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IP International Journal of Forensic Medicine and Toxicological Sciences

Journal homepage: http://www.ijfmts.com/

Review Article

A review on effects of dimethoate

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ARTICLE INFO

Article history:
Received 11-01-2023
Accepted 28-01-2023
Available online 04-05-2023

Keywords:
Organophosphates
Dimethoate
Insecticides
Mammals

ABSTRACT

A set of man-made chemicals that have a tendency to poison insects and mammals are organophosphates. Organophosphates are a broad category of chemicals and are widely used insecticides. Now a days, they have various applications such as in agriculture, home, gardens etc. Dimethoate is an insecticide that falls under the category of organophosphates. Dimethoate is a white crystalline solid with robust odor resembling the form of sand or sugar. It was first produced in 1956 to kill or manage insects such as house fly, termites, etc and various countries use it for farming. We have less knowledge about the levels of dimethoate in the environment as, there are no residues found in soil nor it has been found in drinking water. Insecticides that contain Dimethoate are used on various crops such as cotton, tobacco, olives also on various fruits and vegetables. Rapid and fatal poisoning could be caused if it is inhaled or if it comes in contact with skin. The symptoms include headache, sweating, nausea, vomiting, diarrhea, loss of coordination and muscle twitching. Individuals with long term exposure to higher levels of Dimethoate are found to suffer from personality changes including- depression, anxiety, irritability etc. These harmful effects could last for months or years in human central nervous system. Developing fetus are also affected due to the inhalation of gaseous Dimethoate by pregnant women. In this review we are going to discuss about the toxicological aspects and properties of Dimethoate and its effects on humans and the future aspects related to this insecticide.

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1. Introduction

The review is based on the probable risks of dimethoate due to its direct exposure from post yield of fruits and vegetables. It was patented and introduced in 1950s by American scientists for the purpose of killing houseflies. Various organophosphates are commercially used worldwide for pest control in the agrochemical industry. The review focuses on the various effects related to the Organophosphate Dimethoate which is generally used as an insecticide. Dimethoate is a registered insecticide for over 30 years and is being used widely all over the world. There are various approved sources and products, and is used as a pre-harvest as well as post-harvest in various crop production.

Now let us discuss about the toxicological effects of dimethoate, Dimethoate is both, direct and systemic toxin and its extensive toxicological effect on animals and human is that it inhibits the activity of acetylcholinestrase (AChE), an inhibitor which inhibits cholinesterase, an enzyme responsible for the better functioning of central nervous system (CNS). The active ingredient of the Organophosphate Dimethoate is Dimethoate 0,0-DimethylS-(N-methylcarbamoylmethyl) phosphorodithioate. The direct exposure of dimethoate can cause various effects on humans including rapid and

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https://doi.org/10.18231/j.ijfmts.2023.001
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fatal organophosphate poisoning with headache, nausea, sweating and vomiting, diarrhea, loss of coordination, muscle twitching and death. Dimethoate may also cause some serious genetic mutations.[1–4]

1.1. Toxicological aspects: (absorption, distribution, metabolism and excretion

The absorption of radio-labeled dimethoate was seen from the gastrointerstinal tract (from gut) through oral administration in rats. Following the dose of 10 mg/kg body weight in both the sexes of rats showed similar distribution in the concentration of plasma and tissues within 0.5 hours of administration. The liver and kidneys showed the presence of radioactivity, and the lowest levels were found in brain and fats. The excretion was seen majorly through urine (85-91%), with lesser amounts excreted through feces (1.2- 1.6%) and through expiration (2.1- 2.2%).

