2-Mercaptobenzothiazole

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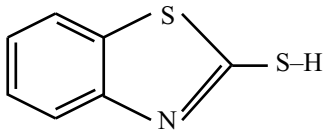
I. IDENTIFICATION

Chemical Name 2- Benzothiazolethiol
Synonyms: Mercaptobenzothiazole, 2-Mercaptobenzothiozole; 2(3H)-benzothiazolethione; 2- benzothiazolyl mercptan; benzothiazole-2-thione; MBT; 2-MBT
CAS Number: 149-30-4
Molecular Formula: $C_7H_5NS_2$
Structural Formula:



II. CHEMICAL AND PHYSICAL PROPERTIES[1–5]

Physical State arid Appearance: Cream to light yellow to brownish flakes or powder
Odor Description: No detectable odor to strong disagreeable odor; odor may be due to impurities.
Molecular Weight: 167.25
Conversion Factors: 1 ppm (v/v) = 6.9 mg/m^3
1 mg/ m^3 = 0.145 ppm
Melting Point: 177°C (351°F)
Boiling Point: >250°C (482°F)at 101.3 kPa
Specific Gravity: 1.49 at 25°C(77°F)
Vapor Pressure: <0.000003 kPa at 25°C (77°F)[6]
Flash Point: 243°C (469°F)
Partition Coefficient: 2.3–2.5[6]
Solubility: Soluble in acetone and dilute alkali; slightly soluble in alcohol and benzene. At 25°C (77°F), insoluble in water for 51 ppm at pH 5, 118 ppm at pH 7 and 900 ppm at pH 9.[6]
Stability: Stable at ambient temperatures; dangerous when heated to decomposition.
Reactivity: Reacts with acids to produce highly toxic fumes of sulfur compounds. Reacts with oxidants. With metals, it forms salts or metal complexes.

III. USES[7–9]

MBT is used as an accelerator and antioxidant in rubber processing. Minor uses are as a corrosion inhibitor in cutting oils, as an extreme-pressure additive in greases, as a metal scavenger in wastewater treatment, arid as a component in pesticide formulations MBT is an unisolated intermediate in the production of compounds used in rubber processing, including the dimer (MBTS) and the MBT sodium and zinc salts. NaMBT is also used as a corrosion inhibitor in antifreeze. Commercially available volumes of MBT and NaMBT may reach in excess of 6.7 million pounds[8,9] and 45 million pounds a year[7] respectively.

IV. ANIMAL TOXICITY DATA

A. Acute Toxicity and Irritancy

1. *Oral Toxicity*

Rats: LD_{50} = 3800 mg/kg, as 20% suspension in corn oil[3]

Rats: LD_{50} = 1800 mg/kg~0)

Mice: LD_{50} = 2000–2300 mg/kg[11,12]

2. *Eye Irritation*

Rabbits: 100 mg of MBT powder placed in the conjunctival sac produced only slight irritation; the score was 3.2/110[3]

3. *Skin Absorption*

Rabbits: LD_{50} > 7900 mg/kg[3]

4. *Skin Irritation*

Rabbits: 0.5 g powder in continuous 24-hr contact with intact or abraded skin showed no irritation in any of 8 animals tested.[3]

5. *Skin Sensitization*

Guinea pigs: After induction (0.1% MBT),an optimization procedure produced strong sensitization reaction in 109/20 animals.[13]

Guinea pigs: MBT was tested using three methods (maximization test, single adjuvant test, and modified Buehler test). Only the maximization test was positive (6/10 animals were sensitized) and MBT was classified as a moderate sensitizer.[14]

A modified Buehler method was used to determine the skin sensitization potential of MBT[15], 5% and 10% MBT (w/w in petroleum) produced the skin irritation reactions. 7 of 10 guinea pigs reacted to a challenge with 2% MBT; 2/10 to 0.5% ; and 0/10 to 0.1%. Based on cross reactivity tests also performed in the study, it was conjectured that the sensitization potential of MBT is directly related to the sulfhydryl group on the benzothiazole structure.

MBT was evaluated for its ability to induce a sensitization reaction in the mouse local lymph node assay.[6] The results of the assay indicated that MBT induced moderate proliferative response in lymphz cells.

MBT has induced a sensitization reaction in guinea pigs in the maximization test.[17]

An *in vitro* lymphocyte transformation assay was performed with MBT.[18] Based on the data, MBT did not induce a T cell response.

