ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–2003–0127; FRL–7321–6]

2,6-Diisopropylnaphthalene; Temporary Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a temporary tolerance of 0.5 parts per million (ppm) for 2,6-Diisopropylnaphthalene (2,6-DIPN) in or on potatoes, and 3 ppm in or on potato peels. Platte Chemical Company requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (FQPA). The temporary tolerance will expire on May 31, 2006.

DATES: This regulation is effective August 8, 2003. Objections and requests for hearings, identified by docket ID number OPP–2003–0127, must be received on or before October 7, 2003.

ADDRESSES: Written objections and hearing requests may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit VII. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Driss Benmhend, Biopesticides and Pollution Prevention Division (7511C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–9525; e-mail address: *Benmhend.driss@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

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

B. How Can I Get Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0127. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*. A frequently updated electronic version of 40 CFR part 180 is available at *http://www.access.gpo.gov/nara/cfr/cfrhtml__00/ Title__40/40cfr180_00.html*, a beta site currently under development. To access the OPPTS Harmonized Guidelines referenced in this document, go directly to the guidelines at *http://www.epa.gov/opptsfrs/home/guidelin.htm.*

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

II. Background and Statutory Findings

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

In the **Federal Register** of September 21, 2001 (66 FR 48677) (FRL–6798–3), EPA issued a notice pursuant to section 408 of the FFDCA, 21 U.S.C. 346a, as amended by FQPA (Public Law 104–170), announcing the filing of a pesticide petition (PF–1043) by Platte Chemical Company, 7251 4th Street, Greely, CO 80632. This notice included a summary of the petition prepared by the petitioner Platte Chemical Company.

The petition requested that 40 CFR 180.1208 be amended by establishing a temporary tolerance for residues of the plant growth regulator 2,6-DIPN, in or on potatoes at 3 parts per million (ppm) for the peels, 0.5 ppm for potato (whole). The tolerance will expire on May 31, 2006. EPA received comments on this petition submitted by John Forsythe, General Manager, on behalf of D-I-1-4, Inc. (Meridian, ID). The issues raised by Mr. Forsythe related to the following: (1) The classification of 2,6-DIPN as a biochemical pesticide; (2) the lack of chronic toxicity data; and (3) the public's exposure to this chemical through its use as an industrial chemical. Mr. Forsythe's comments are discussed individually below, along with EPA's response.

Comment 1. Mr. Forsythe requested that the Agency re-evaluate the biochemical classification determination for 2,6-DIPN and provide any publicly available information regarding the natural occurrence of 2,6-DIPN in any food source.

EPA Response. A biochemical pesticide, by definition, is a naturally occurring substance which controls target pests by a non-toxic mode of

action. However, there are products that are not naturally occurring, yet they are registered by the Agency as ''biochemical-like,'' insofar as data requirements are concerned. Thus, while 2, 6-DIPN, is synthetic and does not occur naturally in any food or non-food plants, it is structurally similar to three compounds (1-isopropyl- 4,6-dimethylnaphthalene, 1-methyl-7-isopropylnaphthalene, and 4-isopropyl-1,6-dimethylnaphthalene) that occur naturally in potatoes, and 2,6-DIPN is functionally identical to the naturally occurring plant growth regulator in potatoes.

Comment 2. Mr. Forsythe expressed concern that the Agency had not presented any public documentation demonstrating that the mode of action of 2,6-DIPN is non-toxic.

EPA Response. The new active ingredient, 2,6-DIPN, is a plant growth regulator (PGR) intended to inhibit sprouting in stored potatoes. PGRs may stimulate or retard ripening, maturity of whole plants and/or fruits, enhance growth, yield, enhance or counteract the activities of other PGRs, and/or change plant architecture (amongst other processes). PGRs are not toxic to the target plant, especially at the application rate. Tests conducted during the experimental use permit showed no toxicity to potatoes. None of these actions are directly lethal to the plants upon which they are applied, which supports a determination that 2, 6-DIPN operates through a non-toxic mode of action. Diisopropylnapthalene is similar in molecular structure, and functions as three sprout inhibiting compounds naturally occurring in potatoes (1-isopropyl-4,6-dimethylnaphthalene, 1-methyl-7-isopropylnaphthalene, and 4-isopropyl-1,6-dimethylnaphthalene). The three compounds found in potatoes and 2,6 DIPN are all isopropyl napthalene, a sprout inhibitor in a manner comparable to natural PGRs found in potato plants (as described above). In addition, acute toxicity studies conducted on animals indicated Toxicity Category IV for all routes of exposure and chronic studies were not triggered following the data requirements for biochemical pesticides as given in 40 CFR 158.690(c). EPA therefore has concluded that its mode of action can be classified as ''non-toxic.''

Comment 3. Mr. Forsythe expressed concerns regarding dietary intake of 2,6-DIPN, due to: (1) The synthetic nature of the compound; and (2) the lack of toxicity information to support an assessment of dietary exposure to 2,6-DIPN.