When 1% aqueous solution of [14C] – dimethoate was administered dermally keeping the dosage of 10 mg/kg body weight, the recovery of radio labeled substance was approximately 9- 11% in the form of urine, feces and corpse. Very good absorption was seen when applied to the skin of rats in the form of spray dilutions of the radio labeled dimethoate. There were variations in the ratio of radio label absorbed from 1- 42% which relied upon the dilution, dosage and frequency of administration. At day 5th, the radio label dimethoate was mainly excreted through urine, feces and the remaining from the corpse. Administered skin sites were also showing significant amount of metabolites. Similar metabolites were seen in this case as were seen the oral and dermal administrations.[5–8]

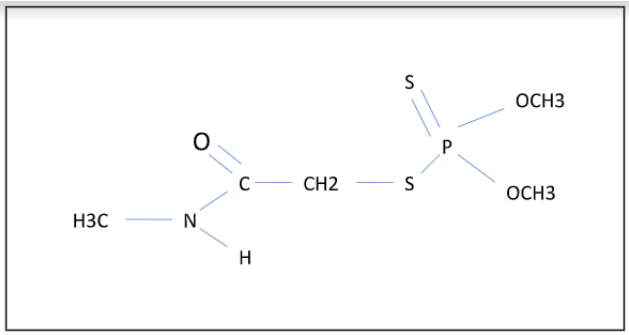

Fig. 1: Chemical structure of dimethoate.[1]

During the metabolism of dimethoate in the liver it behaves as an anti-cholinestrase as it gets converted into four short lived active metaboloites, and when these metabolites get hydrolysed their by-products are excreted very rapidly mainly in the form of urine. The preliminary detectable effect of the dimethoate poisoning is cholinesterase inhibition leading to dis-functioning of the central nervous system (CNS).

The radioactive metabolite always partitioned less in favor of chloroform from water than dimethoate itself in all tissues and excreta, demonstrating degradation to more selectively water-soluble compounds had already taken place (Davies, 1999).[9]

Separated form of the radio label material was investigated that was formed. The chemicals in the mixture were first separated by continuous water extraction of chloroform at, when non-ionic chemicals were removed; the pH was 7 while the ionic chemicals and chloroform remained in the water. The latter, made up of a combination of the products created when P-O-C is hydrolyzed, the P-S-C, amide and dimethoate linkages before or were not further separated after the ono- oxidation. 74- 98% of the discharged materials in rat and human urine were in radioactive form. These results of ionic hydrolysis were all recognized to be of low mammalian toxicity and without anti-cholinesterase action.

2. Toxicological Properties of Dimethoate

Dimethoate is a systemic and contact organophosphate (OP) pesticide that has been approved for use as an insecticide and acaricide in Australia for over 30 years. It is included-in, Schedule 6-of-the-Standard-for Uniform Drug and Poison_Programming (SUSDP). The Australian Acceptable Daily Intake (ADI) value for dimethoate of 0.02 mg/kg body weight/day was established in1988. This ADI was set to the NOEL of 0 by applying a 10x safety factor.2 mg/kg body weight/day for whole blood (ChE) inhibition in a 14- to 57-day repeat dose human study. No acute reference dose (ARfD) has been established for dimethoate. The current health value of dimethoate in Australian drinking water is 0.05 mg/l. Safety information for dimethoate is detailed in the First Aid Guide and Safety Information. There are currently eight approved sources of the active ingredient dimethoate. Revised in 1988, new information has become available that addresses a variety of toxicological endpoints that have not been previously studied or evaluated. In particular, there are behavioral studies that quantify the extent of functional impairment (task performance) in rats-exposed to dimethoate. It is considered important to review these studies and any other new data to determine whether existing health standards are still adequate to protect the general public and workers handling products containing dimethoate. After being given to rats orally, radio labeled dimethoate was efficiently absorbed from the digestive system. Following a dosage of 10 mg/kg body weight, the highest plasma and tissue concentrations were reached 0.5 hours later and had a comparable distribution in both sexes. The liver and kidney were the primary tissues where radioactivity was found, with the brain and fat having the lowest levels. The radiolabel was mostly excreted in the urine (85-91% of the administered dose) by day five (89–95%), with minor amounts being found in the faeces