6. *Inhalation Toxicity*

Rats: LC_{50} (4 hr) = > 127 mg/L[19]

7. *Intraperitoneal Toxicity*

Mice: LD_{50} = 437 mg/kg[11]

Guinea pigs: LD_{50} = 300 mg/kg[12]

B. Subchronic Toxicity

Mice fed MBT for 1 week at doses of 55, 110, and 350 mg/kg/day exhibited severe histopathological liver damage. An increase in sleeping time was also observed. At doses greater than 350 mg/kg/day, central nervous system (CNS) stimulation was observed.[11]

In a 13-week gavage study, doses of MBT in corn oil varied from 95 mg/kg to 1500 mg/kg forB6C3F and from 188 mg/kg to 3000 mg/kg for rats.[20] Deaths occurred in mice dosed at 750 mg/kg and 1500 mg/kg, and in rats dosed at 3000 mg/kg. Based on lethality and weight loss, the maximum tolerated dose for subsequent chronic tests was established at 375 mg/kg for both rats and mice.

MBT administered to male and female Sprague-Dawley rats in the diet at exposure levels of 0 (control), 5000, 10,000, 15,000, 20,000, and 25,000 ppm (0, 357, 714, 1071, 1428, and 1785 mg/kg body weight/day) for 4 weeks resulted in statistically significant body weight gain decreases in males at 15,000 ppm and higher, and for females at 20,000 ppm and higher.t2~Reduced food consumption was also observed in these animals. Slightly increased liver weights were noted in all MET-treatment groups.

C. Chronic Toxicity/Carcinogenicity

Fischer-3441~lrats and B6C3F1 mice received MET by gavage in acorn oil vehicle 5 days/week for 2 years.[22] Dosage levels were 0, 375, or 750 mg/kg for male rats and for male and female mice; female rats were dosed at levels of 0, 188, or 375 mg/kg. Survival over the period of the study was decreased for male rats and high-dose female mice compared with those of controls. Survivals for other dosing groups were similar to those of controls. No effect on body weight gain was noted in rats, and minor reductions only were noted in mice between Weeks 3 and 64. Non-neoplastic histopathological changes included forestomach lesions in high-dose male and female rats and nephropathy in male rats only. Neoplastic changes included mononuclear cell leukemia and pancreatic acinar cell adenomas in low-dose male rats only, adrenal gland pheochromocytomas and malignant pheochromocytomas in high-dose male rats, adrenal glandpheochromocytomas and pituitary glandadenomas in high-dose female rats, and hepatocellular adenomas or carcinomas in low-dose female mice only.

The authors of the report concluded that “there was some evidence of carcinogenic activity” in male and female rats, “equivocal evidence for carcinogenic activity” in female mice, and no evidence forcarcinogenic activity in male mice.

D. Reproductive/DevelopmentalToxicity

Rats were administered 200 mg/kg MBT intraperitoneally on Days 1–15 gestation.[23] There were no chemically related histopathological effects in maternal tissues, and no maternal toxicity, fetal toxicity, or teratogenesis was observed.

In a range-finding teratology study, MBT was administered to female rabbits by gavage in a methylcellulose vehicle at dosages. of 0, 150, 300, 600, 1000, or 1500 mg/kg/day on Gestation Days 6–18.[24] Maternal mortality was observed at 600 mg/kg/day Clinical signs of toxicity and body weight loss occurred in all treatment groups; however, the effects on body weights were reversible

in the 150 and 300 mg/kg/day groups following cessation of treatment. The number of viable fetuses in the 150 and 300 mg/kg/day groups were comparable with concurrent controls and were decreased at 600 mg/kg and above. Fetal body weights were reduced in all treatment groups in a dose-related pattern. No treatment-induced external abnormality was noted in fetuses from MBT-treated dams.

MBT was also administered to pregnant rabbits by gavage in a methylcellulose vehicle at dosages of 0, 50, 150, or 300 mg/kg/day on Gestation Days 6–l8.[25,26] A slight but statistically insignificant reduction in body weight gains was noted for 300 mg/kg/day dams during treatment. Absolute and relative liver weights were also slightly increased at 300 mg/kg/day. There was no indication of fetotoxicity or teratogenicity when comparing treatment to concurrent control groups.

In a range-finding teratology study, MBT was administered to female rats by gavage in a corn oil vehicle at dosage levels of 0, 300, 600, 1000, 1500, or 2200 mg/kg/day on Gestation Days 6–l5.[27] Two dams died at 2200 mg/kg/day and clinical signs of toxicity and body weight losses were also noted at this dose level. Bodyweight losses were also noted at 1500 mg/kg/day. The body weight losses were reversible following cessation of treatment. Fetotoxic effects were not observed in this study.

