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lnterregional Research Project Number 4 (IR-4)

Petition Number: 3E9061

EPA has received a pesticide petition **(3E9061)** from the Interregional Research Project No. 4 (IR-4), North Carolina State University, 1730 Varsity Drive, Suite 210, Venture IV, Raleigh, NC 27606 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180.629 by:

- establishing tolerances for the residues of flutriafol, [(±)-α-(2-fluorophenyl)-α-(4-fluorophenyl)-1H-1,2,4-triazole-1-ethanol], including its metabolites and degradates in or on the following commodities: Brassica, leafy greens, subgroup 4-16B at 7 parts per million (ppm); Celtuce at 10 ppm; Cottonseed subgroup 20C at 0.5 ppm; Fennel, Florence, fresh leaves and stalk at 10 ppm; Fruit, pome, group 11-10 at 0.4 ppm; Fruit, stone, group 12-12 at 1.5 ppm; Kohlrabi at 1.5 ppm; Leafy greens subgroup 4-16A, except head lettuce and radicchio at 10 ppm; Leaf petiole vegetable subgroup 22B at 10 ppm, Tropical and subtropical, small fruit, edible peel, subgroup 23A at 0.01 ppm, and Vegetable, Brassica, head and stem, group 5-16 at 1.5 ppm. Compliance with the tolerances is to be determined by measuring flutriafol only.
- amending 40 CFR 180.629, upon the approval of the aforementioned tolerances, by removing the established tolerances for residues of the fungicide flutriafol, including its metabolites and degradates in or on the following commodities: Brassica, head and stem (subgroup 5A) at 1.5 parts per million (ppm); Brassica, leafy greens (subgroup 5B) at 7.0 ppm; Cotton, undelinted seed at 0.50 ppm; Fruit, pome group 11-09 at 0.40 ppm; Fruit stone, group 12-10 at 1.5 ppm; and Vegetable, leafy, except Brassica, crop group 4, except head lettuce and radicchio at 10 ppm.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408 (d)(2) of FDDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The nature of the residue in plants is adequately understood. The major residue in apples, oilseed rape, sugar beet and cereals treated with flutriafol is the parent compound.

2. *Analytical method.* Adequate enforcement analytical methods for determining flutriafol in/on appropriate raw agricultural commodities and processed commodities are available for the established and proposed tolerances.

3. *Magnitude of residues.* Appropriate residue field trials have been conducted to support the tolerances of flutriafol on the representative crops of23A (olive) as proposed in this petition. All raw agricultural commodities and processed commodities where applicable in all field trials were analyzed for the parent compound flutriafol. The flutriafol residues reported in these field trials support the proposed tolerances.

B. Toxicological Profile

1. *Acute toxicity.* The acute oral LD50 is 1140 mg/kg bw and 1480 mg/kg bw in male and female rats, respectively. The acute dermal LD50 of flutriafol is >2000 mg/kg bw in rats. The acute inhalation LC50 in rats is >5.20 mg/L. Flutriafol displayed mild eye irritation in rabbits. It is not a skin irritant to rabbits. Flutriafol is not a skin sensitizer.

2. *Genotoxicity.* Flutriafol was evaluated for possible mutagenic/genotoxic effects in in vitro and in vivo test systems. In these studies, flutriafol was not mutagenic in either bacterial or mammalian cells at concentrations that were not highly cytotoxic in the presence and absence of metabolic activation in vitro. No clastogenic potential in in vitro and in vivo cytogenetic studies was demonstrated, and no induction of forward mutations was observable in vitro. Furthermore, flutriafol did not induce unscheduled DNA synthesis in vivo, and no clastogenicity was observable in germ cells of mice. Overall, the data indicate that flutriafol has no genotoxic/mutagenic potential in vitro and in vivo.

In three genotoxicity studies flutriafol was not mutagenic in either bacterial or mammalian cells at concentrations that were not highly cytotoxic in the presence and absence of metabolic activation in vitro. No clastogenic potential in the in vitro chromosome aberration study was demonstrated. These results are consistent with the conclusions of the originally submitted genotoxicity studies.

