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BROMOPROPANE; A REVIEW ARTICLE

Noor M. Mohammed[1] , Ahmed M. Al-Yassen[2]
Department of pharmacy , *Al-Farabi university college* , Iraq Baghdad Al-Dora
noor.moner@alfarabiuc.edu.iq , Dr.ahmed.mohamed.mahmood@alfarabiuc.edu.iq

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Abstract:

Bromopropane (BP) was introduced into the workplace as an alternative to ozone depleting solvents and increasingly used in manufacturing industry, bromopropane can be absorbed by inhalation, ingestion, or dermal exposure. Several studies have monitored urine and blood samples in workers to establish biomarkers of exposure. 1-bromopropane metabolism show that CYP catalyzed oxidation (primarily via CYP2E1) reactions and glutathione conjugation are the primary metabolic pathways. Bromopropane has many reactive intermediate metabolites (bromoacetone, glycidol, and -bromohydrin). Workers that exposed to BP showed different systems toxicities, animal studies showed toxicities as well. BP cause disruption of estrous cycle and change ovarian shape and weight, there is a general agreement that male reproductive organs are particularly susceptible to the deleterious effects of reactive oxygen species (ROS) and lipid peroxidation, which ultimately lead to impaired fertility. Human cases of BP toxicity showed ataxia, sensory deficit and hyperreflexia in lower extremities. The reduction of hepatic GSH produced by BP was associated with increased activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Other toxicities further reported like reduction in mean body weight of rats, pancytopenia and bone marrow hypoplasia.

Keywords: Bromopropane, Toxicities, Animal studies, Worker toxicity, Reproductive toxicity

I. IDENTIFICATION

Terminology Chemistry Abstract. Serv. Registration. Number.: 106-94-5 Chemistry. Abstract. Serv. Titled: Propane,
International Union of Pure and Applied Chemistry Logical Title: 1-Bromopropane, Alternative word: 1-Propyl bromide; n-propyl bromide
Abbreviations: BP.
Molecular design: C3H7Br. Relative molecular quantity: 122.99

II. CHEMICAL AND PHYSICAL PROPERTIES

Explanation: Monochrome to nimble yellow and a solid, specific fragrant smell. BP: 71°Celsius , 760 millimetre of mercury. Melting"point" ; ((−110)) °Celsius. Density1: 1.353 in 20 °Celsius. (1)

Vapor mass about 4.25, Solubility: weakly soluble with H2O (2.45 mg/Liter at 20 ° Celsius); soluble in CH3COCH3, C2H5OH, CH3CH2OCH2CH3, C6H6, CHCl3 and CCl4, "Volatility": Vapor density, 110.8 millimetre of mercury at 20 °Celsius; Steadiness: not polymerize1. Highly combustible liquid and vapors; risky when heated or visible to fire or oxidizers; Auto-lighting temperature: 490 °C; Decay: Combustion by1-product releases HBr. Octane/water partition1 coefficient: log Kow1 2.10. (2)

III. PRODUCTION

Manufacture of cost-effective grade bromopropane yields little pollution ranks (0.1–0.2%), (2-BP). Modern developed methods, result in less than 0.1% 2-bromopropane contamination in neats1-bromopropane(1-BP) domestics. (3)
Worldwide production was likely to be 20000–30000 tons in year 2007. (4) But precise capacities formed in each land could not accessible. The US EPA described in 2006 countrywide pool manufacture found to be 1–10 million pounds [More 450–4500 tons]. (5)
In 2011, the EPA listed the general production volume as more than 15.3 million pounds [About 7000 tons]. (6)

IV. USES

Bromopropane used as a solvent "resin, waxes and fats" and principally as intermediary in the pesticides manufacture, ammonium compounds (quaternary), flavours, colognes, and medications in shut courses. (7)
It introduced as safe, fast-drying agents that makes no residue for cleanings ingredients metals, plastics, electrical or electronic constituents. (8)
It was advertised as an auxiliary agent for ozone-depleting and other agents like C2HCl3, perchloroethylene and CH2Cl2. 1-BP is consumed in degreasing and soaking bath, runny and spume adhesive presentations, cloth dry cleaning, and aerosol spray goods. (9)

