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# Ecotoxicological effects of Methyl parathion on living things and environment

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**Methyl parathion; is an insecticide which is commonly used in our country in the aim of agriculture and it is known that some environmental problems arise due to the chronic usage of this pesticide. For this reason, it is very important to raise the awareness the public in terms of the risk factors because of the usage of methyl parathion. The researches showed that methyl parathion inhibits cholinesterase enzyme in the human brains, cause genotoxic effect in mammalian somatic cells in culture medium and increase the anomalies in chromosomal structure. It was determined that this insecticide causes a decrease in the body weights of experimental animals and an increase in the weight of liver, cellular damages in the livers, various neurological problems by affecting reproduction, nervous and cardiovascular systems. Since methyl parathion causes the formation of a toxic medium for human beings, birds, fresh water fish, other hydrophilous organisms as well as sea organisms by mixing to air, soil and water, it is well known that the usage of methyl parathion as an insecticide is a threatening factor for the ecosystem. The usage of methyl parathion has been forbidden in EU (European Union) countries since 2003 and some restrictions have been applied in United States of America. There is no limiting or forbidding enforcement related with the usage of methyl parathion which is in the 3<sup>rd</sup> order among commonly used pesticides in our country. To bring new arrangements for the usage of methyl parathion which constitutes various risk factors in terms of ecological factors may minimize the ecotoxicological effects of this pesticide.**

**Key words:** Methyl parathion, insecticide, ecotoxicological effect.

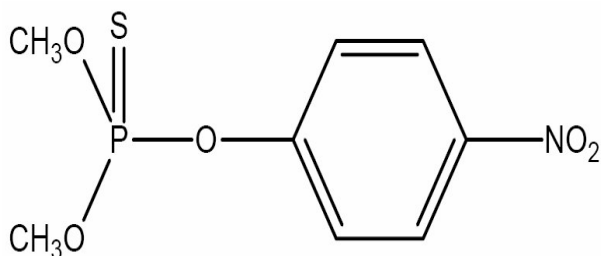
## INTRODUCTION

Methyl parathion (*O,O*-dimethyl *O*-4-nitrophenyl phosphorothioate) has a wide usage of area for the aim of agriculture. Methyl parathion which is an acaricide and an insecticide with organophosphate was firstly introduced to the market in United States of America in 1952 (Ma et al., 2003; ATSDR, 1990) and although it is commonly used in production of cotton, it is also used in order to kill the insects in different products such as corn, apple, bean, rice, wheat, peach, clover and sunflower

(United States Geological Survey, 1997; Ruckart et al., 2004; Uzunhisarcikli, 2008). Since the cost of this pesticide is low and its effect is high, it can be used commonly (Garcia et al., 2003).

Toxic effects of compounds with organophosphor may come out in muscarinic actions such as salivary, sweat, tear and stenosis in bronchus and excess secretion, nausea, vomiting, diarrhea, abdominal pains as cramps, urinary and stools incontinence and excess slowdown in heartbeats, in nicotinic actions emerging with muscle contraction and twitches and causing paralysis, central nervous system actions such as excess discontent, nervousness, dizziness, prostration, dullness, decline in memory, depression, sudden and uncontrolled motions, contractions, coma and respiratory standstill. Moreover, it

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**Figure 1.** Chemical structure of Methyl parathion.

is also known that organophosphates cause the destruction of membrane structures of lysosomes in lens fibers and result in immunotoxicity by inhibiting the lens fiber secretion (Tafari and Roberts, 1987; Sharma and Reddy, 1987; Taylor, 1996; Lefkowitz et al., 1996; Tuovinen, 2004).

The insecticides with organophosphates which also includes methyl parathion indicate their possible effects in target tissues as inhibiting acetylcholinesterase (AChE) enzyme by binding it which provides the destruction of acetylcholin in synapse regions. As a result, they cause acute or chronic poisonings which have no recycling in living things (Kappers et al., 2001; Abu-Qare and Abou-Donia, 2001). It was determined that parathion in the structure of parathion methyl similarly inhibits acetylcholinesterase after metabolising paraoxon (Vural, 2005). Methyl paraoxon makes acetylcholinesterase enzyme inactive by phosphorylating the active region of it. Although its reversible reaction is possible in a few hours after chemical binding of enzyme with methyl paraoxon, irreversible chemical reactions occur with quite strong covalent bonds after 24 - 48 h. The researches showed that alkyl side chain in phosphate group of acetylcholinesterase enzyme is removed and a hydroxyl group is bound instead of it. It was determined that re-activity of acetylcholinesterase enzyme having such a variation is impossible (Dalkılıç, 2006). Other studies favor these findings and it was determined that the plasma, erythrocyte and brain AChE activities in rats exposed to organophosphate insecticides were inhibited (De Blaquiére et al., 2000; Yano et al., 2000; John et al., 2001; Sun et al., 2003; Hazarika et al., 2003; Uzunhisarcikli, 2008).

