technologies that can achieve compliance with the existing regulations. For future regulations, EPA must determine affordable treatment technologies that can achieve compliance for each of the size categories. Within 2 years of the SDWA Amendments of 1996, EPA must list technologies that achieve compliance with all existing regulations. The List of Compliance Technologies for Small Drinking Water Systems to meet the present SWTR is required to be published by August 1997.

B. Request for Stakeholder Involvement

The upcoming meeting deals specifically with EPA's efforts to compile the initial list of compliance technologies for the SWTR. EPA would like to review the initial list of compliance technologies with stakeholders as well as obtain their inputs on additional technologies that should be considered when EPA updates this list in a year.

The meeting will be divided into two parts. The first part involves getting feedback from stakeholders on the EPA proposed list of Compliance Technologies for the SWTR which will be distributed in the background materials to those registered for the meeting. The second part involves getting ideas and insights from stakeholders on approaches to the national level affordability criteria that will be used to determine which pathway (compliance technology or a variance) a system will proceed along and which technologies would be available for the system. The issues on affordability criteria do not apply to the first list of technologies for the SWTR; however, they will apply to future rules and EPA therefore wants to begin to get input on these issues.

The specific issues for discussion at the meeting will be based on the above-mentioned material and will include (but may not be limited to) the following:

1. The compliance technologies for the filtration component of the SWTR will include some technologies that would fall under the ''other filtration technologies'' as per § 141.73(d). The pilot testing for viability would be waived for those technologies on the compliance technology list. These technologies would be treated like the filtration technologies in § 141.73(a)–(c). Testing to ensure that the system is capable of operating the treatment technology may still be required for these other filtration technologies and the technologies directly identified in the SWTR. What are the stakeholder's opinions about this approach for the other filtration technologies?

2. Are there Point-Of-Entry units available that could be used to meet the requirements of the Surface Water Treatment Rule? Is it a manageable option?

3. The primary role of the national-level affordability criteria is to direct a system either into a compliance technology pathway or a variance technology pathway. If the national-level affordability criteria are set very high, then the variance technology pathway will be limited or eliminated and systems will need to install compliance technologies. If the national-level affordability are set very low, the compliance technology pathway will be limited or eliminated and more systems will operate under small system variances. What components should be included in the national-level affordability criteria? What is the best measure of national-level affordability?

4. The initial list of compliance technologies will be similar to the list of disinfection and filtration technologies in the SWTR. What level of detail would stakeholders like to see on the compliance technologies when the list is updated in August, 1998? Is the compliance technology list the best mechanism to incorporate applicability ranges?

5. Do stakeholders recommend any specific criteria for distinguishing treatment applications, in relation to the 3 small system categories specified under SDWA? Would design, operational and management capability, chemical reactivity and/or a hazard posed by some technologies (e.g., chlorine dioxide, chlorine gas) be good parameters to consider within the <10,000 population PWS categories?

The public is invited to provide comments on the issues listed above and other issues related to the List of Compliance Technologies for Small Drinking Water Systems and the Affordable Criteria during the July 22–23, 1997 meeting or in writing by August 12, 1997.

Dated: June 19, 1997.

Cynthia Dougherty,

Director, Office of Ground Water and Drinking Water, Environmental Protection Agency.

[FR Doc. 97–16653 Filed 6–24–97; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–736; FRL–5719–6]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–736, must be received on or before July 25, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI 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 record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager/Regulatory Leader	Office location/telephone number	Address
Elizabeth Haeberer	Rm. 207, CM #2, 703–308–2891, e-mail:haeberer.elizabeth@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Indira Gairola (Reg. Leader).	4th floor, CS #1, 703–308–8371, e-mail: gairola.indira@epamail.epa.gov.	2800 Crystal Drive, Arlington, VA

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–736] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–736] and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Food additives, Feed additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: June 9, 1997.

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Gaylord Chemical Corporation

PP 5E4592

EPA has received a Supplement to a Petition (PP 5E4592) from Gaylord Chemical Corporation, P.O. Box 1209, Slidell, LA 70459-1209, proposing, pursuant to section 408(d)(3) the Federal Food, Drug and Cosmetic Act, 21 U.S. C. section 346a(d), to amend 40 CFR 180.1001(d) to extend the existing exemption from a tolerance for residues of the inert ingredient DMSO [dimethyl sulfoxide] by permitting its use in pesticide formulations applied to the edible parts of food or feed crops. DMSO may currently be used as a solvent or cosolvent in end-use pesticides that are applied before crop emergence or prior to the formation of edible parts of food plants.

Dimethyl sulfoxide (DMSO) is widely used as a solvent in industry, in chemical and biochemical research, and in medicines. DMSO readily penetrates the skin and has proven to be an effective carrier of various pharmaceutical agents into the body. It is currently used in veterinary medicinal formulations as well as being used medicinally in its own right. DMSO has been shown to relieve pain and reduce swelling when applied dermally to acute sprains and strains. It is approved for a variety of human prescriptions in over 125 countries. In the United States, DMSO is FDA-approved for treatment of musculoskeletal injuries in horses, acute or chronic otitis in dogs, and interstitial cystitis in humans. In Canada, DMSO is approved for the treatment of scleroderma while in Germany it is approved for the treatment of sports injuries and in the United Kingdom for treatment of herpes zoster.

On August 21, 1995, Gaylord Chemical Corporation (Gaylord) submitted to the EPA a tolerance exemption petition (PP 5E4592) entitled ''Petition for Extension of Existing Exemption from Tolerance for the Inert Ingredient, DMSO''. That petition proposed to amend 40 CFR part 180.1001(d) by allowing DMSO to be applied to the edible parts of food and feed crops when used in end-use pesticide formulations as a solvent or a cosolvent at up to 10 percent of finished sprays or tank mixes. Gaylord now proposes to amend their petition to clarify that DMSO is intended for applications at not more than 5 lbs. DMSO per acre when used as a solvent or cosolvent in end-use pesticide formulations applied to the edible parts of food and feed crops.

Pursuant to the section 408(d)(2)(A)(i) of the FFDCA, as amended, Gaylord Chemical Corporation has submitted the following summary of information, data and arguments in support of their tolerance exemption petition. This summary was prepared on behalf of Gaylord Chemical Corporation and EPA has not fully evaluated the merits of the petition. The summary may have been edited by EPA if the terminology used was unclear, the summary contained extraneous material, or the summary was not clear that it reflected the conclusion of the petitioner and not necessarily EPA.

Based on petition PP 5E4592, as amended, by the supplemental information presented herein, Gaylord Chemical Corporation concludes that the expanded use of DMSO in pesticide end-use formulations applied to the edible parts of food and feed crops will not result in DMSO dietary exposures of toxicological consequence for the following reasons: (1) DMSO is widely distributed and naturally-occurring in plants and the environment; (2) DMSO is extensively metabolized by plants following either root or foliar uptake; (3) When ingested or dermally applied,

DMSO is practically non acutely toxic, nor is it genotoxic or carcinogenic; (4) DMSO is rapidly metabolized and excreted by animals and humans without any evidence of bioaccumulation; (5) DMSO is not anticipated to cause any cumulative effects; and (6) There is no evidence that DMSO is an endocrine disruptor.

