## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Part 372

**[EPA–HQ–TRI–2013–0393; FRL 9903–44–OEI]**

## Chlorsulfuron; Community Right-to-Know Toxic Chemical Release Reporting

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Denial of Petition.

**SUMMARY:** EPA is denying a petition to remove chlorsulfuron from the list of chemicals subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 and section 6607 of the Pollution Prevention Act (PPA) of 1990. EPA has reviewed the available data on this chemical and has determined that chlorsulfuron does not meet the deletion criterion of EPCRA section 313(d)(3). Specifically, EPA is denying this petition because EPA's review of the petition and available information resulted in the conclusion that chlorsulfuron meets the listing criterion of EPCRA section 313(d)(2)(C) due to its toxicity to aquatic plants.

**DATES:** EPA denied this petition on November 18, 2013.

**FOR FURTHER INFORMATION CONTACT:** Daniel R. Bushman, Environmental Analysis Division, Office of Information Analysis and Access (2842T), Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460; telephone number: 202–566–0743; fax number: 202–566–0677; email: *bushman.daniel@epa.gov,* for specific information on this notice. For general information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Hotline, toll free at (800) 424–9346 (select menu option 3) or (703) 412–9810 in Virginia and Alaska or toll free, TDD (800) 553–7672, *http://www.epa.gov/superfund/contacts/infocenter/.*

**SUPPLEMENTARY INFORMATION:**

## I. General Information

*A. Does this notice apply to me?*

You may be potentially affected by this action if you manufacture, process, or otherwise use chlorsulfuron. Potentially affected categories and entities may include, but are not limited to:

| Category | Examples of potentially affected entities |
|---|---|
| Industry | Facilities included in the following NAICS manufacturing codes (corresponding to SIC codes 20 through 39): 311*, 312*, 313*, 314*, 315*, 316, 321, 322, 323*, 324, 325*, 326*, 327, 331, 332, 333, 334*, 335*, 336, 337*, 339*, 111998*, 211112*, 212324*, 212325*, 212393*, 212399*, 488390*, 511110, 511120, 511130, 511140*, 511191, 511199, 512220, 512230*, 519130*, 541712*, or 811490*.<br>*Exceptions and/or limitations exist for these NAICS codes.<br>Facilities included in the following NAICS codes (corresponding to SIC codes other than SIC codes 20 through 39): 212111, 212112, 212113 (correspond to SIC 12, Coal Mining (except 1241)); or 212221, 212222, 212231, 212234, 212299 (correspond to SIC 10, Metal Mining (except 1011, 1081, and 1094)); or 221111, 221112, 221113, 221119, 221121, 221122, 221330 (Limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce) (correspond to SIC 4911, 4931, and 4939, Electric Utilities); or 424690, 425110, 425120 (Limited to facilities previously classified in SIC 5169, Chemicals and Allied Products, Not Elsewhere Classified); or 424710 (corresponds to SIC 5171, Petroleum Bulk Terminals and Plants); or 562112 (Limited to facilities primarily engaged in solvent recovery services on a contract or fee basis (previously classified under SIC 7389, Business Services, NEC)); or 562211, 562212, 562213, 562219, 562920 (Limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. 6921 et seq.) (correspond to SIC 4953, Refuse Systems). |
| Federal Government | Federal facilities. |

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Some of the entities listed in the table have exemptions and/or limitations regarding coverage, and other types of entities not listed in the table could also be affected. To determine whether your facility would be affected by this action, you should carefully examine the applicability criteria in part 372 subpart B of Title 40 of the Code of Federal Regulations. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding **FOR FURTHER INFORMATION CONTACT** section.

*B. How can I get copies of this document and other related information?*

EPA has established a docket for this action under Docket ID No. EPA–HQ–TRI–2013–0393. All documents in the docket are listed in the *www.regulations.gov* index. Although listed in the index, some information is not publicly available, e.g., CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in *www.regulations.gov* or in hard copy at the OEI Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC. This Docket Facility is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OEI Docket is (202) 566–1752.

## II. Introduction

Section 313 of EPCRA, 42 U.S.C. 11023, requires certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels to report their environmental releases and other waste management quantities of such chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106. Congress established an initial list of toxic chemicals that comprised more than 300 chemicals and 20 chemical categories.

EPCRA section 313(d) authorizes EPA to add or delete chemicals from the list and sets criteria for these actions. EPCRA section 313(d)(2) states that EPA may add a chemical to the list if any of the listing criteria in Section 313(d)(2) are met. Therefore, to add a chemical, EPA must demonstrate that at least one criterion is met, but need not determine whether any other criterion is met. EPCRA section 313(d)(3) states that a chemical may be deleted if the Administrator determines there is not sufficient evidence to establish any of the criteria described in EPCRA section 313(d)(2)(A)–(C). The EPCRA section 313(d)(2)(A)–(C) criteria are:

- The chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases.
- The chemical is known to cause or can reasonably be anticipated to cause in humans:
  - cancer or teratogenic effects, or
  - serious or irreversible—
    - reproductive dysfunctions,
    - neurological disorders,
    - heritable genetic mutations, or
    - other chronic health effects.
- The chemical is known to cause or can be reasonably anticipated to cause, because of:
  - its toxicity,
  - its toxicity and persistence in the environment, or
  - its toxicity and tendency to bioaccumulate in the environment, a significant adverse effect on the environment of sufficient seriousness, in the judgment of the Administrator, to warrant reporting under this section.

EPA often refers to the section 313(d)(2)(A) criterion as the ''acute human health effects criterion;'' the section 313(d)(2)(B) criterion as the ''chronic human health effects criterion;'' and the section 313(d)(2)(C) criterion as the ''environmental effects criterion.''

Under section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479) to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compounds categories. EPA published in the **Federal Register** of November 30, 1994 (59 FR 61432) a statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals.