In a definitive rat teratology study, MBT was administered to pregnant females by gavage in a corn oil vehicle at dosage levels of 0, 300, 1200, or 1800 mg/kg/day on Gestation Days 6–15.[26–28] Salivation, dark red material around the mouth, urine-staining, decreased activity, body weight losses, and reduced food consumption were noted between Days 6–9 in the 1800 mg/kg/day group. Salivation, urine-staining, and dark red material about the mouth were observed at 1200 mg/kg/day. There were no indications of maternal toxicity at 300 mg/kg/day.

A statistically significant increase in post-implantation loss was noted at 300 and 1800 mg/kg/day, but the finding was judged to be equivocal since it was not observed in rats at 1200 mg/kg/day. There were no other indications that MBT was fetotoxic or teratogenic in this species.

In a range-finding reproduction study, doses were 5000, 10,000, and 15,000 ppm (357, 714, and 1071 mg/kg body weight/day.[29] One group of F_0 rats received MBT in the diet at exposure levels of 15,000 ppm during gestation and lactation. F_1 pups from these dams were exposed to 15,000 ppm post-weaning. The second group of F_0 dams received MBT in the diet at a level of 15,000 ppm during gestation and the first week of lactation, 10,000 ppm during the second week of lactation, and 5000 ppm during the third week of lactation. Pregnant females exposed to 15,000 ppm had reduced body weight gains and food consumption throughout gestation and lactation. F_1 pup body weights were reduced compared with those of controls throughout lactation in both MBT-treatment groups. No other differences between treatment and control animals were observed in this study.

Sprague-Dawley rats received MBT in the diet at exposure levels of 0, 2500, 8750, and 15,000 ppm (179, 625, and 1071 mg/kg body weight/day) in a 2-generation reproduction study/30~Food intake was significantly reduced for F_0 animals at 8750 ppm and 15,000 ppm during the first week of the study.

Body weight gain during the first week was statistically significant and dose-dependently reduced for all MBT-treated F_0 males and for mid- and high-exposure F_0 females. Body weights were significantly reduced in the F_1 pups from the mid- and high-exposure groups and in F_2 pups from the MBT-treatment groups 2 beginning on day 14 of lactation. F_1 pup body weights were significantly reduced at 2500 ppm following weaning.

Brown pigmentation was observed in the convoluted tubules of the kidneys in mid- and high-exposure level F_0 and F_1 males and females. Absolute and relative kidney weights were significantly increased for F_0 and F_1 males in the mid- and high-exposure level groups. F_1 animals displayed hepatocyte hypertrophy at 8750 ppm and 15,000 ppm, and mid- and high-exposure F_1 males and high-exposure F_1 females had increased liver weights. Based on a lack of adverse findings in any parameter examined in the study, there was no indication that MBT was a reproductive toxicant.

E. Genotoxicity

MBT was not mutagenic, with and without metabolic activation, in the Ames test using Salmonella lyphimurium strains TA98, TA100, TA1535, or TAl537.~31~Another study reported the TA98 strain to be weakly positive in the presence of, but not in absence of, metabolic activation.[22] MBT caused significant increases in chromosomal aberrations and sister-chromatid exchanges (SCEs) in Chinese hamster ovary (CHO) cells when metabolic activation was present.[22]

Intraperitoneal administration of MBT at 300 mg/kg did not result in increased numbers of bone marrow cell micronuclei.[32] Therefore, MBT was not considered clastogenic in this assay.

Doses of MBT up to 300 μg/mL in a CHO/HGPRT forward mutation assay, conducted both with and without exogenous metabolic activation, resulted in negative findings.[32]

MBT at doses of 100 μg/mL and 150 μg/mL in the mouse lymphoma assay showed mutagenic activity in the absence of exogenous metabolic activation, and with concomitant extreme toxicity.[33] With S-9 activation, however, toxicity was reduced and there was significant increase in mutation at 80 μg/mL and 100 μg/mL.