EPA Response. As discussed in the previous response, the data support the classification of 2,6-DIPN as a biochemical, based on its structural similarity to naturally occuring PGRs. In addition, the registrant has conducted a series of toxicity tests according to the requirements listed in 40 CFR 158.690, in support of experimental use permits (EUPs) and for product registration. Dietary exposure estimates were based on the assumption that 100% of the crop will be treated, and other worst-case assumptions were applied to overestimate the typical dietary exposure likely under normal conditions of use.

A 90–day oral toxicity study (MRID 450493-01) demonstrated that rats did not exhibit immune system effects, demonstrated by no changes in spleen or thymus weights and absence of lesions in spleen, thymus, and lymph nodes. The 90–day oral no observable adverse effects level (NOAEL) was 100 milligrams/kilogram/day (mg/kg/day), and the lowest observable adverse effects level (LOAEL) was 200 mg/kg/day, based on decreased body weight gain and food consumption. In a developmental toxicity study (MRID 4500010–01) in rats, the test animals did not exhibit increased fetal susceptibility to 2,6-DIPN when compared to untreated animals. The prenatal developmental toxicity NOAEL was 150 mg/kg/day and the LOAEL was 500 mg/kg/day, based on decreased fetal body weight and a possible treatment-related cartilage anomaly.

The toxicity data on 2,6-DIPN does not indicate extra sensitivity of offspring when compared with that of adult animals, but the data base does not represent a complete assessment of potential age-related sensitivity or acute effects other than lethality. The absence of a developmental toxicity study in a second species, a multigeneration reproduction toxicity study, or a range of doses adequate to induce a full range of toxic responses, especially potential acute effects in any of the available studies, required that the FQPA 10-fold safety factor be retained in defining EPA's level of concern.

Studies submitted to test the potential genotoxicity or mutagenicity of 2,6-DIPN included a reverse mutation (Ames) assay (MRID 446141–11), an unscheduled DNA synthesis assay in rat primary hepatocytes (MRID 446141–10), and a mouse micronucleus assay (MRID 446141–12); all of these were negative. A mouse lymphoma assay (MRID 454388–01) was positive at higher concentrations for mutagenicity, but since 2,6-DIPN was cytotoxic (killed the test cells) at the those concentrations where the positive results occurred (with and without metabolic activation), the test results are considered as being equivocal, or falsely positive. As a group, these four studies demonstrated that 2,6-DIPN is not a mutagen.

Information supplied by the commenter (Ref. 5) noted that ''Di-Isopropylnaphthalene(s) contained no chemical groups that would be structurally alerting for potential mutagenicity.'' Additionally, in spite of the equivocal study (MRID 454388–01), ''there was no evidence for a mutagenic effect in other *in-vitro* mutagenicity tests or in an adequately performed *in vivo* micronucleus assay in mice. The Committee agreed that no further mutagenicity testing was required.''

Based on the absence of effects on the immune system in the 90–day subchronic study, no effects on developing rats at doses below those causing maternal effects, and no genetic toxicity, Tier II and Tier III toxicity data requirements were not triggered. The Agency does not require any additional toxicity studies at this time although a livestock feeding study must be conducted as a condition of registration (see EPA Response to Comment 4).

Comment 4. Mr. Forsythe stated that, in the absence of any chronic toxicity data, ''it would be inappropriate to disregard the safety factor'' (referring to the FQPA 10-fold margin of safety to account for effects on sensitive populations, such as infants and children), and that ''threshold effects cannot be fully determined, and a safety factor would seem appropriate to address this lack of a complete data set regarding dietary exposure and chronic toxicity.''

EPA Response. As stated above, the Agency has retained the FQPA safety factor in its assessment of the dietary exposure to 2,6-DIPN.

Comment 5: Mr. Forsythe stated that the Agency should consider non-dietary and non-occupational sources of human exposure to 2,6-DIPN. The commenter submitted an EPA document (Ref. 5), in which 2,6-DIPN is described as an ''emerging pollutant'' in Lake Michigan. The document also states that polychlorinated biphenyl (PCB) substitute compounds (which include 2,6-DIPN), are ''detected in effluent, sediment, and fish in the basin; bioaccumulative and toxic.''

Additionally, the commenter provided information that European governments have expressed concerns regarding public exposure to DIPNs via the paper industry. In studies conducted by the United Kingdom Joint Food Safety and Standards Group (JFSSG), it was determined that DIPNs could be present in recycled food packaging and in packaged food (Ref. 5). DIPNs were

detected in 30 of 34 samples of retail packaging at up to 44 mg/kg, and in 6 of 10 food samples at 0.04–0.89 mg/kg.

EPA Response. Section 408(b)(2)(A)(ii) explicitly requires the Agency to find that ''there is a reasonable certainty that no harm will result from aggregate exposures, including all anticipated dietary exposures and all other exposures for which there is reliable information.'' (emphasis added). As discussed below, EPA has considered all available information on non-dietary and non-occupational exposures in establishing this temporary tolerance.

