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

Catalogue Number	CS-T-19517
Product Name	Dimethenamid
CAS No.	87674-68-8
Category	Pesticide Standards
Synonyms	2-Chloro-N-(2,4-dimethyl-3-thienyl)-N-(2-methoxy-1-methylethyl)acetamide
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:

Acute toxicity (Category 4)

2.2 Label Elements

Signal Word: Warning

Hazard Statement(s)

Code	Statement
H302	Harmful if swallowed.
H317	May cause an allergic skin reaction.
H332	Harmful if inhaled.
H400	Not available

H410	Not available
H411	Toxic to aquatic life with long lasting effects.
H351	Not available
H372	Not available
H401	Not available

Precautionary Statement(s)

Code	Statement
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P270	Not available
P271	Use only outdoors or in a well-ventilated area.
P272	Not available
P273	Not available
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P301+P317	Not available
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P317	Not available
P321	Specific treatment (see … on this label).
P330	Not available
P333+P317	Not available
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Not available
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P203	Not available
P260	Not available
P318	Not available
P319	Get medical help if you feel unwell.
P405	Store locked up.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Dimethenamid
CAS Number : 87674-68-8
Molecular Formula : C12H18ClNO2S
Molecular Weight : 275.79
Parent Chemical : -
Synonyms : 2-Chloro-N-(2,4-dimethyl-3-thienyl)-N-(2-methoxy-1-methylethyl)acetamide
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
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: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and

dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations, /GENOTOXICITY/ ... Isolated and cultured peripheral lymphocytes (mostly T cells) were used from two human donors to study the effects of the chloroacetanilides and their metabolites on primary human cells. In tests at 10 uM, the SCE frequency was increased by alachlor and possibly acetochlor but not by butachlor, metolachlor, dimethachlor (a 2,6-dimethyl analog) and dimethenamid (an analog based on 2,4-dimethyl-3-thienylamine). At 0.3 microM in cultured human lymphocytes, alachlor, the corresponding chloroacetanilide (N-dealkyl-alachlor) and aniline metabolites (and their 4-hydroxy derivatives), and diethylbenzoquinone were inactive or active in only one of the two donors whereas at 0.1-0.3 uM the SCE ratio for treated cells divided by the controls was always higher for diethylbenzoquinoneimine than for ethylmethyl- and dimethylbenzoquinoneimines. All the tested compounds were toxic to lymphocytes, but the depression of the mitotic index and increased duration of the cell cycle were not directly linked with SCE induction. Previous investigations have suggested that chloroacetanilide herbicides such as alachlor derived from 2,6-dialkylanilines are metabolized to 2,6-dialkylbenzoquinoneimines and the present study provides the first direct evidence that these metabolites are genotoxic in human lymphocytes.
- Skin corrosion/irritation: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats

and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations, /HUMAN EXPOSURE STUDIES/ The Sandoz Agro manufacturing facility at Beaumont, Texas, USA, produced three batches of Frontier herbicide in 1991 and one batch in 1992. Between five and ten people were involved in the process, which included charging raw materials, process sampling and manually filling containers with the final formulated material. In 1991, approximately 750 containers with a volume of 1 L and 475 containers with a volume of 0.5 L were filled, and in 1992 800 containers of 4 L in volume were filled. Employees wore protective gloves and clothing. Approximately five to seven people in the Research and Development Formulations group handled racemic dimethenamid and its formulations between 1986 and 1992. Activities included material transfer, drying and packaging. Employees wore protective gloves and clothing and were often working under protective hoods. During product development field trials, 20 people worked with racemic dimethenamid products between 1984 and 1991. Activities including mixing, loading and application of the formulation product for application and spraying of the product diluted in water. Most applications were made with a backpack hand sprayer and some applications were with a tractormounted sprayer. These activities were conducted 7-15 times per year. Personnel wore protective gloves and clothing during handling of the product. In none of the three groups surveyed were there any cases of skin irritation, skin rash or other signs of allergic response. In addition, no general signs of adverse health effects were reported. In addition, the sponsor states that: (1) no poisoning incidents are known; (2) no observations regarding health effects after exposure of the general public are known; (3) methods for determination of active substance or metabolites in biological fluids are not established; (4) specific signs of poisoning or clinical tests are not known; (5) no specific antidote is known and (6) expected effects of poisoning (irritation of exposed eyes and skin, dermatitis and eczema) were derived from studies in animals.