Forward mutations in the mouse L51784 lymphoma cells were induced by 2-mercaptobenzothiazole only in the presence of Aroclor 1254-induced male F-344 rat liver S-9.[22]

In vivo binding experiments of ^{14}C-Iabeled to DNA demonstrated (after male and female Fischer rats were gavaged with 375 mg/kg) no indication that MET binds to the DNA of liver, pancreas, bone marrow, adrenal gland, or pituitary gland in rats.[34]

F. Metabolism/Pharmacokinetics (and Special Studies)

Radio-labeled MBT applied to shaved and wrapped skin of guinea pigs exhibited at least 9% of the MBT to be absorbed.[35] Radioactivity was observed in the blood, internal organs (primarily thyroid), gasirointestinal tract, feces, and urine. Urine accounted for more than 90% of the absorbed dose after 48 hr. When injected subcutaneously, after 1 hr most of the MBT was found in the kidney, liver, and thyroid. After 6 hr. 92.6% of the dose had been recovered in the urine.

The urinary metabolites of [35S-mercapto] 2-mercaptobenzothiazole in rats dosed by intraperitoneal injection consisted of conjugates of glutathione, glucuronic acid, and inorganic sulfate.[36]

Biochemical studies indicate that MBT was capable of enzyme inhibition *in vivo* and *in vitro*.[37,38]

MBT is an inhibitor of beta-hydroxylase; as a result, reduced endogenous levels of norepinephrine have been reported in man.[38]

Male and female F-344 rats were dosed orally with 0.592 mg/kg or 55.5 mg/kg of ^{14}C-labeled MBT, then sacrificed post-exposure at 8, 24, 48, 72, or 96 hr.[39,40] Seventy-two percent of the radioactivity was excreted in the urine and 4% in the feces within 96 hr; 0.4% remained associated with erythrocytes. Most of the other unaccounted-for activity was in the serum.

Male and female rats, and female guinea pigs, were topically exposed to [^{14}C]- MBT at approximately 36.1 μg /animal.[39,40] A separate set of rats was also dosed orally for 14 days with unlabeled MBT at 0.510 mg/kg/day prior to a single dosing with 0.503 mg/kg of radio-labeled material. A third set of rats was intravenously administered radio-labeled MBT at 0.602 mg/kg.

In the skin exposure studies, guinea pigs absorbed a greater percentage of the MBT than rats (33.4% vs. 16.1%–17.5%), but the disposition of the radioactivity was similar for the two species. Urine excretion of the absorbed dose was 91% in male rats, 94% in female rats, and 98% in guinea pigs, whereas fecal excretion in rats was 4%–9% and in the guinea pig it was l%–2%. In the intravenous study at 72-hr post-exposure, urine excretion of radioactivity was 91%–96% of the administered dose, with a small amount excreted in the feces; less than 2% remained associated with red blood cells. Similar findings were observed at 96 hr following oral dosing.

Male and female rats were dosed intravenously with 0.602 mg/kg of [^{14}C]-labeled MBT.[41] Four rats/sex were dosed and sacrificed at 5 min, 15 min, 1 hr, 2hr, 4hr, 24 hr, and 72 hr. Between 90.9% and 101% was excreted in the urine within 72 hr; 3.79%–1S.l% was excreted in the feces within 72 hr; arid .52%–1.96% remained associated with the erythrocytes at 72 hr. Only blood tissue seemed to be associated with radioactivity at 72 hr.

G. Neurotoxicity Studies

Male and female Sprague-Dawley rats in a range-finding study were given MBT by gavage in a corn oil vehicle at dosage levels of 0 mg/kg or 2750 mg/kg and then observed for 24 hr in a motor-activity assessment.[42] Peak decreased motor activity was noted for males 7 hr to 16 hr post-treatment and for females at 7–21 hr post- treatment. Based on these data, 12 hr was selected as the point to monitor peak effects of acute MBT exposure in a subsequent acute study.

Male and female Sprague-Dawley rats were dosed once by gavage with MBT in a cornoil vehicle at levels of 0, 500, 1250, or 2750 mg/kg and then observed for 14 days.[43,44] Motor activity testing and a functional observational battery (FOB) were performed. Rats at 2750 mg/kg had statistically significant decreases in body weights compared with that of controls. Transitory differences were noted in treated animals compared with controls in the FOB evaluation. These differences include salivation in 2750 mg/kg males and in all MET-treated female groups at 1 hr post-treatment; decreased vocalization in 1250 mg/kg and 2750 mg/kg males at 1 hr and 6 hr post-treatment; and increased urinary staining in 2750 mg/kg females. Statistically significant decreases in motor activity were noted

in 2750 mg/kg males and in 1250 mg/kg and 2750 mg/kg females at 12 hr post-dosing. Based on these findings, the authors concluded that the effects might be related to an acute, nonspecific toxicity without apparent neurotoxicity.