## III. What is the description of the petition?

On May 18, 2012, EPA received a petition from DuPont Crop Protection (DuPont), Technology Sciences Group Inc. (TSG) requesting EPA to delete chlorsulfuron (Chemical Abstracts Service Registry Number (CASRN) 64902–72–3) from the list of chemicals subject to reporting under EPCRA section 313 and PPA section 6607 (Reference (Ref. 1)). Chlorsulfuron was added to the EPCRA section 313 chemical list on November 30, 1994, based on concerns for developmental and reproductive toxicity (59 FR 61432). DuPont contends that newer studies show that chlorsulfuron does not cause developmental or reproductive toxicity and therefore no longer meets the EPCRA section 313(d)(2) criteria for listing. While the petition addressed the acute human health effects criterion of section 313(d)(2)(A) and chronic human health effects criterion of section 313(d)(2)(B), it did not address the environmental effects criterion of section 313(d)(2)(C).

## IV. What is EPA's evaluation of the human health toxicity of chlorsulfuron?

EPA's evaluation of the toxicity of chlorsulfuron included a review of the original 1994 listing decision (59 FR 1788, January 12, 1994 and 59 FR 61432, November 30, 1994), the 2002 Chlorsulfuron Toxicology Chapter (Ref. 2), the **Federal Register** Notice for Chlorsulfuron Pesticide Tolerance (67 FR 52866, August 14, 2002), and the Reregistration Eligibility Decision (RED) for Chlorsulfuron (Ref. 3). EPA also reviewed the findings of relevant studies published since the RED for chlorsulfuron was published (Ref. 4). Unit IV.A. below outlines evidence of human health toxicity from these existing EPA hazard characterizations and Unit IV.B. provides a brief summary of the findings from recently published studies. Unit IV.C. provides a summary of the ecological toxicity of chlorsulfuron from the existing EPA hazard characterizations.

### *A. Review of the Reregistration Eligibility Decision for Chlorsulfuron*

1. Kinetics and Metabolism

EPA concluded that chlorsulfuron is rapidly absorbed, metabolized, and eliminated when administered orally to rats (Ref. 2). There are no differences in absorption, distribution, and elimination of chlorsulfuron related to sex, dose, or treatment regimen. In one study, the major routes of elimination were found to be urine (58–72%) and feces (20–35%) with small amounts (0.1–0.2%) remaining in tissues (primarily in the liver and whole blood) three days after dosing (Ref. 5). This same study identified the major metabolic pathway of chlorsulfuron as the contraction of the sulfonylurea linkage followed by oxidation and hydroxylation to form IN–70941, IN–70942, Metabolite P5 (desmethyl IN–70942), and Metabolite P4 (OH-desmethyl IN–70942). The cleavage of the sulfonylurea linkage to form Metabolite IN–E9260 was identified as the minor metabolic pathway. No additional information on the absorption, distribution, metabolism, and excretion of chlorsulfuron was found in the literature.

2. Effects of Acute Exposure

EPA concluded that chlorsulfuron has no significant acute toxicity (Ref. 2). The conclusion was based on the results of a dermal study (Ref. 6), an inhalation study (Ref. 7) and on an oral study (Ref. 8).

3. Effects of Repeated Exposure

*a. Effects of subchronic exposure.* As stated in the 2002 Chlorsulfuron Toxicology Chapter (Ref. 2), there are few subchronic studies of chlorsulfuron in the literature. No 21- or 90-day dermal toxicity studies or 90-day inhalation studies were identified. Two subchronic oral toxicity studies were identified and summarized in the 2002 Chlorsulfuron Toxicology Chapter (Ref. 2). In a 90-day oral toxicity study, Smith et al. (Ref. 9) administered chlorsulfuron (100%) to 10 ChR–CD®-1 mice/sex/dose at dietary concentrations of 0, 500, 2,500, 5,000, and 7,500 ppm (equivalent to 0, 150, 783, 1,557, 2,130 milligrams/kilogram/day (mg/kg/day) in males and 0, 220, 1,214, 2,134, 3,176 mg/kg/day in females). The authors reported a lowest-observed-adverse-effect level (LOAEL) of 2,130 mg/kg/day based on increased incidence of retinal dysplasia. This study, however, lacked clinical chemistry and organ weight data. In a 6-month oral toxicity study, Schneider et al. (Ref. 10) administered chlorsulfuron (95%) to purebred Beagle dogs (4/sex/dose) in the diet at dose levels of 0, 100, 500, and 2,500 ppm (equivalent to 0, 3.7, 18.5, and 82.3 mg/kg/day). The authors reported a LOAEL of 82.3 mg/kg/day based on decreased body weight gain in females. Female body weight decreases were slight (91%, 93%, and 87% of control group in the low, mid, and high dose groups, respectively) and body weight decreases were observed in the treatment groups prior to treatment. The authors also noted that high-dose females also exhibited a lower food intake. Additionally, it does not appear that the animals were randomized by body weight at the beginning of the study, which makes these body weight findings more difficult to interpret. No other treatment-related effects were observed in any hematological, clinical chemistry, organ weights, or gross and microscopic pathology in animals of either sex. EPA concluded that the subchronic oral database does not identify toxicity to any particular target organ (Ref. 2).

*b. Carcinogenicity.* EPA concluded there is no evidence of carcinogenicity in rats or mice following oral exposure to chlorsulfuron (Ref. 2). In a chronic toxicity study, Wood et al., (Ref. 11) administered chlorsulfuron (95%) to 80 CD® rats/sex/dose in the diet at dose levels of 0, 100, 500, and 2,500 (equivalent to 0, 5, 25, and 125 mg/kg/day) for two years. The authors reported that the unilateral incidence of interstitial cell tumors was within the known spontaneous range for CD® rats and that there were no other changes suggestive of a treatment-related tumorigenic effect in the testes. In a similar carcinogenicity study, Wood et al., (Ref. 12) administered chlorsulfuron (91.9–95%) to 80 CD–1 mice/sex/dose in the diet at dose levels of 0, 100, 500, and 5,000 ppm (equivalent to 0, 15, 108, and 750 mg/kg/day) for two years. The authors reported no treatment-related increase in tumor incidence. No additional carcinogenicity studies were identified in the literature.