(1.2–1.6%) and expired air (2.1-2.2%). After receiving dosages of 10 and 100 mg/kg body weight of dimethoate, respectively, approximately 69-72% and 52-59% of the radio label were found in the urine within six hours (Kaplanis, 1959). Rats extensively metabolised dimethoate, mostly by first cleaving the C-N bond to produce dimethoate carboxylic acid and then producing a variety of phosphate and thiophosphate esters. The oxidative metabolism of dimethoate to form the oxygen analogue, omethoate, is the quantitatively minor route of elimination. One to two percent of the dose that was eliminated in urine as the parent compound (Kaplanis, 1959). A dose of 10 mg/kg body weight of [14C]- dimethoate administered topically resulted in approximately 9-11% of the radio label being retrieved in the carcass, urine, and faeces. At the treated skin sites, 13–17% of the radio label was found after five days. When a formulation and representative spray dilution of radio labeled dimethoate were sprayed to the skin of rats, they were effectively absorbed. Depending on the dilution, dose, and duration of treatment, the percentage of radio label that was absorbed ranged from 1-42%. Smaller levels of radio label were found in the faeces and corpse at day 5, but the radio label was largely eliminated in the urine. The majority of the dose was in the skin washing fractions, but significant amounts of radio label were recovered from the treated skin locations (Kaplanis, et.al.,1959), (Kruegar 1958). In an in vitro experiment, the amount of radio label absorbed via the skin of rats was much higher than that of humans (from a 400 g/L EC formulation or 1/200 aqueous dilution.[4]

3. Effects of Dimethoate

Fathia A. Khogali, et.al., examined the venomous effects of the chemical Dimethoate 40EC on the tissues of chosen organs, as well as the liver, kidney, stomach, and viscus of Swiss anomaly mice, moreover as on some blood constituents, as well as RBC and WBC count, Hb content, hematocrit price, and plasm macromolecule. The amounts of Dimethoate four EC was provided in doses that were sixteen mg/kg and 4 mg/kg, severally, and were proportional to field dosages.

When compared to controls, treated mice considerably diminished their Hb and hematocrit values, however there was no amendment in their total protein levels. In comparison to the controls, treated mice's hepatocytes had a histochemically noticeable depletion in polyose concentrations. Hepatocyte disease, condition, blood coagulation, and important white corpuscle infiltration were among the histopathological alterations within the liver that were seen. The central vein was additionally affected.

4. Effects of Dimethoate on Humans

A systemic insecticide and acaricide, dimethoate (DIM) is an organophosphorothionate (OPT) pesticide used all over the world. It has low to high acute mammalian toxicity, and like the other OPT pesticides, it works by inhibiting acetylcholinesterase (AChE), which is mediated via its poisonous metabolite dimethoate-oxon or omethoate (OME), which is also employed as a direct acting pesticide (Aprea et al., 1998, 2004).[2] Human c-DNA expressed CYPs and human liver microsomes (HLM), along with CYP-specific pharmacologic inhibitors, were used to describe human hepatic DIM bioactivation to the hazardous metabolite OME utilising a technique based on AChE inhibition. Comparing the measured kinetic parameters and AChE IC50 to those previously achieved with other OPTs reveals decreased DIM desulfuration reaction efficiency and potency in AChE inhibition. Results revealed that, similarly to other OPTs investigated thus far, OME production is mostly catalysed by CYP1A2 at low DIM concentrations, while 3A4 plays a significant role at high DIM levels. Unlike the other OPTs, the DIM desulfuration process displayed an unusual kinetic profile, which was probably caused by CYP3A4 auto activation. The degree of the activity curve's sigmoidicity increased as CYP3A4 levels in HLM rose or vanished in the presence of a CYP3A4 pharmacologic inhibitor (Krieger and Thongsinthusak, 1993; Aprea et al., 2004). The recent findings that among patients hospitalised after OPT intoxication, DIM ingestion gave different symptoms and more severe poisoning (23.1% of fatal cases versus total) than chlorpyrifos (8% of deaths), which has a lower LD50 value, can be considered one of the possible explanations for this atypical kinetic behaviour. Pralidoxime was not well-tolerated by DIM-poisoned patients, hence the use of CYP3A4 inhibitors should be investigated as an adjunctive therapy.

