Available online at www.joac.info

ISSN: 2278-1862

Journal of Applicable Chemistry

2013, 2 (4):832-840

(International Peer Reviewed Journal)

Chemical and Biochemical Aspects, Decomposition and Environmental Impacts of Monocrotophos-Preview

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Received on 22nd May and finalized on 21st June 2013.

ABSTRACT

Monocrotophos is the Asia's most selling insecticide is a systemic and highly toxic organophosphorus pesticide that has been banned in many Countries and known to produce cholinergic and non-cholinergic effects. It listed as a Prior Informed Consent chemical, and it is still used extensively in developing Countries. Asian Countries are top user of monocrotophos because of its low cost, but same time people are using it to committing the suicides. This particular review involved in the study of toxicity, decomposition and fate in environment.

Keywords: Monocrotophos, Insecticide, Toxicity, Decomposition.

INTRODUCTION

Monocrotophos (MCP) (dimethyl (E)-1-methyl-2-(methylcarbamoyl) vinyl phosphate) is a systemic insecticide and acaricide belonging to the vinyl phosphate group [1]. It controls pests on a variety of crops. Insecticide MCP is existing in *cis* and *trans* form [2]. The main trade names of MCP is; Azodrin, Bilobran, Crisodrin, Monocil, Monocron, Nuvacron, Pillardrin, Plantdrin, Susvin, Ulvair, Dominator, Macabre, Suncrotophos, Monopaz etc. Approximately 30,000 tons of MCP are used annually. As per data's Asia is the top user of MCP as; India (43%), South America (26%), China (15%), and Southeast Asia (9%) account for 90% of the use, internationally (figure 1). In India Andhra Pradesh and Punjab are the chief consumers of MCP (figure 2) [3,4].

Same time MCP is misused by the peoples especially in the suicides cases. Now a day in Asia MCP is also available in the form of microcapsules (water-soluble) in which polyurethane acts as a carrier material [5]. MCP is an extremely toxic to aquatic invertebrates, birds and mammals. It is toxic to most organisms and in particular to birds [6]. The most likely route of exposure to MCP for the public is via residues in food. The prolonged or continued use of MCP in plant protection may lead to significant dermal exposure with an impact on cholinesterase, genotoxicity and cardiotoxicity activity [7-12].

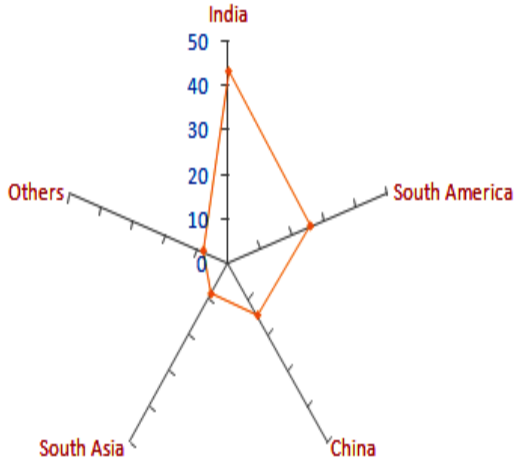

Figure 1. *MCP worldwide consumption (% wise).*

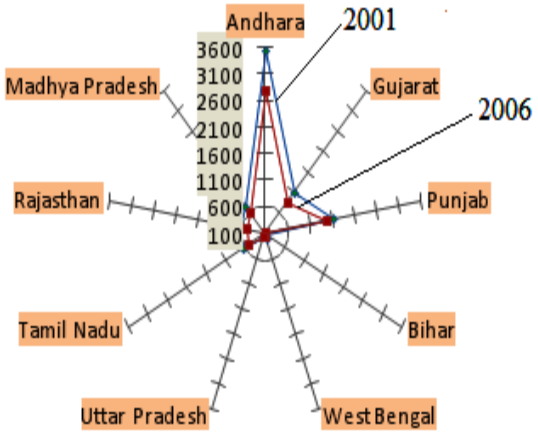

Figure 2. *Consumption of MCP (Metric Tons) in India at year 2001 and 2006.*

Toxicity- MCP is a water-soluble organophosphate insecticide with high oral and moderate dermal toxicity. The toxicologically relevant mode of action is the inhibition of **acetylcholinesteras**e (AChE) activities [13]. MCP is neurotoxic in nature and caused a significant inhibition of the brain acetylcholinesterase activity [14-17].There is age-related differences in the inhibition of brain, but not necessarily red blood cell and AChE [18].

