SECTION-1: Identification of the substance / mixture and the company / undertaking

Catalogue Number	CS-T-23746
Product Name	Flufenoxuron
CAS No.	101463-69-8
Category	Pesticide Standards
Synonyms	Not available
Brand	Clearsynth Labs Ltd.
Identified uses	Laboratory Chemicals
Uses advised against	Not available
Company	Clearsynth Labs Ltd. Mumbai, India
Emergency Phone #	+91-22-245045900
REACH No.	Not available

SECTION 2: Hazards identification

Disclaimer: This is sample MSDS. Please email **sales@clearsynth.com** for more details.

2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Not available

2.2 Label Elements

Signal Word: Warning



Hazard Statement(s)

Code	Statement
H362	Not available
H400	Not available
H410	Not available

Precautionary Statement(s)

Code	Statement
P203	Not available
P260	Not available
P263	Not available
P264	Wash hands thoroughly after handling.
P270	Not available
P273	Not available
P318	Not available
P391	Not available
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Flufenoxuron
CAS Number : 101463-69-8
Molecular Formula : C21H11ClF6N2O3
Molecular Weight : 488.77
Parent Chemical : -
Synonyms : Not available
Concentration : Not available

SECTION 4: First aid measures

Not available

SECTION 5: Firefighting measures

Not available

SECTION 6: Accidental release measures

Not available

SECTION-7: Handling and storage

Not available

SECTION 8: Exposure controls / personal protection

Not available

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	No data available
IR spectrum	No data available
pH	No data available
Solubility	No data available

Property	Value
a) Physical State	No data available
b) Color	No data available
c) Odor	No data available
d) pH	No data available
e) Vapour Pressure	No data available
f) Viscosity	No data available
g) Initial Boiling Point and boiling range	No data available
h) Melting Point / Freezing Point	No data available
i) Auto Ignition Temperature	No data available
j) Flash Point	No data available
k) Explosion Limit, Lower	No data available
l) Explosion Limit, Upper	No data available
m) Decomposition Temperature	No data available
n) Loss on Drying	No data available
o) Relative Density	No data available
p) Solubility (in DMSO)	No data available

Property	Value
q) Oxidizing Properties	No data available

SECTION 10: Stability and reactivity

Not available

SECTION 11: Toxicological information

11.1 Information on toxicological effects
- Acute toxicity: /CASE REPORTS/ A 72-year-old woman was brought to the emergency department by ambulance. The person accompanying her brought an empty 100 mL bottle of an insecticide (Cascade), which was found at the scene. The active ingredient of the product is flufenoxuron and the other components include surfactants and solvents. A detailed composition obtained from the manufacturer was flufenoxuron, ethoxylated nonylphenol phosphate, polyoxyethylene nonylphenol, N-methyl-2-pyrrolidone, and cyclohexanone. Upon arrival at the intensive care unit (ICU), her arterial pH was 7.093, her bicarbonate level was 7.4 mEq/L, and the anion gap was 33.8 mEq/L. Her lactic acid concentration was 16.5 mmol/L. Lactic acidosis was not considered to be a consequence of circulatory shock, because there was no clinical sign of shock other than lactic acidosis, and cardiac output was never below 4.5 L/min. Her acid-base status began to improve and returned to near normal on the next day. It can be hypothesized that the toxicity of the product includes inhibition of the oxygen utilization mechanism at the cellular level. The product is composed of a number of components, similar to many other herbicide products. It is not possible to identify which of the ingredients was specifically responsible for the toxic effects in this case. /GENOTOXICITY/ In a mammalian cell cytogenetics assay (Chromosome aberration), human lymphocyte (whole blood) cultures were exposed to WL115110 (/flufenoxuron/ 98.1% ai) in dimethyl sulfoxide (DMSO) at concentrations of 0, 78.4, 112, or 160 ug/mL for three hours with and without metabolic activation (S-9 mix). Cells were harvested 17 hours following termination of treatment both with and without S-9 mix. Cells were also harvested 41 hours following termination of treatment in another experiment at the highest concentration with S-9 mix. An additional assay was conducted by exposing cells to test material concentrations of 0, 78.4, 112, or 160 ug/mL without S9-mix for 20 hours with immediate post-treatment harvest. Yet another assay was conducted by exposing cells to test material concentrations of 0 or 160 ug/mL without S9-mix for 44 hours with immediate posttreatment harvest. Blood was obtained from a healthy male donor. The S9-fraction was obtained from Aroclor 1254 induced male Sprague-Dawley rat liver. Flufenoxuron was tested up to concentrations limited by solubility. The maximum mitotic inhibition detected was a decrease of 42% in the mitotic index following the 20-hour treatment at the highest concentration in the absence of S-9 mix. Precipitate was seen in the culture media at all concentrations tested. There were no statistically significant increases in the percentage of cells with structural aberrations including or excluding gaps or in polyploidy over the solvent control values at any test material concentration with or without S9-mix. The solvent and positive control values were appropriate, and solvent control values were within the testing laboratory's historical control ranges in all assays. There was no evidence of chromosome aberrations induced over background.
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: /GENOTOXICITY/ In a mammalian cell cytogenetics assay (Chromosome aberration), human lymphocyte (whole blood) cultures were exposed to WL115110 (/flufenoxuron/ 98.1% ai) in dimethyl sulfoxide (DMSO) at concentrations of 0, 78.4, 112, or 160 ug/mL for three hours with and without metabolic