EPA reviewed the LaMP study (Ref. 5), and found that these ''emerging pollutants'' were only included in a list of chemical stressors in the lake ''as a precautionary measure, either because of their widespread use in the basin, the fact that these chemicals are beginning to show up in monitoring data, or both.'' The list of emerging pollutants listed includes: Mineral and silicone oils, di(2-ethylhexyl)phathalate (DEHP), isopropylbiphenyls, diphenylmethanes, butylbiphenyls, dichlorobenzyldichlorotoluene, phenylxylyl ethane, and diisopropylnaphthalene. The article does list PCB substitute compounds as being ''detected in effluent, sediment, and fish in the basin; bioaccumulative and toxic'' (Ref. 5). According to the Michigan LaMP (Ref. 5), ''Following the 1979 restrictions on PCB use, [these] compounds began being used in dielectric fluids, hydraulic system lubricants, and in solvents and carriers in the carbonless paper industry. Little was known about the potential impact of these (PCB) substitutes on the basin; therefore (they) were designated an emerging pollutant needing further evaluation.'' With the exception of DHEP, the Michigan LaMP goes on to state that ''other PCB substitutes (such as DIPN) have not been extensively studied; therefore, information on releases to the environment are limited.'' The article further states that information regarding the actual loading of PCB substitutes into Lake Michigan and their impact on the lake ecosystem were unknown (Ref. 5).

An environmental sampling study (Ref. 5), indicated that DIPNs and three other PCB substitutes were identified in effluent from: A de-inking/recycling paper plant and a wastewater treatment facility that received waste water from a carbonless paper manufacturing plant; fish collected near discharge points; and sediments, all of these samples were collected from the Fox River in Wisconsin. However, it is unknown whether all four PCB substitutes were found nor what concentrations were measured in each, and the study lacked environmental fate and transport data for DIPNs. Based on the statements in the LaMP study, EPA concluded that although DIPNs have been detected in a few environmental matrices, it has not been associated with any adverse effects to human health or the environment.

EPA also reviewed the JFSSG Food Surveillance Information Sheet, No. 169, January 1999. The conclusion reached by the JFSSG was that although varying amounts of DIPNs can be carried through the papermaking process to the finished product, there was no correlation between DIPN levels in food and that found in the food packaging materials.

Data was reviewed that demonstrated that 2,6-DIPN does not pose any significant bioaccumulation risk. A summary of metabolism studies/data in support of a temporary tolerance exemption on stored potatoes (PP 8G05008; Ref. 3; MRIDs 451632–01 and 451632–02) was submitted by the registrant, Platte Chemical Co., that demonstrated orally administered DIPNs were rapidly metabolized and excreted by experimental animals, and exhibited little potential for bioaccumulation (Ref. 5). Additionally, experimental animals exposed to DIPNs via inhalation did not exhibit any clinical signs of toxicity or mortality (Ref. 5). Necropsies were negative in experimental animals dosed with DIPNs in all of the aforementioned studies.

III. Aggregate Risk Assessment and Determination of Safety

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

Consistent with section 408(b)(2)(D) of the FFDCA, EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of and to make a determination on aggregate exposure, consistent with section 408(b)(2) of the FFDCA, for a tolerance for residues of 2,6-DIPN on potatoes at 3 ppm for the peels and 0.5 ppm for potato (whole) ppm. EPA's assessment of exposures and risks associated with establishing the tolerance follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children.

The classification of 2,6-DIPN as a biopesticide was based on its structural and functional similarity to 1-isopropyl-4,6-dimethylnaphthalene, 1-methyl-7-isopropylnaphthalene, and 4-isopropyl-1,6-dimethylnaphthalene which are naturally occurring plant growth regulators found in plant tissues. In addition, 2,6-DIPN is a sprout inhibitor, with a non-toxic mode of action. Therefore, the toxicity data reviewed include acute oral, dermal and inhalation toxicity studies, eye and skin irritation studies, a dermal sensitization study, subchronic feeding and developmental toxicity studies and genetic toxicity studies.

2,6-DIPN is classified in Toxicity Category IV for mammalian acute oral toxicity (lethal dose $(LD)_{50}$ > 5,000 mg/kg; OPPTS Harmonized Guideline 870.1100; 152–10; MRID 446141–04), acute dermal toxicity (LD_{50} > 5,000 mg/kg; OPPTS Harmonized Guideline 870.1200; 152-11; MRID 446141–05), and acute inhalation toxicity (lethal concentration $(LC)_{50}$ >2.60 mg/L; OPPTS Harmonized Guideline 870.1300; 152–12; MRID 446141–06), eye irritation (OPPTS Harmonized Guideline 870.2400; 152–13; MRID 446141–07) and dermal irritant (OPPTS Harmonized Guideline 870.2500; 152–14; MRID 446141–08). The active ingredient was not allergenic on skin (not a dermal sensitizer; OPPTS Harmonized Guideline 870.2600; 152–15; MRID 446141–09).