V. HUMAN USE AND EXPERIENCE

For humans, MBT has been identified as a skin sensitizer.[45–47] Several human studies, however, have elicited little or no response to MBT. Patch testing 50 volunteers with serial applications and subsequent challenge produced no positive response.[3] In a comparison of MBT with 15 other active skin sensitizers, MET was the least active — only 1% of the 1088 subjects tested positive. The combination of Thiram (another rubber chemical) and MBT might be sensitizing to some individuals, however.[48]

Reports on workplace dermatitis were included in a survey of 800 dermatitis patients studied between 1955 and 1958. Of 29 cases determined to be occupationally related to the rubber indus~y,16 were rubber workers — all of whom tested positive to MBT.[49] In a European study of 300 patients suspected of having occupational contact dermatitis, 3% tested positive to MBT.~50~In a 1978 NIOSH workplace evaluation at a rubber plant, wheal-type skin lesions were observed and were attributed to exposure to Kargarax-A that contained MBT Air sampling did not detect MBT (detection limit 0.035 mg/m^3 based on a 60-L air sample)[51] In 1983, NIOSH conducted a workplace evaluation at another rubber plant where skin rashes were noted. MBT and Thiram were among the chemicals present. The authors suggested that airborne dust could be responsible for the dermal effects.[52]

Comments from producers of MBT indicate that there apparently are no significant problems associated with exposure when good hygiene practices are followed.

VI. EPIDEMIOLOGY

A study performed in a Wales chemicals production plant between 1955 and 1984 consisted of 2 subcohorts, including about 2410 employees.[53] Workers may have attracted zero exposure; very low (0–1 mg/rn^3); low (1–2.5 mg/m^3); medium (2.5–6 mg/m^3); or high exposure (6–20 mg/ m3). Observed and expected numbers of deaths for all neoplasms and cancer in individuals exposed to MBT suggests that overall mortality experience was near (or below) the expectation for all workers. Estimated cumulative exposure to MBT was found not to be a risk factor. The trend of standardized mortality ratios (SMR) in this study suggest that there was no significance to the mortality for cancer in the workers.

A study involving 1059 production workers in a U.S. rubber chemicals plant between 1955 and 1977 resulted in the observation that MBT workers who were not in a job assignment with exposure to para-amino biphenyl (PAB) did not show excesses for most malignant neoplasms.[54] Categories for possible exposure were very low (0–0.5 mg/m^3); low (0.5–2 mg/m^3); medium (2–5 mg/m^3); and high (5–20 mg/m^3). The SMR for bladder cancer was raised, although there were too few deaths to evaluate. Confounding the results were the MET workers who were not in plant jobs directly related to departments with exposure to PAB. There were no deaths from bladder cancer among MBT workers hired after the end of the plant's PAB use, although only a 0.03 death ratio was expected.

VII. RATIONALE

2-Mercaptobenzothiazole does not have high acute toxicity. In animal and human tests, MBT was not irritating to the skin or eyes, and it would not be expected to irritate the respiratory tract. MBT is a skin sensitizer. Overall, it is not genotoxic. At high doses over time, liver damage was observed in mice.

In the 2-year gavage study with MBT, there was some evidence of carcinogenic activity in rats, but less than the strength of response desired by the National Toxicology Program for clear evidence of a carcinogenic response. There was no evidence of carcinogenic activity in male mice and equivocal evidence in female mice. Considering these effects, MBT may have a weak carcinogenic potential, at most.

The fact that the lowest dose of 188 mg/kg (NOEL) in the 2-year gavage study did not negatively affect survival, one could translate this as an inhalation exposure of 1300 mg/m^3 for a human weighing 70 kg. Since there is some small potential for carcinogenic activity, an 8-hr time- weighted average (TWA) of 5 mg/m^3 should provide ample protection from the small opportunity for exposure.

VIII. RECOMMENDED OEL

8-hr TWA: 5 mg/rn^3, skin

IX. REFERENCES

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NOTE(1): The following databases were searched in developing the 1997 WEEL:

CAS
EMIC
MEDLINE
RTECS
TOXLINE

NOTE(2): The following databases were searched for this 2010 WEEL Revision:

MEDLINE (1989 – 2008)
IPA (1970 – 2008)
CURRENT CONTENTS (1990 – 2008)
CAB (Global Health)(1973 – 2008)
CINAHL (1982 – 2008)
TOXNET (2006 – January 2010)