西亚试剂® Xiya Reagent 说明书

www.xiyashiji.com

化 学 品 安 全 技 术 说 明 书

填表时间 2020-02-25

打印时间 2025-12-14

MSDS标题

HEXACONAZOLE MSDS报告

产品标题

(RS)-2-(2,4-二氯苯基)-1-(1H-1,2,4-三唑-1-基)-己-2-醇

CAS号

79983-71-4

化学品及企业标识

PRODUCT NAME

HEXACONAZOLE

NFPA

Flammability	1
Toxicity	2
Body Contact	2
Reactivity	1
Chronic	2

SCALE: Min/Nil=0 Low=1 Moderate=2 High=3 Extreme=4

PRODUCT USE

Systemic fungicide with protective and curative action for broad spectrum treatment of many pathogens such as Ascomycetes, Basidiomycetes. Inhibits ergosterol biosynthesis. Regeant

SYNONYMS

C14-H17-Cl2-N3-O, "1H-1, 2, 4-triazole-1-ethanol, alpha-butyl-alpha-(2, 4-dichlorophenyl)-, (+/-)-", "1H-1, 2, 4-triazole-1-ethanol, alpha-butyl-alpha-(2, 4-dichlorophenyl)-, (+/-)-", "alpha-butyl-alpha-(2, 4-dichlorophenyl)-1H-1, 2, 4-triazole-1-ethanol (+/-)-", "alpha-butyl-alpha-(2, 4-dichlorophenyl)-1H-1, 2, 4-triazole-1-ethanol (+/-)-", "(RS)-2-(2, 4-dichlorophenyl)-1-(1H-1, 2, 4-triazol-1-yl)hexan-2-ol", "(RS)-2-(2, 4-dichlorophenyl)-1-(1H-1, 2, 4-triazol-1-yl)hexan-2-ol", Anvil, PP-523, "azole pesticide/ fungicide"

CANADIAN WHMIS SYMBOLS

EMERGENCY OVERVIEW

RISK

Harmful if swallowed.
May cause SENSITIZATION by skin contact.
Toxic to aquatic organisms, may cause long- term adverse effects in the aquatic environment.

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

SWALLOWED

Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Aromatase inhibitors produce several side effects including mood swing, depression, weight gain, hot flushes, vaginal dryness, bloating, early onset of menopause. Long-term use may result in bone weakness, increased risk of blood clots, gastrointestinal disturbance,and sweats. Aromatase inhibitors lower the level of oestrogen in post-menopausal women who have hormone-receptor-positive breast cancers. Prior to menopause oestrogen is mostly produced in the ovaries. Post-menopausal women produce oestrogen from another hormone, androgen. Aromatase inhibitors prevent the enzyme, aromatase from catalysing this reaction. Breast cancer cell growth in post-menopausal women is stimulated by oestrogen.

EYE

Limited evidence or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals. Prolonged eye contact may cause inflammation characterized by a temporary redness of the conjunctiva (similar to windburn).

SKIN

Skin contact is not thought to produce harmful health effects (as classified using animal models). Systemic harm, however, has been identified following exposure of animals by at least one other route and the material may still produce health damage following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Open cuts, abraded or irritated skin should not be exposed to this material. Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

INHALED

The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified using animal models). Nevertheless, adverse effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled.

CHRONIC HEALTH EFFECTS

Skin contact with the material is more likely to cause a sensitization reaction in some persons compared to the general population. There has been some concern that this material can cause cancer or mutations but there is not enough data to make an assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.. Based on experience with animal studies, there is a possibility that exposure to the material may result in toxic effects to the development of the fetus, at levels which do not cause significant toxic effects to the mother. Azole fungicides show a broad antifungal activity and are used either to prevent fungal infections or to cure an infection. Therefore, they are important tools in integrated agricultural production. According to their chemical structure, azole compounds are classified into triazoles and imidazoles; however, their antifungal activity is due to the same molecular mechanism. The cell membrane assembly of fungi and yeast is disturbed by blocking the synthesis of the essential membrane component ergosterol. This fundamental biochemical mechanism is the basis for the use of azole fungicides in agriculture and in human and veterinary antimycotic

