Protective Effect of Garlic on Some Biochemical Changes
In Liver and Kidney of Male Rats Induced –Malathion

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ABSTRACT

This study was investigate the protective effect of garlic against toxicity induced-malathion in liver and kidney of male albino rats. The acute oral toxicity of malathion (Malatox 60 % EC) was determined and the oral median lethal dose (LD50) value to male rats was found to be 893.36 mg/Kg b. wt. The treatments were carried as follow: control (C), garlic (G) as +ve control (200 mg/Kg b. wt.), malathion (M) (1/20 LD50, 44.7 mg/Kg b. wt.), garlic + malathion (GM). Tissue samples were obtained after 28 days of treatment. Biochemical investigations revealed that, malathion induced significant suppression of γ-glutamyl transferase (γ GT) activity, and albumin level in liver. However, in kidney noticed significant increase in creatinine levels, but urea level was decreased. The presented data has also exposed a wide range variation in protein patterns in liver tissue. Malathion residues in tissues were nondetected in M and GM groups. Histopathological examination revealed degeneration of hepatocytes and cholelengitis in liver also, capsular hemorrhage associated with vacuolation of renal tubules in kidney of M group while, GM group revealed slight lessions. Finally, garlic supplement may improve the detrimental effects of malathion in rats.

Key words: Garlic, Malathion, Protection, Liver Function, Kidney Function, Protein Electrophoresis, Residues, Histopathology.
1 - INTRODUCTION

The rapid development agriculture in Egypt in the last few years required increase uses of agriculture chemicals and pesticides. Organophosphorus pesticides are widely used for the control of pests; which are neurotoxic insecticides selectively inhibiting acetylcholinesterase. Recent studies have demonstrated that free radicals are also, implicated in the deleterious effects of many environmental contaminants, such as organophosphates to mammals (Rodriguez et al., 2005). Malathion (S-1,2-bis (ethoxycarbonyl) ethyl O,O-dimethyl phosphorodithioate) was extensively used to control of crop pests, major arthropod disease vectors in public health programmes, household insects, and for the protection of stored grain (Abou-Donia, 1994).

Humans and animals in environment exposed to many chemicals that can impose stress on the organism and caused tissue damage by numerous biochemical mechanisms. Since the interaction between contaminants and biomolecules is the first step in the generation of toxic effects, understanding biochemical alterations induced by the exposure to pollutants may contribute to the prediction of toxic effects that may occur later at higher levels of biological organisations. Liver is a primary site for biotransformation so; many ingested xenobiotics are detoxified and eliminated by the liver. While others are bioactivity to reactive intermediates, which may lead to liver damage and other disorders, including cancer. In this direction, some antioxidant enzymes have been proposed as biological indicators of pollutant exposure in non-target organisms. Liver cells have relatively high antioxidant defence levels including enzymatic and non-enzymatic systems (Aruoma, 1998).

Garlic is known for its therapeutic properties since beginning of the recorded history and is probably the most widely studied medicinal plant (Block, 1985). The active substances of garlic are consistent with reports that in addition to sulfur containing compounds such as diallyl sulfide, flavinoids/isoflavinoids, polysaccharides, prostaglandins, saponins, and terpenes (Pinto and Rivlin, 2001). The biological responses of garlic include reduction of risk factors for cardiovascular diseases and cancer, a stimulation of immune function, enhanced foreign compound detoxification, radioprotection, restoration of physical strength, resistance to various stresses and potential antiaging effects (Amagase et al., 2001). In addition, Garlic is known for
its antibacterial, anticarcinogenic, hypolipidemic, hypoglycemic, antifungal, and anti-atherosclerotic properties, and an antioxidant against free radicals (Hassan et. al., 2009).

This work conducted to assess the protective mechanism of garlic against malathion insecticide on some biochemical indicators of liver and kidney functions as well as protein patterns and histopathological changes also, residue levels in rats.

2 - MATERIALS and METHODS

2.1- Pesticide and Antioxidant Used:

Malatox (60 % EC) formulated form of malathion purchased from El-Helb Co. for pesticide industry, Cairo, Egypt. Also, garlic used as EC tablet contains 200 mg of specially prepared garlic powder produced by ATOS Pharma, and purchased from Sekem Company Egypt.