A. Proposed Use Practices of DMSO

DMSO is a pesticidally inert ingredient that currently is exempted [40 CFR (180.1001(d)] from the requirement of a tolerance for residues when used as a solvent or cosolvent in pesticide formulations applied before crop emergence from the soil or prior to formation of edible parts of food plants. There are no other limits for DMSO expressed in 40 CFR (180.1001(d). The proposed amended use would allow DMSO applications at not more than 5 lbs. DMSO per acre when used as a solvent or cosolvent in end-use pesticide formulations applied to the edible parts of food and feed crops.

B. Natural Occurrence of DMSO

Researchers have estimated that approximately 20 - 60 billion pounds of DMSO are created in the atmosphere each year from naturally-occurring, atmospheric dimethyl sulfide (DMS). DMSO is also found in natural waters, where it is believed to be produced by photochemical oxidation of dimethyl sulfide (DMS) generated by algae and phytoplankton. There is also evidence that DMSO is found naturally in soils and is metabolized by a variety of microorganisms, resulting in volatilization of sulphur from soil.

Naturally-occurring DMSO has been identified in alfalfa, asparagus, barley, beans, beets, cabbage, corn, cucumbers, oats, onions, Swiss chard, tomatoes, apples, raspberries, spearmint, beer, milk, coffee and tea. DMSO concentrations in fresh fruit, vegetables and grains ranged from undetectable (<0.05 parts per million (ppm) to 1.8 ppm. In processed products such as sauerkraut or tomato paste, concentrations of DMSO ranged from <0.05 to 3.7 ppm. DMSO was also found in milk (0.13 ppm), lager beer (1.4 ppm), coffee (2.6 ppm) and black tea (16.0 ppm). In forage crops such as alfalfa and corn silage, DMSO levels were 0.17 and 0.31 ppm, respectively.

C. Product Identity/Chemistry

1. *Identity of inert compound and corresponding residues.* Dimethyl sulfoxide (CAS number 67–68–5) is commonly known and abbreviated as DMSO. Other names for DMSO are sulfinylbismethane and methyl sulfoxide. The molecular weight of DMSO is 78.13, the empirical formula is C2H6SO, and the structural formula is (CH3)2SO. DMSO is a very hygroscopic liquid with practically no odor or color. Residues of DMSO include DMSO2 (dimethyl sulfone) and DMS (dimethyl sulfide).

2. *Plant metabolism*. The metabolism of DMSO in plants is well understood. Extensive studies have shown that: (1) DMSO is absorbed by plant roots and foliage; (2) translocation of DMSO is primarily upward and associated with the transpirational stream; (3) metabolism of DMSO is primarily occurs in the foliage; (4) DMSO is metabolized to DMSO2 by oxidation, to volatile DMS by reduction and to components that are incorporated into sulfur-containing amino acids and proteins; (5) DMSO does not accumulate in plant tissues; and (6) the amount of residue is dependent on the time since application.

3. *Analytical methods*. Validated analytical methods for residues of DMSO in or on plant and animal tissues are available. DMSO is extracted from the samples, analyzed by gas chromatography using a flame photometric detector operating in the sulfur mode and quantified by comparison to external standards.

4. *Magnitude of the residues*. In 1 study, 15 food or feed crops were treated with DMS35O at a rate of 5 lbs per acre 24 hours before harvest. The maximum total radioactive residue (TRR) found in forage crops was 39.16 ppm. Among the food crops, grain from fall-planted barley had maximum total S35 residues (5.38 ppm), while red raspberries had residues of 1.81 ppm. All of the other treated crops had residues less than 1 ppm with those in or on sweet corn, cabbage, apples, onions and dried beans at less than 0.01 ppm.

A series of studies were also conducted to determine the types of residues and the level of S35 in milk and tissues of lactating goats and in eggs and tissues of chickens fed 20, 60 or 200 ppm DMS35O in the diet for 28 days. Summary results are: (1) the maximum amounts of DMS35O in milk, eggs, and goat and chicken tissues from the 20 ppm DMS35O feeding level were 0.06, 0.28, 0.20 and 0.44 ppm, respectively, and TRR was 0.64, 3.00, 3.86 and 2.13 ppm, respectively ; (2) most of the DMS35O activity fed to the test animals was eliminated or metabolized to DMS35O2 and higher molecular weight S35-bearing compounds; (3) total S35 and DMS35O activities in milk and eggs remained fairly constant within each feeding level for the 28-day feeding period (i.e., no accumulation of S35 activity with time); (4) there was no accumulation of total S35 activity in chicken and goat tissues at any feeding level; and (5) the largest amounts of total S35 activity were found in goat liver and kidney and in chicken liver and muscle.

D. Toxicological Profile

1. *Acute toxicity*. DMSO has low acute toxicity and is practically non-toxic (LD_{50} > 5 g/kg) by ingestion or dermal application. Rat oral LD_{50}s are reported from 14.5 to 28.3 g/kg, whereas LD_{50}s for mice have been reported from 16.5 to 24.6 g/kg. The acute dermal LD_{50} is 40 g/kg for the rat and 50 g/kg for the mouse, while dermal LD_{50}s > 11 g/kg are reported for both dogs (beagles) and primates (rhesus monkeys). The acute rat inhalation LC_{50} > 1.6 mg/l, the only dose level tested, and which is also the no-observed-effect-level (NOEL). Although DMSO can cause skin and eye irritation, it is not a skin sensitizer.

2. *Genotoxicity*. DMSO is not mutagenic to Salmonella, Drosophila, and fish cell cultures. Because DMSO is not considered to be mutagenic, it is widely used as a solvent in mutagenicity testing. Although DMSO is bacteriostatic or bactericidal at concentrations of 5-50 percent, there is no evidence that DMSO causes chromosomal aberrations at levels that are not directly toxic to cells. In vivo cytogenetic studies with primates receiving orally or dermally administered DMSO showed no abnormalities in bone marrow smears. There are no documented adverse genetic effects reported as a result of medicinal DMSO uses (including quasi-medicinal uses for treatment of arthritis or sprains and strains). Additionally, no adverse genetic effects have been reported from occupational exposure to DMSO in over 40 years of industrial use.

3. *Reproductive and developmental toxicity*. A mouse teratology NOEL of 12 g/kg/day has been established based on research with a 50 percent DMSO solution administered orally. Additional teratogenicity studies of orally administered DMSO to pregnant mice, rats, rabbits and guinea pigs have demonstrated that DMSO is not a teratogen in mammals except at high levels that cause overt maternal toxicity and are coincident with the maximum tolerated dose. The data suggest that DMSO is not teratogenic at low levels regardless of the route of administration. Finally, the teratogenic potential of DMSO is dependent on the route of administration, the dose level and gestation stage at exposure.

4. *Subchronic toxicity*. A subchronic rat inhalation study established a NOEL at 200 mg/m^3 (0.2 mg/l), the only concentration tested. Extensive monitoring of human patients have shown that DMSO does not affect human renal function. DMSO is a diuretic but no sign of kidney damage has been found in humans or laboratory animals after repeated DMSO treatment.