4.1. Result on prostate

Prostate cancer is one among the foremost typical cancers in males. varied studies have advised that farming might give a big risk for glandular carcinoma. chemical applicators seasoned a disproportionately high variety of occurrences and fatalities relative to the final population. glandular carcinoma has been connected to specific individual organochlorine and insect powder insect powder exposures. Dimethoate users had a way higher likelihood of developing aggressive glandular carcinoma than nonusers, in line with analysis on pesticides to work out their role within the development of the unwellness.

With extra changes for extremely correlate pesticides with the other pesticides coupled to glandular carcinoma, the elevated risk for dimethoate remained unchanged. In comparison to people while not a case history of glandular carcinoma, those with a positive history of the unwellness

showed a bigger relationship between dimethoate use and aggressive glandular carcinoma.

Larissa A. and Laura E showed in their study that men UN agency have ever used the OP insect powder dimethoate have a considerably redoubled risk of developing aggressive glandular carcinoma. During this analysis, we have a tendency to found a better risk for aggressive PCa among applicators UN agency reported exploitation dimethoate. Dimethoate (O,O-dimethyl S-methylcarbamoylmethyl Phosphorodithioate) could be a dithioate OP insect powder. In some strains of rats and mice, dimethoate showed cancer effects, with neoplasms occurring within the endocrine organs, liver, and humour it's additionally in keeping with known uses for dimethoate that chemical applicators UN agency reported exploitation it were a lot of possible to say animal-related application techniques. Though this mechanism has not been specifically coupled to a better risk of glandular carcinoma, the pesticidal activity of OP pesticides is caused by their ability to inhibit the catalyst that breaks down the neurochemical neurotransmitter.

The study done by them has incontestable that there's a perceptibly redoubled risk of pesticides interacting with genetic variations in signal transduction and cellular communication pathways that ar wedged by neurotransmission. Therefore, a association between glandular carcinoma and acetylcholinesterase suppression is probably going.

5. Metabolism of Dimethoate

Dimethoate is largely metabolised in liver. Dimethoate is metabolized into 2 toxic metabolites i.e., dimethoate-oxon and omethoate (OME). so as to verify the method of metabolism Associate in Nursing experiment was conducted during which they gave a healthy adult some sugar peas with dimethoate residues. Giving a healthy male sugar peas having permissiveness dimethoate residues (17 ppm; V-day oxon) Associate in Nursingd a bolus indefinite quantity of dimethoate provided information to assist with an exposure analysis. The tolerance for dimethoate in peas remains a pair of ppm. Serial samples of whole piddle were taken and examined for the presence of dimethoate and its oxons, dimethylphosphate, dimethylphosphorothioate (DMTP), and dimethyl- phosphorodithioate. the quantity of dimethoate that was given was around zero.1 mg/kg weight, and it had no harmful effects. among 2 hours, dimethylphosphates were detected within the piddle. regarding hour of the overall metabolites were DMTP. solely minute amounts (less than zero.5%) of dimethoate and oxon were found within the piddle. though piddle metabolites were conspicuous and acetylcholinesterase inhibition wasn't, this means that they're a stronger signal of acute exposure than enzyme inhibition. There ar numerous opposing dotes that ar used for insect powder and toxic condition like mydriatic oximes and benzodiazepines. Charcoal can even be used for treatment of those quite poisoning.