- Serious eye damage/eye irritation: /LABORATORY ANIMALS: Acute Exposure/ The acute oral toxicity of racemic dimethenamid (purity, 97.1%) was investigated in groups of five male and five female fasted Sprague-Dawley rats given undiluted herbicide at a dose of 150, 300 or 600 mg/kg bw by gavage. The rats were observed for 14 days after dosing. Clinical observations on the day of dosing included oral and ocular discharges and hypoactivity at all doses. In addition, at 600 and 300 mg/kg bw, some rats developed nasal discharge, wet rales, fecal staining, soft stools and abdominal griping. Signs seen only at 600 mg/kg bw were irregular gait, coarse and fine tremors, hypopnea, irregular breathing, urinary staining and prostration. Clinical signs seen after the day of dosing included hypoactivity and decreased food consumption and, in one rat at 150 mg/kg bw, a red ocular discharge. All surviving rats were free of clinical signs by day 4 after dosing. Most surviving rats had gained weight by day 7, and all survivors had gained weight by day 14. Treatment-related effects on gross pathology were observed only in rats that died and consisted of redness in the region of the thymus, fluid in the thoracic cavity, red lungs, black mucosa or brown fluid in the stomach and red testes. Observations made at autopsy on four rats found dead consisted primarily

of gastric and intestinal discoloration or thickening of the walls and the presence of red fluid and test material. In addition, at 600 and 300 mg/kg bw some rats that died had discolored lungs or lungs with red foci. At 600 mg/kg bw, all male rats and four out of five females died within 1 day of treatment; at 300 mg/kg bw, one out of five males died on day 2. No mortality occurred at 150 mg/kg bw. The LD50 was 371 mg/kg bw in male and 427 mg/kg bw in female Sprague-Dawley rats.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations, /GENOTOXICITY/ ... Isolated and cultured peripheral lymphocytes (mostly T cells) were used from two human donors to study the effects of the chloroacetanilides and their metabolites on primary human cells. In tests at 10 uM, the SCE frequency was increased by alachlor and possibly acetochlor but not by butachlor, metolachlor, dimethachlor (a 2,6-dimethyl analog) and dimethenamid (an analog based on 2,4-dimethyl-3-thienylamine). At 0.3 microM in cultured human lymphocytes, alachlor, the corresponding chloroacetanilide (N-dealkyl-alachlor) and aniline metabolites (and their 4-hydroxy derivatives), and diethylbenzoquinone were inactive or active in only one of the two donors whereas at 0.1-0.3 uM the SCE ratio for treated cells divided by the controls was always higher for diethylbenzoquinoneimine than for ethylmethyl- and dimethylbenzoquinoneimines. All the tested compounds were toxic to lymphocytes, but the depression of the mitotic index and increased duration of the cell cycle were not directly linked with SCE induction. Previous investigations have suggested that chloroacetanilide herbicides such as alachlor derived from 2,6-dialkylanilines are metabolized to 2,6-dialkylbenzoquinoneimines and the present study provides the first direct

evidence that these metabolites are genotoxic in human lymphocytes.
- Carcinogenicity: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations,
- Reproductive toxicity: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic

dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations, /AQUATIC SPECIES/ Agricultural runoff is a non-point source of chemical contaminants that are seasonally detected in surface water and sediments. Agrichemicals found within seasonal runoff can elicit endocrine disrupting effects in organisms as adults, juveniles and larvae. The objectives of this study were (1) to determine if exposure to water, sediment or the water-sediment combination collected from an agricultural runoff event was responsible for changes in endocrine-responsive gene expression and development in fathead minnow larvae, and (2) whether such early life exposure leads to adverse effects as adults. Larvae were exposed during the first month post-hatch to water and sediment collected from the Elkhorn River and then allowed to depurate in filtered water until reaching sexual maturity, exemplifying a best-case recovery scenario. Gas chromatography mass spectrometry (GC/MS) analysis of the water and sediment samples detected 12 pesticides including atrazine, acetochlor, metolachlor and dimethenamid. In minnow larvae, exposure to river water upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Results from this study indicate that both water and sediments found in agricultural runoff elicit responses from minnow larvae, and larvae can recover following early life exposure under a best-case scenario. /Mixture/

- STOT-single exposure: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg

bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations,

- STOT-repeated exposure: IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations,
- Aspiration hazard: No data available.

