there are no tolerance actions related to this proposal, EPA believes the FQPA considerations are appropriate to include in the 1,3-D risk assessment. Although there are no residues in crops grown in treated soils, there is dietary risk since 1,3-D can migrate to ground water that is used for drinking water.

Both the 1,3-D risk and benefits assessments are weakened by numerous uncertainties, despite efforts by both EPA and Dow AgroSciences to develop specialized and comprehensive data on exposures, carcinogenicity and use and usage information. EPA also considered whether additional data could be developed to assign a mitigation value to the measures that have been incorporated into 1,3-D registrations or to overcome other weaknesses in the data base. Given that many of the factors that have a substantial influence over 1,3-D exposures are uncontrollable in normal field settings, the potential for improving the current risk assessment with additional data is minimal. Instead, EPA evaluated both the nature of the uncertainties and the current data base to weigh the risks and benefits of 1,3-D use.

B. Summary of Mitigation Measures on 1,3-D Labels and Risk

In 1992 and in 1995, Dow AgroSciences requested label changes to reduce levels of 1,3-D which volatilize into the atmosphere during fumigant transfers, application and the post-fumigation time period. Measures added to 1,3-D labels were shut-off valves to prevent 1,3-D from spilling at row turns, closed loading systems, soil sealing, a 300–foot no-treatment buffer from occupied structures, improved product stewardship, a phase-out of drum delivery, and reduced application rates. These measures reduced exposures not only for workers, but for anyone in the vicinity of treated fields.

On September 30, 1998, Dow AgroSciences requested additional modifications to the terms and conditions of 1,3-D registrations to include a use prohibition in certain northern tier states (ND, SD, MN, NY, ME, NH, VT, MA, UT, MT, WI) where ground water is less than 50 feet from the surface and soils are Hydrogeologic Type A, a 100–foot no-treatment buffer around drinking water wells, prohibition of use in areas overlying karst geologies and additional monitoring to confirm that use of 1,3-D does not pose unreasonable risks.

EPA has determined that 1,3-D is a probable human carcinogen. The quantified portion of the risk assessment for 1,3-D shows that inhalation cancer risk estimates for workers are estimated

to be in the 10^{-5} to 10^{-6} range. Residents who live near treated fields are also exposed to 1,3-D as it volatilizes from treated fields. Not taking into account any of the mitigation provided for on 1,3-D labels, studies show that risks for area residents who live within 300 feet of treated fields can be as high as 6×10^{-5}. EPA views this as an overestimate of exposures under typical use patterns and believes that the label measures such as soil sealing, lowered rates, soil moisture, and deeper injection, reduce exposures to an acceptable level. EPA has determined that 1,3-D and its degradates can migrate to ground water under normal use conditions. Using the results of the on-site wells in the Florida prospective ground water study, lifetime cancer risk estimates are 4×10^{-6} from drinking water. Because the new labels will require a 100 foot setback from drinking water wells, EPA believes this drinking water risk is an overestimate. From these estimates, EPA calculated the aggregate risk (oral plus inhalation) to be 1×10^{-5}.

EPA also recognizes aspects of the assessments that may understate risk. An increase in 1,3-D use since the 1991 assessment could result in higher risk if a worker's exposure duration is increased based on handling more product. Although the 1,3-D studies were designed to mimic higher-end exposure scenarios, they never measured exposure from application at more than one site at a time. Thus, EPA was not able to assess the impact on air and water levels in areas experiencing multiple 1,3-D treatments. Dow AgroSciences is conducting air monitoring in California where multiple fields undergo simultaneous treatment. EPA has arranged to obtain this information to assess the impact on air levels.

Although the final risk estimates were derived from an assessment that does not consider the reduction offered by several mitigation measures, EPA believes that cumulatively all of the measures on the 1,3-D labels adequately reduce exposures.