therapies. The enzyme involved is sterol 14[alpha]-demethylase, which is found in several phyla. In mammals, it converts lanosterol into the meiosis-activating sterols (MAS) which regulate or modify cell division. These precursors of cholesterol have been discovered to moderate the development of male and female germ (sexual) cells. Several metabolites of lanosterol have been regarded only as precursors of cholesterol without any biological function in animals. This view dramatically changed recently with the observation that FF-MAS isolated from human follicle fluid and T-MAS isolated from bull testis as well as the MAS- 412 and MAS-414 induced resumption of meiosis in cultivated mouse oocytes (Byskov et al. 1995). Aromatase is another target enzyme of azole compounds. In steroidogenesis, it converts androgens into the corresponding estrogens. The importance of androgens and estrogens for the development of reproductive organs, for fertility, and in certain sex steroid- dependent diseases is well known. Therefore, azole compounds can be directed against aromatase to treat estrogen-responsive diseases. Based on the inhibitory activity of azoles on key enzymes involved in sex steroid hormone synthesis, it is likely that effects on fertility, sexual behavior, and reproductive organ development will occur depending on dose level and duration of treatment of laboratory animals. Several azole compounds were shown to inhibit the aromatase and to disturb the balance of androgens and estrogens in vivo. In fact, the clinical use of azole compounds in estrogen-dependent diseases is based on this effect. Additionally, azole antifungals developed to inhibit the sterol 14[alpha]-demethylase of fungi and yeast in agriculture and medicine are also inhibiting aromatase. Therefore, these antifungals may unintentionally disturb the balance of androgens and estrogens. Until now, it is not clear whether this effect is compensated by an increased expression of aromatase or by other unknown mechanisms. The broad use of biologically active compounds in human therapy as well as in nonhuman applications may involve some risks, as exemplified by emerging antibiotic resistance. In agriculture, fungi and yeast are well known to develop resistance to azoles, and some molecular mechanisms of resistance development have been described. The significance of the agricultural azole resistance for human clinical antimycotic therapies has been discussed in Europe, but is not clarified yet. The actual target enzyme of azole antifungals, the fungal sterol 14[alpha]-demethylase, is expressed in many species including humans, and it is highly conserved through evolution. Hence, it seems reasonable to assume that most of the azole antifungals used in agriculture and medicine as well as azoles used in management of breast cancer also act as inhibitors on human sterol 14[alpha]-demethylase to an unknown extent. The toxicologic profiles of individual azole fungicides provide evidence for endocrine effects. In fact, many of these fungicides have effects on prostate, testis, uterus, and ovaries as well as on fertility, development, and sexual behavior. The current database does not allow us to establish causal relationships of these effects with inhibition of sterol 14[alpha]-demethylase and/or aromatase, but the overall view strongly suggests a connection with disturbed steroidogenesis. Zam et al; Environmental Health Perspectives - 3/1/2003 . Triazole pesticides all contain a triazole ring with nitrogen atoms at the 1,2 and 4 positions. 1,2,4-Triazole (1,2,4-T) and its conjugates, triazole alanine (TA), triazole acetic acid (TAA), triazole pyruvic acid, and triazole lactic acid are the metabolic products of plant and animal bioconversion. These compounds with