Approximately 3 millions of poisoning events and 200,000 death events occur every year depending on organophosphates used for the aim of agriculture (Kwong, 2002). It is a known fact that the wrong usage of a pesticide named Folidol which includes methyl parathion as an active material and is used for killing parasites such as phthirus in our country causes many poisonings resulting in deaths. Similarly, consumption of vegetables-fruits and other foods including pesticide residuals having methyl parathion causes many acute and chronic poisonings. The usage of medicines including methyl parathion was forbidden in European Union (EU)

countries in 2003 and some restrictions were applied for its usage in United States of America. On the other hand, methyl parathion which is permitted to be used with licence in our country is the third commonly used pesticide after methamidophos and chlorpyrifos-ethyl according to the data of Ministry of Agriculture and Rural Affairs between 1999 and 2002 (Delen et al., 2005). The possible ecological effects of methyl parathion which has still been commonly used in our country for various aims are not known well. In this study, it was aimed to investigate the environmental and ecological problems that methyl parathion was estimated to cause.

## METHOD OF THE RESEARCH

For the analysis of possible environmental and ecological problems depending on the usage of methyl parathion which has been commonly used against agricultural pests and house insects all over the world, investigation of the experimental studies and literatural information about methyl parathion was searched.

## Chemical properties of methyl parathion

The molecular formula of methyl parathion is  $C_8H_{10}NO_5PS$  and its molecular weight is 263.21. Its commonly used names are parathion-methyl, methyl parathion and metaphos. The chemical structure of methyl parathion is given in Figure 1. Methyl parathion is found as colorless crystals at room temperature in its pure form and has the ability to dissolve in many organic solvents rapidly. Its melting point is 35 - 36°C and it hydrolyzes rapidly in alkaline mediums and is divided into its structural isomers depending on the heat intake. Methyl parathion can be found as emulsions, as powder and liquid forms that can dissolve in water. It was first synthesized in 1949 and it was first introduced to the market commercially in United States of America in 1952. The common commercial names of methyl parathion which is sold in different concentrations and forms as commercially are known as Bladan M, Cekumethion, Dalf, Dimethyl parathion, Devithion, E 601, Folidol M, Fosfero M50, Gearphos, Kilex parathion, Metacide, Metaphos, Metron, Nitrox 80, Partron M, Penncap-M, Tekwaisa, Dithon 63, Ketokil 52, Seis-tres 63, Metaspray 5E and Paraspray 6 - 3 (Yıldırım, 2006).

## Pharmacokinetics of methyl parathion

Methyl parathion is a toxic material having the property of high toxicity according to the classification of U.S. Environmental Protection Agency (EPA) and high solubility property in lipids. For this reason, it can be absorbed rapidly by skin, respiration and digestion in human beings. Most of methyl parathion is discharged with urinary and some of it is discharged with stools (Yıldırım, 2006). Since it is discharged as p-nitrophenol in the urinary, p-nitrophenol is also examined in both blood and urinary as well as AChE activity in the situation of parathion poisoning (Vural, 2005). The methyl parathion which is inhaled into the body is completely and rapidly absorbed by gastrointestinal system. Methyl parathion which is taken in 50 mg/kg dosage orally by rats is determined in blood plasma of rats after 6 - 8 min following the application and after 90 s following i.v. application, it was determined in brain tissue of rats (Yamamoto et al., 1983). Da Silva et al. (1993) applied Folidol 600 including methyl parathion to *Callichthys callichthys* which is a kind of fish and it was observed that the plasma cholinesterase enzyme (ChE) activity in the fish was inhibited in the rate of 90% right after

**Table 1.** Most commonly consumed insecticides in Turkey between 1999 and 2002, Their Acute Oral LD<sub>50</sub> values Their rates in insecticide consumption (Ware, 1994; Delen et al., 2005).