Repeated exposure to MCP in rats may cause behavioral and neurochemical modifications which may persist even after withdrawal [19]. MCP use (alone or combination) had genotoxic and mutagenic effects [20]. MCP may bring about physiological upsets by altering extra-hepatic glutathione & glutathione-S-transferase dependent and by inhibition of ATP synthesis, by oxidative phosphorylation and glycolysis, in addition to inhibition of muscle acetylcholinesterase [21,22]. Muscle mitochondrial ATP synthase activity was inhibited in the rat in acute exposure to MCP while respiration was not affected. This was accompanied by decreased mitochondrial uptake of calcium and increased levels of nitric oxide [23]. Membrane bound enzymes like acetylcholinesterase (AChE), Na^+-K^+-ATPase and Ca^{2+}-ATPase were also inhibited [24,25]. Brain AChE activity was significantly greater in fish followed by pigeon and rat. MCP was found to be a competitive inhibitor of rat, pigeon, and fish brain AChE, thereby, altering the Km (Michaelis constant) widely among the species. Comparatively, least alterations in Km were observed in fish and maximum in pigeon [26].

MCP induced apoptosis in neuronal cells was involved in apoptotic changes correlated with expression of selected cytochrome P450s in PC12 cells as a result a significant induction in reactive oxygen species and decrease in glutathione levels were observed in cells exposed to MCP [27]. In vitro studies have revealed that all the MCP induced apoptosis and necrosis in cultured human peripheral blood lymphocytes [28].

MCP has histopathological effects in liver, kidney and muscles of normal, protein malnourished, diabetic as well as both protein-malnourished, diabetic albino rats and fish [29,30]. Histopathological effects of MCP on the gill, kidney and intestine tissues of the Cirrhinus mrigala were determined by light microscopy [31]. MCP had enzymatic changes in plasma and liver of female albino rats and cellular toxicity of MCP observed even after their sub chronic administration in low doses for a long period [32]. Histological changes in the liver of Tilapia mossambica were observed after exposure to a sub lethal level (2.5 ppm) of MCP [33]. MCP altered the protein, DNA, and RNA contents and the 5'-nucleotidase activity levels as early as 2 and 5 days. However, these changes were reversed by 10 days and after a short period of recovery, the alterations reappeared. This supports our earlier histological observations of hepatic pathology during MCP exposure [34]. Among the mammalian cell lines, MCP showed enhanced DNA

synthesis [35]. Total protein, amino acid, and ammonia contents were decreased in gill, kidney, liver, and muscle tissues, and recovery was slight at the end of 21 days of transfer of fish into freshwater [36,37].
MCP pesticide during sexual development causes the feminization/demasculinization of the reproductive traits [38]. Reproductive toxicity caused by organophosphates (MCP) at cellular and molecular level in the ovaries of rats [39]. Reproductive toxicity of MCP also observed in bobwhite quail [40]. MCP had significant decline in the weight of testes, epididymis, seminal vesicle, ventral prostate and reduction in sperm counts both in epididymis and testes in toxicated animals. Pre and post fertility test showed 60 and 70 % -ve results after treatment. A significant reduction in the testicular glycogen and sialic acid was also noticed. Contrary to these, protein and cholesterol contents of testes were significantly increased. In addition, acid phosphatase activity was significantly increased, while alkaline phosphatase and testosterone levels were diminished. Thus obtained results collectively indicated that MCP possesses toxic effect on male reproductive functions [41]. MCP adversely affects the fish by twofold mechanisms: (a) MCP exposure enhanced the mRNA expression of gonadal aromatase, the enzyme that converts androgens into estrogens, consequently reducing plasma testosterone levels and increasing plasma concentrations of 17betaestradiol and (b) MCP treatment increased follicle-stimulating hormone beta subunit mRNA expression and secretion and decreased luteinizing hormone beta subunit mRNA expression and secretion, leading to the disruption of reproductive endocrine control and androgen and estrogen balance [42,43].
MCP is genotoxic on Meretrix ovum and also induces a pollution stress related retardation of somatic growth of this mussel [44,45]. Genotoxic mechanism in fish taken placed by DNA damage and measured in the gill, kidney and lymphocytes as the percentage of DNA in comet tails of fish exposed to different sublethal and nonlethal concentrations of MCP [46,47]. The Salmonella lactam test is a newly developed method for detecting genotoxins. This technique is based on the ability of DNA damaging agents to reverse expression of the beta-lactamase gene, an important gene that enables microbes to resist beta-lactam antibiotics. MCP, acephate and carbofuran proved highly mutagenic in strain (JK947) [48].
Hyperglycemic and stressogenic effects of MCP has been observed, hyperglycemia and hyperlactacidemia induced by MCP were abolished by pre-treatment with atropine [49,50].

