

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)phthalate (DEHP), isopropylbiphenyls, diphenylmethanes, butylbiphenyls, dichlorobenzylchlorotoluene, phenylxylyl ethane, and diisopropyl-naphthalene. 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-isopropyl-naphthalene, 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)<sub>50</sub> > 5,000 mg/kg; OPPTS Harmonized Guideline 870.1100; 152–10; MRID 446141–04), acute dermal toxicity (LD<sub>50</sub> > 5,000 mg/kg; OPPTS Harmonized Guideline 870.1200; 152–11; MRID 446141–05), and acute inhalation toxicity (lethal concentration (LC)<sub>50</sub> > 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