3. *Reproductive and developmental toxicity.* A two-generation reproductive toxicity study in rats was conducted with flutriafol at doses of 0, 60, 240 or 1000 ppm in the diet. The parental NOAEL was 240 ppm (calculated by EPA to be 20.6 mg/kg bw/day in males and 21.9 mg/kg bw/day in females) based on reduced body weight gain and food consumption, and effects on the liver (increased liver weights, centrilobular hypertrophy and fatty changes). The NOAEL for developmental and reproductive toxicity was 240 ppm (20.6 mg/kg bw/day for males and

21.9 mg/kg bw/day for females) based on reduced litter sizes and effects on the liver (fatty change/vacuolation).

In a second two-generation reproduction study, flutriafol was administered to groups of rats at concentrations of 0, 30, 80, 150 and 300 ppm in the diet. In the P and F1 parental generations, dietary exposure to flutriafol resulted in centrilobular hepatocellular hypertrophy at 300 ppm. These findings correlated with increased liver to body weight ratios in P parental animals, which are indicative of an adaptive change to metabolic activation by a xenobiotic and not a toxic response due to the administration of flutriafol. At 150 ppm, 80 ppm and 30 ppm, no test item-related effects were noted.

There were no treatment-related differences in reproductive parameters between treated animals and controls. There were no treatment-related differences in litter parameters between treated groups and controls. Based on these results, 300 ppm (15.6 mg/kg bw/day for males and 20.7 mg/kg bw/day for females) is the NOAEL for parental toxicity and is the NOEL for effects on reproductive function. The NOEL for parental toxicity is 150 ppm. The results of this new study confirm the earlier study that flutriafol is not a reproductive toxicant to rats.

In the rat developmental toxicity study, flutriafol was orally administered at doses of 0, 2, 5, 10 or 75 mg/kg bw/day. The maternal toxicity NOAEL was 10 mg/kg bw/day based on reduced body weight gain and food consumption. The developmental toxicity NOAEL was 10 mg/kg bw/day based on increased late resorptions and specific malformations/variations, including hyoid arch absent, interrupted, and/or misshapen.

In the rabbit developmental study, flutriafol was administered at doses of 0, 2.5, 7.5 or 15 mg/kg bw/day. The NOAEL for maternal toxicity was 7.5 mg/kg bw/day based on reduced body weight gain and food consumption. The NOAEL for developmental toxicity was 7.5 mg/kg bw/day based on an increase in post-implantation losses and a reduction in the number of viable fetuses.

Flutriafol is neither a reproductive nor developmental toxicant in the absence of paternal/maternal toxicity.

4. *Subchronic toxicity.* The short-term toxicity of flutriafol was investigated in several subchronic studies including a 90-day feeding study in rats and a 90-day study in dogs. A 28-day dermal toxicity study in rats was also performed.

The 90-day rat feeding study was conducted with dietary concentrations of 0, 20, 200 or 2000 ppm. The NOAEL of 200 ppm (14 mg/kg bw/day for males and 22 mg/kg bw/day for females) was based on decreased body weight gain and food consumption and liver toxicity (increased absolute and relative liver weights, increased endoplasmic reticulum proliferation in the male rats and increased APDM activity).

In the 90-day study in dogs, flutriafol was administered at concentrations of 0, 1, 5 or 15 mg/kg bw/day via capsule. The NOAEL was 5 mg/kg bw/day based on reduced body weight gain in females, changes in hematology and clinical chemistry parameters in both sexes, and effects in the livers of males and females and in the spleens of the males at the 15 mg/kg bw/day.

In the 28-day dermal toxicity study in rats, there was no evidence of systemic toxicity, and the NOAEL was >1000 mg/kg bw/day.

5. *Chronic toxicity.* A one-year oral study in the dog was conducted at concentrations of 0, 1, 5 or 20 mg/kg bw/day. The NOAEL was 5 mg/kg bw/day based on adverse liver findings (increased liver weights, increased lipid content of hepatocytes, and increased alkaline phosphatase, albumin and triglycerides), increased adrenal cortical vacuolation of the zona fasciculate, and increased hemosiderin deposition in the spleen and liver of both sexes; decreased body weight gain, and increased adrenal weights in females.