4. Reproductive and Developmental Toxicity

While the rabbit toxicity study cited in the 1994 **Federal Register** TRI listing of chlorsulfuron (59 FR 1788, January 12, 1994) reported a treatment-related increase in fetal resorptions and decrease in the mean incidence of live fetuses per litter at 75 mg/kg/day (Ref. 13), the results were not strongly indicative of a dose-response effect, were not able to be replicated in a follow-up study, and have recently been reanalyzed with improved methods. In this 1980 study cited in the original TRI listing of chlorsulfuron, Hoberman (Ref. 13) administered chlorsulfuron to pregnant female New Zealand white rabbits by oral intubation at doses of 0, 10, 25, and 75 mg/kg on days 6–19 of gestation. The author reported no significant changes in maternal body weight, uterine weight, ovarian weight, corpora lutea, visceral anomalies, or implantations. The author concluded that the increased mean incidence of resorptions in the highest dose group (31.3% versus 11.6% in the control group) and the decreased mean of live fetuses in the highest dose group (59.8% versus 88.5% in the control group) were significant treatment-related effects. A subsequent 1991 study performed in accordance with EPA guidelines (Ref. 14), however, was not able to replicate these findings using similar methods and higher doses (Ref. 15). This study concluded that chlorsulfuron does not cause an increase in fetal resorptions or decrease in fetal viability in rabbits up to 1,000 mg/kg/day, the highest dose tested (Ref. 15). Moreover, the 2002 Chlorsulfuron Toxicology Chapter (Ref. 2), the **Federal Register** Notice on Chlorsulfuron Pesticide Tolerance (67 FR 52866, August 14, 2002), and the Reregistration Eligibility Decision for Chlorsulfuron (Ref. 3) do not include fetal resorptions or decreased fetal viability as a part of the chlorsulfuron hazard characterization.

The 1994 **Federal Register** TRI listing of chlorsulfuron (59 FR 1788, January 12, 1994) also cited a slight treatment-related decrease in maternal fertility in the F3 generation observed in a rat chronic toxicity study (Ref. 11), but these findings have since been questioned. The design of this study is briefly summarized in the above Carcinogenicity Section (Unit IV.A.3.b.). The 2002 Chlorsulfuron Toxicology Chapter (Ref. 2) and the **Federal Register** Notice on Chlorsulfuron Pesticide Tolerance (67 FR 52866, August 14, 2002) concluded that the findings of this study are of questionable significance due to several study deficiencies. This study did not satisfy the current guideline (Ref. 14) requirements and contains numerous deficiencies including but not limited to: (1) No assessment of estrous cyclicity, (2) no assessment of male reproductive performance, (3) no gross pathology or histopathology examination of parental animals, (4) no assessment of developmental landmarks, and (5) histopathology examinations were conducted only for the F3B generation (Ref. 2; 67 FR 52866, August 14, 2002). As such, EPA classified this study as unacceptable.

EPA concluded that developmental toxicity was observed in both the rabbit (Ref. 15) and rat (Ref. 16), as evidenced by decreased fetal body weight (Ref. 2). However, treatment-related fetal body weight decreases in the rabbit study (Ref. 15) were slight (10%), occurred at a moderately high dose (LOAEL of 400 mg/kg/day), and were observed in the absence of other developmental effects. Additionally, decreased fetal body weight occurred in the presence of decreased maternal body weight. Adjusted maternal body weight gains throughout the study (days 0–29) in the highest treatment groups (original study: 200, 400 mg/kg/day; supplemental study: 400, 1,000 mg/kg/day) were substantially lower than those in the control group (78%, 54%, 43%, and 43% of control, respectively). In the original and supplemental studies, however, the adjusted maternal body weight gains in the treatment groups appeared to fall within the range of normal variation of control group animals. Also, the final adjusted maternal body weights in both these studies were not statistically different among treatment and control groups. Furthermore, it is not apparent that the study authors examined food consumption or food efficiency in either study. It is important to note that a dose of 1,000 mg/kg/day resulted in a high percentage of maternal mortality (i.e., much greater than 10%), which makes the developmental data in this dose group unreliable and of limited value based on the EPA Developmental Test Guidelines (Ref. 17). In the rat study, fetal toxicity was limited to decreased fetal weight in the highest dose group (1,500 mg/kg/day) and there were no teratogenic effects observed (Ref. 16). Dams in the highest dose group exhibited vaginal discharge associated with alopecia. Based on these data, the authors determined that the maternal LOAEL was 500 mg/kg/day and the developmental LOAEL was 1,500 mg/kg/day for rats.

5. Mutagenicity

A few mutagenicity studies were identified in the 2002 Chlorsulfuron Toxicology Chapter (Ref. 2), but none of these studies provided evidence of mutagenicity. Therefore, EPA concluded that there is no concern for mutagenicity of chlorsulfuron.

6. Neurotoxicity

There is no evidence of neurotoxicity in any study of chlorsulfuron.

7. Other Chronic Toxicity

In addition to the body weight findings from Alvarez (Refs. 15 and 16) discussed in the above Reproductive and Developmental Toxicity Section (Unit IV.A.4), several other chronic studies derived chlorsulfuron LOAELs based on observed changes in body weight and/or body weight gain. Wood et al. (Ref. 11) reported a LOAEL of 25 mg/kg/day based on decreased body weight in male rats in the highest dose groups (25 and 125 mg/kg/day). The reported decrease in body weight, however, was slight (4–9% and 5–10%, respectively) and decreased body weight gain was not significantly different between the highest treatment group and the control group when measured over the entire study. Additionally, no changes were reported in female rats and no other treatment-related effects were reported in the study. Wood et al. (Ref. 12) reported a LOAEL of 750 mg/kg/day due to decreased body weight and body weight gain in male and female mice. This high LOAEL, however, is indicative of moderately low to low chlorsulfuron toxicity. Atkinson et al. (Ref. 18) reported a LOAEL of 215 mg/kg/day based on

decreased body weight gain in female Beagle dogs. While these reported changes were observed in the absence of decreased food consumption, they were not found to be statistically significant. Moreover, body weight gains decreased in the highest dose group in the first half of the study (weeks 1–26), but there was no treatment-related effect on overall body weight gain over the entire study (weeks 1–52). Based on these findings, the evidence for body weight and body weight changes is not sufficient to conclude that chlorsulfuron is reasonably anticipated to cause serious or irreversible systemic toxicity.

