

Impact of Methyl Parathion Organophosphate Pesticides Residues on Human Health and Environment as Toxicological Outcome

Rajesh K. Patel^{*1}, P. K. Singh¹, Rajesh Pandey², Sandeep Mishra¹, Sandeep K. Soni², Babbul Patel¹

¹Department of Chemistry, SGS Govt. PG College, Sidhi, Madhya Pradesh, India

²Department of Biochemistry, APS University, Rewa, Madhya Pradesh, India

ABSTRACT

Methyl parathion is one of the Organophosphate pesticides widely employed for crop shield against pests and also to enhance productivity of variable ranges of crops. Methyl parathion (*O, O-dimethyl O-p- nitrophenyl phosphorothioate*), is one such organophosphate pesticide. Extensive utilize of it has led to bioaddition in their surroundings. Therefore causing unsafe health hazards impacts like hang-up of *acetylcholinesterase* enzyme in neuron system concern movement, circulation, distribution. Metabolic pathway exhibited as *O-methyl-o-para* nitrophenyl phosphate, Dimethyl phosphoric acid along with *P-nitrophenol* intermediate. A direct relationship shown between infectivity of methyl parathion pesticide and their residual exposure on human, which causes the toxic impacts to humans along with high risk of contamination in environment. The present review is focused on the direct and indirect impact of methyl parathion by means of degradation through potential associated biotic system as their genotoxic, neurotoxin, carcinogenic and common health effects. Risk measured via organizations, under Food Quality Protection Act (FQPTA), for cumulative chronic hazard, distinctively food, water and residential aspects. Measures decline the exposures with protection during the application.

Article Info

Volume 6, Issue 2

Page Number: 12-23

Publication Issue :

March-April-2021

Article History

Accepted : 05 March 2021

Published : 10 March 2021

Keywords : Methyl parathion, Organophosphate, acetylcholinesterase enzyme, FQPTA, human health

I. INTRODUCTION

Methyl parathion is an organophosphate constrained insecticide. The sharp toxicological effect of methyl parathion is apprehensive to a variety health related complaint. As a result of agricultural applications with extensive exercise of methyl parathion its toxic action originates significant distress on the topic of its

occurrence in ambient air. The contours of toxicological nature of methyl parathion create a huge collection of information. In view of initial registration, in 1954, methyl parathion was employed as wide ranging insecticide severe environment related concerns arises because of widespread exercise of pesticides, from used pesticides the target resulted in contagion of soil and water bodies organisms which

are foremost environmental predicament of current era [1]. The intervallic exercise of pesticides formulates the situation principally disquieting. Inevitably term guides for accretion of pesticides with its filtrates within environment, endangering the entire inhabitants through their comprehensive toxicological effects [2]. A direct correlation established among contagion of various types of pesticides with their enduring exposure systems [3]. There is a high risk of infectivity in ecological unit with initiation of causing toxicological impacts in humans, [4]. Nearby an endure risk for consequences of squirted pesticides is typically smack contractual, with non-contractual vegetation. This causes pollution within air, soil, water along with plants [5]. Major chronic intimidation for mankind is the low quantity disclosure to pesticides which origin hormonal and DNA interruption diminish intellectual power, including reproductive irregularity [6]. Toxicity with methyl parathion organophosphate insecticide leads to cholinergic over encouragement by means of signs of toxicity including vomiting, diarrhea, sweating, convulsions, respiratory arrest etc. Reports of methyl parathion intoxication, usually seen simply in field pesticide applicators, have amplified throughout the abroad as a result of unauthorized submission of methyl parathion inside residences. The health concerns of utilize of methyl parathion have outcome in termination of its employ in most food crops in the United States [4-6].

II. Chemistry and Structure

Methyl parathion chemically *ortho*-dimethyl *ortho*-*para* nitrophenyl phosphorothioate, is an organophosphate insect killer extremely exercised in agriculture activities. Across various countries methyl parathion got proscribed due to its privileged toxicological nature in human, animals also including birds [7]. It is extremely poisonous insecticide having commercial tag name mepaton, mepatox, methyl E-605, dimethyl parathion, and methylthophos

exhibiting insect repelling action for wide variety of insect along with pests [8, 9]. Methyl parathion mixed and suspended in different insecticides as like acephate, camphechlor, carbaryl, carbophenothion, dicofol, ethylparathion, ethion, cypermethrin, lindane, methoxychlor, monocrotophos, phosal, propargite and all that. Under critical condition of scientific evaluation methyl parathion is a white crystalline substance, liquid chemical having light or dark color, pungent odour having more than 50% dynamic constituent, including xylene, with still factors.