activation (S-9 mix). Cells were harvested 17 hours following termination of treatment both with and without S-9 mix. Cells were also harvested 41 hours following termination of treatment in another experiment at the highest concentration with S-9 mix. An additional assay was conducted by exposing cells to test material concentrations of 0, 78.4, 112, or 160 ug/mL without S9-mix for 20 hours with immediate post-treatment harvest. Yet another assay was conducted by exposing cells to test material concentrations of 0 or 160 ug/mL without S9-mix for 44 hours with immediate posttreatment harvest. Blood was obtained from a healthy male donor. The S9-fraction was obtained from Aroclor 1254 induced male Sprague-Dawley rat liver. Flufenoxuron was tested up to concentrations limited by solubility. The maximum mitotic inhibition detected was a decrease of 42% in the mitotic index following the 20-hour treatment at the highest concentration in the absence of S-9 mix. Precipitate was seen in the culture media at all concentrations tested. There were no statistically significant increases in the percentage of cells with structural aberrations including or excluding gaps or in polyploidy over the solvent control values at any test material concentration with or without S9-mix. The solvent and positive control values were appropriate, and solvent control values were within the testing laboratory's historical control ranges in all assays. There was no evidence of chromosome aberrations induced over background.
- Carcinogenicity: /LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ In a 52-week oral chronic toxicity study, WL 115110 (/flufenoxuron/ 98 % ai) was administered to four beagle dogs/sex/dose in the diet at dose levels of 0, 10, 100, 500, or 50,000 (> limit dose) ppm (0, 0.75, 7.5, 37.5 or 3750 mg/kg/day). There were no differences between the controls and the flufenoxuron treated groups in body weights, food and water consumption, ophthalmology, electrocardiography, clinical chemistry, or urinalysis. There were no treatment related mortalities and no treatment related clinical observations. One female dog in the 500 ppm group diagnosed with "autoimmune hemolytic anemia" was humanly sacrificed. Males that received 50,000 ppm flufenoxuron showed increases in absolute liver weight of 136% of the control ($p<0.001$). In males in the 50,000 ppm group, the levels of methemoglobin were increased from 208 to 296% of the control ($p<0.05$ or $p<0.01$) during Weeks 5 to 40. The levels of sulfhemoglobin were also increased from 214 to 680% of the control ($p<0.05$ and 0.001) in the same group through out the study period. In addition, in the 500 ppm group, there were increases of methemoglobin (159% of the control, $p<0.05$) but only in Week 27 and sulfhemoglobin (267 to 286% of the control, $p<0.05$) during Weeks 27 and 40. These changes were associated with a decrease in hemoglobin and erythrocyte values in the 50,000 ppm group and to a lesser extent, in the 500 ppm group. In addition, these changes also correlated with an increase in mean cell volume of red blood cells in the 50,000 ppm group in Weeks 13 to 40 and in the 500 ppm group at Week 27. The effects of flufenoxuron on hematology parameters at 50,000 ppm was first observed at Week 5 and continued. The destruction of red blood cells by flufenoxuron was compensated by the increase of red blood cell production and platelets. These effects on the blood also correlated with increases in brown pigment deposition in the liver Kupffer cells, hemosiderin deposition in the spleen, and in bone marrow hypercellular activity. Increased reticulocyte and platelet counts ranged from 213 to 350% and 151 to 191% of the controls, and attained statistical significance for dogs that received 50,000 ppm from Week 5 to the end of the study, respectively. In females, similar hematological effects as seen in males , were observed, however, to a lesser extent. The LOAEL is 37.5 mg/kg/day (500 ppm), based on decreased erythrocytes counts, and mean cell hemoglobin concentration and increased mean cell volume, platelet counts in males, methemoglobinemia and sulfhemoglobinemia in males and females. The NOAEL is 7.5 mg/kg/day (100 ppm).
- Reproductive toxicity: No data available.
- STOT-single exposure: No data available.
- STOT-repeated exposure: /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a 90-day oral toxicity study, WL 115110 (/flufenoxuron/ 98 % ai) was administered to four beagle dogs/sex/dose in the diet at dose levels of 0, 0.05% (500 ppm), 0.5% (5000 ppm), or 5% (50,000 ppm) (0, 38, 375 or 3750 (> 3.5 times limit dose) mg/kg/day, respectively). A mistake in the dose preparation of the 0.5% group was corrected in the second week of the study, and therefore the study was extended to 15 weeks. There were no differences between the controls and

