Material Safety Data Sheet

BASIC MANUFACTURER

CHANGZHOU BOOMING CROP SCIENCE CO.,LTD
NO.2, BUILDING 53,HUNDSUN SCIENCE &TECHNOLOGY PARK,BEITANGHE ROAD,TIANNING DISTRICT, CHANGZHOU, JIANGSU, CHINA
Phone: 86-519-68208602
Fax: 86-519-85851009

TRADE OR OTHER NAMES

The active ingredient acetochlor is found in a variety of commercial herbicides. Some trade names for products containing acetochlor include Acenit, Guardian, Harness, Relay, Sacemid, Surpass, Top-Hand, Trophy and Winner. Most formulations come as emulsifiable concentrates.

REGULATORY STATUS

Acetochlor has a conditional registration in the United States to the Acetochlor Registration Partnership (ARP) composed of Monsanto Co., The Agricultural Group and ZENECA Ag. Products. The registration allows for automatic cancellation if use of several alternative corn herbicides is not reduced by a specified amount. Groundwater contamination may also be grounds for cancellation. Acetochlor is considered a restricted use pesticide (RUP) with a toxicity classification of I (Highly toxic). Check with specific state regulations for local restrictions which may apply. Products containing acetochlor must bear the signal word "Danger" on their label

INTRODUCTION

Acetochlor is used for control of most annual grasses and certain broadleaf weeds and yellow nutsedge. Crops include cabbage, citrus, coffee, corn (all types), cotton, green peas, maize, onion, orchards, peanuts, potatoes, rape, soybeans, sugarbeets, sugarcane, sunflower, and vineyards. Acetochlor is applied preemergence, preplant incorporated and is compatible with most other pesticides and fluid fertilizers when used at recommended rates. Usually 0.3-0.6 inches of rainfall will activate the product if it occurs within 7-10 days

TOXICOLOGICAL EFFECTS

ACUTE TOXICITY

Studies reported the oral LD50 for technical acetochlor when fed to rats ranged from 1426 mg/kg to 2148 mg/kg and formulated acetochlor in male rats ranged up to 2953 mg/kg. The oral LD50 for the formulated product Harness in female rats was reported to be 1700 mg/kg, while dermal LD50 was greater than 5000 mg/kg. Acetochlor is considered moderately toxic by ingestion.

The inhalation LC50 in rats exposed to acetochlor was greater than 3.85 mg/L, practically nontoxic.

The acute oral LD50 of technical acetochlor when administered to bobwhite quail was 1260 mg/kg.

In a study done on rabbits exposed to both technical acetochlor and the formulated product Harness (7.5 lb/gal EC), toxicity to skin, eyes, and possible danger through inhalation was tested. Rabbits exposed to technical acetochlor had a dermal LD50 of greater than 4166 mg/kg, an eye irritation classification of "practically non-irritating" and a skin irritation classifiction of "practically non-irritating." Rabbits exposed to Harness had a dermal LD50 of 3667 mg/kg, an eye irritation classification of "moderately irritating" and a skin irritation classifiction of "practically non- irritating".

Acute toxicology data submitted to EPA places technical acetochlor in toxicity category II for eye irritation, toxicity category III for acute oral, acute dermal, and acute inhalation. Technical acetochlor is in toxicity category IV for primary skin irritation and is considered a skin sensitizer.

CHRONIC TOXICITY

A 3-month feeding study submitted by Monsanto with rats fed dosages of 0, 40, 100, and 300 mg/kg/day resulted in a no-observed-effect-level (NOEL) of 40 mg/kg/day based on loss of body weight and decreased food consumption at 100 mg/kg/day.

A 3-week dermal study submitted by ZENECA with rats fed dosages of 0.1, 1.0, 10, or 100 mg/kg/day resulted in minimum to mild skin irritation after 21 days. Signs of systemic toxicity were not apparent at any level. Higher doses were not possible because of severe dermal toxicity at higher doses.

In a 90-day chronic toxicity study in rats, no significant toxicological effects were observed at dietary levels of 800 ppm.

The 4-hour inhalation LC50 for technical acetochlor in rabbits was 1.5 mg/L. Rabbit eye irritation was substantial but temporary; skin irritation was considered severe.

Reproductive Effects

In a two-generation reproduction study submitted by Monsanto, with rats fed dosages of 0, 30.4, 74.1, and 324.5 mg/kg/day (males) or 0, 44.9, 130.1, and 441.5 mg/kg/day (females), the reproductive NOEL was 30.4 mg/kg/day for males and 44.9 mg/kg/day for females based on decreased body weight gain of F2b pups at 74.1 mg/kg/day for males and 130.1 mg/kg/day for females. A NOEL for systemic effects was not established.