Insecticides	LD <sub>50</sub> values (mg/kg)*	The rates of insecticide consumption by years (%)			
		1999	2000	2001	2002
Methamidophos	13	19.35	17.24	12.99	14.52
Chlorpyrifos-ethyl	135	13.72	14.09	31.94	12.76
Parathion-methyl	9	10.95	12.97	9.81	10.96
Dichlorvos (DDVP)	25	7.72	10.22	8.91	8.08
Endosulphan	18	7.20	-	6.14	-
Carbaryl	307	-	5.80	-	-
Azinphos-methyl	5	-	-	-	7.08
TOTAL		58.94	60.32	69.79	53.40

4 h following the application and this inhibition was continued for 4 days .

Liver is the first organ where methyl parathion is metabolized and detoxified as a result of poisoning, a death case was observed after tachycardia, dizziness, headache, heart failure (WHO, 1993). In another study which was carried out with chickens exposed to 14% LD<sub>50</sub> dosage of methyl parathion, it was informed that the insecticide was rapidly absorbed by gastrointestinal way. After eight hours following the application, it was determined that methyl parathion was spread to plasma, liver, kidney, brain and gastrointestinal tissues and methyl parathion and its metabolite were present in both liver and kidney (Abu-Qare and Abou-Donia, 2001). The studies showed that four different mechanisms have a role in metabolism of methyl parathion. These are oxidation, hydrolysis, dearilization and dealkylation. It was determined that desmethyl methyl parathion was occurred from methyl parathion and O - methyl - 4 - nitrophenol was occurred from methyl paraoxon in dealkylation (Dalkilic, 2006).

#### Its toxic effects

It was determined that minimum lethal dosage of methyl parathion for human beings is 100 milligram (mg) and LD<sub>50</sub> value by skin is 20 mg/kg. Even after a short period (about 15 min) following the application of parathion that is soluble in lipids, it was determined that it couldn't be removed from the skin by water with soap. It is a known fact that parathion that is in contact with the skin during application is absorbed through the skin and causes acute poisoning and deaths in human beings (Vural, 2005). The maximum permitted daily amount of methyl parathion among insecticides with organic phosphor is 0.001 mg/kg/day as in the residues of foods (Kalayanova and El Batavvi, 1991). Its acute oral LD<sub>50</sub> value, on the other hand, is 9 mg/kg (Delen et al., 2005). Oral LD<sub>50</sub> dosage of methyl parathion for male rats was determined as 14 mg/kg whereas for female rats it was determined as 24 mg/kg (Gains, 1960). In the studies, it was showed that methyl parathion caused a decrease in the weights of experimental animals (Zhu et al., 2001; Chung et al., 2002; Joshi et al., 2003; Narayana, 2005; Yildirim, 2006; Kahraman 2006). Uzunhisarcikli (2008) described that methyl parathion caused anorexia in experimental animals and indicated that this might be related with the damage occurred in gastrointestinal system. Zhu et al. (2001) determined that a decrease in the weight of rats was observed after seven days following 1 mg/kg of methyl parathion application as dermally.

In some studies, it was suggested that some pesticides cause a decrease in the weight of the liver related with the decrease in body weight (Dwivedi et al., 1998; Tamura et al., 2001) and some pesti-