In a two-year chronic toxicity and oncogenicity study, flutriafol was administered in the diet to rats at doses of 0, 20, 200 or 2000 ppm. The NOAEL was 200 ppm (calculated by EPA to be 10.0 mg/kg bw/day in males and 12.2 mg/kg bw/day in females) based on reduced body weight, body weight gain, and food consumption in both sexes and adverse liver effects (increased liver weights, fatty change, bile duct proliferation/cholangiolarfibrosis, hemosiderin accumulation in Kupffer cells and centrilobular hypertrophy) and clinical chemistry findings. There was no evidence of carcinogenicity in this study.

In a two-year oncogenicity study, flutriafol was administered in the diet to mice at doses of 0, 10, 50 or 200 ppm. The NOAEL was 50 ppm (calculated by EPA to be 5.9 mg/kg bw/day in males and 7.4 mg/kg bw/day in females) based on reduced body weight and body weight gain in males and females, increased liver weight, and hepatocellular hypertrophy and centrilobular fatty change in the liver. There was no evidence of carcinogenicity in this study.

6. *Neurotoxicity.* In an acute neurotoxicity study, rats were administered flutriafol by gavage at doses of 0, 125, 250 or 750 mg/kg bw. EPA concluded that the systemic NOAEL was

250 mg/kg bw based on decreased body weight gain and food consumption and clinical signs of toxicity indicative of a moribund condition. The neurotoxicity NOAEL was >750 mg/kg bw.

In a subchronic neurotoxicity study, flutriafol was administered in the diet to rats at doses of 0, 500, 1500 or 3000 ppm. The systemic NOAEL was 1500 ppm (84.3 mg/kg bw/day in males and 97.6 mg/kg bw/day in females) based on decreased body weight gain and decreased absolute and relative food consumption. The neurotoxicity NOAEL was >3000 ppm (>172.1 mg/kg bw/day in males and >185.0 mg/kg bw/day in females).

7. *Immunotoxicity*. In an immunotoxicity study, flutriafol was administered to 10 Crl:CD1(ICR) female mice/group in the diet at dose levels of 0, 50, 250, 500 and 1000 ppm (equivalent to 0, 9.8, 46.8, 94.0 and 208.0 mg/kg bw/day, respectively) for 28 consecutive days. Immunotoxicity was evaluated by assessment of the spleen IgM antibody response to the T-dependent antigen, sheep red blood cells (sRBCs). The NOAEL for general toxicity in female CD-1 mice was 50 ppm (9.8 mg/kg bw/day) based on increased absolute and relative liver weights and hematology effects (decreased mean corpuscular volume). There were no significant effects on absolute and relative spleen weights, spleen cell number and spleen IgM antibody response to the T cell-dependent antigen, sheep erythrocytes at any dose level. Therefore, the immunotoxic NOEL is 1000 ppm (208 mg/kg bw/day), the highest dose tested.

8. *Animal Metabolism*. Based on a series of studies, flutriafol is extensively metabolized in the rat and the metabolic processes are well understood.

Flutriafol was quantitatively absorbed after oral administration to rats and the extent of absorption was in the range of 90% to 99% of the administered dose. Flutriafol is rapidly distributed, metabolized and eliminated in rats for all dosing regimens after oral administration. After single oral administration 78% and 91% of the dose given was excreted in urine and bile/feces within 72 hours and 47% to 79% of the dose was eliminated with bile. Only 0.8% to 10.4% of the dose given was directly excreted with feces. After repeated oral administration, excretion in urine and feces 24 hours after the first and last dose was comparable. The major route of excretion was urinary accounting for approximately 50% to 61% of the daily dose excreted within 24 hours after dosing, while fecal excretion accounted for approximately 30% to 40%. Residues in carcass 168 hours after the last of 14 daily doses of flutriafol were found to be less than 3% of administered doses. Flutriafol is distributed systemically resulting in highest tissue levels in whole blood, kidneys, liver, and muscle within the range of 0.7% to 1.46 % of daily dose.

The metabolites identified indicated that the 2-fluorophenyl ring of flutriafol was the main site for biotransformation. The initial metabolic step probably involved epoxidation followed by either rearrangement to form the dihydrodiol isomers of flutriafol or to form

hydroxy or dihydroxy metabolites. The hydroxyl groups on these primary metabolites may then be either conjugated with glucuronic acid or methylated. A second, minor route, for the metabolism of flutriafol was via the removal of the triazole ring to form 1-(2 fluorophenyl)-1-(4-fluorophenyl)-ethandiol, which is then conjugated with glucuronic acid.