II. METHYL PARATHION FORMULATION

Methyl parathion kills the insects and mites through interaction with stomach along with respiratory mechanism including inhalation specially. High toxic behavior of Methyl parathion expressed intended for animals concern to warm blood includes mammals and birds. Methyl parathion represents as potent inhibitors of acetyl cholinesterase enzyme, therefore nervous system and associated mechanism affected indirectly. These pesticides apply on agricultural crops by means of above ground or ground spraying tools. Methyl parathion has been identified in surface waters in addition to residues, rainwater, aquatic organisms, pulses and cereals [10].

Methyl parathion devised as innumerable and diverse commercial products. The universally existing formulation comprises a wettable powder, emulsified concentrate, dustable powder, and least volume of liquid and micro summarize product. Such insecticides is worn for controlling pests and bugs which have mouth for chewing and sucking, including weevils, aphids as well as leafhopper within wide range of crops for instance cereals, vegetables, fruit, nuts, cotton with many field crops [11].

IV. Response of methyl parathion in environment

With practical application in soil methyl parathion captures numerous months for its degradation however its practical application with higher concentration in soil, degradation takes more than a few years. In natural water the half life period of methyl parathion is approximate one month that is a protracted period to generate toxicity within living organisms in environments [12]. In soil methyl parathion has quite more portability. In swamped that means anaerobic earth, it humiliate quickly wherever half life period is nearly a week other than in aerobic hectic soil half life period got estimated about two months. Within water the half life period of methyl parathion is about three to four week [13]. Numerous chemical, physical as well as biochemical factors manipulate the distribution of methyl parathion in biotic and non biotic factors in environment. Methyl parathion acts as pollutant when it gets accompanied with air, water and soil. Disproportionate and unremitting exploit of pesticides with all the above facts being basic reason for pollution. Various dissimilar paths such as fortuitous trickle, direct purpose enduring may cause penetration of these pesticides in the environment causing pollution [14]. A series of slice like soil, water, air, micro biotic along with macro biotic organisms are enrolled in the environment. After penetration of pesticides it is followed by uninterrupted renovation starting from one element to another. Some natal events determined by the magnitude of enduring pesticides are adjudicated through which it can continue in one booth for a assured duration.

V. Methyl parathion: Metabolism Pathway

After the ingress of methyl parathion organophosphates within the body cells, it gets a part of metabolism after which it is disseminated along assorted region of body. There they may get able to harm the proper biochemical activities and thus

conclude its toxicants. Enzymatically methyl parathion renovate in their oxon outward appearance that may act in response with existing cholinesterase showing its toxic nature later [15]. Recent study and investigation accounted for metabolism array of methyl parathion to unite with acetyl cholinesterase along with, it check the aqueous detraction of acetylcholine [16]. Another review of health impacts to mankind ensuing from methyl parathion revelation delineates the metabolic pathways of methyl parathion. Passing in course of two dependent stages organophosphate experiences metabolic biochemical alteration with phase-I hepatic and phase-II advance hepatic stage [17]. Phase-I effect embraced of methyl parathion desulfuration through cytochrome P₄₅₀ [16, 17]. When methyl parathion ingress in the body cells, whichever it gets triggered. So far after oxidation it structured methyl *p*-oxon, tremendously which is extra intoxicating [18-19]. Therefore after completion of desulfuration oxidation, methyl parathion organophosphate by hydrolysis converted to *p*-nitrophenol along with one different metabolites [16, 20]. Thus, it clarifies that P₁₅=S₁₆ derivative compounds are transformed to P₁₅=O₈ form which is strong inhibitor for acetyl cholinesterase.

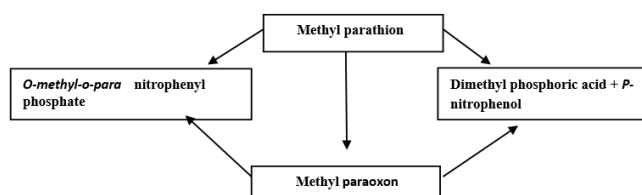


Figure 1. Methyl parathion metabolic pathway

VI. Methyl parathion: Transport and Circulation

Transport and Circulation of methyl parathion organophosphates get throughout the atmosphere within diversified pathways. Methyl parathion after pertaining like insecticide or pesticide, it principally degrades through photolysis or by biological breakdown carried out by microorganisms in deposited aqueous medium [21, 22]. Rain water, wind,