5. *Chronic toxicity*. DMSO is not listed as a carcinogen by IARC, NTP, OSHA or ACGIH, based on reviews of numerous studies. In fact, a study supported by the US Public Health Services concluded that DMSO was not a carcinogen and is a safe carrying agent analogous to mineral oil. An 18–month study with rhesus monkeys established an oral NOEL of 3 g/kg/day. No tumors were observed and bone marrow smears from the monkeys that received oral or topical doses of DMSO at up to 9 g/kg/day for 18 months showed no DMSO effects. A 78–week rat study revealed no increases in mortality or tumors and established an oral NOEL of 3.3 g/kg/day based on hematology and ocular effects. If one considers the rhesus monkey to be the most appropriate model for extrapolation to humans, the oral monkey NOEL of 3 g/kg/day is comparable to an average human (70 kg) consuming approximately 210 g DMSO per day. Continuing research has demonstrated that the ocular effects reported from DMSO treatment of dogs, rabbits, guinea pigs and swine are species-specific and not reproducible in primates, including humans. In fact, 84 humans that have received daily topical treatment of 2.6 g DMSO/kg/day for up to 3 months showed no DMSO-related effects beyond occasional skin irritation and garlicky breath and body odor.

6. *Human and animal metabolism*. DMSO is metabolized in humans by oxidation to DMSO2 or by reduction to DMS. DMSO and DMSO2 are excreted in the urine and feces. DMS is eliminated through the breath and skin with a characteristic garlicky or oyster-like odor. Human excretion of orally administered DMSO is complete within 120 hours, with up to 68 percent as unchanged DMSO and 21-23 percent as DMSO2 excreted in the urine. The rate of renal clearance has been shown to be similar for chronic and singly administered doses regardless of dose concentration. No residual accumulation of DMSO has been reported in humans or lower animals who have received DMSO treatment for protracted periods of time, regardless of route of dose administration.

7. *Metabolite toxicity*. The metabolites of DMSO are DMSO2, which is naturally-occurring at low levels in human urine, and DMS, which is naturally-occurring in plants, the atmosphere, and lakes and oceans. Both of these metabolites are readily excreted from the body. Based on their widespread natural occurrence and ready degradation and/or excretion, the production of these metabolites from the proposed use of DMSO on food producing plants is not expected to pose any toxicological concern.

E. Aggregate Exposure

1. *Dietary exposure*. While potential dietary exposure is usually determined by multiplying the residue tolerance level for each exposed food or feed crop by its dietary consumption data then summing the residue contributions from all dietary sources, this method is not possible for DMSO for the following reasons: (1) because DMSO is naturally-occurring in many plants as well as in natural waters, the daily intake of endogenous DMSO is unknown; (2) residue data are only available for some of the raw agricultural commodities (RAC) that may potentially be exposed to DMSO from its proposed use in pesticides; and (3) it is unknown at this time which RACs will be exposed to DMSO used in pesticides applied to edible crop parts.

However, one can broadly estimate dietary exposure based on certain assumptions and/or generalizations, the available residue data to estimate conservative residue levels in broad crop groupings, and dietary consumption information for categories of food commodities. For example, information on per capita consumption data provided by food and nutrition specialists allows the following estimate of daily food consumption: meat - 0.5 lbs, dairy - 1.0 lbs, fruit and vegetables - 2.0 lbs and grains - 2.0 lbs, for a daily food consumption of 5.5 lbs or 2.5 kg food per day.

2. *Food*. When DMSO is applied at up to 5.0 lbs/acre to the edible parts of food and feed crops, dietary exposure to DMSO can be estimated from naturally-occurring DMSO levels in various food and feedstuffs in combination with those from crops harvested 24 hours after DMSO application. Maximum theoretical DMSO residues were 0.5 to 4 ppm in or on fruits and vegetables, up to about 10 ppm in or on small grains, and up to about 40 ppm in or on forage grasses and legumes.

Theoretical residues of DMSO in the human diet from meat and dairy products were determined from theoretical animal diets, the available crop residue data converted to dry weight basis and residue data from animal feeding studies. Based on these estimates of DMSO in bovine and poultry diets, bovine meat (liver) and milk would contribute 19.2 ppm and 8.0 ppm DMSO to the human diet, respectively, while poultry meat (liver) and eggs would contribute 2.1 ppm and 3.0 ppm DMSO to the diet.

Using the available residue data for DMSO in the raw agricultural commodities (RACs) and animal products in concert with dietary consumption information, total daily dietary intake of DMSO in human diet would be 0.0207 g (20.7 mg) DMSO. DMSO levels (ppm) in the human diet from endogenous sources and the proposed uses of DMSO in pesticide formulations are estimated to be 8.66 ppm. For dietary risk calculations, a more conservative value of 10.0 ppm will be used for estimated DMSO levels in human diet.

3. *Drinking water*. Based on the natural occurrence of DMSO in the environment, its chemical and biological characteristics and little-to-no mobility in soil, the expanded agronomic usage of DMSO is not expected to significantly increase drinking water exposures to DMSO. DMSO is found in many natural waters but concentrations are dependent on DMSO producing algae and other natural variables. It is unknown if or at what levels DMSO would be found in municipal or private water systems. Any DMSO that may be oversprayed to the soil from applications to crops would be rapidly metabolized by a wide variety of microorganisms, thereby diminishing ground or surface water exposure to DMSO. Additionally, environmental studies have shown little-to-no mobility of DMSO in the soil. Finally, DMSO is already cleared as a pesticidal inert for use in products applied to crops. Therefore, the proposal to expand the application timing of DMSO from early in the cropping season to include the entire cropping season would not be expected to significantly increase exposure of drinking water sources to DMSO.

4. *Non-dietary exposure*. The only anticipated human exposure to DMSO from non-dietary sources would be through occupational exposure, medicinal or quasi-medicinal uses of DMSO. DMSO applied to plants is rapidly absorbed and metabolized. When oversprayed to soils during agronomic use, DMSO is metabolized by a wide variety of soil microorganisms. DMSO is legally and readily available in health stores in many states and is reportedly used as a unregistered topical treatment for arthritis, muscle strains and sprains and bursitis. However, while these uses are not FDA-approved,

they have been practiced for 30 to 40 years with no documented ill effects beyond skin irritation to humans. Dermal exposure to very low levels of naturally-occurring DMSO may also occur from swimming in lakes or in the ocean.

F. Cumulative Effects

There is no reliable information to indicate that DMSO has a common mechanism of toxicity with any other chemical compound. Therefore, for cumulative exposure considerations, Gaylord believes it is appropriate to consider only the potential risks of DMSO.

Metabolism studies in humans and animals have shown that DMSO is not bioaccumulative. Since DMSO is naturally-occurring in many if not most fruits, vegetables and grains, is readily metabolized and eliminated, and has low toxicity, there would not be any anticipated increased human risk or adverse effects from DMSO applied to edible parts of plants. Plant-eating animals, including humans, ingest endogenous DMSO on a daily basis throughout their life as part of the normal diet. Ingestion of low-level DMSO residues resulting from agronomic use of DMSO will not increase the body burden of this efficiently metabolized and excreted compound.