cides cause an increase in the weight of the liver (Raizada et al., 2001; Johnson et al., 2002; Kang et al., 2004; Sharma et al., 2005; Juberg et al., 2006; Jeong et al., 2006). Dalkilic (2006) observed that there was a significant decrease in the concentration of sulphhydryl group related with the protein present in liver and kidney tissues of rats which were exposed to methyl parathion for three months and it was concluded that this might be due to oxidative stress related with the toxicity of methyl parathion. Uzunhisarcikli (2008) determined that the decrease in total protein and albumin levels after 4 and 7 weeks following methyl parathion application (0.28 mg/kg day) was resulted from disorder occurred in liver functions. He identified that AST, ALT, ALP, GGT and LDH penetrated into the blood and the levels of these enzymes in the plasm and blood serum increased since cellular damage occurred in the livers of rats which were exposed to methyl parathion. At the same time, serious degeneration, necrosis and fibrosis were observed in the light microscopy examination. In another study, Dikshith et al. (1991) determined that LDH levels in the serum increased in the rats which were treated with methyl parathion. Moreover, it was informed that methyl parathion inhibited (Ca<sup>+2</sup>,Mg<sup>+2</sup>)-ATPase activity in erythrocyte membrane and kidney microsomes (Reddy and Rao, 1990; Blasiak, 1995a, 1995b). According to these researchers, inhibition occurred via binding of methyl parathion on a specific region of ATPase and it was resulted in destruction of Ca<sup>+2</sup> homeostasis inside the cell (Reddy and Rao, 1990; Blasiak, 1995a). It was indicated in a research that methyl parathion or its metabolite p-nitrophenol in urinary samples of general population was one of the most commonly determined pesticides (Barr et al., 2005). In another study, it was determined from the variations observed in brain EEG of farmers who were exposed to methyl parathion as water or spray that their brain cholinesterase enzyme was inhibited (Muttray et al., 2005). Moreover, approximately 15% decrease was observed in erythrocyte cholinesterase activity of farmers who applied methyl parathion for two hours as nonprotected by spray (He et al., 2002). *In vitro* studies of methyl parathion showed that neutrophils of human leucocytes inhibited chemiotaxy (Lee et al., 1979). It was asserted that methyl parathion caused DNA damage in rats (Bartoli et al., 1991) and had significant genotoxic effect in DNA of human lymphocyte (Blasiak and Kowalik, 1999). In rats, methyl parathion was applied as 2 mg/kg orally for two years and it as described that there was not a certain increase in tumour formation. However, *in vivo* and *in vitro* studies in mammalian somatic cells showed that methyl parathion had genotoxic effect and increased chromosome abnormalities (Bartoli et al., 1991; Vijayaraghavan and Nararaja, 1994). In one study, it was determined that methyl parathion caused a decrease in the weight of ovary (Asmathbanu et al., 1997) and in another one, it was determined that it caused disorders in

oestrogen cycle (Sortur et al., 1999). Dhondup and Kaliwal (1997) applied methyl parathion intraperitoneally in 2.5, 3.5, 4.0 and 5.0 mg/kg dosages for 15 days and hypertrophy in ovary, significant decrease in the weight of ovary and in the number of healthy follicle were observed. Mathew et al. (1992) applied methyl parathion to rats in 1/2 LD<sub>50</sub>, 1/4 LD<sub>50</sub>, 1/8 LD<sub>50</sub> and 1/16 LD<sub>50</sub> dosages for 5 weeks and found that there was a significant increase in the number of abnormal sperms depending on the dosage. In the study of Joshi et al. (2003), the effect of methyl parathion on the reproduction system of male rats was investigated. Methyl parathion that was applied for 30 days in 30 mg/kg dosage caused a decrease in the weights of testicle, epididymis and seminal vesicle and patomorphological alterations in ventral prostate. At the same time, degeneration occurred in testicles. They pointed out that spermatogenesis ceased in lumen of seminiferous tubules and sperms were not found due to cellular disorders in the lumen. Moreover, an increase in total serum protein and in the number of white blood cells (WBC) and a significant decrease in the number of red blood cells (RBC) were determined. There was an increase in serum total cholesterol and triglyceride concentrations and a decrease in serum phospholipid concentration. Büyükkömürçü (2006) applied methyl parathion to male rats in the rate of 1/50 LD<sub>50</sub> and observed pathological alterations in testicle tissues after 4 and 7 weeks. Narayana et al. (2005) searched genotoxic and cytotoxic effects of methyl parathion on male reproduction cells. In this study, they applied 0.5 and 1 mg/kg methyl parathion to mature Wistar male rats for 12 days and 0.75 and 1.5 mg/kg methyl parathion for 25 days intraperitoneally. After 17, 77 and 130 days following the application, epididymal sperm content, sperm abnormality and testicular ascorbic acid level were measured and a decrease in sperm content and a certain increase in abnormal sperm number were observed. In addition to this, it was also observed that the level of ascorbic acid in testicle decreased. Moreover, the rats in the test group were matched with the female ones in the control group and their reproductive index (fertility) was measured. As a result of this, a decrease in the reproductive index was observed. In new born babies, a decrease in the body weights was determined. Uzunhisarcikli et al. (2007) observed a decrease in the weight of both body and testicle, a decrease in the number and mobility of sperms and an increase in the number of sperms having abnormal sperm morphology.