The subchronic toxicity study in rats (OPPTS Harmonized Guideline 870.3100; 152-20; MRID 450493–01) suggests a no observed effect level (NOEL) of 104 mg/kg/day (104 or 121 mg/kg/day for males and females, respectively). The lowest observed adverse effect level (LOAEL) is 208 mg/kg/day (208 and 245 mg/kg/day for males and females, respectively), based on minimal decreases in body weight gains, food consumption, adrenal effects (including increased absolute and relative organ weights and adrenal cortical hypertrophy) and kidney toxicity (evidence of tubular nephrosis in male rats).

In the rat developmental toxicity study (OPPTS Harmonized Guideline

870.3700; 152–23; MRID 450001–01), the maternal toxicity LOAEL is 150 mg/kg/day based on reduced body weight gains and food consumption. The maternal toxicity NOAEL is 50 mg/kg/day. The developmental toxicity LOAEL is 500 mg/kg/day based on reduced fetal body weights and a slightly increased incidence of a skeletal alteration (fusion of cartilaginous bands in the cervical centra). The developmental toxicity NOAEL is 150 mg/kg/day.

A mouse lymphoma gene mutation assay (OPPTS Harmonized Guideline 870.5300; 152–17; MRID 454388–01) showed that 2,6-DIPN might be mutagenic without metabolic activation at doses between 10–30 µg/mL. With metabolic activation, the results were equivocal at doses between 25–90 µg/mL. Cytotoxicity was observed in tests using the aforementioned doses, with and without metabolic activation. No genotoxicity was observed in other acceptable studies including a reverse mutation (Ames) assay (OPPTS 870.5100; 152–17; MRID 446141–11), *in vivo/in vitro* unscheduled DNA synthesis (UDS) assays in rat primary hepatocytes (OPPTS 870.5550; 152–17; MRID 446141–10), and a mouse micronucleus assay (OPPTS 870.5395; 152–17; MRID 446141–12). The collective data from the four-study mutagenicity battery demonstrates that 2,6-DIPN is not likely to be mutagenic.

B. Toxicological Endpoints

1. *Acute toxicity*. The acute toxicity studies were acceptable in accordance with the guidelines as discussed in Unit III.A. All studies were performed at a single limit dose with no observable (non-lethal) toxic endpoints.

2. *Short-term and intermediate-term toxicity*. Although the rat developmental toxicity study indicates a lower maternal NOEL (50 mg/kg/day) for similar toxicity than the subchronic toxicity study (reduced body weight, weight gain and food consumption), the maternal LOAEL of 150 mg/kg/day falls between the subchronic NOEL of 104–121 mg/kg/day and the subchronic LOAEL of 208–245 mg/kg/day. The maternal NOEL of 50 mg/kg/day from the developmental toxicity study may be appropriate for use in characterization of risks for the subpopulation of women 13–49 years of (child-bearing) age. However, the 104 mg/kg/day NOEL in the subchronic study was selected as the endpoint for short-term and intermediate-term dietary assessments since the effects observed at the subchronic LOAEL (208–245 mg/kg/day) were more thoroughly defined than the developmental effects observed at the LOAEL (500 mg/kg/day) in the developmental toxicity study, which were minimal.

A reference dose (RfD) of 1 mg/kg/day is established by dividing the 104 mg/kg/day NOEL by a 100-fold uncertainty factor (10X for interspecies extrapolation and 10X for intraspecies variability). Available developmental toxicity data on 2,6-DIPN does not indicate extra sensitivity of offspring when compared with that of adult animals, but a developmental toxicity study in a second species and a multigeneration reproduction toxicity study are needed to fully determine age-related differences in response. In addition, residues have been detected in treated potatoes under laboratory and field conditions. Therefore, the default safety factor of 10X is retained, and acute and chronic population adjusted doses (aPAD and cPAD) for dietary risk characterizations are established by dividing the RfD by 10X (accounting for age-related sensitivity for the subpopulations of infants and children). Therefore, the aPAD and cPAD are 0.1 mg/kg/day.

3. *Chronic toxicity*. An extra 10-fold uncertainty factor for the absence of chronic toxicity data were not applied to determine a RfD because 2,6-DIPN has been classified as a biochemical pesticide having a non-toxic mode of action with biological activity more specific to plants than animals. Acute toxicity studies on animals indicated Toxicity Category IV for all routes of exposure. Chronic studies are not required to support registration of biochemical pesticides unless all of the following are true:

i. Has subchronic toxicity.

ii. Its use pattern involves a significant rate, frequency or site of application.

iii. The frequency and level of human exposure are significant (40 CFR 158.690(c)).

These criteria were evaluated in the Agency's risk assessment (Refs. 1 and 2) which compared the cPAD to worst-case estimates of dietary exposure. The use pattern and exposure associated with 2,6-DIPN on potatoes in storage does not trigger chronic studies. Since the conservative exposure estimates did not result in risk characterizations exceeding the defined level of concern (exposure >100% of the cPAD).