G. Endocrine Effects

In light of the ubiquitous natural occurrence of this compound and the absence of any reported endocrine effects from any of the toxicity studies (even at very high dose levels), DMSO is not considered to be an endocrine disruptor. DMSO is found naturally in the environment, in natural waters and in most foods and feeds. Studies have shown that DMSO applied to plants is metabolized and incorporated into amino acids and other sulfur-containing plant components. Animal and human metabolism studies have shown that DMSO is predominantly eliminated ''as is'' or metabolized to DMSO2 and DMS prior to elimination. Several studies in which different species (i.e. rat, mouse, rabbit, hamster) were administered DMSO at high levels (up to lethal levels) have shown no effect on the time-to-mating or on mating and fertility indices. Radiolabeled DMS35O fed to chickens (laying hens) for 28 days had no effect on the ability of the hens to produce eggs. This wealth of data suggests that there are no effects on the estrous cycle, on mating behavior, or on male or female fertility. Chronic and subchronic studies in rhesus monkeys, mice, rats and dogs have not demonstrated any evidence of toxicity to the male or female reproductive tracts.

H. Safety Determination

1. *US population*. Based on the human NOEL of 2.6 g/kg/day and very conservative assumptions about DMSO residue levels in food/feed from natural occurrence and from the proposed expanded agronomic usage of DMSO, it would be impossible for humans to ingest toxicologically consequential levels of DMSO. DMSO is naturally present in most edible plants and animal products (i.e. milk, eggs, etc.). The proposed use of DMSO on edible parts of food crops would not add appreciably to naturally-occurring DMSO levels except for forage crops. Even when residues in or on forage crops and maximum anticipated residues from animal tissues/products are considered, total theoretical maximum levels of DMSO in the diet are still considerably below levels that would be of toxicological concern.

There is ample information to determine a reference dose (RfD) of 0.03 g DMSO/kg body weight/day based on data from chronic oral studies with rhesus monkeys. NOELs established by chronic oral studies vary from 3.0 g/kg/day for a monkey oral study to 12 g/kg/day for a mouse teratology study. Since dogs are the most sensitive specie tested using the oral route of exposure, based on lenticular effects, it would seem appropriate to use a dog study to establish the RfD for conducting a dietary exposure assessment. However, since rhesus monkeys are physiologically more closely related to humans than dogs, and the lenticular effect observed in dogs has never been documented in primates or humans in over 30 years of testing, the primate oral NOEL of 3 g/kg/day would be more relevant for use in human dietary risk assessments. Since the NOEL was established in a non-human it is appropriate to use an uncertainty factor (UF) of 100X (using current EPA criteria of 10X for intra-species variability and 10X for inter-species variability, 10 x 10 = 100). The data from the multigeneration studies indicate that there is no increased risk to neonates or young when DMSO is administered orally; therefore, an extra safety factor for the protection of infants and children is not warranted. This would result in a UF of 100X and a RfD of 0.03 g/kg/day or 30 mg/kg/day DMSO. For an average adult (70 kg) this is equivalent to 2.1 g DMSO/day, which is lower than therapeutic levels (i.e., 2.6 g/kg/day) that have shown no adverse effects in humans.

Since the RfD of 0.26 g/kg/day calculated from human data is based on a 3–month exposure period, the more conservative RfD of 0.03 g/kg/day calculated from monkey data, based on a 18–month exposure period, will be used in conducting the DMSO lifetime risk assessment. Using the compounded and extremely conservative exposure assumptions described above and the very conservative RfD of 0.03 g DMSO/kg/day, the aggregate human exposure to DMSO from its proposed agronomic use will utilize only 0.99 percent [(0.0207 g DMSO/day in diet) ((0.03 g/kg/day x 70 kg body wt) = 0.0207g DMSO/day anticipated (2.1 g/day DMSO allowed = 0.00985] of the RfD for the US population (based on estimated average consumption of 2.5 kg food/day for an average 70 kg adult). EPA generally has little concern for exposures below 100 percent of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable health risks to humans. Thus, based on the natural occurrence of DMSO in the human diet, DMSO's low toxicity, the ability of humans to readily metabolize DMSO, and very low aggregate dietary exposure, Gaylord concludes with reasonable certainty that no harm will result from aggregate human exposure to residues from the proposed use of DMSO in pesticide products applied to the edible parts of food and feed crops.

2. *Infants and children*. The proposed use of DMSO in pesticide products applied to the edible parts of plants will pose no additional risk of adverse effects to infants or children. Human infants and children are exposed to endogenous levels of DMSO and readily metabolize and excrete this compound. Even so, when assessing the potential for additional sensitivity of infants and children to DMSO and its residues, it is appropriate to consider the results of the developmental and reproductive studies, chronic studies and human health studies. The available data provide a clear picture of possible toxic effects and indicate that there is no increased risk to neonates or young when DMSO is ingested. Therefore, Gaylord concludes that an additional safety factor for the protection of infants and children is not needed and that the RfD of 0.03 g/kg/day is appropriate for assessing DMSO risks to infants and children.

Using the conservative exposure assumptions previously described, the percent RfD utilized by the aggregate human exposure to residues of DMSO from natural occurrence and from the proposed use would be 1.2 percent

[(0.0207 g DMSO/day in diet) x (0.25 percent of adult intake) ((0.03 g/kg/day x 14 kg body wt) = 0.0052 g DMSO/day anticipated (0.42 g/day DMSO allowed = 0.0123] for children 1 to 6 years old, based on estimated average consumption of 0.625 kg food/day (1/4 of adult consumption) and average body weight of 14 kg. Therefore, based on this conservative exposure assessment, Gaylord concludes with reasonable certainty that no harm will result to infants and children from aggregate human exposure to residues from the proposed use of DMSO in pesticide products applied to the edible parts of food and feed crops.

I. Existing Tolerances

DMSO is a pesticidally inert ingredient that currently is exempted [40 CFR (180.1001(d)] from the requirement of a tolerance for residues when used as a solvent or cosolvent in pesticide formulations applied before the crop emergence from the soil or prior to formation of edible parts of food plants. There are no other limits for DMSO expressed in 40 CFR (180.1001(d).

J. International Tolerances

There are no Codex maximum residue levels established for residues of DMSO on food or feed crops.