In the study of Undeger et al. (2000), genotoxic and immunotoxic effects of dimethoate (28.2, 14.1 and 7.04 mg/kg dosages) which is an insecticide having organophosphates and methyl parathion (0.872, 0.436 and 0.218 mg/kg dosages) were investigated by applying them to Wistar rats for 28 days orally. It was found that methyl parathion in high dosages resulted in an increase in the weight of liver and a dosage-depending decrease occurred in the cellular volume of erythrocytes at the minimum dosage. Abu-Qare and Abou Donia (2001) applied methyl parathion (10 mg/kg) to pregnant rats dermally as a single dosage and determined that cholinesterase enzyme was inhibited in the rats and their fetus. In the study of Guney et al. (2007), the subchronic effect of methyl parathion on lipid peroxidation and serum ChE activities and the protective role of vitamin E and C combination were investigated. In the group treated with methyl parathion, the level of malondialdehyde (MDA) increase while ChE activity decreased. In the group treated with methyl parathion together with vitamin combination, on the other hand, the level of MDA decreased while ChE activity increased. Ho et al. (2001) applied methyl parathion to rats in 6.25, 12.50 and 50 mg/kg dosages as a single dosage and observed 51, 77 and 88% decrease in the blood cholinesterase activity, respectively. Rao and Rao (1984) suggested that oxidative phosphorylation and active carrying were inhibited with the effect of methyl parathion and indicated that total adenosine triphosphatase (ATPaz) activity decreased in all tissues. In the study of Liu et al. (2006), a mixture of cypermethrin which is a piretroid and methyl parathion which is an insecticide having organophosphate in

different dosages (1/600, 1/135 and 1/30 LD<sub>50</sub> dosage) was applied to the rats for two months and investigated its effect on the level of hormones. As a result, an alteration wasn't observed in the body weights of the rats, however, a decrease was observed in relative adrenal gland weight. Yildirim (2006) treated methyl parathion in the rate of 1/50 LD<sub>50</sub> to male rats in his study and determined that pathological alterations (necrosis, infiltration and glomerular atrophy) were observed in kidney tissues after 4 and 7 weeks following the application.

### Its possible ecological effects

The residuals of pesticides having organophosphates were determined in the soil, vegetables, seeds, living things in water and in various foods (IARC, 1983; John et al., 2001). After using methyl parathion for the aim of agriculture which is one of the most commonly used pesticides, it is permeates to natural environment and remains in the nature for a few weeks to a few months (ATSDR, 1990). Remaining of methyl parathion in the applied region at least one week causes contamination of human beings and other living things with this toxic compound in various ways. In addition to this, the usage of compounds with methyl parathion for the aim of cleaning in the houses also causes people to be affected from this compound. Every year, thousands of people are exposed to methyl parathion for various reasons and experience various health problems depending on this (Leveridge, 1998). When ecotoxicity of methyl parathion is taken into consideration, its LD<sub>50</sub> value for bees is 0.17 mg and for birds, it is between 3 and 8 mg/kg body weight. For most of the fish, it is 6 - 25 mg/l (IPCS, 1993). Acute and chronic studies showed that methyl parathion is quite toxic for mammals. The mammals are exposed to methyl parathion orally, in dermal way and respiratory systems (Garcia et al., 2003). Methyl parathion has a toxic effect not only for mammals but also for fish, birds and other nontarget invertebrates (Solecki et al., 1996; US EPA, 1999; Fanta et al., 2003). As a result of illegal usage of methyl parathion in the houses and farms in America, it was indicated that cats and dogs died by poisoning, children get sick and depression was observed in people who were exposed to methyl parathion for a long time (Environmental Science and Technology News, 1997; ATSDR, 1997; Rehner et al., 2000).

In a research carried out by Fanta et al. (2003), histopathological alterations occurred in gills, intestines and liver of fish (*Corydoras paleatus*) which were exposed to sublethal dosage of methyl parathion as a result of its contamination in water and foods were investigated. They observed that hyperplasia, oedema and separations occurred in gills, respiration lamella and epithelial and this situation was observed more in the group which was exposed to it from water. After application with both ways, vacuolization in cells, focal necrosis, atrophy and embolization in the bile duct in the liver were determined.