In acute toxicity studies higher concentrations of MCP caused marked increase in mobility of cells exhibiting rocking movements within two mins of exposure but were decreased after 30 mins. Cytotoxic effects of MCP revealed non-repair of necrosis [51-52]. Acute exposure of MCP caused significant induction of the heat shock proteins (HSP60 and HSP70) in a tissue and dose-dependent manner, suggesting their importance as molecular indicators (biomarker) in the assessment of cellular toxicity caused by MCP [53-54]. MCP affects the intermediary metabolism of fish (Oreochromi mossambicus) by depleting in gluthathione-S-transferase activity in all the tissues, thereby enhancing the lipid peroxidation resulting in cell damage[55]. MCP has the propensity to augment the oxidative stress and further disrupt glucose homeostasis in diabetic rats [56]. MCP has strong influence on thyroid gland of dams as the thyroid profile was altered significantly and the hepatotoxic effects of MCP were also observed [57]. Acute exposure to organophosphates induces a delayed neurodegenerative condition, this was accompanied by decreased activities of the mitochondrial enzymes; NADH dehydrogenase, succinate dehydrogenase, and cytochrome oxidase [58-59]. Vitellogenin mRNA and protein indicated that MCP had significant estrogenic properties and was thus a potential endocrine disruptor [60].

MCP binding affinity with hepatic microsomal cytochrome P-450 is strongly correlated with their LD50 value and has a substantial co-relationship with the cytochrome P-450 level in the liver [61]. MCP induces irreversible cataractous changes in the lens of fish (C. carpio communis). The fish visual system has many similarities to mammals and may serve as a good model for comparative toxicologic and ophthalmologic studies [62]. The order of acute toxicity and hatching success of four organophosphorus insecticides in order; chlorpyrifos > profenofos > MCP > acephate was studied in a short-term bioassay using brine shrimp, Artemia salina [63]. The extent of damage in gill was dependent on the duration of exposure. The findings revealed that inhibition in brain AChE activity and structural alteration in gill were responsible for altering the locomotor behavior of exposed fish [64]. The sensitivity of the tissue AChE was in the order

gill>brain>muscle [65]. Acute toxicity of MCP to subterranean termites, Odontotermes obesus (Rambur), has been studied by a paper contact method reveled the 88% reduction in locomotor behavior [66].
MCP had serious consequences on the performance and haematological parameters of rats [67]. MCP was highly toxic to Pristhesancus plagipennis (Walker) under both laboratory and field conditions even at low concentrations and left persistent harmful residues [68]. A higher protein level may prove efficient and significant for alleviating pesticide (MCP) toxicity [69]. MCP and 2,4-D (herbicide) on the juveniles of *Etroplus suratensis* enhanced potential of the individual toxicant for pollution in the natural bodies when they are applied sequentially and even simultaneously [70]. Studies revealed that the toxic threshold of MCP towards Daphnia magnais higher than its reported highest residue (4 micro g/L) in the ordinary aquatic environment [71].Chronic exposure of chicks to small amount of monocrptophos leads to deleterious effects on metabolism and immune system of birds [72].

Repeated oral administration of MCP in doses of 0.5 and 2.0 $mg.kg^{-1}.d^{-1}$ produced a significant reduction in the total number of protozoa (31-40%) in the rumen of buffalo calves has been observed MCP had interruption in estrous cycle [73], decrease in healthy follicles and increase in atretic follicles may be due to harmonal imbalance or toxic effects MCP inhibited monoamine oxidase activity both in vitro and in vivo in liver, kidney [74] and brain areas of albino rats [75]. MCP has significant effects on protein and carbohydrate metabolism in different tissues. As per studies the protein content was increased in liver, serum and spleen of albino rats after treatment with MCP. The protein content decreased in muscle and kidney, and overall the free sugar level decreased in all tissues. The glycogen content increased in muscle, serum and kidney after treatment with MCP, and the glycogen content and reducing sugar level decreased in liver and spleen [76].

Decomposition

Metabolic Decomposition

As organophosphates decomposition is a linear function of pH and temperature, MCP may involve in the changing process of absorption, distribution and redistribution [77]. MCP on alkaline hydrolysis yield N-methyl acetoacetamide. A study revealed that dimethyl phosphate is a metabolite of MCP that was found in all of the patient's samples in urine, even after storage at -20^0C for up to 3 years [78].
Three different metabolic reactions are involved in the initial biotransformation: N-demethylation, O-demethylation, and cleavage of the vinyl phosphate bond. The compound is completely degradable, ultimately leading to CO_2. All carbon atoms of the molecule have the potential to enter the carbon pool [89]. The main mechanism of biotransformation of MCP was hydrolysis of the P-O vinyl linkage, to give dimethyl phosphate and *N*-methylacetoacetamide as major metabolites. The mechanisms involved in the absorption, distribution, metabolism, and elimination of MCP seem to be largely species independent. In the initial biotransformation, three different metabolic reactions occur: hydroxylation of the *N*-methyl group, demethylation of the *N*-methyl group, and hydrolysis of the phosphate-vinyl linkage [80].