2. Gustafson Incorporation

PP 5F4584

EPA has received a pesticide petition (PP 5F4584) pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, as amended, 21 U.S.C. 346a(d), by the Food Quality Protection Act of 1996 (Pub. L. 104-170, 110 Stat. 1489) from Gustafson, Inc., 1400 Preston Road, Suite 400, Plano, Texas 75093 requesting that the time limited tolerances for wheat, barley and sugar beet RACs be made permanent for residues of the insecticide, imidacloprid: 1-[(6-chloro-3-pyridinyl)methyl]-*N*-nitro-2-imidazolidinimine and its metabolites containing the 6-chloro-pyridinyl moiety. In September 1995, the EPA revised Table II of the Pesticide Assessment Guidelines, Subdivision O, Residue Chemistry. At that time, forage was removed as a raw agricultural commodity of barley. It is proposed that tolerances of 0.20 ppm for wheat, hay, and 0.20 ppm for barley, hay, be added. It is proposed that the tolerance for barley, straw, be increased from 0.20 ppm to 0.30 ppm. It is proposed that the tolerance for beets, sugar (tops) be increased from 0.20 ppm to 0.30 ppm. The original time-limited tolerances were published in the December 13, 1995 and in the August 30, 1995 **Federal Registers**. Imidacloprid is a broad-spectrum insecticide with excellent systemic and contact toxicity characteristics which is used primarily for sucking insects. The nature of the imidacloprid residue in plants and livestock is adequately understood. The analytical method for determining residues is a common moiety method for imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety using oxidation, derivatization, and analysis by capillary gas chromatography with a mass-selective detector. Pursuant to section 408(d)(2)(A)(i) of the FFDCA, as amended, Gustafson has submitted the following summary of information, data and arguments in support of its pesticide petition. The summary was proposed by Gustafson, and EPA has not yet fully evaluated the merits of the petition. The conclusions and arguments presented are those of the petitioner and not of the EPA although the EPA has edited the summary for clarification as necessary.

A. Plant Metabolism and Analytical Method

The metabolism of imidacloprid in plants is adequately understood for the purposes of these tolerances. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety, all calculated as imidacloprid. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. This method has successfully passed a petition method validation in EPA labs. There is a confirmatory method specifically for imidacloprid and several metabolites utilizing GC/MS and HPLC-UV which has been validated by the EPA as well. Imidacloprid and its metabolites are stable for at least 24 months in the commodities when frozen.

B. Magnitude of the Residue

1. *Wheat.* When the conditional registrations and the time-limited tolerances were issued for wheat grain, wheat forage and wheat straw, the EPA requested additional residue field trials and residue testing to support a tolerance for wheat hay. Wheat seed was treated with imidacloprid, formulated as Gaucho 480 FS at a rate of 2.0 oz. a.i./cwt seed. Field trials were conducted at seven locations: Colorado, Nebraska (two locations), North Dakota, Oklahoma, Texas and Wyoming. The wheat seed was planted and the RACs were harvested at the appropriate growth stages. Maximum residues in wheat grain, wheat forage and wheat straw were less than the time-limited tolerances. The maximum residue level in wheat hay was 0.187 ppm. A tolerance of 0.20 ppm for wheat hay is proposed.

2. *Barley*. When the conditional registrations and the time-limited tolerances were issued for barley grain, barley forage and barley straw, the EPA requested additional residue field trials and residue testing to support a tolerance for barley hay. Barley seed was treated with imidacloprid, formulated as Gaucho 480 FS at a rate of 2.0 oz. a.i./cwt seed. Field trials were conducted at five locations: Colorado, Nebraska, North Dakota, Pennsylvania and Wyoming. The barley seed was planted and the RACs were harvested at the appropriate growth stages. The maximum residue in barley grain was less than the time-limited tolerance. The maximum residue level in barley straw was 0.221 ppm, which was above the time- limited tolerance of 0.20 ppm. A revised tolerance of 0.30 ppm for barley straw is proposed. The maximum residue level in barley hay was 0.181 ppm. A tolerance of 0.20 ppm for barley hay is proposed.

3. *Sugar Beets*. When the conditional registrations and the time-limited tolerances were issued for beets, sugar (tops); beets, sugar (roots); and beets, sugar, molasses; the EPA requested additional residue field trials. Sugar beet seed was treated with imidacloprid, formulated as Gaucho 75 ST at a rate of 90 g ai/kg raw seed. Field trials were conducted at four locations: California, Colorado, Idaho and Nebraska. The sugar beet seed was planted and the RACs were harvested at the appropriate growth stages. The maximum residue in the sugar beet roots was less than the time-limited tolerances. The maximum residue level in the sugar beet tops was 0.255 ppm, which was above the time-limited tolerance of 0.10 ppm. A revised tolerance of 0.30 ppm for sugar beet tops is proposed.

C. Toxicological Profile of Imidacloprid

1. *Acute toxicity*. The acute oral LD_{50} values for imidacloprid technical ranged from 424 - 475 mg/kg bwt in the rat. The acute dermal LD_{50} was greater than 5,000 mg/kg in rats. The 4–hour inhalation LC_{50} was less than 69 mg/m^3 air (aerosol). Imidacloprid was not irritating to rabbit skin or eyes. Imidacloprid did not cause skin sensitization in guinea pigs.

2. *Genotoxicity*. Extensive mutagenicity studies conducted to investigate point and gene mutations, DNA damage and chromosomal aberration, both using *in vitro* and *in vivo* test systems show imidacloprid to be non-genotoxic.

3. *Reproductive and developmental toxicity*. A 2–generation rat reproduction study gave a no-observed-effect level (NOEL) of 100 ppm (8 mg/kg/bwt). Rat and rabbit developmental toxicity studies were negative at doses up to 30 mg/kg/bwt and 24 mg/kg/bwt, respectively.

4. *Subchronic toxicity*. 90–day feeding studies were conducted in rats and dogs. The NOELs for these tests were 14 mg/kg/bwt/day (150 ppm) and 5 mg/kg/bwt/day (200 ppm), for the rat and dog studies, respectively.

5. *Chronic toxicity/oncogenicity*. A 2–year rat feeding/ carcinogenicity study was negative for carcinogenic effects under the conditions of the study and had a NOEL of 100 ppm (5.7 mg/kg/bwt in males and 7.6 mg/kg/bwt in females for noncarcinogenic effects that included decreased body weight gain in females at 300 ppm and increased thyroid lesions in males at 300 ppm and females at 900 ppm. A 1–year dog feeding study indicated a NOEL of 1,250 ppm (41 mg/kg/bwt). A 2–year mouse carcinogenicity study was negative for carcinogenic effects under conditions of the study and had a NOEL of 1,000 ppm (208 mg/kg/day).

Imidacloprid has been classified under ''Group E'' (no evidence of carcinogenicity) by EPA's OPP/HED's Reference Dose (RfD) Committee. There is no cancer risk associated with exposure to this chemical. The reference dose (RfD) based on the 2–year rat feeding/carcinogenic study with a NOEL of 5.7 mg/kg/bwt and hundredfold uncertainty factor, is calculated to be 0.057 mg/kg/bwt. The theoretical maximum residue contribution (TMRC) from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7 percent of the RfD.

6. *Endocrine effects*. The toxicology database for imidacloprid is current and complete. Studies in this database include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short or long term exposure. These studies revealed no primary endocrine effects due to imidacloprid.

7. *Mode of action*. Imidacloprid exhibits a mode of action different from traditional organophosphate, carbamate, or pyrethroid insecticides. Imidacloprid acts by binding to the nicotinergic receptor sites at the postsynaptic membrane of the insect nerve. Due to this novel mode of action, imidacloprid has not shown any cross resistance to registered alternative insecticides and is a valuable tool for use in IPM or resistance management programs.