6. Future Aspects

6.1. Star gene

It has been demonstrated that the widely used organophosphate insecticide dimethoate interferes with animal reproduction. Serum testosterone levels are believed to be a key factor in the development of Dimethoate-induced infertility, even though the aetiology of Dimethoate-induced reproductive damage is yet unknown. The mouse MA-10 Leydig tumour cell line was utilised to examine whether dimethoate can directly block steroid hormone biosynthesis and to pinpoint the site of steroidogenic inhibition because Leydig cells, which produce testosterone, are essential for male reproductive function. Without influencing total protein synthesis or protein kinase A activity, Even while it reduced the P450 side chain cleavage (P450 scc) enzyme's activity, this alone did not explain the degree to which Bu2cAMP hindered progesterone synthesis. Our findings instead suggest that Dimethoate largely prevented the StAR gene from being transcribed, inhibiting steroidogenesis. This discovery is crucial since the transfer of cholesterol from the outer to the inner mitochondrial membrane, the rate-limiting and sensitively controlled phase in steroidogenesis, is mediated by StAR protein (Clark, et. al., 1994). This work suggests that StAR may be a crucial target for contaminants in the environment that interfere with steroidogenesis and harm reproductive function.[10]

6.2. Methylene blue to reverse the effect of dimethoate

Organophosphate agrchemicals sold commercially for pest control have the potential to be misused for intentional self harm. A case study presented by Youssefi ND described case of refractory hemodynamic instability brought on by the suicidal consumption of dimethoate, a moderately toxic organophosphate insecticide, and the successful use of methylene blue to treat this complication.

The case mentioned a 47 year old man who had consumed dimethoate, fell unconscious and was brought to the emergency room. The emergency medical services report, the patient was discovered in a barn with comatose, mouth foaming and with a foul stench. According to his medical history as described by his family, comprised peptic ulcer disease, a cerebrovascular accident event with no lasting damage, hypertension, gout, depression, and suicidal tendencies. Although having suicidal tendencies the patient never tried to commit suicide before. He experienced severe hypotension very quickly, which was resistant to all treatments, including norepinephrine, epinephrine, and vasopressin as well as antidotes and resuscitative fluids. High cardiac output and low peripheral vascular resistance during pulmonary artery catheterization were indicative of

distributive shock. When Methylene blue was started for the patient, his hemodynamic condition improved. On-admission plasma cholinesterase levels and dimethoate serum levels were found to be 2247 U and 56 g/mL, respectively, according to laboratory examinations. The patient needed a two-week stay in the intensive care unit eventually requiring a tracheotomy for ventilation (Pardo et.al., 2020).[11]

Due to its extreme toxicity, dimethoate stands out among anticholinergic medications. Despite identical animal toxicity, acute self-poisoning with dimethoate has a three-fold higher human case fatality rate than poisoning with chlorpyrifos. Its half-life for elimination in plasma is 30.4 hours, whereas that in urine is 23.8 hours. The blood content of dimethoate in deadly and suicidal poisonings varies from 2 to 100 g/Ml. The patient's serum dimethoate level in this was 56 g/mL. Dimethoate as an OP can decrease baroreceptor reflexes, causing a significant drop in blood pressure, by blocking nicotinic transmission at sympathetic and parasympathetic ganglia. Through the action of acetylcholine on muscarinic receptors on vascular endothelium, it may also cause peripheral vasodilatation. Additionally, an increase in nitric oxide (NO) generation may be the cause of the peripheral vasodilation caused by OP chemicals.

Methylene blue is a hetercyclic dye used for a number of toxicological crises. It uses suppression of NO-induced vasodilation mechanism to treat hypotension. The activity of NO synthase causes the production of NO in vascular smooth muscle cells and endothelial cells.Once guanylate cyclase is activated by NO it results in vasodilation. Methylene blue blocks NO synthase, vasodilation is reduced and systemic vascular tone is improved.