The results demonstrated that absorption, distribution, metabolism, and excretion of flutriafol was comparable in both sexes and also similar after single low, single high, or repeated low-dose scenario. The data indicate that flutriafol and/or its metabolites do not bioaccumulate.

9. *Metabolite Toxicology.* Data regarding the potential toxicity of the three metabolites common to triazoles have been developed and submitted by the US Triazole Task Force. The Task Force has entered into a data access agreement with Cheminova A/S by which it has authorized Cheminova to rely on all of the studies, data and information that the Task Force has submitted to EPA. This includes the comprehensive risk assessment of these three metabolites. Cheminova hereby incorporates and relies on the Task Force studies, data and information in support of its pesticide action. This includes relying on the T-D metabolite risk assessment submitted to the EPA by the Task Force.

10. *Endocrine Disruption.* All guideline studies conducted to characterize the toxicological profile showed no endocrine-related toxicity or tumorigenicity.

C. Aggregate Exposure

1. *Dietary exposure.* Tolerances are proposed for residues of flutriafol on several crop commodities or crop (sub)groups as described above. For the purposes of assessing the potential dietary (food and drinking water) exposure, an exposure assessment including the proposed crops in addition to all of the registered crops was conducted using the Dietary Exposure Evaluation Model-Food Consumption Intake Database (DEEM-FCID) version 4.02 software, which used consumption data derived from the 2005-2010 National Health and Nutrition Examination Survey/ "What We Eat in America" (NHANES/WWEIA). Food residues from crops were assumed at tolerance level (all crops for acute analysis and most crops for chronic analysis) or average field trial residues (a few crops for chronic analysis). Available empirical or default processing factors as well as translation among similar crops following HED's 2022 memorandum on default processing factors (18-Feb-2022) were used. Refined percent crop treated data were used for certain crops in the chronic analysis. Livestock residues were estimated from calculated dietary burdens and livestock feeding studies. Estimated potential concentrations in drinking water were incorporated directly into the dietary exposure assessments to provide total aggregate dietary exposure estimates.

For the purpose of dietary exposure assessment, it is assumed that the residues of concern (ROC) are parent flutriafol and those metabolites assumed to be toxicologically similar to flutriafol. Adjustment factors to account for potential residues of conjugated or defluorinated flutriafol metabolites are included as appropriate in risk assessment calculations for certain commodities. Separate risk assessments are required for the triazole metabolites T and TA/TAA/TLA, which have been determined to be toxicologically different from flutriafol.

a. *Acute Dietary Exposure.* The acute population adjusted dose (aPAD) for females 13-49 years old is 0.075 mg/kg/day (NOAEL of 7.5 mg/kg/day from the rabbit developmental toxicity study with a 100-fold uncertainty factor). The aPAD for other subpopulations is 2.5 mg/kg/day (NOAEL of 250 mg/kg/day from the acute neurotoxicity screening battery with a 100-fold uncertainty factor). These endpoints were used to characterize the risk associated with acute dietary exposures.

Cheminova has conducted a Tier 1 acute dietary exposure analysis with DEEM-FCID (ver. 4.02). The acute analysis assumed tolerance-level residues or tolerance-level residues adjusted to account for the residue of concern for risk assessment, 100% crop treated, livestock residue estimates based on maximum reasonable dietary burden (MRDB), and modeled drinking water estimates.

Potential residues in water were included in the exposure assessment using the peak concentration of 630 ppb in ground water estimated by Pesticide Root Zone Model-Groundwater (PRZM-GW) for the registered use on turf. The worst-case turf use scenario included a maximum annual rate of 1.5 lb a.i./A..