soil be the most important carrier for transport of methyl parathion organophosphate to adjoining regions [21]. The deprivation of organophosphates can be found out through preferred chemical agents along with other chemicals which were bared to different water bodies under environmental conditions [21]. Recent research informed that humilation of methyl parathion be prejudiced by microbial deprivation, aqueous hydrolysis, under alkaline condition. For methyl parathion degradation half life period and photolysis invariable are the some significant issue affecting biochemical activity. Since hydrolysis of methyl parathion occur rapidly, by the reason of physical and chemical temperament its properties are somewhat indifferent, it never relocate throughout the food chain of environment [22]. Methyl parathion may have an environmental half life period from some days to week if it is practiced accurately within farming background where it interpreted to water and harmful ultraviolet radiation. Conversely, when is applied within homes, its half-life amplify significantly [23]. Some of the analytical studies also conclude the end product with relative humidity, temperature and reactivity on methyl parathion and shown that the half-live period for methyl parathion may be two to three months at 0°C, at 20°C it may be more than one week and from 20-28 hours at 40°C. Across ecological aspects together with chemical and biological issues, it terminates the collapse of methyl parathion organophosphate to frequently affected environmental and ecological factors [24].

VII. Methyl parathion residues toxicity in human

Methyl parathion exhibits the high oral and moderate dermal toxicity with a half life of 14 to 21 days. The toxicologically relevant mode of action is the inhibition of choline esterase activities and also various clinical effects occur due to organophosphate poisoning in human. Methyl parathion is lipid soluble and it can penetrate in skin also. It also enters the

body through the respiratory and gastrointestinal tracts and when absorbed through alimentary canal, it is stored in adipose tissue. Its primary mechanism for toxicity is its slow release into the bloodstream and subsequently to the nervous system [25]. Methyl parathion is classed as an extremely hazardous pesticide with a rodent LD50 of 6 to 24 mg/kg. It has been banned in numerous countries, but there are few reports of acute methyl parathion poisoning. Plasma cholinesterase and acetyl cholinesterase were measured in blood. Methyl parathion and the major metabolite 4-nitrophenol where measured in serum as well as urine also. Based on accessible concentration data, the pharmacokinetic constraints of methyl parathion were calculated approximately intended for patient. Results under the human male, which was ingested (12 to 24g) 50 to 100mL concentration of methyl parathion causing delayed repression of acetyl cholinesterase [26]. Various investigations exposed that methyl parathion organophosphate is a source of damage and alteration in DNA with uncontrolled strain in different organ systems. Potential of acute exposure to organophosphorus and organochlorine pesticide were also documented in terms of mutation similarly the bioassay of methyl parathion for possible carcinogenicity [27, 28].

Human contact so premeditated like riveted dosage. As the ratio of breathing rate with respect to body weight changes from group to group so that the riveted dosage may be different among infants, children, teenager females, and males per unit of body weight. This is the reason why human contact estimation is differentiated in four groups. Among all four group infants whose age is less than six to eight months usually they have ceiling exposure outstanding because of the premier breathing rate when come contact of methyl parathion exposure [29].

VIII. Genotoxic and Neurotoxin Impacts

Current researchers observed that methyl parathion shows genotoxic effects in vitro and in vivo resulting gene alterations in different microorganisms such as bacteria, mammalian cells, sister chromatid exchange with activist consequence within sexual decisiveness in *Drosophila*. Being able of binding acetyl cholinesterase to methyl parathion it has to prevent the hydrolysis of acetylcholine. Toxicological behavior within nervous system is associated to high rank disclosure of methyl parathion and neuro degenerative diseases. Methyl parathion in vitro, illustrated direct binding to the sugar unit of DNA [30]. In path of resolving chromosomal impacts coupled by means of methyl parathion revelation. The involvement of insect repellent publicity causes Parkinson's syndrome with one to seven creases up in hazard [31]. Among the statistics collected through controller along with experimental group no noteworthy dissimilarity was analysed [32]. The considerable enhance to regularity of constant chromosomal abnormality within patients intensely showing to organophosphate [33]. The regularity resolutions of male sperm aneuploidy in addition to their interaction by way of organophosphate urinary metabolites within farming human resources were observed. Analysis incorporated dimethyl dithiophosphate, dimethyl phosphate, diethyl phosphate, dimethyl thiophosphate and dimethyl thiophosphate [32, 33].

Chemical outcomes coupled with neurons or nervous system because of frequent exposure of methyl parathion within mature and neonatal rats was premeditated in order to resolve some growth related results intended for methyl parathion. Every day for about weeks the organophosphate chemicals was over seeded in neonatal and mature rats within neuro chemical endpoints, cholinesterase reticence and receptor binding in anterior cortex then deliberated at instant during initial calendar day's hours of

sunlight [34]. The connection involving extent of ChE inhibition with decrease of cortical muscarinic binding receptor move toward between the mature groups by means of high embraced diminution illustrious to neonates as conflicting into adults at available stage of ChE reticence [34]. The muscarinic binding receptor along with ChE action is further reduced within neonatal regions of brain at the same time as divergent action stage was deliberated in fully developed brain expanse with recurring contact.

