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TOXIC EFFECTS OF DIMETHOATE ON ANIMAL

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ABSTRACT

Dimethoate, (o, o - dimethy l S – methy l – carbamoyl – methy l phosphorodithioate) (fig.1) a broad spectrum organophosphorus insecticide, has been extensively utilized in agriculture to control a wide variety of pests. It has systemic, contact and stomach mode of action. Its mechanism of action involves the inhibition of acetylcholinesterase, a pivotal enzyme in neurotransmission, resulting in the accumulation of acetylcholine and subsequent disruption of nerve impulses. This mode of action accounts for its potent insecticidal properties but also poses significant risks to non-target organisms, including mammals, due to potential neurotoxic effects. Studies investigating the toxicological profile of dimethoate reveal adverse impacts on various systems, including the nervous, respiratory, and digestive systems and affect many vital organs including kidney, liver and gonads. Acute exposure to high doses manifests in symptoms such as nausea, vomiting, diarrhea, abdominal pain, and, in severe cases, convulsions and respiratory distress. Chronic exposure to lower doses may lead to cumulative effects, potentially resulting in long-term neurological and health complications. Furthermore, research underscores the environmental persistence of dimethoate and its potential to leach into water sources, posing threats to aquatic ecosystems and non-target organisms. Regulatory measures aimed at mitigating risks associated with its use have been implemented, emphasizing proper handling, application, and adherence to safety protocols. Understanding the toxicological profile of dimethoate is crucial in delineating its risks and implementing effective strategies to minimize human and environmental exposure. Further research endeavors are warranted to comprehensively assess its impact on diverse ecosystems and to develop safer alternatives for pest control practices.

KEYWORDS: Dimethoate, insecticide, digestive system, respiratory system, kidney, liver.

INTRODUCTION

Pesticides are crucial to both public health and agriculture. They contribute significantly by raising food and fibre output and enhancing human health by lowering the incidence of vector-borne illnesses. In addition to causing harm to crops, bugs can also create harmful compounds that affect domestic animals and humans. In this regard, up to the year 2000, 812 active substances that are referred to as "pesticides" were registered, according to the Pesticide Manual. Currently, over 10,400 pesticides are authorised globally. According to reports, two million tonnes of insecticides are consumed annually globally. On the other hand, the term "pesticides" refers to a wide variety of pesticides, including herbicides, bactericides, nematicides, acaricides, fungicides, molluscicides, and rodenticides. Each type of pesticide is effective against a particular pest, such as a rat, snail, weed, bacteria, nematode, fungus, or insect.[1] Let's now talk about dimethoate's toxicological consequences. As an acetylcholinesterase inhibitor, dimethoate prevents the neurotransmitter acetylcholine from breaking down normally. This may result in an overstimulation of nerve cells, which might give rise to cholinergic symptoms such tremors, twitches, and weakening in the muscles. The dimethoate 0, 0- Dimethy l S - (N-methylcarbamoylmethyl) phosphorodithioate is the active component of the organophosphate dimethoate. Direct human exposure to dimethoate can have a variety of negative consequences, such as an organophosphate poisoning that can happen quickly and fatally and induce headaches, nausea, vomiting, sweating, diarrhoea, lack of coordination, jerking muscles, and even death. Additionally, dimethoate has the potential to seriously alter genes. Dimethoate has a harmful effect on male mice's reproductive system. gestating mice.[3] Research on the insecticide dimethoate's carcinogenicity in animals was reviewed.[4] Histopathological observations of the treated rats under low light microscopes showed that dimethoate caused dose-related testicular damage, which was manifested as moderate to severe seminiferous tubule degeneration as sloughing, atrophy, degeneration of germ cells, and partial arrest of spermatogenesis.[5] Testicles are harmed by dimethoate. The treated groups showed a drop in plasma testosterone levels. Dimethoate produced testicular lesions, which were histologically examined and showed partial halt of spermatogenesis and mild to severe degenerative alterations of spermatogonial cells. Hepatic sinusoids and central veins showed dilated in sections of the liver, with some places showing bleeding. Dimethoate concentrations were lowest in skeletal muscle and greatest in the liver and testis. Dimethoate has a significant carcinogenic potential.[7] Oxidative stress brought on by dimethoate results in genotoxicity.[8] In male rats, dimethoate decreased fertility.[9] An extremely water soluble organophosphorous insecticide is