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During 2003, the US National Institute for Occupational Safety and Health (NIOSH) supplied an authenticated analytical way for 1-BP in air that advises adsorption on activated charcoal, desorption with CS2 and study by (GC) with spark ionization recognition; the limit of detection for NIOSH technique 1025, 1 Microgram by 12 Liter air sample. (OSHA) technique 1017 gathers 1-BP in activated charcoal then customs CS2 (99%) and N,N-dimethylformamide for elution and GC with electron captive recognition; it totally authorized, measureable bound of 5.9 µg/m3 of 12 Liter air model. (10)

To minimize the risk of 1-BP uses and contrariwise, EPA showed extensive investigation and Manages. This included EPA's analysis of published writings and online databanks including recent information exists from EPA's Chemical Data1Reporting program (CDR) and Safety Data Sheets (SDSs). EPA also showed online research by studying company webs of potential constructors, shippers, retailers, and other users of 1-BP and probed government and commercial trade sources. EPA also received comments on the Latitude of the Risk Estimation for 1-BP (Scope Document; EPA-HQ-OPPT-2017-0741-0049) that were used to determine the situations of use. Some of the observations received were just relevant to the hazard evaluation process. In addition, EPA assembled meetings with companies, manufacturing sets, chemical consumers, environmental groups, and shareholders to support in identifying situations of use and checking conditions of in identified by EPA. Those conventions included a February 14, 2017 public seminar with relevant persons and an October 25, 2017 visit to CRCCIndustries. (11)

Revisions on humans and Lab animals showed that BP could be absorb during inhalation, ingestion, or dermal contact. Workers exposure arises by inhalation and dermal contact, studies on worker's demonstrated a good relation between urine levels of 1-BP, B-1, and Nacetyl-S-(n-propyl)-L-cysteine (AcPrCys) with their 1-BP inhalation zone air. Many studies have tested urine and blood in workers to launch markers of exposure. These revisions always, showed that nonmetabolized 1-BP excreted to urine in people but not reported with animal readings. Animal studies tell relatively great part of 1- BP is absorbed, quickly spread, mainly removed during exhalation (40% - 70%), but it also removed in the urine and feces. 1-BP taken by intra-venous dosed in rodents exhaled unaffected or by way of CO2 in four hours of contact. Kidney metabolites hold about 13% - 23% of the directed amount after about 48 hr

V. METABOLISM

The current studies on 1-BP metabolism illustration that CYP450 catalyzed oxidation (mostly via CYP2E1) with glutathione conjugation as the main metabolic paths. At least 16 urinary metabolites were recognized in rodents (either rats or mice), containing several reactive intermediate metabolites (such as bromoacetone, glycidol, and -bromohydrin). While, the differences in results between rodents and humans the results are limited for humans and some differences seem to be associated to the extent of check. The outcome reflects that no reason to undertake humans are markedly diverse from animals in the kinetics of 1-BP. (12)

[14C]1-BrP given to (rats), exhaled unaffected Radio-Carbon Dioxide (14CO2) and 1-BrP, mercapturic acid (MA) with related sulfoxide cognizant as metabolites in the urine of tested animal with 1-BP. MA of the metabolites 2-hydroxy BP, 3-hydroxy BP, and C3H5BrO2 also included within exposed animal to 1-BrP urine. (13)

According to C.E. Garner et al, the kinetics of 1-BrP have been tested in animals. The means of kinetics of Carbon-14 -1-BrP revealed to be differ in high (100 mg/kg) vs. small doses (20 mg/kg). Urine metabolites of animals was recognized by 13C NMR and mass-spectra. The statistics elaborate the 1-BrP oxidation via CYP-450 at the C2 anticipated linked to the C1 and C3. So the references submit the major metabolite of 1-BP, "1-bromo-2-hydroxypropanol" is integrate with glutathione or glucuronic acid or others. (14)