*B. Review of Studies Published Since the Reregistration Eligibility Decision for Chlorsulfuron*

EPA identified and reviewed all relevant studies on chlorsulfuron that have been published since the RED for Chlorsulfuron (Ref. 3) was issued. After review of the recent literature, EPA concluded that there were no acceptable studies that strongly suggest either acute or chronic toxicity of chlorsulfuron (Ref. 4). Below are brief summaries of the findings from these studies identified in the recent literature.

1. Mylchreest Reproductive Study

In a 2-generation reproduction study, Mylchreest (Ref. 19) administered chlorsulfuron Crl:CD®(SD)IGS BR rats via the diet. The administered dose levels were 0, 100, 500, 2,500, and 7,500 ppm (average daily doses of 0, 6, 30, 151, 456 mg/kg/day in males and 0, 7, 39, 188, 591 mg/kg/day in females) throughout the 10-week premating period and throughout gestation and lactation. This study replicated the design of the Wood (Ref. 11) study with updates to ensure compliance with new EPA guidelines (Ref. 14) and good laboratory practices.

No treatment-related effects were reported in litter size, live birth index, number born dead, viability and lactation indices, clinical examinations, sex ratio, sexual maturation, organ weights, and gross or microscopic observations. The first generation (F1) sex ratio was significantly higher in the highest dose group (55% versus 45% males in the control group), but the authors did not consider this a treatment-related effect because it fell within the historical control range (45–59%). Lower offspring body weights were observed in the highest dose group in both generations, but these differences were not considered adverse because the magnitude of body weight changes was slight (5–7%) and the potential effect of larger litter size on pup weight. The authors reported an offspring no-observed-adverse-effect level (NOAEL) of 456 mg/kg/day in males and 498 mg/kg/day in females (note: the administered dose of 591 mg/kg-day was adjusted for decreased intake during gestation), the highest dose tested.

There were no treatment-related effects on ovarian follicles counts in F1 females, sperm and estrous cycle parameters in parental (P) and F1 adults, mating, precoital interval, fertility, gestation length, number of implantation sites, and implantation efficiency in either generation. As such, the authors reported a reproductive NOAEL of 456 mg/kg/day in males and 498 mg/kg/day in females (**note:** the administered dose of 591 mg/kg-day was adjusted for decreased intake during gestation), the highest dose tested. These results demonstrate that chlorsulfuron did not cause any treatment-related reproductive toxicity and its effects on parental body weight and food efficiency indicate moderately low to low toxicity.

2. Other Studies

In addition to the Mylchreest (Ref. 19) study, three other recent chlorsulfuron toxicity studies were identified in the literature. It is difficult to draw conclusions about these studies' findings, however, due to the lack of basic information provided by the authors. The studies contained numerous deficiencies including, no details on animal species or strain, the body weights of study animals were not reported (only an overall range was given), the age of the test animals was not reported, analytical methods were not described nor was their methodology for the different tests, etc. Given these deficiencies, findings from these studies were of very limited use in the determination of hazard for chlorsulfuron.

In an acute oral toxicity study, Rudaya et al. (Ref. 20) administered chlorsulfuron potassium salt intragastrically in male and female non-pedigreed white rats, male and female mice, and male rabbits of the Chinchilla line. The authors concluded that the $LD_{50}$ (i.e., the dose of a chemical that is lethal to 50 percent of the test organisms) was 5,580 ± 1,002 mg/kg for male rats, 5,500 ± 729 mg/kg for female rats, 2,050 ± 367 mg/kg for male mice, 2,460 ± 312 mg/kg for female mice, and 3,900 ± 451 mg/kg for male rabbits.

In a chronic oral toxicity study, Rudaya et al. (Ref. 21) examined the effect of chlorsulfuron potassium salt administered intragastrically in male white rats. Chlorsulfuron potassium salt was administered orally at dose levels of 0, 0.558, 5.58, and 55.8 mg/kg over 9 months. The authors reported several effects of chlorsulfuron potassium salt on the liver, kidneys, heart, and thyroid gland, and on behavior, but it is unclear from the study whether any of these effects were statistically or biologically significant. Based on these findings, the authors concluded that the no-effect dose of chlorsulfuron potassium salt was 0.558 mg/kg.

Rakitsky and Beloyedova (Ref. 22) studied the acute and chronic effects of several sulfonylurea herbicides, including chlorsulfuron, in rats, mice, dogs, and rabbits. The authors measured central nervous function, liver, kidney, and hematologic function up to several months after exposure. The authors reported an oral $LD_{50}$ in rats of 5,545–6,293 mg/kg in males and females, respectively, and a dermal $LD_{50}$ of 2,500 mg/kg in rabbits. The authors also reported an $LC_{50}$ (4 hours) of >5,900 mg/$m^3$ in rats. The authors reported a chronic no-observed-effect level (NOEL) of 0.2–5 mg/kg/day for rats, 108 mg/kg/day for mice, and 60.6 mg/kg/day for dogs, but they did not indicate from which health endpoints these NOELs were derived.

*C. Review of Ecological Effects*

1. Environmental Fate and Degradation

Chlorsulfuron is likely to be persistent and highly mobile in the environment. It may be transported to non-target areas via runoff and/or spray drift (Ref. 3). Degradation in the aquatic environment occurs primarily through hydrolysis at low pH (23 day half-life at pH 5) but it is stable in neutral to basic environments (Ref. 23). Aerobic aquatic metabolism data are not available; however, aerobic soil metabolism data suggest that aerobic aquatic metabolism may occur. Soil degradation half-lives have been reported to vary from 12 to 183 days (Ref. 24).