The acute dietary assessment was conducted for the overall US population and select subpopulations (i.e., all infants <1 year old, children aged 1-2 years old, children aged 3-5 years old, children aged 6-12 years old, youths aged 13-19 years old, adults aged 20-49 years old, adults 50+ years old, and females aged 13-49 years old). The results of this Tier 1 acute analysis indicate that the most highly exposed population is all infants (<1 year old), with an estimated acute exposure of 00.1402 mg/kg/day at the 95th percentile, which corresponds to 5.6% of the aPAD of 2.5 mg/kg/day. For the overall US population, the acute exposure at the 95th percentile is 0.0638 mg/kg/day, which corresponds to 2.6% of the aPAD. For females 13-49 years old, the acute exposure at the 95th percentile is 0.0501 mg/kg/day, which corresponds to 66.8% of the population-specific aPAD of 0.075 mg/kg/day and presents the highest percent of the aPAD among all population subgroups.

b. *Chronic Dietary Exposure.* The chronic population adjusted dose (cPAD) of 0.05 mg/kg/day (NOAEL of 5 mg/kg/day from the one-year chronic toxicity study in the dog with a 100-fold uncertainty factor) was used to characterize risk associated with chronic dietary exposures for all population groups.

Cheminova has conducted a partially refined chronic dietary exposure analysis with DEEM-FCID (ver. 4.02). The chronic analysis assumed tolerance-level residues or tolerance-level residues adjusted to account for the residue of concern for risk assessment, excluding wheat, apple, grape, rice and peach, where average field-trial residues were assumed and for apple, grape and raisin the screening-level usage analysis (SLUA) percent crop treated estimates were assumed (100% crop treated assumed for the remaining crops). The chronic analysis also incorporated refinements to the livestock residue estimates through incorporation of median residues in feed in calculation of the dietary burden estimates (100% crop treated assumed) and through the incorporation of average residues in animal matrices from the livestock feeding study.

In addition, potential residues in water were included using the post-breakthrough average concentration of 540 ppb in ground water estimated by Pesticide Root Zone Model-Groundwater (PRZM-GW) for the registered use on turf. The worst-case turf use scenario included a maximum annual rate of 1.5 lb a.i./A.

The chronic dietary assessment was conducted for the overall US population and select subpopulations (i.e., all infants <1-year-old, children aged 1-2 years old, children aged 3-5 years old, children aged 6-12 years old, youths aged 13-19 years old, adults aged 20-49 years old, adults 50+ years old, and females aged 13-49 years old). The results of this chronic analysis including refined food residue exposures indicate that the most highly exposed population is all infants (<1 year old), with an estimated chronic exposure of 0.0486 mg/kg/day, which corresponds to 97.2% of the cPAD. For the overall US population, the chronic exposure is calculated to be 0.0.0176 mg/kg/day, which corresponds to 35.2% of the cPAD.

2. *Non-dietary exposure*. Flutriafol is currently registered for the following uses that could result in residential exposures: golf course turf. There is the potential for post-application exposure for individuals exposed as a result of being in an environment that has been previously treated with flutriafol. The quantitative exposure/risk assessment for residential post-application exposures is based on the following scenario: Dermal exposures for children (6 to < 11 years old), children (11 to < 16 years old), and adults contacting residues deposited on turf resulting from broadcast golf course applications. The life stages (i.e., adults, children 11 < 16 years old, and children 6 < 11 years old) selected for each post-application scenario are considered health protective for the exposures and risks for any other potentially exposed life stages. Residential post-application exposure is expected to be short-term in duration due to the intermittent nature of the applications and areas being restricted to non-residential turf (i.e. golf course). With an application rate of 0.5 lb ai/A on golf course, the post-application residential exposure and risk estimates indicate that the short-term dermal margin of exposure (MOE) on “day 0” ranging from 2,500 to 2,900 are not of concern to HED (i.e., MOEs ≥ 100).

Flutriafol is not currently registered for residential use on turf and ornamentals, nor is it registered for use by homeowners. However, application for registration for these uses have been submitted to EPA and are undergoing review.