dimethoate. It smells like mercaptan and is a white, crystalline solid when pure. It is stable at room temperature in pH 2–7 aqueous solutions and unstable in alkaline environments. It has a moderate affinity for organic matter and a low affinity for soils. Its low vapour pressure indicates that it is non-volatile, moderately stable to microbial degradation, and susceptible to hydrolysis in acidic environments. There is a greater chance of runoff into surface waters and/or leaching into groundwater because of the higher water solubility and lower soil persistency. Animals in agricultural environments may come into contact with dimethoate through a variety of routes, including crop residues, tainted water sources, drift and spray, and secondary poisoning.[10] When exposed to, dimethoate, an organophosphorus pesticide, can be toxic to mammals, including humans. The following is a summary of some of the harmful effects dimethoate has on mammals.

Neurotoxicity

The main way that dimethoate causes harm is by blocking the enzyme acetylcholinesterase, which is in charge of degrading the neurotransmitter acetylcholine. As a result, endogenous acetylcholine builds up at the nerve ends, overstimulating nerve cells and subsequently activating nicotinic, muscarinic, and cholinergic receptors.[11,12] Headaches, lightheadedness, tremors, twitching of the muscles, and in extreme situations, seizures and paralysis, are examples of neurological symptoms. Elevated exposure to dimethoate has been linked to neurodegenerative diseases and has been shown to have harmful oxidative and neurotoxic effects.[13]

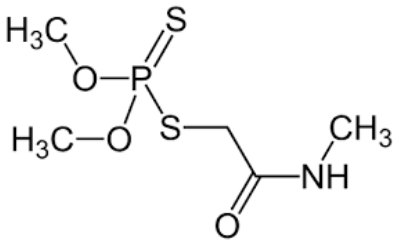

Fig. 1: O, O-dimethyl S-methyl - carbamoyl - methyl phosphorodithioat.

Effects of dimethoate on Respiratory system

Exposure to dimethoate can lead to respiratory distress, including difficulty in breathing, in coughing, and bronchochontriction.[14] Most result from acute respiratory failure due to central respiratory depression, respiratory muscle weakness, and/or direct pulmonary effects (bronchospasm and bronchorrhoea).[15]

Effects of dimethoate on Reproductive system

The effects of dimethoate on the reproductive system of animals can vary depending on the species, doses, exposure duration, and other factors. Generally, organophosphate pesticides have been associate with adverse effects on reproductive health in animals. Animals exposed to dimethoate may have lower fertility. Hormonal imbalances and abnormalties in the reproductive system are to blame for this. Following dimethoate treatment, sperm count, motility, and viability significantly decreased in mice, while spermatozoa with aberrant morphology significantly increased.[16] Fish treated with dimethoate (Ghousia) also showed a notable decline in fertility. Dimethoate's effects on the male reproductive system may be brought on by dimethoate. Hormonal imbalance may results in histopathological changes, testicular and epididymal weight loss, and a reduction in sperm count.

High dosages of dimethoate can lower leutinizing hormonal cells and levels of leutinizing hormone.[17,18,19,20,21] They can also lower testosterone levels and testicular decline.[22,23]

It has been documented that dimethoate is toxic to rodent reproductive systems in both sexes.

It has been shown that exposure to dimethoate altered serum gonadotrophin levels and altered the estrous cycle.[24]

Effects on liver

Research has demonstrated that liver tissue may undergo histopathological alterations as a result of exposure to organophosphates, such as dimethoate. These modifications could include hepatocyte hypertrophy, necrosis, cellular damage, changes in the structure of the liver cells, hepatocyte cytoplasm becoming vacuolar, dilation of sinusoidal spaces, irregular nuclear shape, and lymphocytic infiltration in the central vein.[25, 26, 27,28, 29] Serum total protein, albumin, and globulin levels are decreased while those of gama-glutamyl transferases, phosphatases, aminotransferases, urea, creatinine, and uric acid are elevated in response to dimethoate.[30] Total lipid (TL), cholesterol, triglyceride (TG), and low density lipoprotein (LDL), were significantly. increased, while high density lipoprotein (HDL) level was decreased. NSO.