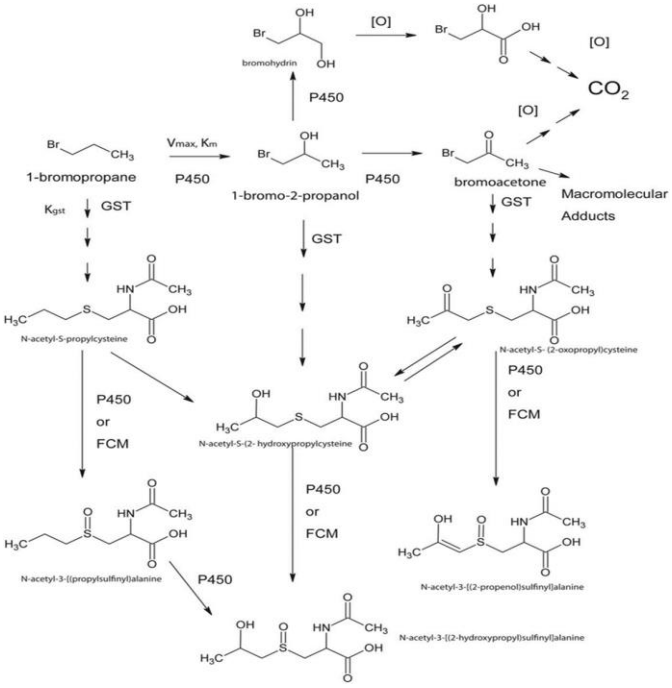

FIG. 1. Proposed metabolism of (1-BP) metabolism in male F344 rats following inhalation exposure (Garner et al. 2007, 2006). Both P450-mediated oxidation (V max , K m) and direct glutathione (GSH) conjugation (K gst) occurred during the metabolism of 1-BP.

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VI. Reproductive System Toxicity

Many revisions presented 1-BP could cause grave toxic things on CNS, and Reproductive system. (15)

The process of damage of 1-BP on sexual organs is quite vary from 2-BP in male animal (rat). Previous readings have showed that a ten weeks' exposure to 1-BP at 750 ppm lead to ovarian weight decline and amounts of corpora lutea, and also extend the length of estrous cycle. (16)

There is elongation within estrous cycle on rats exposed to 500 ppm cluster in the same time the ovarian weigh was not altered. The ovaries gain weights after 1-BP exposure was elaborated in different study. (17)

From above studies and discussion this agent considered to be toxic to male's and female's sexual organs. It's important to know that there are no enough revisions on histopathological alterations on ovaries, and it's good to check these deteriorations on both ovaries and its follicles after 1-BP exposure and stand that in front of 2-BP toxicity as comparative study. (18) Meanwhile old data presented the target differences on testis among 2-BP and 1-BP. (19)

2-BP toxicity on sexual function diverge from 1-BP in animals (female rat). Remarkably, primordial follicles clearly rise at 800-ppm cluster. Also, notes the primordial follicles increment with decline in growing and antral follicles further contact stage, the consequence explains the slice of interference of the follicular growth style. According to this finding, that its reveal the modification of age at necropsy. In 21 weeks 800ppm animal checked and study after sacrificed also other groups (21weeks) were in necropsy. Primordial follicle is not developed with aged animal, but, younger and newer rat may gain more primordial follicle. (20)

In chronic 2-BP contact, estrous cycle will be deteriorated (1000ppm female rat). (21) Ovarian follicles declines when exposed to previous dose according to different paper. (22) Thus, primordial follicles markedly affected with 2-BP. chemicals like C6H9CH=CH2 and dimethylbenzanthracene also harm primordial follicles. Ater all, early menopause may will clearly settled. (23)