IX. Common health effects

Headaches, vomiting, night wakeup are some Common health sign for toxicological impacts of methyl parathion. With its overexposure it can cause sleeplessness, diarrhea, impatience, complexity in inhalation, wooziness, abdominal contraction, high amount of sweating, excessive saliva loss, psychological mystification in addition with cardiovascular disorder. [35]. Recent articles and investigations shown that respiratory disorder, cardiovascular disorder, swelling in liver, pulmonary artery blockage, cardiovascular tissues weakness, acute nephritis in kidney, hepatic laceration and different other kidney related impacts caused due to gulp of air having of methyl parathion. Blockage of the esophageal region and internal bleeding within petechial are also related due to exposure of toxicological chemical, *methyl parathion organophosphate* [36].

X. Carcinogenic Effects

Although reviewed research has indicated that there is no evidence that methyl parathion causes cancer to individuals regularly exposed to the chemical, other studies have shown that carcinogenic endpoints have been observed in rats [37]. Methyl parathion was administered to F344 rats and B6C3F1 mice in feed intended for aim of determining whether or not any impacts of carcinogenic. In starting group members

were given about hundred mg per kilogram of methyl parathion. Afterward doses were reduced as an outcome of declining average body weight. There was not a considerable raise in the occasion of tumors moreover in rats in assessment to organize groups. Therefore it is accomplished that methyl parathion is not carcinogenic for any F344 rats or B6C3F1 mice [38]. Although it has been concluded that methyl parathion is not cancer causing to animals and mankind, a assemblage of identified adverse effects of chemicals used in agricultural activities was finished and studies the links among facsimile and physical condition impacts of these chemicals [39] Various study revealed that low level chronic disclosure to methyl parathion from early years may later cause cancer development [40].

XI. Methyl Parathion absorption and distribution

Methyl Parathion absorption and distribution transpire in the course of gastrointestinal zone, the respiratory zone along with cell membranes [41, 42]. Earlier reviews specified that crucial path of methyl parathion exposure is the skin incorporation; also this revelation occurs as a result of industrial or agriculture practices. Direct or indirect contact with methyl parathion caused due to breathing and making contact with unhygienic stuff. Disclosure of this insecticide can be calculated within blood stream, urine samples, saliva and amniotic fluid. The entire sample is used to analyze the degree of disclosure to methyl parathion or such insecticides [42]. Once the body cells get exposed to methyl parathion, high amount of absorbed chemical was analyzed in adipose tissue, huge concentration observed in hepatic cells along with kidney after long time exposure [43]. Researches be evidence that particular dose of 10 milligram per kilogram of methyl parathion in dermal tissues with maximum concentrations of methyl parathion in adipose tissue 67,532 µg per gram, in kidney 1571 µg per gram, in spleen 1004 µg per gram, in heart 729 µg per gram, in liver 706 µg per gram, in

brain 546 µg per gram [42, 43]. The investigation also indicate that methyl parathion organophosphate participate in basic mechanism toxicity passing through its accumulation in adipose tissue where it discharge into body circulatory system. Subsequently it passes into nervous system that possibly responsible for toxicological impacts.

XII. Methyl parathion: Risk Assessment

Different organization reflect on assistance for risk assessment from different disclosure under Food Quality Protection Act, for cumulative chronic hazard, distinctively food, water, and residential. There is no any registered accommodate application of methyl parathion. This is the reason only for its exposure through food and water consumption was considered in the methyl parathion risk assessment. Therefore, the comprehensive risks are equivalent to those activities that cause excessive exposure of methyl parathion. Methyl parathion is prohibited pesticide that should be practiced by expert applicators, residential and farmers only as its revelation may occur through squirting flow at time of appliance of methyl parathion in farming area. Always squirting flow has a possible source of revelation to occupants thereby during spraying maneuver. Predominantly this is the case with airborne function, to a less significant extent, could also be a prospective resource of contact through basic applicable method. The agencies with spray drift task force working with local offices and state agencies in order to control the standard pesticide regulation system with significant manner. Third parties also develop the paramount squirt flow management with observation. Under this policy the agency can enforce additional refinements for spray drift management practices to reduce and risks connected to airborne as well as other submission [44].