Metal Based

Decomposition rate of MCP was directly proportional to ozone concentration and inversely proportional to pH. The presence of carbonate and bicarbonate species in basic conditions inhibited the decomposition of MCP. The presence of Fe^{2+} and Mn^{2+} ions interfered with the decomposition of MCP in acidic solutions. The decomposition pathway of MCP was proposed and the breakage of the carbon-carbon double bond by ozonation was found to occur at first to form various nitrogen-and phosphorus-containing compounds, and subsequently decomposed to be H_2O, CO_2, NO_3^-, and PO_4^{3-} species [81-83].
Mg-doped TiO_2 decomposition of MCP was demonstrated with different Mg^{2+} concentrations by preparing the sol-gel method; it was observed that the rate of degradation of MCP over Mg-doped TiO_2 is better than pure TiO_2 and Degussa P-25. XRD, UV, SEM, FT-IR and XPS spectra demonstrated that the Mg^{2+} ions are present in the TiO_2 lattice as substitutional dopant [84]. List of insecticides by TiO2 photocatalytic mineralization of MCP was performed in the presence of TiO2. Decarboxylation was the main reaction pathways *S*-isomer of phosphate ester observed as a decomposed moiety [85].

Photocatalytic degradation of MCP was study in water by UV/TiO2 (80% anatase, 20% rutile) irradiation at different pH values, values, TiO_2 dosages, light intensities and dissolved oxygen levels. The company of oxygen inhibited the recombination of electrons and holes, enhancing the photo-degradation of MCP. Surplus of oxygen had no effect. The degradation was more efficient in acid solution than in alkaline medium. Increasing the light intensity improved the decomposition [86]. Heterogenous photocatalytic Oxidation with TiO2 catalysis for pesticide degradation in an annular slurry reactor. The pesticide (MCP) degradation was higher at low pesticide concentration and in acidic pH [87].

Environmental Effects

Organophosphates including the MCP have adverse effects on nontarget soil microorganisms and their activities [88]. It was expected to undergo *E*/*Z* isomerization and cleavage of the P-O vinyl linkage [89]. The fate of MCP in the aqueous and soil environment was examined. Hydrolysis rates for MCP are pH-dependent and follow first-order kinetics. The half-lives of MCP in pH 3 and 9 buffer solution at 25^{o}C are 131 and 26 days, respectively [90]. Hydrolysis of MCP was found only predominant on plant surfaces without any contribution from photolysis conducted a metabolism study in cotton using the *cis*- and *trans*-isomers of ^{14}C reported faster degradation of the *cis*-isomer on foliage, which would be due to its easier hydrolysis. MCP do not have any chromophore to absorb sunlight and thus direct photolysis was most unlikely. The study made use of the gas liquid chromatography (GLC) for the analysis of MCP in extracts. The result of the study shows that there is no degradation of MCP occurred in the dark while 72.8% of the applied MCP could be recovered when exposure to sunlight for eight hours is done [91].

MCP has a low environmental persistence. It does not accumulate in soil because it is biodegradable. Its half-life is less than 7 days in soil exposed to natural sunlight. The degradation of MCP in black vertisol and red alfinsol soils was rapid accounting for 96-98% of the applied quantity and followed the first-order kinetics with rate constants of 0.0753 and 0.0606 day^{-1} and half-lives ($t_{1/2}$) of 9.2 and 11.4 days, respectively. Degradation of MCP in soils proceeded by hydrolysis with formation of N-methylacetoacetamide [92]. MCP show the dissipation and leaching behavior in soil and water, a study of ^{14}C-MCP was studied for 365 days under field conditions using PVC cylinders. ^{14}C-MCP dissipated faster, up to 45% in first 90 days in columns treated with only MCP compared to 25% in columns that received MCP along with other insecticides. After 180 days of treatment, 46% radio labeled residues were observed, which reduced up to 39.6% after 365 days. Leaching of ^{14}C-MCP to 15-30 cm soil layer was observed in both the experimental setups. In the 15-30 cm soil layer of both soil columns, up to 0.19 mg ^{14}C-MCP kg^{-1}d wt. soil was detected after 270 days [93]. Study reveal about the persistence of MCP in soils at different temperatures and decay in the microbial activities in the presence of less organic substances in soils [94].MCP is unlikely to undergo photochemical reactions on soil due to lack of chromophores in their molecules [95].