4. *Carcinogenicity*. Based on the 90–day oral toxicity study and the genotoxicity/mutagencity studies, there were no results to indicate potential neoplastic changes, and the genetic toxicity studies did not suggest carcinogenic potential in mammalian cells.

C. Exposure Assessment

1. *Dietary exposure from food and feed uses*. There is a potential for dietary exposure to 2,6-DIPN, which can occur following its application to stored potatoes. According to the label, the plant growth regulator is to be applied at a rate of 16.6 ppm (weight/weight), and as many as three applications can be used in a storage period with a minimum interval between application and use of the treated potatoes of 30 days.

Residue profile. The submitted residue chemistry data for the use of 2,6-DIPN on potatoes is limited, and important factors in this assessment depend on default assumptions or hypothetical calculations having a low level of confidence.

For purposes of this rule, the regulated residue is considered to be 2,6-DIPN, and a potential for some accumulation of 2,6-DIPN residues in body and subcutaneous fat was observed. These results and the possible use of peels with residues from treated potatoes as livestock feed (processed potato wastes are used for this purpose) suggest that residues of 2,6-DIPN may occur in meat and milk; however, this has not been evaluated in a livestock metabolism study.

Limited field and laboratory residue data suggested tolerance levels as high as 0.5 ppm in/on whole potatoes, 3 ppm on potato peels, 1.35 ppm in meat and meat by-products, and 0.7 ppm in milk.

The analytical method for 2,6-DIPN has a level of quantification (LOQ) of 0.02 ppm and field and laboratory studies suggests that 20 ppm is a likely maximum commercial application rate for 2,6-DIPN. Residue levels expressed as 2,6-DIPN were reported at 3 ppm in potato peels and 0.5 ppm in whole potatoes.

In a published report (MRID 451632–01), the investigators noted that DIPNs could accumulate in the fat of treated rats suggesting a potential for secondary residues in meat and milk from livestock fed treated potatoes, but a livestock metabolism study was not submitted. Worst-case estimates of secondary residues were calculated for meat (1.35 ppm) and milk (0.7 ppm) of beef/dairy cattle fed waste from 2,6-DIPN-treated processed potatoes.

Supplementary metabolism information was submitted on 2,6-DIPN in rats from two published articles (MRID 451632–01). In one study, rats were given either a single dose or 30 daily oral doses, at 100 mg 2,6-DIPN per kg body weight. Residues of 2,6-DIPN were detected in all tissues 2 hours after receiving the test dose. With the

exception of body and subcutaneous fat, DIPN was not detected 48 hours after the single (100 mg/kg) dose. Peak levels in body and subcutaneous fat were found 24 hours after dosing at 75 and 85 µg/g of tissue, respectively; these levels declined to approximately 60 µg/g by 48 hours following the single dose. Results were similar in rats given the repeated doses with the peak levels in body and subcutaneous fat reported to be 150 and 90 µg/g, respectively, at 2 hours following administration of the last dose. By 30 days after this last dose was given, the 2,6-DIPN levels in fat had declined to 5 µg/g. The estimated half-life for 2,6-DIPN in fat was approximately 7 days, and the investigators noted that DIPNs had a small potential for accumulation in fat (levels increased from 2 to 7% over those found after a single dose in subcutaneous and body fat, respectively). Worst-case estimates of secondary residues were calculated for meat (1.35 ppm) and milk (0.7 ppm) of beef/dairy cattle fed waste from 2,6-DIPN-treated processed potatoes. These tolerance provide a reasonable certainty of no harm and livestock feeding studies will allow further refinement of these estimates.

In the second article, it was noted that 2,6-DIPN was metabolized in rats primarily by way of an oxidative pathway involving the isopropyl groups. Five metabolites were identified in urine from rats given an oral dose of 240 mg 2,6-DIPN per kg body weight, and the majority of the DIPN residues recovered in the urine (23% of the dose at 24 hours) was represented by 2-[6(1-hydroxy-1-methyl)ethylnaphthalen-2-yl]-2-hydroxypionic acid (17.5% of the dose). This study did not explain the fate of the remaining 77% of the administered dose. The livestock feeding study should determine the fate of the administered dose, but because worst-case estimates were used to establish the tolerances, there is a reasonable certainty of no harm.

Acute and chronic dietary exposure assessments were conducted using the Dietary Exposure Evaluation Model software (DEEM™ version 1.30) which incorporates consumption data from USDA's Continuing Surveys of Food Intakes by Individuals (CSFII, 1994–1996/1998).