XIII. Measures to Reduce Exposures with Protection during Application

WHO in order to reduce direct and indirect counsel to facilitate for health and safety measures intended for employees in addition to the common inhabitants, the conductance as well as relevance of methyl parathion be supposed to hand over barely to proficiently administered and tractable applicators, which should trail satisfactory safety events with application of the chemical contents in insecticides as per superior practices [45, 46]. Workers could practice all necessary clothing counting a respirator. During application of pesticides in stifling climatic conditions it require strategy for delicate security [47]. Respirator must be used by blender at the time of squirting major harvesting products. At that time we should pass up the flaggers. After use every part of

protective equipment along with cloths has to be washed meticulously.

The application practices on agricultural employ and discarding of methyl parathion should be cautiously managed to minimize infectivity of the surroundings. To diminish risks for all individuals, a 48-hour gap stuck between re-entry in addition to spraying keen on some sprayed part is recommended. The findings suggested with good work practices, measures of hygienic some safety precautions; methyl parathion is differently to in attendance a danger for those occupationally interpretation. DNAs evaluating the methyl parathion in a some country will require to believe whether the necessary preventative measures can be ensured in the country as part of risk evaluation of the utilize of the methyl parathion products marketed as various formulations subject to guidance and technical health related support [46-48].

Table 1. Analytical method and their detection limit

S. No.	Sample	Analytical method	Detection limit	Ref.
1.	Marine sediment	Combined capillary column gas		[49]
2.	Water	Dispersive liquid liquid micro extraction gas chromatography with electron capture detector	0.083 ppb	[50]
3.	Rice	Headspace solid phase micro extraction gas chromatography mass spectrometry	0.026 ppb	[51]
4.	Mango	High performance liquid chromatography	0.005 ppm	[52]
5.	Gourd	Gas chromatography	0.1 ppm	[53]

XIV. Regulation with technical support, suggestion and Recommendation

Regulatory technical suggestion and recommendation that build up guidelines for lethal matters are formulated by United States Federal Agencies,

Environmental Protection Agency, Food and Drug Administration and the Occupational Safety and Health Administration. In order to care for community fitness from the injurious smash up through toxic natured chemical complexes, implementation in dogmatic course of action had been brought through Safety and Health

Administration Bureau. According to the federal insecticide, fungicide, and rodenticides act each insect repellent means insecticides or pesticides merchandise produced in the United States of America be obliged to be off the record either for common use or constrained exploit. When any insecticide or pesticide is used as per instructions, generally it does not cause harmful and diverse impacts in ecological factors and no cautions will originate in the environment and environmental factors. Conversely generally the application of constrained chemical pesticide or insecticides devoid of supplementary supervisory body may cause bad-tempered impacts in the environment. After 1978, methyl parathion organophosphate has been prohibited in different nations considering it a restricted use pesticide for the reason that it has a large number of side effects on organisms having contact of such chemicals [54, 55].

According to EPA's water confines it comprises (for children with 1-10 days of contact 0.3 mg/L), (for children more than 10 days contact of 0.03 mg/L and for adults with lifetime contact 0.002 mg/L) [54]. EPA established charitable extinction of organophosphate methyl parathion relevance on confident stuff for dietetic use as well as non dietetic uses. Across different nations application of methyl parathion is prohibited for some agricultural crops and certain food stuff for within emergent period. In different crops and fruits like carrots, tomatoes, moist beans, moist peas, cauliflower, spinach, turnips and in decorative plants or grasses, EPA allocates maximum usage of 1-2 ppm of insecticides or methyl parathion in crops which are officially standardized for methyl parathion application [54].

A large number of confrontation due to experience of methyl parathion happened because of industrial or working during in farms exposure. Along with Occupational Safety and Health management, American Conference of Industrial Hygienist

prescribed tolerable disclosure limit, it is considered as authentic stricture, its entrance boundary that suggested are restriction with scientific data and research [56, 57]. At primary level, Occupational Safety and Health management hadn't defined any tolerable disclosure limit for methyl parathion. In case of methyl parathion the standard entrance limit is about 0.2 mgm⁻³ [73]. On behalf of an equivalence of methyl parathion with parathion of 0.2 mgm⁻³ Occupational Safety and Health management had later suggested a ultimate tolerable disclosure limit [74]. Workers or farmers within place of work be supposed not to be uncovered in tolerable disclosure limit exceeding 0.2 mgm⁻³ of organophosphate methyl parathion [58]. Human beings are uncovered to methyl parathion due to airborne disclosure or through dietary sources. The adequate day by day inhalation or exposure limit has been decided by the WHO which is 0.003 mgkg⁻¹ of body mass. On behalf of unsympathetic impact level which is 5mgkg⁻¹ WHO has ascertained regular inhalation or exposure limit [55-58].