Effects on kidney

Organ damage, including renal damage, can result from the interference of organophosphorus compounds, such as dimethoate, with cellular functions. According to the current study, the

effects of dimethoate on the kidney included glomerular degeneration, haemorrhage, compression of blood vesicles, tubuar degeration, glomerular shrinkage, cell rupture and hydropic changes, and changes in the cellular lining of the Bowman capsule.[31, 32] Rats given dimethoate showed a variety of renal dysfunctional symptoms, including elevated urine production and altered levels of creatinine, urea, and uric acid.[33, 43]

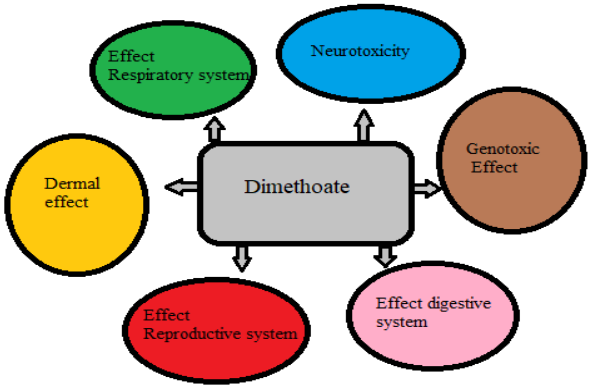

Figure 2: showing toxic effects of dimethoate.

Genotoxicity

According to Silva[34], the term "genotoxicity" describes a chemical agent's capacity to either interact directly with DNA or act indirectly, causing damage to DNA by interfering with spindle apparatus or enzymes involved in DNA replication and consequently causing mutations. The literature has extensively examined how oxidative stress can trigger multiple pathways that result in an oxidative imbalance that harms mammalian cells and eventually encourages the development of cancer.[35,36]

Dermal effects

Skin contact with dimethoate can result in irritation, redness, and in severe cases, chemical burns. At high doses, pesticides increase DNA replication and cell prolification which can give to rise neoplastic cells; the target organ depends on the route of absorption and metabolization.[36] Dimethoate can absorb easily through various route due to their lipophilic structure.[37] Dimethoate may causes dermatitis and sensitization in close contact with skin.[38,39]

Gastro intestinal disturbance

Dimethoate insecticide affect the digestive system of rats if ingested in toxic amounts. Exposure of dimethoate, to rat disrupt their digestive process by interfering with the normal

functioning of acetylcholinesterase, an enzyme that regulates the neurotransmitter acetylcholine. Rats exposed to dimethoate might experience nausea, which can lead to vomiting. This could be a result of the disruption in the normal digestive processes caused by the toxic effects of the chemical. Dimethoate exposure might lead to diarrhea in rats. This is often as a result of the disruption in the gut's normal motility and function due to the toxic effects of the chemical on the nervous system. Rats might exhibit signs of abdominal discomfort or pain due to the impact of dimethoate on the nervous system, leading to altered gut function and sensations. Prolonged exposure to high doses of dimethoate might lead to gastrointestinal ulceration or inflammation due to its toxic effects on the lining of the digestive tract.[40] It's important to note that the severity of these effects can vary based on the dose and duration of exposure. Moreover, these effects are based on toxicological studies and might not fully reflect individual variations or other potential impacts of dimethoate on the digestive system. Always remember that the use of pesticides and chemicals should be handled with extreme caution, following safety guidelines and regulations to prevent unintended exposure to humans and animals. Dimethoate causes histological changes in liver and kidney of mice that may worsen digestive system.[31] Pancreas is the target organ for insecticide, that deteriorate pancreatic functions and disturb digestive function.[41] Pancreatic damage may lead to pancreatic cancer.[42]

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