D. Aggregate Exposure

Imidacloprid is a broad-spectrum insecticide with excellent systemic and contact toxicity characteristics with both food and non-food uses. Imidacloprid is currently registered for use on various food crops including seed treatments, tobacco, turf, ornamentals, buildings for termite control, and cats and dogs for flea control. Those potential exposures are addressed below:

1. *Dietary*. The EPA has determined that the reference dose (RfD) based on the 2–year rat feeding/carcinogenicity study with a NOEL of 5.7 mg/kg/bwt and hundredfold uncertainty factor, is calculated to be 0.057 mg/kg/bwt. As published in the **Federal Register** June 12, 1996 (61 FR 29674) (petition to establish tolerances on leafy green vegetables (PP 5F4522/R2237)), the theoretical maximum residue contribution (TMRC) from published uses is 0.008358 mg/kg/bwt utilizing 14.7 percent of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1 percent of the RfD.

The TMRC for wheat is calculated to be 0.000066 mg/kg/bwt/day for the general population, which represents 0.1 percent of the RfD. The TMRC for the most highly exposed subgroup in the population, children 1 to 6 years of age, is 0.000149 mg/kg/bwt/day, which represents 0.3 percent of the RfD. The TMRC for nursing infants is 0.000009 mg/kg/bwt/day, which represents 0.0 percent of the RfD, and for non-nursing infants is 0.000033 mg/kg/bwt/day, which represents 0.1 percent of the RfD. Therefore, dietary exposure from wheat will not exceed the reference dose for any subpopulation (including infants and children).

The TMRC for barley is calculated to be 0.000004 mg/kg/bwt/day for the general population, which represents 0.0 percent of the RfD. The TMRC for the most highly exposed subgroup in the population, non-nursing infants, is 0.000009 mg/kg/bwt/day, which represents 0.0 percent of the RfD. The TMRC for nursing infants is 0.000000 mg/kg/bwt/day, which represents 0.0 percent of the RfD. The TMRC for children 1 to 6 years of age is 0.000001 mg/kg/bwt/day, which represents 0.0 percent of the RfD. Therefore, dietary exposure from barley will not exceed the reference dose for any subpopulation (including infants and children).

The TMRC for sugar beets is calculated to be 0.000012 mg/kg/bwt/day for the general population, which represents 0.0 percent of the RfD. The TMRC for the most highly exposed subgroup in the population, children 1 to 6 years of age, is 0.000027 mg/kg/bwt/day, which represents 0.0 percent of the RfD. The TMRC for non-nursing infants is 0.000017 mg/kg/bwt/day, which represents 0.0 percent of the RfD. The TMRC for nursing infants is 0.000005 mg/kg/bwt/day, which represents 0.0 percent of the RfD. Therefore, dietary exposure from sugar beets will not exceed the reference dose for any subpopulation (including infants and children).

The additive TMRC from exposure to wheat, barley and sugar beets for the general population, is 0.000082 mg/kg/bwt/day, which represents 0.1 percent of the RfD. The additive TMRC from exposure to wheat, barley and sugar beets to children, 1 to 6 years of age, is 0.000177 mg/kg/bwt/day, which represents 0.3 percent of the RfD. For non-nursing infants, the additive TMRC is 0.000029 mg/kg/bwt/day, which is 0.1 percent of the RfD. For nursing infants, the additive TMRC is 0.000014 mg/kg/bwt/day, which is 0.0 percent of the RfD.

2. *Water*. Although the various imidacloprid labels contain a statement that this chemical demonstrates the properties associated with chemicals detected in groundwater, the Registrant is not aware of imidacloprid being detected in any wells, ponds, lakes, streams, etc. from its use in the United States. Imidacloprid is hydrolytically stable at pH 5 and 7 with photolytic degradation in water having a half-life of 4.2 hours. Under aerobic soil conditions in laboratory studies, imidacloprid has a half-life of 188 to >366 days. Under laboratory anaerobic aquatic conditions, the half-life was 27 days. Adsorption/desorption studies indicate that aged imidacloprid residues do not leach into the soil. Imidacloprid dissipates under actual field conditions with a half-life of 7 to 196 days. Imidacloprid remained in the top six inches of the soil in U.S. tests for the duration of nine of ten field dissipation studies. The presence of growing vegetation significantly increased the rate of degradation of imidacloprid. In studies conducted in 1995, imidacloprid was not detected in seventeen wells on potato farms in Quebec, Canada. In addition, groundwater monitoring

studies are currently underway in California and Michigan. Therefore, contributions to the dietary burden from residues of imidacloprid in water would be inconsequential.

3. *Non-occupational*— i. *Residential turf*. Bayer Corporation has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children.

Margins of safety (MOS) of 7,587 - 41,546 for 10 year old children and 6,859 - 45,249 for 5 year old children were estimated by comparing dermal exposure doses to the imidacloprid no-observable effect level of 1,000 mg/kg/day established in a 15–day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10 year old children ranged from 5.6 - 38.2 g/cm^2 and for 5 year old children from 5.1 - 33.3 g/cm^2. This compares with the average imidacloprid transferable residue level of 0.080 g/cm^2 present immediately after the sprays have dried. These data indicate that children can safely contact imidacloprid-treated turf as soon after application as the spray has dried.

ii. *Termiticide*. Imidacloprid is registered as a termiticide. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by EPA's Occupational and Residential Exposure Branch (OREB) and Bayer Corporation. Data indicate that the Margins of Safety for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0 x 10^7 and 2.4 x 10^8, respectively, and exposure can thus be considered negligible.

iii. *Tobacco smoke*. Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only 2 percent of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOEL of 5.5 mg/m^3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet treatment*. Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA's Occupational and Residential Exposure Branch (OREB) who have concluded that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

4. *Cumulative Effects*. No other chemicals having the same mechanism of toxicity are currently registered, therefore, there is no risk from cumulative effects from other substances with a common mechanism of toxicity.

E. Safety Determinations

1. *U.S. Population in general*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 15 percent of the RfD for the U.S. population. EPA generally has no concerns for exposures below 100 percent of the RfD, because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. The TMRC from exposure to wheat, barley and sugar beets for the general population, is 0.000082 mg/kg/bwt/day, which represents 0.1 percent of the RfD. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2–generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through 2 generations, as well as any observed systemic toxicity.

FFDCA Section 408 provides that the EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and post-natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2–year rat feeding/carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a hundredfold uncertainty factor is already used to calculate the RfD, it is surmised that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg/bwt/day is appropriate for assessing aggregate risk to infants and children.

Using the conservative exposure assumptions described above, EPA has concluded that the TMRC from use of imidacloprid from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7 percent of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1 percent of the RfD. The additive TMRC from exposure to wheat, barley and sugar beets to children, 1 to 6 years of age, is 0.000177 mg/kg/bwt/day, which represents 0.3 percent of the RfD. For non-nursing infants, the additive TMRC is 0.000029 mg/kg/bwt/day, which is 0.1 percent of the RfD. For nursing infants, the additive TMRC is 0.000014 mg/kg/bwt/day, which is 0.0 percent of the RfD. Thus, it can be concluded that there is a reasonable certainty that no harm will result from additional exposure of infants and children.