For acute exposure assessments, individual 1-day food consumption data define an exposure distribution which is expressed as a percentage of the aPAD (aPAD is 0.1 mg/kg). For chronic exposure and risk assessment, an estimate of the residue level in each food or food-form on the commodity residue list is multiplied by the average daily consumption estimate for the food/food-form. The resulting residue consumption estimate for each food/food-form is summed with the residue consumption estimate for all other food/food-forms on the commodity residue list to arrive at the total estimated exposure. Exposure estimates are expressed as mg/kg body weight/day and as a percent of the cPAD (0.1 mg/kg/day). It is just as likely that the exposure estimates are appropriate, given that it is not uncommon for the peels to be eaten. These procedures were performed for each population subgroup.

As a condition of registration, the registrant will be required to submit livestock feeding studies and enforcement analytical methods for livestock and potatoes; however, EPA believes that its analyses, which rely on the available data, supplemented with conservative assumptions, are sufficient to support a tolerance for the short period during which these studies are conducted.

2. *Dietary exposure from drinking water*. Pesticide residues in drinking water are not expected to result from this use. The use is restricted to application in a commercial warehouse to stored potatoes. In addition, the label will restrict users from contaminating water supplies when cleaning equipment or disposing of equipment wash waters.

3. *From non-dietary exposure*. The term ''residential exposure'' is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiticides, and flea and tick control on pets).

2,6-DIPN is not registered for use on any sites that would result in residential exposure, but is restricted to use in commercial warehouses.

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

EPA does not have, at this time, available data to determine whether 2,6-DIPN has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, 2,6-DIPN does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that 2,6-DIPN has a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the final rule for Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997).

D. Safety Factor for Infants and Children

1. *In general*. Section 408 of the FFDCA provides that EPA shall apply an additional ten-fold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base on toxicity and exposure unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans.

2. *Prenatal and postnatal sensitivity*. The toxicity data on 2,6-DIPN does not indicate extra sensitivity of offspring when compared with that of adult animals, but the data base does not represent a complete assessment of potential age-related sensitivity or acute effects other than lethality. The following data would be necessary to allow for a complete assessment: A developmental toxicity study in a second species, a multigeneration reproduction toxicity study, or a range of doses adequate to induce a full range of toxic responses, especially potential acute effects in any of the available studies.

3. *Conclusion*. In light of the absence of a developmental toxicity study in a second species, a multigeneration reproduction toxicity study, or a range of doses adequate to induce a full range of toxic responses, especially potential acute effects in any of the available studies, EPA has retained the default 10-fold safety factor

IV. Aggregate Risks and Determination of Safety for U.S. Population, Infants and Children

1. *Acute risk*. Acute dietary exposure estimates were based on the available residue data and worst-case assumptions (Refs. 1 and 2). For the U.S. population, acute dietary exposure was estimated to be 0.023113 mg/kg. These

values represented 23.11% of the aPAD. The subpopulation with the highest acute dietary exposure estimate was children 1 to 6 years of age (0.053492 mg/kg; 53.49% of the aPAD). The acute dietary exposures to all the subpopulations in the analysis did not exceed EPA's level of concern (> 100% of the aPAD).

2. *Chronic risk*. Using the exposure assumptions described previously for chronic exposure, EPA has concluded that the chronic dietary exposure for the general population was estimated to be 0.006939 mg/kg/day, 6.9% of the cPAD. The subpopulation with the highest chronic dietary exposure estimate was children 1 to 6 years of age, with estimated exposures of 0.023247 mg/kg/day, which constitutes 23.25% of the cPAD.

3. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to the general population, and to infants and children from aggregate exposure to 2,6-DIPN residues. This includes all anticipated dietary exposures and all other exposures for which there is reliable information.

V. Other Considerations

A. Analytical Enforcement Methodology

A liquid chromatography (HPLC) method was used to measure the levels of 2,6-DIPN in the residue study.

Adequate enforcement methodology (for example, gas chromatography) is available to enforce the tolerance expression. The method may be requested from: Chief, Analytical Chemistry Branch, Environmental Science Center, 701 Mapes Rd., Ft. Meade, MD 20755–5350; telephone number: (410) 305–2905; e-mail address: *residuemethods@epa.gov*.

B. International Residue Limits

There are no Codex Alimentarius Commission (Codex) maximum residue levels for residues of 2,6-DIPN.

VI. Conclusion

Based upon the risk assessment, residue data and use pattern described above, a temporary tolerance is established for residues of 2,6-DIPN in raw potatoes and potato peel at 0.5 ppm and 3 ppm respectively.

VII. Objections and Hearing Requests

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

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

You must file your objection or request a hearing on this regulation in accordance with the instructions provided in this unit and in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket ID number OPP–2003–0127, in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk on or before October 7, 2003.

1. *Filing the request*. Your objection must specify the specific provisions in the regulation that you object to, and the grounds for the objections (40 CFR 178.25). If a hearing is requested, the objections must include a statement of the factual issues(s) on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the objector (40 CFR 178.27). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

Mail your written request to: Office of the Hearing Clerk (1900C), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001. You may also deliver your request to the Office of the Hearing Clerk in Rm. 104, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. The Office of the Hearing Clerk is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Office of the Hearing Clerk is (703) 603–0061.