the flufenoxuron treated groups in body weights, food and water consumption, ophthalmology, and electrocardiography. There was no mortality during the study. The clinical observations were confined to pallor of the gums or sclera which occurred late in the study in the mid-and high-dose groups. In all treated males, the levels of methemoglobin were increased in a dose-related manner and were significantly higher ($p<0.001$) in the mid-and high-dose groups than the controls. The effect was seen in males of the 50,000 ppm group by Week 2 ($p<0.05$). The levels of sulfhemoglobin were also increased ($p<0.05$ and $p<0.001$) in the mid-and high-dose groups. These changes were associated with the decreases in hemoglobin, hematocrit and erythrocyte counts in the mid-and high-dose groups at Week 9, with recovery by Week 15. In addition, these changes also correlated with the increase in mean cell volume of red blood cells in the mid-dose group (at Week 9) which persisted to Week 15 in the high dose group. The effect of flufenoxuron on hematology parameters at doses > or = 0.05% was first observed at Week 9, followed by some compensatory effect and recovery in the low-and mid-dose groups. The effect persisted to Week 15 in male beagle dogs that received the mid- and high doses. Similar effects of flufenoxuron were observed in the treated females but to a lesser extent than males in the mid-and high-dose groups. In females, the presence of methemoglobinemia in the mid-and high-dose groups had no effect on red blood cells parameters. The destruction of red blood cells by flufenoxuron was compensated by increased red blood cells production and was associated with the increased incidence of yellow pigment deposition in liver Kupffer cells and in the bone marrow. Histologically, bone marrow hyperplasia was observed in the males and females at doses > or = 0.05% of flufenoxuron. Males showed increases in absolute and relative liver weight. Males in the high dose group exhibited a slight reduction in body weight gain. In contrast, no remarkable changes were found in females. There were a number of sporadic changes in clinical chemistry analyses found, such as the elevated cholesterol levels in mid-and high-dose groups of males. No remarkable changes were found in the females. The systemic toxicity LOAEL is 38 mg/kg/day (500 ppm) based on decreased hemoglobin, hematocrit levels and erythrocyte counts in males; increased absolute liver weights, bone marrow hyperplasia and methemoglobinemia in males and females. The systemic toxicity NOAEL was not established. /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In a 90-day oral study, flufenoxuron (96.6% ai) was administered in the diet to groups of 10 male and 10 female mice in the treated groups and 20 male and 20 female mice in the control groups. An additional subgroup of ten mice/sex/dose group in the treated groups and twenty mice/sex for controls were used to provide blood for clinical chemistry. The concentrations administered were 0 (controls), 50, 500, 5,000, 10,000, or 50,000 ppm (equivalent to 0, 7.5, 75, 750, 1500 or 7500 mg/kg/day, respectively, based on a food conversion factor of 0.15). ... Five animals either died or were sacrificed prematurely during the study. These included two females in the 5,000 ppm group, two males in the 10,000 ppm group and one female in the 50,000 ppm subgroup used for clinical chemistry bleeding. The causes of death were not determined in necropsy and did not appear to be treatment-related. The only clinical sign observed in any group, treated or controls, was the presence of pale feces in the highest-dosed group in both males and females throughout the study which appeared to be unabsorbed compound. Mean body weight was decreased in the highest-dose (50,000 ppm) males compared to controls throughout the study, although these decreases were <10% of controls. Mean body weight gain was also decreased in the 50,000 ppm males starting at week three and ranged from 14-28% less than controls without a time-response. Overall, the high-dose males had a total weight gain that was 22% less than controls. Treated females had body weight and weight gain comparable to controls throughout the study. Food consumption data were marginally collected. From the limited data endpoints, males did not exhibit any treatment-related differences and no conclusions could be made on the females. A statistically significant ($p < or = 0.01$) decrease in some erythrocyte parameters including: red blood cell count, erythrocyte cell size variability (red cell distribution width), mean hemoglobin, and mean hematocrit, were observed in high-dose males. High-dose females also demonstrated a statistically significant ($p < or = 0.01$) decrease in red cell width distribution. Bilirubin concentrations were statistically increased ($p < or = 0.01$) in both the treated male and female mice compared to controls at doses above 50 ppm and exhibited a dose response, although the increases were slight and did not increase to the extent expected to correspond with the increase in dose