As a result of the study, it was informed by the researchers that serious health problems were observed in the fish which were exposed to sublethal dosage of methyl parathion. In the study of De Aguiar et al. (2004), the fish (*Brycon cephalus*) were exposed to 1/3 LC<sub>50</sub> concentration of Folidol 600 (methyl parathion) for 96 h. AChE activity in brain, liver, muscle and plasm and the levels of ALT, AST, LDH were measured. It was determined that AChE activity in brain and plasm (64% plasm, 87% brain) was significantly inhibited. ALT decreased 59.4% in the liver whereas it increased 92.2% in the plasm. AST activity increased as an indication of tissue damages in the heart and plasm.

Monteiro et al. (2006) applied methyl parathion to fresh water trout *Brycon cephalus*. After 96 hours following the application, the effect of 2 mg/L single dosage of methyl parathion's commercial formula (MPc) on the catalase (CAT), glutathion peroxidase (GPx), super-oxide dismutaz (SOD), glutathion S-transferase (GST), reduced glutathion (GSH) and lipid peroxidation (LPO) of white

muscle and intestines was evaluated.

In the study of Bianchini and Monserrat (2007), methyl parathion was applied to yengeçlere (*Chasmagnathus granulatus*) in 10/100 LD<sub>50</sub> dosage for 72 h. As a result of the application, an increase was indicated in the damage percentage of hepatopancreatic tubules. Granules similar to melanin were observed in the connective tissue between hepatopancreatic tubules. Moreover, antioxidant enzyme (CAT and GSH) activities and lipid peroxidation (LPO) level increased in the hepatopancreas of crabs in which methyl parathion was injected.

Maitra and Sarkar (1996) applied methyl parathion to birds (*Lonchura malabarica* (Aves: Passeriformes) in sublethal dosages (5, 10 and 20 µg/100 g body weight) orally. After 10 days following the application, it was informed that a decrease was observed in the content of healthy reproduction cells of seminiferous tubules depending on the dosage and a significant decrease was observed in the weights of testicle with 10 µg and 20 µg dosages. As a result of methyl parathion application, AChE activity in the brains and testicles of birds decreased with respect to dosage and time. In the study of Solecki et al. (1996); 14, 20, 28 and 40 ppm methyl parathion were applied to quails for 6 weeks and it was determined that ChE level decreased in the brain and plasm. Moreover, the effect of methyl parathion on the reproduction was investigated and they informed that there was a decrease in the weights of eggs and in the thickness of egg shells.

## RESULTS AND SUGGESTIONS

This study was carried out in order to examine the ecotoxic risk factors of methyl parathion in terms of environmental and human health in the light of literature. The findings of animal experiments carried out with methyl parathion indicated that parathion methyl caused a decrease in the body weight of experiment animals and an increase in the weight of livers. A significant decrease in the rate of sulphydryl group related with protein in the tissues of liver and kidneys of rats and a cellular damage in the liver were observed. *In vivo* and *in vitro* studies in somatic cells of mammals showed that methyl parathion was genotoxic and increased the abnormalities of chromosomes. Moreover, it was found that it resulted in the inhibition of cholinesterase enzyme in the brains of human beings. This pesticide caused pathological alterations in the testicle of male rats, a decrease in the number of sperms and sperm mobility and an increase in abnormal sperm morphology. In addition to this, hypertrophy in ovary and a significant decrease in the number of healthy follicles were observed.

As a result of long-period exposure to methyl parathion, various neurological problems, muscle weakening, short period loss of memory and depression were determined (Rehner et al., 2000; Garcia et al., 2003). Moreover, fear, sleep withdrawal, not staying immobile, tremor related with central nervous system; excess working of secretory glands, diarrhea, nausea and vomiting related with environmental nervous system were observed in the methyl parathion poisoning (Coye et al., 1986). As it is the case in other pesticides, it is known that parathion methyl has some toxic effects directly or indirectly for the environment and so for the ecosystem by mixing to the air, soil and water. Parathion methyl was proved to have toxic

effect especially for birds, fresh water fish, hydrophilous organisms as well as sea organisms in a low rate. The studies carried out about this exhibits that the usage of methyl parathion as an insecticide is extremely detrimental for the ecosystem. It attracts attention that methyl parathion, the usage of which was forbidden in European Union (EU) countries in 2003 and was limited in United States of America, however, which is commonly used in our country has no restrictive or prohibitive enforcement. To bring new arrangements, to forbid or to limit the usage of methyl parathion both in our country and in other countries where it is freely used seem to be an obligatory.

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