F. Other Considerations

The nature of the imidacloprid residue in plants and livestock is adequately understood. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloropyridinyl moiety, all calculated as imidacloprid. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. There is an additional confirmatory method available. Imidacloprid and its metabolites have been shown to be stable for at least 24 months in frozen storage.

G. International Tolerances

No CODEX Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time.

3. Gustafson Incorporation

PP 6F4682

EPA has received a pesticide petition (PP 6F4682) pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, as amended, 21 U.S.C. 346a(d), by the Food Quality Protection Act of 1996 (Pub. L. 104-170, 110 Stat. 1489) from Gustafson, Inc., 1400 Preston Road, Suite 400, Plano, Texas 75093 requesting that tolerances be established for residues of the insecticide, imidacloprid: 1-[(6-chloro-3-pyridinyl)methyl]-*N*-nitro-2-imidazolidinimine and its metabolites containing the 6-chloro-pyridinyl moiety. It is proposed that tolerances of 0.05 parts per million (ppm) for field corn, grain, 0.02 ppm for field corn, fodder and 0.10 ppm for field corn, forage be established. The nature of the imidacloprid residue in plants and livestock is adequately understood. The analytical method for determining residues is a common moiety method for imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety using oxidation, derivatization, and analysis by capillary gas chromatography with a mass-selective detector.

Imidacloprid is a broad spectrum insecticide with excellent systemic and contact toxicity characteristics which is used primarily for sucking insects. Pursuant to section 408(d)(2)(A)(i) of the FFDCA, as amended, Gustafson has submitted the following summary of information, data and arguments in support of its pesticide petition. The summary was proposed by Gustafson, and EPA has not yet fully evaluated the merits of the petition. The conclusions and arguments presented are those of the petitioner and not of the EPA although the EPA has edited the summary for clarification as necessary.

A. Plant Metabolism and Analytical Method

The metabolism of imidacloprid in plants is adequately understood for the purposes of these tolerances. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety, all calculated as imidacloprid. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloro-pyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. This method has successfully passed a petition method validation in EPA labs. There is a confirmatory method specifically for imidacloprid and several metabolites utilizing GC/MS and HPLC-UV which has been validated by the EPA as well. Imidacloprid and its metabolites are stable for at least 24 months in the commodities when frozen.

B. Magnitude of the Residue

Corn seed was treated with imidacloprid, formulated as Gaucho 480 FS at a rate of 8.0 oz.ai/cwt seed. Field trials were conducted at twenty locations, one in Region 1, one in Region 2, seventeen in Region 5, and one in Region 6. The corn seed was planted and the RACs were harvested at the appropriate growth stages. The highest average residue level found in field corn forage was 0.064 ppm. The highest average residue level found in the field corn grain was less than the Limit of Quantitation, which was 0.05 ppm. The highest average residue level found in the field corn fodder was 0.150 ppm. The proposed tolerance for field corn forage is 0.10 ppm. The proposed tolerance for the field corn fodder is 0.20 ppm. The proposed tolerance for the field corn grain is 0.05 ppm.

Since there were no quantifiable residues in the field corn grain RAC samples analyzed in the processing study or in the RAC study, neither a section 409 food/feed additive tolerance or a section 701 maximum residue level is required for the processed commodities.

C. Toxicological Profile of Imidacloprid

1. *Acute toxicity*. The acute oral LD_{50} values for imidacloprid technical ranged from 424 - 475 mg/kg bwt in the rat. The acute dermal LD_{50} was greater than 5,000 mg/kg in rats. The 4 hour inhalation LC_{50} was less than 69 mg/m^3 air (aerosol). Imidacloprid was not irritating to rabbit skin or eyes. Imidacloprid did not cause skin sensitization in guinea pigs.

2. *Genotoxicity*. Extensive mutagenicity studies conducted to investigate point and gene mutations, DNA damage and chromosomal aberration, both using *in vitro* and *in vivo* test systems show imidacloprid to be non-genotoxic.

3. *Reproductive and developmental toxicity*. A 2–generation rat reproduction study gave a no-observed-effect level (NOEL) of 100 ppm (8 mg/kg/bwt). Rat and rabbit developmental toxicity studies were negative at doses up to 30 mg/kg/bwt and 24 mg/kg/bwt, respectively.

4. *Subchronic toxicity*. 90–day feeding studies were conducted in rats and dogs. The NOELs for these tests were 14 mg/kg/bwt/day (150 ppm) and 5 mg/kg/bwt/day (200 ppm), for the rat and dog studies, respectively.

5. *Chronic toxicity/oncogenicity*. A 2–year rat feeding/ carcinogenicity study was negative for carcinogenic effects under the conditions of the study and had a NOEL of 100 ppm (5.7 mg/kg/bwt in males and 7.6 mg/kg/bwt in females for noncarcinogenic effects that included decreased body weight gain in females at 300 ppm and increased thyroid lesions in males at 300 ppm and females at 900 ppm. A 1–year dog feeding study indicated a NOEL of 1,250 ppm (41 mg/kg/bwt). A 2–year mouse carcinogenicity study was negative for carcinogenic effects under conditions of the study and had a NOEL of 1,000 ppm (208 mg/kg/day).

Imidacloprid has been classified under ''Group E'' (no evidence of carcinogenicity) by EPA's OPP/HED's Reference Dose (RfD) Committee. There is no cancer risk associated with exposure to this chemical. The reference dose (RfD) based on the 2–year rat feeding/carcinogenic study with a NOEL of 5.7 mg/kg/bwt and hundredfold uncertainty factor, is calculated to be 0.057 mg/kg/bwt. The theoretical maximum residue contribution (TMRC) from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7 percent of the RfD.

6. *Endocrine effects*. The toxicology database for imidacloprid is current and complete. Studies in this database include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short or long term exposure. These studies revealed no primary endocrine effects due to imidacloprid.

7. *Mode of action*. Imidacloprid exhibits a mode of action different from traditional organophosphate, carbamate, or pyrethroid insecticides. Imidacloprid acts by binding to the nicotinergic receptor sites at the postsynaptic membrane of the insect nerve. Due to this novel mode of action, imidacloprid has not shown any cross resistance to registered alternative insecticides and is a valuable tool for use in IPM or resistance management programs.

D. Aggregate Exposure

Imidacloprid is a broad-spectrum insecticide with excellent systemic and contact toxicity characteristics with both food and non-food uses. Imidacloprid is currently registered for use on various food crops including seed treatments, tobacco, turf, ornamentals, buildings for termite control, and cats and dogs for flea control. Those potential exposures are addressed below:

1. *Dietary*. The EPA has determined that the reference dose (RfD) based on the 2–year rat feeding/carcinogenicity study with a NOEL of 5.7 mg/kg/bwt and hundredfold uncertainty factor, is calculated to be 0.057 mg/kg/bwt. As published in the **Federal Register** June 12, 1996 (61 FR 29674) (petition to establish tolerances on leafy green vegetables (PP 5F4522/R2237), the theoretical maximum residue contribution (TMRC) from published uses is 0.008358 mg/kg/bwt utilizing 14.7 percent of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1 percent of the RfD.