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

EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding the waiver of these fees, you may contact James Tompkins by phone at (703) 305–5697, by e-mail at tompkins.jim@epa.gov, or by mailing a request for information to Mr. Tompkins at Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001.

If you would like to request a waiver of the tolerance objection fees, you must mail your request for such a waiver to: James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001.

3. *Copies for the Docket*. In addition to filing an objection or hearing request with the Hearing Clerk as described in Unit VI.A., you should also send a copy of your request to the PIRIB for its inclusion in the official record that is described in Unit I.B.1. Mail your copies, identified by docket ID number OPP–2003–0127, to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001. In person or by courier, bring a copy to the location of the PIRIB described in Unit I.B.1. You may also send an electronic copy of your request via e-mail to: opp-docket@epa.gov. Please use an ASCII file format and avoid the use of special characters and any form of encryption. Copies of electronic objections and hearing requests will also be accepted on disks in WordPerfect 6.1/8.0 or ASCII file format. Do not include any CBI in your electronic copy. You may also submit an electronic copy of your request at many Federal Depository Libraries.

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

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

VIII. References

1. EPA Memorandum. Roger Gardner to Manying Xue. ''Addendum to a previous review of a petition for an exemption from the requirement of a tolerance (PP# 1F06338) for 2,6-DIPN (PC 055803) in/on stored potatoes (EPA File Symbol No. 34704–IUE; DP Barcodes D276743 and D276753; Submission Nos. S601233 and S601234).'' March 7, 2003.

2. EPA Memorandum. Roger Gardner to Driss Benmhend. ''Petition for an exemption from the requirement of a tolerance (PP# 1F06338) for 2,6-DIPN (PC 055803) in/on stored potatoes (EPA File Symbol No. 34704–IUE; DP Barcodes D276743 and D276753; Submission Nos. S601233 and S601234).'' December 10, 2002.

3. EPA Memorandum. Russell S. Jones to Driss Benmhend. ''Renewal Request for an Experimental Use Permit for Amplify® Sprout Inhibitor (EPA Symbol No. 034704–EUP–13), containing 99.7% 2,6-Diisopropyl-napthlalene [2,6-DIPN; (Chemical No. 055803)] as its Active Ingredient; and a Petition to Extend the Temporary Exemption from the Requirement of a Tolerance on Stored Potatoes (PP# 8G05008). Review of Toxicity, Metabolism, and Residue Chemistry Studies. DP Barcodes D267369 and D267587; Case Nos. 062532 and 290334; Submission Nos. S581969 and S582755; MRIDs 451632–01 and –02.'' August 3, 2000.

4. EPA Memorandum. Russell S. Jones to Driss Benmhend. ''Amplify® Sprout Inhibitor (EPA Symbol No. 034704–EUP–13), containing 99.7% 2,6-Diisopropyl-napthlalene [2,6-DIPN; (Chemical No. 055803)] A New Active Ingredient; and a Petition For Exemption from the Requirement of Tolerances for 2,6-DIPN on Food Commodities (PP# 1F06338). Response to Comments Received Following Publication of an FR Notice Regarding a Request for a Tolerance Exemption for 2,6-DIPN. DP Barcode D278840; Case No. 070700; Submission No. S601234; No MRID Nos.'' August 7, 2000.

5. Lake Michigan Lakewide Management Plan (LaMP Study). United States Environmental Protection Agency, Office of Water, Chapter 5 pp 5-125. April 2000.

IX. Statutory and Executive Order Reviews

This final rule establishes a temporary tolerance under section 408(d) of the FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). Because this rule has been exempted from review under Executive Order 12866 due to its lack of significance, this rule is not subject to Executive Order 13211, *Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use* (66 FR 28355, May 22, 2001). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4). Nor does it require any special considerations under Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994); or OMB review or any Agency action under Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). Since tolerances and exemptions that are established on the basis of a petition under section 408(d) of the FFDCA, such as the temporary tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. In addition, the Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999). Executive Order 13132 requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.'' This final rule directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of section 408(n)(4) of the FFDCA. For these same reasons, the Agency has determined that this rule does not have any ''tribal implications'' as described in Executive Order 13175, entitled *Consultation and Coordination with Indian Tribal Governments* (65 FR 67249, November 6, 2000). Executive Order 13175, requires EPA to develop an accountable process to ensure ''meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.'' ''Policies that have tribal implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on one or more Indian tribes, on the relationship between the Federal Government and the Indian tribes, or on the distribution of power and responsibilities between the Federal Government and Indian tribes.'' This rule will not have substantial direct effects on tribal governments, on the relationship between the Federal Government and Indian tribes, or on the distribution of power and responsibilities between the Federal Government and Indian tribes, as specified in Executive Order 13175. Thus, Executive Order 13175 does not apply to this rule.

X. Congressional Review Act

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General

of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this final rule in the **Federal Register**. This final rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

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

Dated: July 31,2003.