potentially significant toxicological properties. Following application of a triazole-derivative fungicide, biological and/or chemical processes may cause the triazole ring to be released from the parent compound. In rats and livestock, 1,2,4-triazole is relatively stable and is the terminal form of the triazole ring. In plants, the 1,2,4-triazole molecule may become conjugated to serine. The resulting compound, triazole alanine, may be oxidised to form triazole acetic acid. Triazole alanine and triazole acetic acid are the primary terminal forms of the triazole ring in plants, though some 1,2,4-triazole may remain. The degree of formation of any given form of the triazole ring is highly dependent on the nature and properties of the parent compound. Although other triazole conjugates such as triazole lactic acid and triazole pyruvate have been observed in plant metabolism studies, TA and TAA are the predominant conjugates that need to be included in the dietary risk assessment. Although for most pesticides, mammals convert only a small proportion to free triazole (less than 25%), two compounds (tetraconazole and flusilazole) demonstrate relatively high conversion (68-77%) in rat metabolism studies. Available acute data indicate that 1,2,4-triazole is slightly toxic by the oral route (with oral LD50 values ranging from 666 mg/kg in rabbits to 3650 mg/kg in mice) and slightly to moderately toxic by the dermal route (dermal LD50s were less than 2000 mg/kg in rabbits, and 3000-4000 mg/kg in rats). Limited available information indicates that 1, 2,4-triazole is slightly irritating or non-irritating to the skin, but severely irritating to the eye. Based on the limited acute toxicity data, as well as the available developmental toxicity data (see below), it appears that rabbits may be substantially more susceptible to 1,2,4-triazole than are rats or mice Studies indicate that 1,2,4-triazole affects the central and peripheral nervous systems, reproductive tissues of both sexes, and the hematological system. Developmental and reproductive effects have been noted for this compound. Based on the available metabolism data from rats and livestock, 1,2,4-triazole may form in humans following exposure to parent triazole compounds. Relative to triazole alanine, fewer studies are available depicting the toxicological effects of the other triazole conjugates. It is assumed that the triazole conjugates are all toxicologically equivalent to triazole alanine. The available studies found developmental skeletal effects, decreased body weight and body weight gain, and decreased leukocytes and triglycerides. A number of target organs and critical effects have been identified. 1,2,4-triazole targets the nervous system, both central and peripheral, as brain lesions (most notably in the cerebellum) were seen in both rats and mice, and peripheral nerve degeneration was also seen in the subchronic neurotoxicity study in rats. In addition, brain weight decreases were seen in several studies, including in the offspring in the reproductive toxicity study. In the subchronic/neurotoxicity study, there is evidence that effects progress over time, with an increase in incidence of clinical signs (including tremors and muscle fasciculations) during weeks 8 and 13 that were not seen during earlier evaluations. There is no evidence that exposure to triazole alanine results in neurotoxicity. No clinical signs of neurotoxicity, changes in brain weights, changes in brain gross or microscopic pathology, or any other neurotoxic effects were observed in the short-term rat studies, the subchronic rat and dog feeding studies, the rat developmental toxicity study, or the two-generation reproduction study Effects were also seen on

reproductive organs in both sexes, most notably ovaries (in rats) and testes (in rats and mice), in both the reproductive toxicity and subchronic toxicity studies. Hematological changes, including slightly decreased hemoglobin and/or hematocrit, have also been seen in multiple studies and species (in rats at doses of 33 mg/kg/day and above, and in mice at doses of 487 mg/kg/day and above). 1,2,4-triazole also causes developmental toxicity in both rats and rabbits, including malformations, at doses similar to those inducing maternal toxicity (decreased body weight gain in rats and clinical signs and mortality in rabbits). Developmental toxicity was also seen in the reproductive toxicity study, with offspring showing adverse effects on multiple endpoints (including decreased brain and body weight) at doses lower than those at which effects were seen in parents. In addition, reproductive toxicity was seen in both sexes: at the highest dose (3000 ppm), only two F1 litters (one pup/litter) were produced, and neither survived to adulthood. Triazole alanine showed increased incidences of skeletal findings in the offspring at the mid and high doses, while no treatment-related effects were seen in the dams up to the limit dose. The skeletal findings included unossified odontoid processes at 300 and 1000 mg/kg/day, with partially ossified transverse processes of the 7th cervical vertebra (bilateral), unossified 5th sternebra, and partially ossified 13th thoracic centrum observed only at 1000 mg/kg/day. Available mutagenicity data are limited but negative. A large number of parent triazole- derivative pesticides have been classified as carcinogens (most also non-mutagenic), but the relevance of that finding to expected effects of free triazole may be limited. The types of tumors associated with exposure to the parent chemicals are most commonly hepatocellular adenomas/carcinomas in mice. Other tumor types vary considerably (including liver tumors, thyroid tumors, ovarian tumors, testicular tumors, and bladder tumors). None of the tumor types are clearly associated with the proportion of free triazole formed in available rat metabolism studies. Evidence indicates that the parent triazole compounds appear to result in a tumor response subsequent to perturbation of liver metabolism, specifically xenobiotic and fatty acid metabolic pathways. In addition the thyroid response appears to be secondary to perturbation of thyroid homeostasis. Thus, the conazoles appear to drive a tumor response secondary to epigenetic effects and not from direct interaction with the DNA. An epigenetic mode of action would be consistent with a nonlinear process.