C. Summary of Benefits

1,3-D is registered for use on all vegetable, field, fruit and nut and nursery crops. As a fumigant, it is considered more effective than other fumigant and non-fumigant alternatives, except for methyl bromide, and certain uses of aldicarb and metam sodium. As a pre-plant fumigant, 1,3-D treatments are only applied once per crop planting; whereas the non-fumigant alternatives may require multiple applications, including to growing crops.

Nematode infestations typically lead to lowered yields and, in the case of root crops, may also lead to smaller and disfigured roots. Other types of pests also controlled by 1,3-D, such as certain soilborne diseases, generally cause similar types of yield impacts. Because residues in crops and rotational crops are not an issue, growers have an option in selecting which crops to plant after soils have been treated with 1,3-D.

Although methyl bromide is considered an effective alternative, its production and importation are scheduled to be completely phased out by the year 2005. It is anticipated that 1,3-D will be used to replace an unknown amount of the current methyl bromide soil fumigation usage when the phase-out occurs. Additionally, all the fumigant and non-fumigant alternatives pose acute risks, including potentially unacceptable dietary risks.

EPA has estimated that if 1,3-D were not available, annual losses to growers resulting from yield losses and/or increased treatment costs would range from $37–89 million (or higher depending on the availability of alternatives). Significant impacts would be incurred by growers of Irish potatoes, tobacco, sugar beets, curcubits, onions, strawberries and peppers. The regions most affected would be the Pacific Northwest and south-eastern states.

The main weaknesses in the benefits case are that the information used is several years old and there are uncertainties associated with the anticipated phase-out of methyl bromide use and the regulatory status of the remaining nematicides. Restrictions on the alternatives are likely to substantially increase the benefits related to 1,3-D use.

D. Summary of Risk/Benefit Determination

In assessing the risk/benefit balance for 1,3-D, EPA evaluated the mitigation provided by all of the mitigation measures included on 1,3-D labels. The Agency has sought a wide variety of measures, including those which can be both qualitatively and quantitatively assessed, to reduce risks to the greatest extent possible. EPA has determined that the exposure reduction derived from quantitative and qualitative risk mitigation measures, taken together, provide acceptable exposure reduction for those who handle 1,3-D products, as well as for those who live near treated fields. EPA used this determination in 1998 to support the Agency's decision that all uses of 1,3-D are eligible for reregistration.

Accordingly, EPA has determined that the benefits of 1,3-D use outweigh the risks, taking into account mitigation measures on the labels, lack of safe, effective alternatives and benefits associated with 1,3-D's use. Therefore, EPA is proposing to terminate the 1,3-D Special Review.

Nothing in today's proposal affects EPA's ability to seek additional data or changes to the terms and conditions of 1,3-D registrations should the need arise. On-going reviews of studies being conducted for reregistration, such as the tap water monitoring program, present opportunities to review the status of 1,3-D registrations in the future. Should those data, or any other information, show that 1,3-D use poses unreasonable risks to the environment, EPA could seek additional mitigation, and if appropriate, initiate regulatory action involving 1,3-D.

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List of Subjects

Environmental protection, pesticides and pest.

Dated: December 17, 1999.

Susan H. Wayland,

Deputy Assistant Administrator for Prevention, Pesticides and Toxic Substances.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–30471A; FRL–6399–9]

Pesticide Products; Registration Approvals

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces Agency approval of applications to register the pesticide products Trifloxystrobin Technical, Flint, Stratego, and Compass containing an active ingredient not included in any previously registered products pursuant to the provisions of section 3(c)(5) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended.

FOR FURTHER INFORMATION CONTACT: By mail: Cynthia Giles-Parker Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–7740; and e-mail address: giles-parker@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

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

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

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

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

To access a fact sheet which provides more detail on this registration, go to the Home Page for the Office of Pesticide Programs at http://www.epa.gov/pesticides/, and select ''fact sheet.''

2. *In person*. The Agency has established an official record for this action under docket control number OPP–30471A. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are also available for public inspection. Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of