MCP had significant increase in the population density of cultures of Azospirillum sp., isolated from insecticide treated soils hence exhibited greater nitrogen-fixing activity [96]. The interaction of insecticide combinations of MCP and quinalphos on Anabaena Torulosa had three types of interaction were noticed for total protein, DNA and RNA. Interestingly, the insecticide combinations at lower concentrations yielded all interaction responses for heterocyst frequency and nitrogenase activity. But, higher concentrations, in combination, resulted in synergism for heterocyst differentiation and nitrogen fixation [97].

Nitrification and phosphatase activity in two groundnut (Arachis hypogeae L.) soils was studied by taking MCP with other pesticides. The oxidation of ammonical nitrogen was significantly enhanced under the impact of selected pesticides alone and in combinations at 2.5 kg ha^{-1} in black soil, and furthermore, increase in concentration of pesticides decreased the rate of nitrification, whereas in the case of red soil, the nitrification was increased up to 5.0 kg ha^{-1} after 4 weeks, and then decline phase was started gradually from 6 to 8 weeks of incubation. The activity of phosphatase was increased in soils, which received the

MCP alone and in combination with mancozeb up to 2.5 and 5.0 kg ha(-1), whereas the application of chlorpyrifos singly and in combination with carbendazim at 2.5 kg ha^{-1} profoundly increased the phosphatase activity after 20 days of incubation, in both soils. But higher concentrations of pesticides were either innocuous or inhibitory to the phosphatase activity [98]. MCP had a direct knockdown effect on soil microarthropods Cyphoderus sp. but another collembolan species, Xenylla sp., appeared to be somewhat resistant [99].
Rhizospheric soil of soybean treated with MCP and phorate show no significant change in the total viable count of any kind of bacteria due to application of pesticides has been found showing their ability to degrade this pesticides [100]. In the cotton crop MCP had no side effect on the parasitoid pupae of T. pretiosum developing in E. kuehniella eggs [101].

Significant variations in the bacterial population were evident between the treatments in sugarcane field soil and tomato field water exposed to MCP and kinado plus, respectively. In addition, significant variations between heterotrophic bacteria, Staphlyococci and Enterococci population were also evinced in both the sugarcane and tomato fields. The dominant pesticide resistant bacteria, Staphylococcus aureus, Enterococcus faecalis and Pseudomonas aeuroginosa harboured plasmids and the resistant trait observed were found to be plasmid borne [102].

Antagonistic interactions were more pronounced toward soil microflora and dehydrogenase activity when the two (MCP or quinalphos + cypermethrin) insecticides were present together in the soil at highest level (25 + 25 micro g^{-1}), whereas synergistic or additive responses occurred at lower level with the same combination of insecticides in soil [103]. Interaction effects of the insecticides MCP and quinalphos and cypermethrin on microbial activities in two agricultural soils-black vertisol soil and red alfinsol soil were tested for 30 days under laboratory conditions, interaction responses were persistent even for 30 days [104]. Though moth larvae (Helicoverpa armigera Hübner) has resistance to most pesticides but least resistance for MCP [105]. The fungus *Aspergillus* sp. was soil fungus capable of utilizing or breaking down MCP. GC- mass spectroscopy used to detect the mechanism [106]. The bioremediation of MCP has been already tried using *Bacillus* sp. [107] several species of algae and by the soil fungus, *Penicillium corylophilum* [108]. Two microbial isolates, *Pseudomonas aeruginosa* and *Clavibacter michigenese* utilize MCP as the sole source of phosphorous [109].

CONCLUSIONS

MCP is an old product that is still very much appreciated as a low-cost and efficient insecticide. An unfavorable characteristic of MCP is its high mammalian toxicity. The objective of future projects will be to develop less toxic formulations [110]. There are number of gaps observed after the broad study of MCP, mode of bonding of acephate (H- bonding and characterization), metal based decomposition, nano-material based decomposition, method of detection of MCP, not a broad study about UV-visible, FTIR and NMR based detection of MCP, decomposition in soil, biotechnical decomposition, interaction with soil bacteria, effects of MCP and decomposed moieties of MCP (N-methyl acetoacetamide, HPO_4^{3-} and CO_2) on soil fertility and effects on plants growth etc.

Toxicity point of views MCP is considered less toxic for mammalians but its misuse, cocktailed use and decomposed product *N*-methylacetoacetamide highly toxic. Application of MCP should be restricted or avoided in the feed of birds and sandy soils with low microbial activity are more prone to MCP leaching.

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