D. Cumulative Effects

Flutriafol is a member of the triazole class of fungicides. Other members of this class are registered for use in the United States. Although flutriafol and other triazoles may have similar fungicidal modes of action, there are no available data to determine whether flutriafol has a common mechanism of mammalian toxicity with other triazoles or information on how to include this pesticide in a cumulative risk assessment. Therefore, for the purposes of this tolerance petition no assumption has been made with regard to cumulative exposure with other compounds having a common mode of action.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions described above and based on the completeness of the toxicity data, it can be concluded that total food and drinking water exposure to flutriafol from all registered and proposed crop uses will be 2.6% of the aPAD and 35.2% of the cPAD for the overall US population. For females 13-49 years old, the population-specific aPAD is 0.075 mg/kg bw and estimated acute dietary exposures accounted for 66.8% of the aPAD. EPA generally has no concern for exposures below 100% of the PAD because the PAD represents the level at or below which daily aggregate exposures will not pose appreciable risks to human health. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to residues arising from the proposed tolerances that are the subject of this petition, and all approved uses of flutriafol.

2. *Infants and children.* Using the conservative exposure assumptions described above and based on the completeness of the toxicity data, it can be concluded that total food and drinking water exposure to flutriafol from all proposed and registered crop uses will be $\leq$5.6% of the aPAD and $\leq$97.2% of the cPAD for infants and children.

In assessing the potential for additional sensitivity of infants and children to residues of flutriafol, the data from developmental toxicity studies in both the rat and rabbit and a reproduction study in rats have been considered.

The developmental toxicity studies evaluate potential adverse effects on the developing animal resulting from pesticide exposure to the mother during prenatal development. The reproduction study evaluates effects from exposure to the pesticide on the reproductive capability of mating animals through two generations, as well as any observed systemic toxicity.

EPA has concluded that there is no increased quantitative sensitivity to infants and children based on the developmental and reproductive studies conducted with flutriafol. Therefore, infants and children are adequately protected and an additional uncertainty factor for infants and children is not warranted.

F. International Tolerances

Codex Maximum Residue Levels (MRLs) are established for residues of flutriafol for the following commodities: banana (0.3 ppm); brassica vegetable (except Brassica leafy vegetables) (1.5 ppm), celery (3 ppm), cherries (subgroup) (0.8 ppm), coffee beans (0.15 ppm); cotton seed (0.5 ppm), eggs (0.01 ppm), fruiting vegetables, cucurbits (group) (0.3 ppm), dried grapes (= currants, raisins, sultanas) (2 ppm); grapes (0.8 ppm); lettuce, head (1.5 ppm), maize (0.01 ppm), maize fodder (dry) (20 ppm), mammalian fats (except milk fats) (0.02 ppm), meat (from mammals other than marine mammals) (0.02 ppm), milks (0.01 ppm), peaches (including apricots and nectarine) (subgroup) (0.6 ppm), peanut (0.15 ppm); peanut fodder (20 ppm); peppers (subgroup) (1 ppm); peppers chili, dried (10 ppm); plums (including fresh prunes) (subgroup) (0.4 ppm), pome fruits (group) (0.4 ppm); poultry fats (0.02 ppm), poultry meat (0.01 ppm), poultry edible offal of (0.03 ppm), prunes (0.9 ppm), rape seed (0.5 ppm), sorghum grain (1.5 ppm), sorghum straw and fodder, dry (7 ppm), soya bean (dry) (0.4 ppm); strawberry (1.5 ppm), sugar beet (0.02 ppm), sugar beet leaves or tops (dry) (3 ppm), tomato (0.8 ppm), wheat (0.15 ppm); wheat bran, unprocessed (0.3 ppm); and wheat hay and/or straw (8 ppm).

Established crop tolerances for flutriafol in Canada are: berries, blueberries (1.5 ppm), berries, cranberries (1.5 ppm), berries, grapes (1.5 ppm), berries, strawberry (1.5 ppm), corn, field (maize) (0.01 ppm), hops (20 ppm), legume, soybean (dried) (0.4 ppm), oilseeds, peanut (0.15 ppm), pome fruit, apple (0.4 ppm), pome fruit, pear (0.4 ppm), root crop, sugar beet (0.08), stone fruit, cherry (1.5 ppm), stone fruit, Japanese plum (1.5 ppm), stone fruit, nectarine (1.5 ppm), stone fruit, peach (1.5 ppm), stone fruit, plum (1.5 ppm), tree nuts, almond (0.6 ppm), tropical fruit, banana (0.3 ppm).

There are no established tolerances for flutriafol on any crops in Mexico.