The TMRC for corn is calculated to be 0.000055 mg/kg/bwt/day for the general population, which represents 0.1 percent of the RfD. The TMRC for the most highly exposed subgroup in the population, non-nursing infants is 0.000131 mg/kg/bwt/day, which represents 0.2 percent of the RfD. The TMRC for children ages 1 to 6 years is 0.000130 mg/kg/bwt/day, which represents 0.2 percent of the RfD, and for nursing infants is 0.000032 mg/kg/bwt/day, which represents 0.1 percent of the RfD. For children 7 to 12 years of age, the TMRC is 0.000098 mg/kg/bwt/day, which represents 0.2 percent of the RfD. Therefore, dietary exposure from field corn will not exceed the reference dose for any subpopulation (including infants and children).

2. *Water*. Although the various imidacloprid labels contain a statement that this chemical demonstrates the properties associated with chemicals detected in groundwater, the Registrant is not aware of imidacloprid being detected in any wells, ponds, lakes, streams, etc. from its use in the United States. Imidacloprid is hydrolytically stable at pH 5 and 7 with photolytic degradation in water having a half-life of 4.2 hours. Under aerobic soil conditions in laboratory studies, imidacloprid has a half-life of 188 to >366 days. Under laboratory anaerobic aquatic conditions, the half-life was 27 days. Adsorption/desorption studies indicate that aged imidacloprid residues do not leach into the soil. Imidacloprid dissipates under actual field conditions with a half-life of 7 to 196 days. Imidacloprid remained in the top six inches of the soil in U.S. tests for the duration of nine of ten field dissipation studies. The presence of growing vegetation significantly increased the rate of degradation of imidacloprid. In studies conducted in 1995, imidacloprid was not detected in seventeen wells on potato farms in Quebec, Canada. In addition, groundwater monitoring studies are currently underway in California and Michigan. Therefore, contributions to the dietary burden from residues of imidacloprid in water would be inconsequential.

3. *Non-occupational*— i. *Residential turf*. Bayer Corporation has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children.

Margins of safety (MOS) of 7,587 - 41,546 for 10 year old children and 6,859 - 45,249 for 5 year old children were estimated by comparing dermal exposure doses to the imidacloprid no-observable effect level of 1,000 mg/kg/day established in a 15 day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10 year old children ranged from 5.6 - 38.2 g/cm^2 and for 5 year old children from 5.1 - 33.3 g/cm^2. This compares with the average imidacloprid transferable residue level of 0.080 g/cm^2 present immediately after the sprays have dried. These data indicate that children can safely contact imidacloprid-treated turf as soon after application as the spray has dried.

ii. *Termiticide*. Imidacloprid is registered as a termiticide. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by EPA's Occupational and Residential Exposure Branch (OREB) and Bayer Corporation. Data indicate that the Margins of Safety for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0 x 10^7 and 2.4 x 10^8, respectively, and exposure can thus be considered negligible.

iii. *Tobacco smoke*. Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only two percent of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOEL of 5.5 mg/m^3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet treatment*. Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA's Occupational and Residential Exposure Branch (OREB) who have concluded that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

4. *Cumulative effects*. No other chemicals having the same mechanism of toxicity are currently registered, therefore, there is no risk from cumulative effects from other substances with a common mechanism of toxicity.

E. Safety Determinations

1. *U.S. Population in general*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, it can be concluded that total aggregate exposure to imidacloprid from all current uses including those currently proposed will utilize little more than 15 percent of the RfD for the U.S. population. EPA generally has no concerns for exposures below 100 percent of the RfD, because the RfD represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. The TMRC from exposure to field corn for the general population, is 0.000055 mg/kg/bwt/day, which represents 0.1 percent of the RfD. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of imidacloprid, the data from developmental studies in both rat and rabbit and a 2–generation reproduction study in the rat have been considered. The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure of the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through 2 generations, as well as any observed systemic toxicity.

FFDCA section 408 provides that the EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for imidacloprid relative to pre- and

post-natal effects is complete. Further for imidacloprid, the NOEL of 5.7 mg/kg/bwt from the 2–year rat feeding/carcinogenic study, which was used to calculate the RfD (discussed above), is already lower than the NOELs from the developmental studies in rats and rabbits by a factor of 4.2 to 17.5 times. Since a hundredfold uncertainty factor is already used to calculate the RfD, it is surmised that an additional uncertainty factor is not warranted and that the RfD at 0.057 mg/kg/bwt/day is appropriate for assessing aggregate risk to infants and children. Using the conservative exposure assumptions described above, EPA has concluded that the TMRC from use of imidacloprid from published uses is 0.008358 mg/kg/bwt/day utilizing 14.7 percent of the RfD for the general population. For the most highly exposed subgroup in the population, non-nursing infants (less than 1 year old), the TMRC for the published tolerances is 0.01547 mg/kg/day. This is equal to 27.1 percent of the RfD. The TMRC from exposure to field corn to non-nursing infants is 0.000131 mg/kg/bwt/day, which represents 0.2 percent of the RfD. The TMRC for children ages 1 to 6 years is 0.000130 mg/kg/bwt/day, which represents 0.2 percent of the RfD. For nursing infants, the TMRC is 0.000032 mg/kg/bwt/day, which is 0.1 percent of the RfD. For children ages 7 to 12 years, the TMRC is 0.000098 mg/kg/bwt/day, which is 0.2 percent of the RfD. Thus, it can be concluded that there is a reasonable certainty that no harm will result from additional exposure of infants and children.

F. Other Considerations

The nature of the imidacloprid residue in plants and livestock is adequately understood. The residues of concern are combined residues of imidacloprid and its metabolites containing the 6-chloropyridinyl moiety, all calculated as imidacloprid. The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary GC-MS selective ion monitoring. There is an additional confirmatory method available. Imidacloprid and its metabolites have been shown to be stable for at least 24 months in frozen storage.

G. International Tolerances

No CODEX Maximum Residue Levels (MRLs) have been established for residues of imidacloprid on any crops at this time.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–739; FRL–5721–7]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–739, must be received on or before July 25, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI 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 record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The regulatory action leaders listed in the table below:

Product Manager	Office location/telephone number	Address
Sheryl Reilly (PM 90)	Rm. 5-W29, 5th Floor, CS-1, 703-308-8265 e-mail: reilly.sheryl@epamail.epa.gov	2800 Jefferson Davis Hwy., Arlington, VA 22202
Mike Mendelsohn (PM 90).	Rm. 5-W44, 5th Floor, CS-1, 703-308-8715 e-mail: mendelsohn.mike@epamail.epa.gov	Do.
Linda Hollis (PM 90)	Rm 5-J, 5th Floor, CS-1, 703-308-8733 e-mail: hollis.linda@epamail.epa.gov	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–739] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in