James Jones,

Director, Office of Pesticide Programs.

■ Therefore, 40 CFR chapter I is amended as follows:

PART 180—[AMENDED]

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

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

■ 2. Section 180.590 is added to subpart C to read as follows:

§ 180.590 2,6-Diisopropylnaphthalene (2,6-DIPN); tolerances for residues.

(a) *General.* Tolerances are established for residues of 2,6-Diisopropylnaphthalene (2,6-DIPN) in or on the following commodities:

Commodity	Parts per million	Expiration/ revocation date
Meat	1.35	5/31/06
Meat byproducts	1.35	5/31/06
Milk	0.7	5/31/06
Potatoes (peel)	3	5/31/06
Potatoes (whole)	0.5	5/31/06

(b) Section 18 emergency exemptions. [Reserved]

(c) Tolerances with regional registrations. [Reserved]

(d) Indirect or inadvertent residues. [Reserved]

§ 180.1208 [Removed]

■ 3. Section 180.1208 is removed.

[FR Doc. 03–20307 Filed 8–7–03; 8:45 am]

BILLING CODE 6560–50–S

FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 54

[CC Docket No. 96–45; FCC 03–188]

Federal-State Joint Board on Universal Service: Children's Internet Protection Act

AGENCY: Federal Communications Commission.

ACTION: Final rule.

SUMMARY: In this document, the Commission adopts measures to ensure that its implementation of the Children's Internet Protection Act (CIPA) complies with the recent decision of the United States Supreme Court. CIPA requires schools and libraries with ''computer Internet access'' to certify that they have Internet safety policies and technology protection measures, *e.g.*, software filtering technology, to receive discounts for Internet access and internal connections under the schools and libraries universal service support mechanism (e-rate).

DATES: The rule and the revised FCC Forms 479 and 486 in this document contain collection requirements that have not been approved by OMB. Upon OMB approval, the Commission will publish a document in the **Federal Register** announcing the effective date of the rule and the revised FCC Forms 479 and 486.

FOR FURTHER INFORMATION CONTACT: Jennifer Schneider, Attorney, Wireline Competition Bureau, Telecommunications Access Policy Division, (202) 418–7400.

SUPPLEMENTARY INFORMATION: This is a summary of the Commission's Order in CC Docket No. 96–45 released on July 24, 2003. The full text of this document is available for public inspection during regular business hours in the FCC Reference Center, Room CY–A257, 445 Twelfth Street, SW., Washington, DC 20554.

I. Introduction

1. In this Order, we adopt measures to ensure that our implementation of the Children's Internet Protection Act (CIPA) complies with the recent decision of the United States Supreme Court. CIPA requires schools and libraries with ''computer Internet access'' to certify that they have Internet safety policies and technology protection measures, *e.g.*, software filtering technology, to receive discounts for Internet access and internal connections under the schools' and libraries' universal service support mechanism (e-rate).

2. Libraries subject to CIPA's filtering requirements that are not currently in compliance with the CIPA filtering requirements must undertake efforts in Funding Year 2003 to comply by Funding Year 2004 in order to receive e-rate funds. Libraries must be in compliance with the CIPA requirements by Funding Year 2004, except to the extent such libraries are eligible for and receive a waiver of the CIPA requirements pursuant to section 254(h)(6)(E)(ii)(III). We direct the Administrator in consultation with the Wireline Competition Bureau (Bureau) to implement the necessary procedural changes, including changes to the current CIPA-related certifications required of applicants. We take these steps to respond promptly to the Supreme Court's decision and to ensure that the schools' and libraries' universal service support mechanism continues to operate in accordance with federal law.

II. Discussion

3. Consistent with the Supreme Court decision, as of the effective date of this Order, we lift the suspension of enforcement of those § of 54.520 of our rules which implemented the section 254(h)(6) requirement that libraries have Internet filtering technology to receive discounts for Internet access and internal connections under e-rate. Specifically, we lift the suspension of enforcement of §§ 54.520(c)(2)(i) and (iii), 54.520(c)(3), 54.520(d), and 54.520(g)(1) of our rules as applied to libraries. In addition, we modify § 54.520(f) and (g) to conform with the revised timeline for the implementation of section 254(h)(6) of the Act.

4. Consistent with the implementation framework established by Congress, libraries receiving e-rate discounts for Internet access or internal connections shall have one year from July 1, 2003, which is the start of Funding Year 2003, to come into compliance with the filtering requirements of CIPA. When Congress enacted CIPA in 2001, it recognized that it may take libraries a significant amount of time to procure and install the Internet filtering technology required to comply with CIPA. Accordingly, CIPA allows libraries either to certify (1) that they are in compliance with CIPA or (2) that they are ''undertaking such actions, including any necessary procurement procedures, to put in place'' the required policy measures to comply with CIPA for the next funding year. Given that the Supreme Court decision was issued on June 23, 2003 and will be effective no sooner than July 18, 2003,