2. Ecological Toxicity and Hazard

*a. Toxicity to aquatic animals.* Experimental toxicity values are reported only for a few surrogate species. Estuarine data are limited to a crustacean, a mollusk, and a fish. No amphibians or reptiles were tested (Ref. 3). Chlorsulfuron is practically nontoxic to both freshwater and marine/estuarine fish and slightly toxic to estuarine/marine invertebrates when measured under acute conditions (Ref. 3). Chronic exposure of rainbow trout (*Oncorhynchus mykiss*) to chlorsulfuron resulted in a No Observed Effect Concentration (NOEC) of 32 mg/L (Ref. 25). The observed NOEC for water fleas (*Daphnia magna*) was 20 mg/L (Ref. 26).

*b. Toxicity to aquatic plants.* In contrast to the data for aquatic animals, for some species of aquatic plants the toxicity of chlorsulfuron is very high. (Ref. 3). Duckweed (*Lemna gibba*) was the most sensitive aquatic vascular plant (Refs. 27 and 28). Growth rate studies using endpoints for both biomass (dry weight) and the number of normal fronds found 14 day $EC_{50}$'s (concentration at which 50% of the plants are affected) of $3.5 \times 10^{-4}$ milligrams per liter (mg/L) and $4.2 \times 10^{-4}$ mg/L respectively (Table I). The 14 day NOEC for both biomass and the number of normal fronds was 0.24 µg/L (micrograms per liter) (Table I). The most sensitive nonvascular aquatic plant was the green alga *Pseudokirchneriella subcapitata* (formerly *Skeletonema costatum*) (120 hour (hr) $EC_{50}$ = 0.05 mg/L; 120 hr NOEC = 0.0094 mg/L) (Refs. 29 and 30) and measured acute toxicity to the freshwater blue-green alga *Anabaena flos-aquae* was also quite high (Refs. 31 and 32) (Table I).

TABLE I—SUMMARY OF ACUTE AND CHRONIC TOXICITY DATA OF CHLORSULFURON TO FRESHWATER AQUATIC PLANTS AND ALGAE

| Species | Common name | Toxicity | Citation |
|---|---|---|---|
| *Pseudokirchneriella subcapitata* (formerly known as *Selenastrum capricornutum*). | Green Algae | 120 hr $EC_{50}$ = 0.05 mg/L (cell density); 120 hr NOEC = 0.0094 mg/L (cell density). | Refs. 29 and 30. |
| *Lemna gibba* | Freshwater Duckweed | 14 day $EC_{50}$ = $3.5 \times 10^{-4}$ mg/L (biomass); 14 day $EC_{50}$ = $4.2 \times 10^{-4}$ mg/L (number of normal fronds); 14 day NOEC = $2.4 \times 10^{-4}$ mg/L (for both biomass and normal fronds). | Refs. 27 and 28. |
| *Anabaena flos-aquae* | Cyanobacteria | 120 hr $EC_{50}$ = 0.609 mg/L (area under the growth curve); 120 hr $EC_{50}$ = 1.77 mg/L (mean specific growth rate); 120 hr $EC_{50}$ = 0.807 mg/L (cell counts); 120 hr NOEC = 0.236 mg/L (area under the growth curve); 120 hr NOEC = 0.485 mg/L (mean specific growth rate); 120 hr NOEC = 0.236 mg/L (cell counts). | Refs. 31 and 32. |

*c. Toxicity to terrestrial animals.* Chlorsulfuron is practically nontoxic to birds and mammals in acute exposure regimes and chlorsulfuron is also practically nontoxic to birds given subacute dietary exposures (Refs. 3 and 33). Chronic toxicity to northern bobwhite quail (*Colinus virginianus*) included significant reductions in female body weight, decreased 14-day old survival, decreased number of normal hatchlings, decreased number of viable embryos (Ref. 34).

*d. Toxicity to terrestrial plants.* Chlorsulfuron exposure is known to affect nontarget plant fruit or seed production and may cause visible disease symptoms days or weeks after exposure (Ref. 3). Short term symptoms include spotting, leaf puckering or twisting, as well as chlorosis and discolored veins. Developmental and reproductive effects of exposure may not become apparent until three or four months after exposure. Reduced fruit development and decreased seed production due to chlorsulfuron exposure has been observed in canola, smartweed, soybean, and sunflower. Thus these types of chronic toxicity effects may be difficult to recognize in the field due to the time lag inherent in their expression.

Available experimental toxicity data for terrestrial plants (Refs. 33 and 34) reveals $EC_{25}$ (concentration at which 25 percent of the organisms are affected) values as low as $1.0 \times 10^{-5}$ lbs ai/A (pounds active ingredient per acre) measured for vegetative vigor (shoot dry weight) of nontarget plants (sugar beet). NOEC values of $5.4 \times 10^{-6}$ lbs ai/A for vegetative vigor (shoot dry weight) have been measured for onion and sugar beet.

*D. Summary of Human Health and Ecological Toxicity Evaluation*

Based on previous EPA hazard characterizations (Refs. 2 and 3; 67 FR 52866, August 14, 2002), there is sufficient evidence to support a low concern for human toxicity from exposure to chlorsulfuron. A more recent guideline (Ref. 14) study (Ref. 15) was not able to replicate findings from one of the studies upon which the addition of chlorsulfuron to the list of toxic chemicals subject to reporting requirements of EPCRA section 313 and section 6607 of the PPA was based (Ref. 13). Additionally, recent assessments of the studies cited in the listing of chlorsulfuron (Refs. 11 and 13) question the validity of these studies' methods and conclusions (Ref. 2; 67 FR 52866, August 14, 2002).

Additionally, no studies that strongly suggest either acute or chronic toxicity of chlorsulfuron were identified in the literature since the publication of the RED for chlorsulfuron (Ref. 3). A relatively recent guideline (Ref. 14) study (Ref. 19) was not able to replicate findings from another one of the studies upon which the addition of chlorsulfuron to the EPCRA section 313 toxic chemical list was based (Ref. 11). The reported findings from the other additional studies (Refs. 20, 21, and 22) were of very limited use in the determination of hazard for chlorsulfuron due to the study deficiencies previously outlined. While treatment-related body weight changes were observed in the Mylchreest study (Ref. 19), these changes were observed at a relatively high dose level (close to 500 mg/kg/day) and were observed in the absence of any other treatment-related effects.