Likely routes of exposure

- /LABORATORY ANIMALS: Acute Exposure/ The acute inhalation toxicity of racemic-dimethenamid (purity, 91.4%) was investigated in a group of five male and five female Wistar rats. Exposure was for 4 hr to a target atmospheric concentration of 4990 ug/L. Particle size distribution was determined twice during the exposure and the exposure concentration measured gravimetrically five times during the exposure. The rats were monitored for mortality and

clinical signs four times during the first day and daily thereafter. Body weights were recorded before exposure and on days 8 and 15. A gross autopsy was performed on all rats. Measurements of particle size distribution showed that approximately 6% of the aerosol was of size < 1 um. All rats survived the exposure and the 14-day observation period. Clinical signs observed were sedation, dyspnea, curved body position and ruffled fur during the exposure in several rats and during day 4 in one rat. Body weights increased normally and no abnormalities were noted at autopsy. The LC50 at 4 hr for racemic dimethenamid was > 4990 ug/L in male and female Wistar rats.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Dimethenamid is a herbicide and a racemic mixture of the M (or R) and P (or S) stereoisomers. When this compound was originally registered in various countries, all studies of toxicity were conducted with the racemic mixture. Later, it was discovered that only the P (or S) enantiomer has useful herbicidal activity. HUMAN STUDIES: In 50 people handling racemic dimethenamid and its formulated products over 7 years there were no reported cases of skin irritation or other adverse health effects. Dimethenamid did not increase sister chromatid exchange frequency in cultured human lymphocytes. ANIMAL STUDIES: In short-term studies with racemic dimethenamid, the signs of toxicity observed in mice, rats and dogs were similar, with reduced body-weight gain and liver enlargement being common features. Histopathology confirmed the liver as a target organ with observation of hypertrophy of hepatocytes. In addition, however, vacuolization of hepatocytes and dilatation of liver sinusoids occurred in dogs. Long-term feeding studies with racemic dimethenamid in rats and mice demonstrated that the primary target organ was the liver. There was no evidence for a carcinogenic potential in these studies. Apart from an equivocal result in one of three assays for unscheduled DNA synthesis in vitro with racemic dimethenamid, none of the other genotoxicity assays gave any indication that racemic dimethenamid might be genotoxic. The reproductive toxicity of racemic dimethenamid was investigated in a two-generation study in rats and in a study of developmental toxicity in rabbits. Reproductive function was not affected in rats in the two-generation study of racemic dimethenamid. In a study of developmental toxicity, rats were given racemic dimethenamid at doses of up to 425 mg/kg bw per day. Signs of maternal toxicity that were recorded included excess salivation at 215 mg/kg bw per day and 425 mg/kg bw per day, and urine-stained abdominal fur at 425 mg/kg bw per day. Fetal body weights were reduced and the frequency of early deaths was increased at doses of 215 mg/kg bw per day and 425 mg/kg bw. In a study of developmental toxicity in rabbits given racemic dimethenamid at doses of up to 150 mg/kg bw per day, significant maternal toxicity (body-weight loss preceded by reduced food consumption and associated with dry feces) was observed at the highest dose and less severe effects were noted at 75 mg/kg bw per day. Abortions in two rabbits at 150 mg/kg bw per day were considered to be treatment-related, but secondary to the clear maternal toxicity. ECOTOXICITY STUDIES: In minnow larvae, exposure to river water containing a mixture of pesticides including dimethenamid, upregulated androgen receptor gene expression whereas exposure to the sediment downregulated estrogen receptor a expression. Adult males previously exposed to both water and sediment were feminized through the induction of an ovipositor structure whereas no impacts were observed in other reproductive or sex characteristic endpoints for either sex based on exposure history. Dimethenamid induced significant modifications of the phytoplankton populations,

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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