III. CONCLUSION

In view of present review, the indiscriminate employ of pesticides methyl parathion is generating problems related to environment and human health. Major toxicological outcome of methyl parathion organophosphate pesticides residues demonstrated that those dietary products which have been come in contact with methyl parathion drastically increases to causes of direct and indirect toxicities to humans and environment. Consumption of these foods products containing relatively residues causes serious health issues to biotic system via transport, absorption, distribution after and during the application. Some analytical tools and their detection limit are suggested with technical support, regulation and appropriate recommendation, strict rules obliged to be declared by local and international establishment in favor of

consumers therefore decline the chances of exposures in terms of protection during application.

IV. REFERENCES

- [1]. United States Environmental Protection Agency. Interim registration eligibility decision Facts: Methyl parathion. Washington, DC, USA. 1986; EPA-738-F-03-005.
- [2]. Liu YH. Chung YC and Xiong Y. Purification and characterization of a dimethoate-degrading enzyme of *Aspergillus niger* ZHY256, isolated from sewage. *Applied and Environmental Microbiology*, 2001; 67(8):3746-3749.
- [3]. Calderbank A., The occurrence and significance of bound pesticide residues in soil. *Reviews of Environmental Contamination and Toxicology*, 1989; 108:71-103.
- [4]. Veiga, MM, Silva DM., Veiga LBE., and de Castro Faria MV., Pesticide pollution in water systems in a small rural community in Southeast Brazil. *Cadernos de Saude Publication*, 2006; 22(11): 2391-2399.
- [5]. Johnson J. and Ware WG., *Pesticide Litigation Manual*. 1992 Edition. New York: Clark Boardman Callaghan Environmental Law Series. 1992.
- [6]. Gupta, PK., Pesticide exposure Indian scene. *Toxicology*, 2004; 198 (1-3): 83-90.
- [7]. Sharmila M., Ramanand K. and Sethunathan N., Hydrolysis of methyl parathion in a flooded soil. *Bulletin of Environmental Contamination and Toxicology*, 1989; 43:45-51.
- [8]. Fao.org Internet]., Decision Guidance Document: Methyl Parathion. 1997.
- [9]. Orme S. and Kegley S., *Methyl Parathion (PAN) Pesticides Database* San Francisco: Pesticide Action Network. 2006.
- [10]. Kidd H. and James, DR., *The Agrochemicals Handbook*. 3rd edition. Cambridge: Royal Society of Chemistry Information services. 1991.
- [11]. Ortiz Hernandez, ML., Monterrosas BM., Yanez OG. and Sanchez SE., Biodegradation of Methyl Parathion by Bacteria Isolated From Agricultural Soil Revolution of International Contamination., 2001; 17(3):147-155.
- [12]. Pritchard PH., Cripe CR. and Walker WW., Biotic and abiotic dehydration rates of methyl parathion in freshwater and estuarine water and sediment samples. *Chemosphere*. 1987; 16:1509-1520.
- [13]. Castilh JA., Fenzl N., Guillen SM. and Nascimento FS., Organochlorine and organophosphorus pesticide residues in the Atoya river basin, Chinandega, Nicaragua. *Environment Pollution*. 2000;110: 523-533.
- [14]. Ortiz D., Yanez L., Gomez H., Martinez SJA. and Diaz-BF., Acute toxicological effects in rats treated with a mixture of commercially formulated products containing methyl parathion and permethrin. *Ecotoxicology Environment Safe Journal*. 1995; 32:154-158.
- [15]. Mendola P., Use of biomarkers to indicate exposure of children to organophosphate pesticides: Implications for a longitudinal study of children's environmental health. *Environmental Health Perspectives*, 2003;111:1939-1946.
- [16]. Garcia SJ., Abu QAW., Meeker-O'Connel WA., Borton AJ., Abou-Donia MB., Methyl parathion: A review of health effects. *Journal of Toxicology and Environmental Health*. 2003;6:285-210.
- [17]. Abu-Qare AW. Abdel Rahman AA., Kishk AM. and Abou-Donia MB., Placental transfer and pharmacokinetics of a single dermal dose of ¹⁴Cmethyl parathion in rats. *Toxicological Sciences*. 2000; 53:5-12.
- [18]. Chambers JE. and Carr RL., Inhibition patterns of brain acetylcholinesterase and hepatic and plasma aliesterases following exposure to the phosphorothionate insecticides and their oxons