Based on EPA's review of the available data, there is no compelling evidence of the acute toxicity, carcinogenicity, reproductive or developmental toxicity, mutagenicity, or other serious chronic toxicity of chlorsulfuron. While treatment-related body weight changes were observed in some studies, the evidence for these changes is not sufficient to conclude

that chlorsulfuron is expected to cause serious or irreversible systemic toxicity. Therefore, chlorsulfuron is not reasonably anticipated to cause acute or chronic toxicity in humans.

Chlorsulfuron has low toxicity to most aquatic and terrestrial animals. However, chlorsulfuron is highly toxic to some species of aquatic plants. Toxicity values ($EC_{50}$s) for aquatic plants are as low as $3.5 \times 10^{-4}$ mg/L indicating very high toxicity (Ref. 3).

## V. What is EPA's rationale for the denial?

EPA is denying the petition to delete chlorsulfuron from the EPCRA section 313 list of toxic chemicals. This denial is based on EPA's conclusion that chlorsulfuron can reasonably be anticipated to cause toxicity to aquatic plants. Chlorsulfuron has been shown to have an adverse effect on aquatic plant growth at very low concentrations with an $EC_{50}$ of $3.5 \times 10^{-4}$ mg/L for duckweed and an $EC_{50}$ of 0.05 mg/L for green algae as well as $EC_{50}$ of 0.609 mg/L for blue green algae. Therefore, EPA has concluded that chlorsulfuron meets the EPCRA section 313(d)(2)(C) listing criteria based on the available environmental toxicity data.

Because EPA believes that chlorsulfuron is highly toxic to aquatic plants, EPA does not believe that an exposure assessment is appropriate for determining whether chlorsulfuron meets the criteria of EPCRA section 313(d)(2)(C). This determination is consistent with EPA's published statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals (59 FR 61432, November 30, 1994).

## VI. References

EPA has established an official public docket for this action under Docket ID No. EPA–HQ–TRI–2013–0393. The public docket includes information considered by EPA in developing this action, including the documents listed below, which are electronically or physically located in the docket. In addition, interested parties should consult documents that are referenced in the documents that EPA has placed in the docket, regardless of whether these referenced documents are electronically or physically located in the docket. For assistance in locating documents that are referenced in documents that EPA has placed in the docket, but that are not electronically or physically located in the docket, please consult the person listed in the above **FOR FURTHER INFORMATION CONTACT** section.


1. DuPont Crop Protection. 2012. Petition to Delete Chlorsulfuron from TRI List. DuPont Crop Protection (DuPont), Technology Sciences Group Inc. (TSG). May 18, 2012.

2. U.S. EPA. 2002. Toxicology Chapter for Chlorsulfuron. Health Effects Division, Office of Pesticide Programs. July 17, 2002.

3. U.S. EPA. 2005. Reregistration Eligibility Decision for Chlorsulfuron. Office of Pesticide Programs. May 20, 2005.

4. U.S. EPA, OEI. 2013. Memorandum from Jocelyn Hospital, Toxicologist, Analytical Support Branch to Daniel Bushman, TRI Petitions Coordinator and Chemical List Manager, Analytical Support Branch. April 24, 2013. Subject: Review of Chlorsulfuron Studies Published Since Publication of the Reregistration Decision for Chlorsulfuron.

5. Hawking, D., Epsom, L., Garcon, R., et al. 1989. The absorption and Disposition of o carbon 14–DPX–E9636 in the Rat: Lab Project Number: HRC/DPT 190/891138: AMR–1197–88. Unpublished study prepared by Huntingdon Research Centre Ltd. 76p. As cited in Ref. 2.

6. Edwards, D.F. 1979. Acute Skin Absorption Test on Rabbits—$LD_{50}$: Haskell Laboratory Report No. 415–79. (Unpublished study received Sep 1, 1981 under 352–EX–109; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL:245879–I). As cited in Ref. 2.

7. Ferenz, R.L. 1980. $LC_{50}$-Inhalation Test for Pesticide Registration—AlbinoHaskell Laboratory Report No. 129–80. (Unpublished study received Nov 13, 1981 under 352–404; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL:070471–H). As cited in Ref. 2.

8. Trivits, R.L. 1979. Oral $LD_{50}$ Test in Fasted Male and Female Rats; Report No. 399–79. Unpublished study received Jun 16, 1980 under 352–EX–105; submitted by E.I. du Pont de Nemours & Co., Inc., Wilmington, DE; CDL:099460–A). As cited in Ref. 2.

9. Smith, L.W., Kaplan, A.M., Gibson, J.R. et al. 1980. Ninety-Day Range-Finding Feeding Study with 2-Chloro-N-o(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino carbonyl benzenesulfonamide (INW–4189) in Mice: Report No. 69–80. (Unpublished study including pathology report no. 55–78, received Jun 16, 1980 under 352–EX–105l submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL: 099461–B). As cited in Ref. 2.

10. Schneider, P.W., Jr., Smith, L.W., Barnes, J.R., et al. 1980. Six-Month Feeding Study in Dogs with 2-Chloro-N-o(4-methoxy-6-methy-1,3,5-triazin–2yl)amino carbonyl benzenesulfonamide (INW–4189): Report No. 108–80. Final rept. (Unpublished study including pathology report no. 53–79, received Jun 16, 1980 under 352–EX–105; submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:099461–A). As cited in Ref. 2.

11. Wood, C.K., Wollenberg, E.J., Turner, D.T., et al. 1981a. Long-Term Feeding Study with 2-chloro-N-6(4-methoxy-6-methyl-1,3,5-triazin-2-yl)aminocarbonylbenzenesulfonamide (INW–4189) in Rats. E.I. du Pont de Nemours & Company, Haskell Laboratory Report No. 557–81, November 13, 1981. MRID 0086003. Unpublished. As cited in Ref. 2.