In a two-generation reproduction study submitted by ZENECA, with rats fed dosages of 0,1.6, 21, and 160 mg/kg/day, the reproductive NOEL was 21 mg/kg/day based on significant reductions in pup weight at lactational day 21 and total body weight gain during lactation at 160 mg/kg/day, the highest dose tested. The parental NOEL was 21 mg/kg/day based on reductions in body weight, accompanied by slight reductions in food consumption and significant increases in relative organ weights at 160 mg/kg/day, the highest dose tested.

Teratogenic Effects

In a developmental study submitted by Monsanto, with rats fed dosages of 0, 50, 200, and 400 mg/kg/day, acetochlor did not induce developmental toxicity in rats up to 400 mg/kg/day, the highest dose tested. The maternal NOEL was 200 mg/kg/day based on matting and/or staining of the anogenital region, a decrease in mean maternal weight gain during the treatment period, and an adjusted mean weight gain on gestation day 20 at 400 mg/kg/day, the highest dose tested.

In a developmental study submitted by ZENECA, with rats fed dosages of 0, 40, 150, and 600 mg/kg/day, the development NOEL was 150 mg/kg/day based on incereased resorptions, post-implantation loss, and decrease in mean fetal weight at 600 mg/kg/day, the highest dose tested. The maternal toxicity for this study was 150 mg/kg/day based on animals sacrificed approaching death, clinical observations, and decreased body weight gain at 600 mg/kg/day, the highest dose tested.

In a developmental study submitted by Monsanto, with rabbits fed dosages of 0, 15, 50, and 190 mg/kg/day, (females) acetochlor did not induce developmental toxicity in rabbits up to 190 mg/kg/day, the highest dose tested. The maternal toxicity NOEL was 50 mg/kg/day based on loss of body weight during dosing at 190 mg/kg/day, the highest dose tested.

In a developmental study submitted by ZENECA, with rabbits fed dosages of 0, 30, 100, and 300 mg/kg/day, acetochlor did not induce either maternal or developmental toxicity up to 300 mg/kg/day, the highest dose tested.

Mutagenic Effects

In mutagenicity testing submitted by Monsanto, acetochlor was weakly positive in the CHO/HGPRT gene mutation assay with and without activation in the mouse lymphoma assay. Acetochlor was negative in a DNA damage repair assay in rat hepatocytes, a Salmonella assay, and two in vivo chromosomal aberration studies.

In mutagenicity tests conducted by ZENECA, acetochlor induced a repro- ducible, positive, mutagenic response in a strain of Salmonella typhimurium with metabolic activation at 100 ug/plate. Significant increases in number of revertant colonies were not induced in four other strains. Acetochlor was not clastogenic (capable of causing breakage to chromosomes) in a mouse micronucleus test at doses tested (898 and 1,436 mg/kg in males; 1,075 and 1,719 mg/kg in females). Acetochlor was clastogenic in cultured human lymphocytes at both 50 and 100 ug/ml. Acetochlor induced a weak DNA repair (measured by UDS) in rat hepatocytes (liver cells) derived from animals exposed in vivo at 2,000 mg/kg. In a structural chromosome aberration-study, acetochlor at doses 1,000 and 2,000 mg/kg resulted in reduced fertility during weeks 2, 3, and 4 of this study, as shown by reduced pregnancy incidence, decreased implants per pregnancy incidence, increased preimplantion loss, and loss, and decreased time implant per pregnancy. Intrauterine deaths were not affected in this study. There was positive evidence of mutagenicity at the mid- and high-dose levels in this study.

Carcinogenic Effects

In a chronic feeding/carcinogenicity study submitted by Monsanto with mice fed dosages of 0, 75, 225, and 750 mg/kg/day carcinogenic effects noted included increased incidence of liver carcinomas in high-dose males, total lung tumors in females at all dose levels, carcinomas of lungs in females fed 75 and 750 mg/kg/day, uterine histiocytic sarcomas in females at all dose levels, and total benign ovarian tumors in mid-dose females.

In a chronic feeding/carcinogenicity study submitted by ZENECA, with mice fed dosages 0, 1.1, 11, and 116 mg/kg/day in males and 0, 1.4, 13, and 135 mg/kg/day in females, carcinogenic effects noted included an increase in pulmonary adenoma in both male and females at the high dose. Pulmonary tumors were confirmed as adenomas or carcinomas of the lung parenchyma and were all of the alveolar type. The NOEL for systemic toxicity in females was 13 mg/kg/day based on a significant increase in anterior polar vacuoles in the lens of the eye at 135 mg/kg/day.

In a chronic feeding/carcinogenicity study submitted by Monsanto, with rats fed dosages of 0, 22, 69, and 250 mg/kg/day (males) or 0, 30, 93, and 343 mg/kg/day (females), carcinogenic effects noted at 250 mg/kg/day in males and 343 mg/kg/day in females included hepatocellular carcinoma in both sexes and thyroid follicular cell adenoma in males. Nasal papillary adenomas were noted in male rats at 69 mg/kg/day and above and in females at 93 mg/kg/day. A NOEL for chronic effects was not established.