- in rats. *Fundamental and Applied Toxicology*. 1993; 21: 111-119.
- [19]. Hollingworth RM., Metcalf R. L., Fukuto TR., The selectivity of sumithion compared with methyl parathion. *Metabolism in the white mouse. Journal of Agricultural and Food Chemistry*, 1967; 15: 242-249.
- [20]. Abu-Qare AW., Abou-Donia MB., Urinary excretion of metabolites following a single dermal dose of ¹⁴Cmethyl parathion in pregnant rats. *Toxicology*. 2000; 150: 119-127.
- [21]. Sakellarides TM., Siskos MG., Albanis TA., Photodegradation of selected organophosphorus insecticides under sunlight in different natural waters and soils. *International Journal of Environmental Analytical Chemistry*. 2002; 83(1): 33-50.
- [22]. World Health Organization (WHO). Methyl parathion: Environmental health criteria. Geneva, World Health Organization. NLM Classification: WA 240. 1993;145.
- [23]. Barr DB., Turner WE., DiPietro E., McClure PC., Baker SE., Barr JR., ehle K., Grissom Jr RE., Bravo R., Driskell WJ., Patterson Jr DG., Hill Jr RH., Needham LL., Pirkle JL. and Sampson EJ., Measurement of p-nitrophenol in the urine of residents whose homes were contaminated with methyl parathion. *Environmental Health Perspective*. 2002; 110(6):1085-1091.
- [24]. Athanasopoulos PE., Kyriakidis NV. and Stavropoulo P., A study of the environmental degradation of pesticides azinphos methyl and parathion methyl. *Journal of Environmental Science Health*. 2004; 39(2): 297-309.
- [25]. Bradman A. Barr DB., Claus Henn BG., Brumheller T., Curry C. and Eskenazi B., Measurement of pesticides and other toxicants in amniotic fluid as a potential biomarker of prenatal exposure: A validation study. *Environmental Health Perspective*. 2003; 111: 1779-1782
- [26]. Geoffrey KI., Katie M., Lena EF., Mary H., Enda O., Connor RP., Manel A., Michael E., Human methyl parathion poisoning *Clinical Toxicology*. 2007; 45(8):956-960.
- [27]. Vijayaraghavan M. and Nagarajan B., Mutagenic potential of acute exposure to organophosphorus and organochlorine compounds. *Mutation Research archive*. 1994; 321(1-2): 103-111.
- [28]. National Cancer Institute., Bioassay of methyl parathion for possible carcinogenicity. Technical Report Series No. 157. Washington, DC, USA: U.S. Department of Health, Education, and Welfare, Public Health Service. 1979.
- [29]. International Agency for Research on Cancer. Methyl parathion: Summary of data reported 1983; 30.
- [30]. Bradman A., Barr DB., Claus Henn BG., Brumheller T., Curry C. and Eskenazi B., Measurement of pesticides and other toxicants in amniotic fluid as a potential biomarker of prenatal exposure: A validation study. *Environmental Health Perspectives*. 2003; 111:1779-1782.
- [31]. Barr DB., Measurement of organophosphate metabolites in postpartum meconium as a potential biomarker of prenatal exposure: A validation study. *Environmental Health Perspectives*. 2001; 109: 417-420.
- [32]. Van BT., Bzabo I., Ruzicska P. and Czeizel R., Chromosome aberrations in patients suffering acute organic phosphate insecticide intoxication. *Human genetics*. 1974; 24: 33-57.
- [33]. Cebrian ME., Organophosphorus pesticide exposure increases the frequency of sperm sex null aneuploidy. *Environmental Health Perspectives*. 2001; 109: 1237-1240.
- [34]. Liu J., Olivier K. and Pope CN., Comparative neurochemical effects of repeated methyl parathion or chlorpyrifos exposures in neonatal