12. Wood, C.K., Wollenberg, E.J., Turner, D.T., et al. 1981b. Long-Term Feeding Study with INW–4189 in Mice. E.I. du Pont de Nemours & Company, Haskell Laboratory Report No. 836–81, December 28, 1981. MRID 0090030. Unpublished. As cited in Ref. 2.

13. Hoberman, A.M. 1980. Teratology Study in Rabbits. E.I. du Pont de Nemours & Company, Haskell Laboratory, Newark, DE, Report No. HLO 534–80, July 17, 1980. Unpublished. As cited in 59 FR 1788, January 12, 1994.

14. U.S. EPA. 1998. Health Effects Test Guidelines OPPTS 870.3800 Reproduction and Fertility Effects. Washington, DC. EPA 712–C–98–208.

15. Alvarez, L. 1991a. Teratogenicity Study of DPX–W4189 (Chlorsulfuron) in Rabbits. E.I. du Pont de Nemours & Company, Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE, Laboratory Project ID: 306–390, August 12, 1991. Unpublished. As cited in Ref. 2.

16. Alvarez, L. 1991b. Teratogenicity Study of DPX–W4189 (Chlorsulfuron) in Rats. E.I. du Pont de Nemours & Company, Haskell Laboratory for Toxicology and Industrial Medicine, Newark, DE, Laboratory Project ID: 734–90, February 27, 1991. Unpublished. As cited in Ref. 2.

17. U.S. EPA. 1991. Guidelines for Developmental Toxicity Risk Assessment. Risk Assessment Forum, Washington, DC. EPA/600/FR–91/001.

18. Atkinson, J. 1991. A Chronic (1 Year) Oral Toxicity Study in the Dog with DPX–W4189 (Chlorsulfuron) via the Diet: Lab Project Number: 89/3501: 163/91. Unpublished study prepared by Bio/dynamics, Inc. 716 p. As cited in Ref. 2.

19. Mylchreest, E. 2005. Chlorsulfuron (DPX–W4189) Technical: Multigeneration Reproduction Study in Rats. DuPont Haskell Laboratory for Health and Environmental Sciences, Newark, DE, Laboratory Project ID: DUPONT.13495,14601,904. September 11, 2003–June 4, 2004. MRID 46493201. Unpublished. As cited in Ref. 4. Data Evaluation Record: Taylor, Linda (2007), MRID 46493201.

20. Rudaya, P.L., and Zhminko, P.G. 2009. Toxic Properties of Chlorsulfuron Potassium Salt Herbicide Administered Once Orally to Mammals. Modern Problems of Toxicology 2: 59–65. *Translated from Ukrainian.*

21. Rudaya, P.L., Zhminko, P.G., Povyakel, L.I., and Reshavska, O.V. 2010. Toxicodynamics of Chlorsulfuron Potassium Salt Given Orally In Long-Term Experiment on White Rats. Modern Problems of Toxicology 1: 59–63. *Translated from Ukrainian.*

22. Rakitsky, V.N. and Beloyedova, N.S. 2009. Toxicity and Hazardousness of Sulfonylurea Herbicides. Toxicology Herald 4: 25–30. *Translated from Russian.*

23. Dietrich, R. and McAleer, N. (1989) Hydrolysis of Phenyl(U)-$C^{14}$-Chlorsulfuron and Triazine-2-$C^{14}$-Chlorsulfuron: Lab Project Number: AMR–1455–89. 161–1. Unpublished study prepared by E.I. du Pont de Nemours & Co., Inc. 61 pp. MRID: 42156701. As cited in Ref. 3.

24. Hawkins, D., Kirkpatrick, D., Dean, G., et al. (1990) The Photodegradation of $C^{14}$-

Chlorsulfuron on a Silty Clay Loam Soil: Lab Project Number: HRC/DPT 205/90571: AMR–1563–89. Unpublished study prepared by Huntingdon Research Centre Ltd. 58 pp. MRID: 42156703. As cited in Ref. 3.

25. Pierson, K. (1991) Flow-Through 77 Day Toxicity of DPX–W4189–170 to Embryo and Larval Rainbow Trout, *Oncorhynchus mykiss.* Lab Project Number 494–91: MR–4581–866. Unpublished study prepared by E.I. du Pont de Nemours and Co. 471 pp. MRID: 41976405. As cited in Ref. 3.

26. Hutton, D. (1991) Chronic Toxicity of DPX–W4189–94 to *Daphnia magna.* Lab Project Number: 4581–655: 87–89. Unpublished study prepared by E.I. du Pont de Nemours and Co. 92 pp. MRID: 41976408. As cited in Ref. 3.

27. Boeri, R., Wyskiel, D., Ward, T. (2002) Chlorsulfuron (DPX–W4189) Technical: Influence on Growth and Growth Rate of the Duckweed, *Lemna gibba:* Lab Project Number: 2042–DU: DUPONT–4468: ASTM E1415–91. Unpublished study prepared by T.R. Wilbury Laboratories. 38 pp. MRID: 45832901. As cited in Refs. 3 and 28.

28. Bryan, R., Worcester, D., Brichfield, N., Ballaff, D. (2003a) Data Evaluation Report on the acute toxicity of Chlorsulfuron to aquatic vascular plants *Lemna gibba.* 12 pp. MRID: 45832901.

29. Blasberg, J., Hicks, S., Stratton, J. (1991) Acute Toxicity of Chlorsulfuron to *Selenastrum capricornutum* Printz,: Final Report: Lab Project Number: AMR–2081–91: 39427. Unpublished study prepared by ABC Laboratories, Inc., 33 pp. MRID: 42186801. As cited in Ref. 30.

30. Levy, B., Myers, T., Behl, E., Ballaff, D. (2003) Data Evaluation Report on the acute toxicity of Chlorsulfuron to algae *Selenastrum capricornutum* Printz. 12 pp. MRID: 42186801.