The case study demonstrated the effectiveness of Methylene blue in correcting refractory hypotension caused by dimethoate poisoning and preserving the patient's life. If the initial therapy with catecholamines or vasopressors fails Methylene blue should be considered for the treatment (Pardo et.al., 2020).[11]

6.3. Protective effect of date palm fruit extract

People are exposed to toxic substances like organophosphorus insecticides more and more frequently these days. These substances have caused an excessive amount of free radical generation, which is to blame for the organism's various cell changes. Recent studies have demonstrated the critical importance of dietary antioxidants in preventing the harm caused by hazardous substances. The purpose of this study is to determine the function of date palm fruit extract (Phoenix dactylifera L.) in preventing liver toxicity and oxidative damage brought on by subchronic exposure to dimethoate (20 mg/kg/day). By increasing the levels of hepatic marker enzymes (transaminases, alkaline phosphatase, gamma-glutamyl transferase, and lactate dehydrogenase), as well as hepatic malondialdehyde, oral administration of dimethoate caused hepatotoxicity, which led to a significant alteration in the body's antioxidant defence system. Particularly, dimethoate was found to boost the activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx), while considerably decreasing the activity of catalase (CAT). These biochemical changes were accompanied by histological abnormalities in the liver slice that included the development of vacuolization, necrosis, congestion, inflammation, and an expansion of the sinusoids. The liver damage caused by dimethoate was reversed by pretreating with date palm fruit extract, as shown by the reduction of hepatic lipid peroxidation, improvement of SOD, GPx, and CAT activities, and improvement of histopathological alterations. The findings show that date palm fruit may be helpful for preventing oxidative stress-induced hepatotoxicity in living organisms.

6.4. Effects of ZnO nanoparticles on dimethoate induced poisoning

Various commercial products, like toothpaste, pigments, medical equipment, medicine carriers, and bio-imaging probes, contain ZnO nanoparticles (nano ZnO). Due to its antibacterial characteristic, nano ZnO is also frequently used in the food industry as dietary supplements, food additives, and food packaging components.[12] There are several kind of cosmetics and food additives on the market which contain nano ZnO. A potential use for nano ZnO in agriculture is as a fungicide. As a result, compared to other nanomaterials, humans are more likely to be exposed to nano ZnO in food-related items. Due to its high efficiency, the organo-phosphorus pesticide dimethoate is frequently employed in nations to control pests and a wide range of other insects in agriculture. In the soil, in crops, in water, and in meals like cow's milk, the residue and analogue of dimethoate have been discovered. Cholinesterase (ChE) inhibition, that results in dysfunction at the neuromuscular junction and inhibition of nerve conduction, is the main cause of dimethoate toxicity. The risk of human co-exposure to nano ZnO and nano Dimethoate has increased due to the extensive usage of these substances (Yan, et.al., 2015).

Studies done have shown that when ZnO nano particles and dimethoate were introduced separately in the mice they have exhibited different affects. The normal day to day was not affected much by the ZnO but symptoms like anorexia were seen in case of dimethoate. However when the subject were exposed to the combined dose they showed varying degrees of signs such as loss of appetite, mild tremor and piloerection. Co- administration of dimethoate with nano or bulk ZnO had a significantly enhanced adverse e ect on the weight gain. In comparison to the nano ZnO group, co-administration of dimethoate and nano ZnO considerably increased the biodistribution

densities of Zn in the liver and lung. Due to DM's lipophilic nature and propensity for interacting with cell membranes, the bilayer structure of phospholipids is disturbed33, which makes it simple for Zn2+ to pass through the membrane and disperse throughout the tissues. As a result, the bio-distribution density of Zn in mice liver is dramatically increased. Co- administration with nano ZnO and dimethoate significantly increased the levels of alanine aminotransferase, aspartate aminotransferase, alkaline phospatase, lactate dehyrogenase, and bilirubin showing that co-administration with nano ZnO and DM had an amplified influence on the liver dysfunction. It is safe to use nano ZnO in food supplements and foods at modest doses. The studies have shown that nano ZnO significantly increases dimethoate-induced liver damage in mice when it is paired with dimethoate, which is caused by the increased accumulation of Zn and DM in the liver (Yan, et.al., 2015).[13]