- and adult rats. *Toxicology and Applied Pharmacology*. 1999; 158(2): 186-196.
- [35]. Rubin C., Esteban E., Kieszak S., Hill RH., Dunlop B., Yacovac R., Trottier J., Boylan K., Tomasewski T. and Pearce K., Assessment of human exposure and human health effects after indoor application of methyl parathion in Lorain County, Ohio, 1995-1996. *Environmental Health Perspectives*. 2002; 110(6):1047-1051
- [36]. Fazekas GI., Macroscopic and microscopic changes in Wofatox (methyl parathion) poisoning. *Zeitschrift fur Rechtsmedizin (Journal of Legal Medicine)*. 1971; 68:189-194.
- [37]. Agency for Toxic Substances and Disease Registry (ATSDR)., Toxicological profile for methyl parathion. Atlanta, Georgia, USA, September. 2001.
- [38]. National Cancer Institute. Bioassay of methyl parathion for possible carcinogenicity. Technical Report Series No. 157. Washington, DC, USA: U.S. Department of Health, Education, and Welfare, Public Health Service. 1979.
- [39]. International Agency for Research on Cancer. Methyl parathion: Summary of data reported. 1983.
- [40]. Tourmaa TE., Adverse effects of agrochemicals on reproduction and health: A brief review from the literature. *Journal of Nutritional and Environmental Medicine*. 1995; 5(4):353-366.
- [41]. Garcia SJ., Abu-Qare AW., Meeker-O Connel WA., Borton AJ. and Abou-Donia MB., Methyl parathion: A review of health effects. *Journal of Toxicology and Environmental Health*. 2003; 6:285-210.
- [42]. Zhu H., Rockhold RW., Baker RE., Kramer RE., Effects of single or repeated dermal exposure to methyl parathion on behavior and blood cholinesterase activity in rats. *Journal of Biomedical Science*. 2001; 8:467-474.
- [43]. Kramer RE., Wellman SE., Rockhold RW. and Baker RC., Pharmacokinetics of methyl parathion: A comparison following single intravenous, oral or dermal administration. *Journal of Biomedical Science*. 2002; 9:311-320.
- [44]. US Environmental Protection Agency office of pesticide programs case no. 0153. 2006; 1-89.
- [45]. IPCS. Environmental health criteria No. 63: Organophosphorous insecticides: a general introduction. International Programme on Chemical Safety, IPCS/ World Health Organization, Geneva. 1986.
- [46]. IPCS. Environmental health criteria No. 145: methyl parathion. International Programme on Chemical Safety, IPCS/ World Health Organization, Geneva. 1993.
- [47]. FAO. Guidelines for personal protection when working with pesticides in tropical countries. Food and Agriculture Organization, Rome. 1990.
- [48]. WHO. Recommended classification of pesticides by hazard and guidelines to classification 1996-1997. WHO/PCS/96.3. World Health Organization, IPCS, Geneva. 1996.
- [49]. Nielsen PG., Quantitative analysis of parathion and parathion-methyl by combined capillary column gas chromatography negative ion chemical ionization mass spectrometry. *Journal of Biological Mass Spectrometry*. 1985; 12(12): 695-698.
- [50]. Pimenta G., de Queiroz M., Victor R., Noronha L., Neves A., de Oliveira A. and Heleno F., DLLME-GC/ECD Method for the Residual Analysis of Parathion-Methyl and its Application in the Study of the UV-Photodegradation Process. *Journal of the Brazilian Chemical Society*. 2017.
- [51]. Da Silva DF., Paiva Silva FE., Silva FGS., Nunes GS., and Badea M., Direct determination of methyl parathion insecticide in rice samples by headspace solid-phase microextraction-gas

- chromatography-mass spectrometry. *Pest Management Science*. 2014; 71(11): 1497–1502.
- [52]. Rao Ch. R. and Arun KL., Qualitative and Quantitative Analysis of Fenvalerate, and Methyl Parathion pesticides in mango and grapes collected by HPLC method. 2012.
- [53]. Mishra S., Thakur LK., and Richhariya N., Gas Chromatographic Determination of 23 Organophosphorus Pesticides Residue in Bottle Guard Matrix. *International Journal of Pharmacy and Pharmaceutical Sciences*. 2018; 10(1): 53-59.
- [54]. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for methyl parathion. Atlanta, Georgia, USA.2001.
- [55]. Agency for Toxic Substance and Disease Registry (ATSDR). Toxicological profile information sheet. Atlanta, GA, USA.8. Available <http://www.atsdr.cdc.gov/toppro2.html>. 2004.
- [56]. Occupational Safety and Health Administration (OSHA). Sampling and analytical methods. 1987.
- [57]. World Health Organization (WHO). Methyl parathion in drinking-water: Background document for development of WHO Guidelines for drinking water quality. WHO/SDE/WSH/03.04/106. 2004.
- [58]. National Institute for Occupational Safety and Health (NIOSH). Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention Cincinnati, OH. 2001.

Cite this article as :

Rajesh K. Patel, P. K. Singh, Rajesh Pandey, Sandeep Mishra, Sandeep K. Soni, Babbulal Patel, "Impact of Methyl Parathion Organophosphate Pesticides Residues on Human Health and Environment as Toxicological Outcome", *International Journal of Scientific Research in Chemistry (IJSRCH)*, ISSN : 2456-8457, Volume 6 Issue 1, pp. 12-23, March-April 2021.
URL : <https://ijsrch.com/IJSRCH21623>