31. Boeri, R., Wyskiel, D., Ward, T. (2001b) Chlorsulfuron (DPX–W4189) Technical: Influence on Growth and Growth Rate of the Alga, *Anabaena flos-aquae:* Lab Project Number: DU: DUPONT–4466:2044DU. Unpublished study prepared by T.R. Wilbury Laboratories. 38 pp. MRID: 45832903. As cited in Refs. 3 and 32.

32. Bryan, R., Worcester, D., Brichfield, N., Ballaff, D. (2003b) Data Evaluation Report on the acute toxicity of Chlorsulfuron to the freshwater algae *Anabaena flos-aquae.* 14 pp. MRID: 45832903.

33. Hinkle, S. (1979) Final Report—Avian Dietary Toxicity ($LC_{50}$) Study in Bobwhite Quail: Project No. 201–523 (Unpublished study received June 16, 1980 under 352–105; prepared by Hazelton Laboratories America, Inc., submitted by E.I. du Pont de Nemours & Co., Wilmington, DE; CDL:099462–K). MRID: 00035265. As cited in Ref. 3.

34. Beavers, J., Foster, J., Lynn, S. et al. (1992) H–18,053 (Chlorsulfuron): A One-generation Reproduction Study with the Northern Bobwhite (*Colinus virginianus*): Lab Project Number: 112–266: 564–92. Unpublished study prepared by Wildlife International Ltd. 185 pp. MRID: 42634001. As cited in Ref. 3.

35. Porch, J. and Martin, K. (2004a) Chlorsulfuron (DPX–W4189) 75WG: A Greenhouse to investigate the Effects on Vegetative Vigor of Ten Terrestrial Plants Following Foliar Exposure. Project Number: 112/542, DUPONT/13552, 14901. Unpublished study prepared by Wildlife International, Ltd.. 191 pp. MRID: 46326801. As cited in Ref. 3.

36. Porch, J. and Martin, K. (2004b) Chlorsulfuron (DPX–W4189) 75WG: A Greenhouse to investigate the Effects on Vegetative Vigor of Ten Terrestrial Plants Following Soil Exposure. Project Number: 112/541, 14901, 1495. Unpublished study prepared by Wildlife International, Ltd. 264 pp. MRID: 46361801. As cited in Ref. 3.

**List of Subjects in 40 CFR Part 372**

Environmental protection, Community right-to-know, Reporting and recordkeeping requirements, and Toxic chemicals.

Dated: November 18, 2013.

**Arnold E. Layne,**

*Director, Office of Information Analysis and Access.*

[FR Doc. 2013–28365 Filed 12–6–13; 8:45 am]

**BILLING CODE 6560–50–P**

## FEDERAL COMMUNICATIONS COMMISSION

### 47 CFR Part 73

**[MB Docket No. 13–284; RM–11704; DA 13–2241]**

### Radio Broadcasting Services; Evart and Ludington, Michigan

**AGENCY:** Federal Communications Commission.

**ACTION:** Proposed rule.

**SUMMARY:** This document proposes, at the request of Synergy Lakeshore Licenses, LLC (''Synergy''), licensee of Station WMLQ(FM), Manistee, Michigan, the deletion of vacant FM Channel 274A at Evart, Michigan. The document also proposes the return of Stations WMLQ(FM), Manistee, and WMOM(FM), Pentwater, Michigan, to the channels that they previously occupied, and the modification of the construction permit for a new FM station at Ludington, Michigan. *See* **SUPPLEMENTARY INFORMATION**, *supra.*

**DATES:** Comments must be filed on or before January 13, 2014, and reply comments on or before January 28, 2014.

**ADDRESSES:** Secretary, Federal Communications Commission, 445 12th Street SW., Washington, DC 20554. In addition to filing comments with the FCC, interested parties should serve the petitioner as follows: David D. Oxenford, Esq., Wilkinson Barker Knauer, LLP, 2300 N Street NW., Suite 700, Washington, DC 20037–1128.

**FOR FURTHER INFORMATION CONTACT:** Andrew J. Rhodes or Rolanda F. Smith, Media Bureau, (202) 418–2700.

**SUPPLEMENTARY INFORMATION:** This is a synopsis of the Commission's Notice of Proposed Rule Making, MB Docket No.13–284, adopted November 21, 2013, and released November 22, 2013. The full text of this Commission decision is available for inspection and copying during normal business hours in the FCC's Reference Information Center at Portals II, CY–A257, 445 12th Street SW., Washington, DC 20554. This document may also be purchased from the Commission's duplicating contractors, Best Copy and Printing, Inc., 445 12th Street SW., Room CY–B402, Washington, DC 20554, telephone 1–800–378–3160 or via email *www.BCPIWEB.com.* This document does not contain proposed information collection requirements subject to the Paperwork Reduction Act of 1995, Public Law 104–13. In addition, therefore, it does not contain any proposed information collection burden ''for small business concerns with fewer than 25 employees,'' pursuant to the Small Business Paperwork Relief Act of 2002, Public Law 107–198, *see* 44 U.S.C. 3506(c)(4).

Previously, we allotted Channel 274A at Evart, Michigan. In order to accommodate this new allotment, we modified the licenses for Station WMOM(FM), Pentwater, from Channel 274A to Channel 242A, and Station WMLQ(FM), Manistee, from Channel 249A to Channel 282A. We required the ultimate permittee of Channel 274A at Evart to reimburse Stations WMOM(FM) and WMLQ(FM) for their reasonable costs in changing channels. We also substituted Channel 249A for vacant Channel 242A at Ludington, Michigan. See 74 FR 13125, March 26, 2009.

The document solicits comment on whether vacant Channel 274A at Evart should be deleted because it went unsold in Auction 94. Interested parties should file comments expressing an interest in this vacant allotment to prevent its removal and provide an explanation as to why they did not participate in our competitive bidding process.

We issue an Order to Show Cause to the licensee of Station WMOM(FM), Pentwater, to show cause as to why its license should not be modified to specify Channel 274A in lieu of Channel 242A. We also propose to modify the construction permit for a new FM station at Ludington, Michigan, from Channel 249A to Channel 242A in order to accommodate the previously discussed channel changes. It is not