Leydig cells, 2-BP develop ROS and lipid peoxidation (LPO). (24). There is a common agreement that male reproductive system is particularly susceptible to the toxic effects of ROS and LPO eventually target the reproductive system causing sterility. (25) High level of poly un-saturated fatty acids C20:4 n6 and C22:5 n6 with weak anti-oxidant capabilities, LPO damage easily harsh sperm cell. (26) Different ROS affect sperm criterion, thus, testicular damage developed with 2-BP exposure linked to ROS and LPO. (27) Testicular germ cell die by apoptosis included Fas signalling system and Bcl-2 family genes, all induced by 2-BP. (28)

VII. Neuro Toxicity

For nervous system toxicities many reports and studies demonstrate cutoff result, like contact of rats to 1000 ppm 2-BP for 3 months made significant fluctuations in the distal latency and motor nerve transmission velocity. For peripheral nerves there is abnormalities in the myelin sheath. (29)

Different Neuro damage such as degeneration of myelin, swelling in axon, histological changes and weakening of grip strength noted after 3 month of 1-BP exposure. (30)

Ataxia showed within human cases, sensory insufficiencies, hyperreflexia in lower extremities (31). Also, we note clear decline in sensory nerve conduction velocity and elongation motor nerve distal latency in and apart from the previous neoru insufficiencies, disorders of the CNS like, memory deficits, anxiety and depression was recorded. (32). After rats exposured to 1-BP, results showed that there is reduction in noradrenalin axon level prefrontal cortex and amygdala and abnormalities in neurobehavioral. (33)

Lingyi Zhang et al, proved that the dentate gyrus decreased neurogenesis after one month exposure to 1-BP. Neurogenesis decline in the hippocampal may responsible in cerebral orders like mood and cognitive impairments. There is no effect on serotonin level but NE level will fall in striatum after 7 days exposure and one month lead to marked fall in level in prefrontal, cortex and striatum. (34)

According to many papers antidepressants could modulate depression that developed by changes in neurotransmitters (NT) levels after BP exposure. Neurogenesis hippocampus affected according to NT fluctuations. While we couldn't recognize the main mechanism of neurogenesis, but it's clear that NE trigger the neurogenesis in hippocampal. (35) Accordings to Sahabudeen S. et al, the elongation processes that appear in astrocytes, complemented with collapse of granular cells and Purkinje cells in the cerebellum of the exposed animal. Its seems that exposure to 1-BP affect myelination since there is mRNA expression decline with reduction in MBP gene and oligodendrocytes with diminution in of myelin/oligodendrocyte-related genes and NG2 gene. Whereas in cerebellum, the rise in astrocytes thought a sensitive indicator, with no clear mechanism. (36)

VIII. Hepato-Toxicity

Male Wistar rats exposed to vapor of 1-BP as follow, first, 1month exposure or less and 5 days per week and 6 hr a day, at 1500 ppm second, for 1 day, 4 or 12 weeks at 700 ppm, notes that level of liver cytochrome P450, serum alanine aminotransferase (ALT), and serum aspartate aminotransferase (AST) were reduced markedly. (37)

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One shot of 1-BP exposure lead to increase ALT and AST by more than 10 fold in respect to control group. (38) Reactive metabolite will be formed could be the reason of hepatotoxicity triggered by 1-BP exposure.Liver GSH decline according to bromopropane exposure aid in increase of hepatic enzymes. (39)

IX. Other Toxicities

Hypocellular developed after 2-BP exposure in bone marrow: hematopoietic cells decline, and hypoplasia in the area, leading to pancytopenia. Same outcome seen on female stated that 2-BP hematotoxic. (40)
According to Xiangrong SONG et al, BP showed decreased body weight of rats, and erythrocyte, hemoglobin concentration, the total serum cholesterol and triglycerides markedly affected. (41)

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