In a repeat chronic feeding/carcinogenicity study submitted by Monsanto, in rats fed dosages of 0, 2, 10, and 50 mg/kg/day oncogenic effects noted at 50 mg/kg/day, the highest dose tested, included neoplastic nodules of the liver, follicular adenoma/cystadenoma of the thyroids and papillary edema of the mucosa of the nose/turbinates in high-dose animals. The NOEL for chronic effects was 10 mg/kg/day based on decreased body weights and body weight gain in both sexes, high cholesterol levels in males, increased absolute and relative kidney and liver weight in males, and increased testicular weights at 50 mg/kg/day, the highest dose tested.

In a 2-year chronic feeding/carcinogenicity study submitted by ZENECA with rats fed dosages of 0, 0.8, 7.9, and 79.6 mg/kg/day, carcinogenic effects noted at 79.6 mg/kg/day, the highest dose tested, included a significant increase in nasal epithelial adenomas and thyroid follicular cell adenomas in both sexes at 79.6 mg/kg/day. Also, at that dose nasal carcinoma was present in two males and one female rat at this dose. Rare tumors in the form of benign chondroma of the femur and basal cell tumor of the stomach were also observed at 79.6 mg/kg/day. The systemic NOEL was 7.9 mg/kg/day based on decreased body weight gain, decreased food efficiency, increased organ to body weight ratios, increased plasma GGT and cholesterol at 79.6 mg/kg/day, the highest dose tested.

Based on the above data, the EPA has classified acetochlor as a "probable human carcinogen".

Organ Toxicity

In a 1-year feeding study submitted by Monsanto, with dogs fed dosages of 0, 4, 12, and 40 mg/kg/day, the NOEL was 12 mg/kg/day based on decreased body weight gains in males, decreased terminal body weight in females, testicular atrophy with accompanying decreases in absolute and relative testicular weight, increase in relative liver weights in males and females, and clinical chemistry changes at 40 mg/kg/day, the highest dose tested.

In a 1-year feeding study submitted by ZENECA, with dogs fed dosages of 0, 2, 10, and 50 mg/kg/day, the NOEL was 2 mg/kg/day based on increased salivation, ornithine carbamyl transferase, and triglyceride values accompanied by decreased blood glucose levels and liver glycogen levels at 10 mg/kg/day. Interstitial nephritis, tubular degeneration of the testes and hypospermia were reported.

ECOLOGICAL EFFECTS

Effects on Birds

The eight-day dietary LC50 in mallard ducks and bobwhite quail was greater than 5620 ppm.

Effects on Aquatic Organisms

The 96-hour LC50 in rainbow trout exposed to technical acetochlor was 0.45 mg/L and 1.30 mg/L in bluebgill sunfish. The 48-hour EC50 in Daphnia magna was 16.00 mg/L.

Effects on Other Animals (Nontarget species)

Acetochlor is considered moderately toxic to honeybees.

ENVIRONMENTAL FATE

Breakdown of Chemical in Soil and Groundwater

Acetochlor is adsorbed by soil colloids and leaches very little. Low soil moisture has little influence on efficiency.The main method of degradation is microbial breakdown. Acetochlor's average persistence at recommended rates is 8 to 12 weeks, but may vary depending on soil type and climatic conditions. It is very active on heavy or high organic matter soils.

Breakdown of Chemical in Surface Water

No information was found.

Breakdown of Chemical in Vegetation

Acetochlor is absorbed mainly by germinating plant shoots, and secondly by roots. It translocates throughout the plant, with higher concentrations in vegetative parts rather than in reproductive parts. Acetochlor appears to inhibit protein synthesis in susceptible plants.

PHYSICAL PROPERTIES AND GUIDELINES

Technical acetochlor is an oily liquid at room temperature, light amber to violet in color. It is considered slightly corrosive to mild steel and should not be used in mild steel tanks or with PVC or rubber hoses or pipes

Although acetochlor is stable under normal temperatures and pressures, thermal decomposition products may include toxic oxides of nitrogen and carbon and toxic and corrosive fumes of chlorides.

Physical Properties:

CAS #:	34256-82-1
Chemical name:	2-chloro-N-(ethoxymethyl)-N-(2-ethyl-6-methylphenyl)acetamide
Chemical Class/Use:	Acetamide compound used as a selective preplant incorporated and preemergence herbicide
Specific gravity:	1.100 at 30 degrees C, 1.136 at 20 degrees C
Solubility in water:	223 ppm in water at 25 degrees C (2, 4, 5); 23 mg/L at 25 degrees C
Solubility in other solvents:	Soluble in alcohol, acetone, toluene and tetrachloride
Melting point:	less than 0 degrees C
Flashpoint:	greater than 100 degrees C (closed cup)
Vapor pressure:	3.4 x 10 to the minus 8 at 25 degrees C