concentration. In males, other differences observed were decreases in urea nitrogen starting at 500 ppm and decreases in triglycerides starting at 5000 ppm; however, these did not have a dose-response. Females had a statistically significant decrease in urea in the 10,000 and 50,000 ppm groups that was dose-responsive. While some of the differences in the clinical chemistry and hematology results support a treatment-related anemia, additional parameters such as methemoglobulin values, reticulocyte counts, myeloid/erythroid ratios and splenic weights and appearances were within normal range. A statistically significant ($p < or = 0.05$ or 0.01) increase in liver organ weight when adjusted to the terminal body weight was observed in the male and female mice beginning at the 500 ppm dose group; although there were not any corresponding hepatic lesions on gross or histopathological examination. Body weight changes, hematology parameters and bilirubin levels in males and females at the highest dose tested were marginal and were within biological variation for this strain and of mice, therefore, considered to be no biological significance. The NOAEL = 50,000 ppm (7500 mg/kg/day, HDT) which is 7.5x the limit dose. LOAEL was not established.
- Aspiration hazard: No data available.

Likely routes of exposure
- LC50 Rat inhalation >5.1 mg/L/4 hr

Symptoms related to the physical, chemical and toxicological characteristics
- /CASE REPORTS/ A 72-year-old woman was brought to the emergency department by ambulance. The person accompanying her brought an empty 100 mL bottle of an insecticide (Cascade), which was found at the scene. The active ingredient of the product is flufenoxuron and the other components include surfactants and solvents. A detailed composition obtained from the manufacturer was flufenoxuron, ethoxylated nonylphenol phosphate, polyoxyethylene nonylphenol, N-methyl-2-pyrrolidone, and cyclohexanone. Upon arrival at the intensive care unit (ICU), her arterial pH was 7.093, her bicarbonate level was 7.4 mEq/L, and the anion gap was 33.8 mEq/L. Her lactic acid concentration was 16.5 mmol/L. Lactic acidosis was not considered to be a consequence of circulatory shock, because there was no clinical sign of shock other than lactic acidosis, and cardiac output was never below 4.5 L/min. Her acid-base status began to improve and returned to near normal on the next day. It can be hypothesized that the toxicity of the product includes inhibition of the oxygen utilization mechanism at the cellular level. The product is composed of a number of components, similar to many other herbicide products. It is not possible to identify which of the ingredients was specifically responsible for the toxic effects in this case.

SECTION 12: Ecological information

Not available

SECTION 13: Disposal considerations

Not available

SECTION 14: Transport information

Not available

SECTION 15: Regulatory information

Not available

SECTION 16: Other information

Not available

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