7. Forensic Importance

Forensic Entomotoxicology is becoming more popular these days in helping with legal investigation. Insects are being utilized as an alternative source to analyse the death. Multiple studies have demonstrated that susbstances from the insect, blow fly (calliphoridae) can be successfully identified, named, and quantified. In forensic investigations, Calliphoridae flies, which are present at crime scenes and dead bodies, have been used to identify toxic compounds, the minimum post-mortem delay, and whether the body has been moved (Chophi, 2019). Toxicological examinations of forensic cases can make use of carrion flies. Drugs and other chemical substances discovered in the decomposing corpse can be consumed by insects and maggots as they feast on it. The medication can be used to identify and provide concentration in the maggots. It is crucial to ascertain the particular anatomy of the insects and how suicidal substances affect them, understanding how different toxicants affect the flies is crucial. In crime scene investigations, the toxicological examination of the insects may help in identifying the cause of death or create connections that would help to identify the cause. Insects at a crime scene can preserve evidence that is not vulnerable to malicious destruction.

An organophosphate used as a pesticide is called dimethoate. The chemical interferes with the flies' systems commonly found at crime scenes, giving it a common cause of poisoning in cases of accidents, suicide, and murder. Toxins such as dimethoate, that are present in the carrion, can alter an insect's life cycle. Such alterations in blowflies at a murder scene could be a sign of dimethoate poisoning.

Study done by Fahd Mohammed exhibits how the dimethoate affects the life cycle of the insect callphoridae. One batch of fly eggs were grown in a medium containing dimethoate and one without it as the controlled sample for the comparison. Dimethoate clearly had an effect on the duration of the life cycle and the various developmental stages. Depending on the pesticide concentration in the treated samples, the life cycle duration changed. The sample affected with the dimethoate took more time to complete the life cycle than the one which was not infected. The increase in dimethoate concentration was strongly associated with increase in Post-Mortem interval. Flies took longer to grow as a result of dimethoate. Blowflies feeding, post-feeding, and pupal stages of development were all prolonged by dimethoate. Carrion flies found at crime scenes with delayed growth and development could have been poisoned with toxins like dimethoate. Such evidence may create pathways to investigate when figuring out the cause of death. Flies feed on rotting corpses and have been found to consume substances that could have caused to the body's demise. Analysis of the life cycle duration can help in estimating the postmortem interval, which can then help determine when someone had died (Chophi, et.al., 2019).[4]

8. Conclusion

An organophosphate known by the name of Dimethoate which is a human made chemical has poisonous effects on insects and mammals. Now a days, it is being produced by many countries for the purpose of use in farms. We have very little knowledge about dimethoate rightnow, but studies have shown that if it is touched or inhaled can cause toxic effects on the body of humans and may result in fatal poisoning too. Long term exposure to this chemical may lead to nausea, muscle twitching, diarrhea, anxiety and may also cause mutation in developing foetus in case of pregnant women.

The absorption of radio labeled dimethoate was seen from the gastro intestinal tract through oral administration. The liver and kidney also showed presence of this chemical. It also affects the functioning of CNS as shown in studies. In humans it has shown significant effect on the functioning of liver and kidney and also on the prostate by increasing the risk of prostate cancer. Dimethoate is metabolized in liver, forming two metabolites i.e., dimethoate- oxon and omethoate (OME). Star gene and methylene blue have the reversing properties on the effects of dimethoate. In the field of forensics entomotoxicology it is becoming legal for investigation as insects are being used as an alternative source to analyze death. In many cases it has been found that the insects lying in or around the crime scene have presence of dimethoate pesticide. In some studies it also shows the effect of dimethoate in the life cycle of housefly.

9. Conflict of Interest

The authors declare that there is no conflict of interest.

10. Source of Funding

None.

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Cite this article: Roziya S, Negi D, Yadav S, Chandra M. A review on effects of dimethoate. *IP Int J Forensic Med Toxicol Sci* 2023;8(1):1-7.