2.2- Animals and Experimental Design:

Forty adult male rats (150 – 160 g body weight), maintained under constant conditions (12:12 h light / dark cycle, at 30 ± 2 °C). They were acclimatized for 2 week before the start of the experiments. The animals were divided into two main groups. The first main group was used to determined acute oral toxicity as median lethal dose (LD$_{50}$) and divided into four groups in each group 5 animals. The second main group was divided into four groups in each group 5 animals to investigate the protective effect of garlic against toxicity induced-malathion in liver and kidney and the treatment was following: the first group control (C), the second group treated with garlic (G) as +ve control (200 mg / Kg b. wt.) according to Paget and Barnes (1964), the third group (M) treated with 44.7 mg malathion / Kg b. wt. (equal 1/20 LD$_{50}$) and the fourth group treated with garlic + malathion (GM). All animals were treated orally by repeated doses for 28 days according to OCED Guidelines, No. 407 (1992). After the experimental period, all the animals were sacrificed to exit internal organs (liver and kidney) immersed in formalin for histopathological study and another digits of same organs frozen at -20 °C for biochemical analyses and residues determination.
2.3- Determination of Malathion LD$_{50}$:

Acute oral toxicity of malathion (Malatox 60 % EC) was performed to determine the acute oral median lethal dose (LD$_{50}$). The twenty mature male rats were divided into 4 groups of 5 rats for each group. Each group was intubated orally by gavage using stomach tube with different doses of experimental insecticide. Then the treated male rats were kept under observation for 24 hours and symptoms of toxicity and mortality were recorded according to EPA Protocol (1998). The acute oral LD$_{50}$ value was calculated according to the method of Weil (1952).

2.4- Biochemical Assay:

Liver and kidney were homogenized (1:10 w/v) in Potter-Elvehjem glass homogenizer with a Teflon piston in distilled water centrifuged at 3000 g for 20 min. at 4°C according to method of Mukhopadhyay et. al. (1982). The supernatant was collected for the assay biochemical substances. The both of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were measured according to the method adopted by Reitman and Frankel (1957). Alkaline phosphatase activity (ALP) was measured by method of Belfild and Goldberg (1971) and γ- glutamyl transferase (γ-GT) activity was measured by kinetic method of Szasz (1969). Total protein (T.P) and albumin contents were done based the method of Bradford (1976) and method of Doumas (1971) respectively. Urea level was measured by method of Fawcett and Scott (1960) and creatinine was measured by method of Schirmeister (1964).

Electrophoretic separation of liver proteins by SDS–PAGE electrophoresis was carried out according to the method of Laemmli (1970). The wide range of SDS-PAGE molecular weight pre-stained standars mix (Bio-Rad) was applied to the first well. Scanning was applied using gel prosoftware version 3 for Media Sci Image densitometry 700 Biorad, USA. A pool of 5 samples from tissue in each group has been used for the electrophoretic separation.

2.5- Histopathological Examinations:

Histopathological study was carried out according to Drury and Wallington (1980). Liver and kidney were immersed in formalin solution (15%) for 14-18 h, passed in a series of graded ethanol and embedded in paraffin. Paraffin sections were cut with at 5 µm thickness and stained with hematoxylin and eosin for light microscopic examination. The sections were examined and
photographed on an Olympus light microscope (Olympus BX51, Tokyo, Japan) with attachment
photograph machine (Olympus C - 5050, Olympus Optical Co. Ltd., Japan).

2.6- Tissue Residual Levels Determination:

Tissue residual levels were determined according to Mario et. al. (2002) as following:-

Apparatus:

An Agilent 7890N gas chromatography connected with FPD (flame photometric detector),
the column used was a capillary column (HP-5, 30 m × 0.32 mm × 0.25 μm USA).

Reagent:

Acetonitrile (HPLC grade – Merck Germany), acetic acid glacial (analytical grade),
Magnesium sulfate anhydrous (analytical reagent grade) and PSA (Primary secondary amine –
Supelco) were used. Stock standard solution: weight 10 mg malathion standard into a 100 ml
volumetric flask, dissolve and dilute to volume with acetonitrile.

Sample preparation:

One gram tissues (liver and kidney) were spiked with a standard pesticide to concentration
10 ng / g to calculate recovery percentage. The recovery percentage was ranged between 85 – 87
%. Accurately 1 g sample was homogenized for 2 min with 3 g magnesium sulfate anhydrous (1
g tissue + 3 g MgSo4). Transfer the sample to 15 ml polypropylene centrifuge tube and 5 ml
acetonitrile was adding. Vortex the sample at high speed for 2 min. and centrifuged at 3000 rpm
for 5 min. Two ml of supernatant were transferred to another clean 5ml centrifuge tube and add
0.2 g MgSo4 and 0.05 g PSA for clean-up, then the sample vortex at high speed and centrifuged
at 5000 rpm / 5 min. The samples were filtered by 0.45 PTEF filter and introduced to GC-FPD
for analysis.

GC Analysis

GC operating sample conditions as following: column temperature 100 °C hold 1min, at 25
°C / min to 180 °C, at 5 °C / min hold 5 min., port temperature 250 °C, injection volume 1 μl,
injection mode splitless, injection detector temperature 260 °C and carrier gas was Nitrogen by
flow rate 5 ml / min.
2.7- Statistical Analysis Procedures:

All data were subjected to statistical analysis by one – way Analysis of variance (ANOVA) test (Gad, 2001) using SPSS software for Windows version 10. A probability of \( p \leq 0.05 \); as the level of significance unless stated otherwise. Statistical significant differences between all treatments were carried by Least Significant Differences (LSD).

3 - RESULTS

3.1- Determination of the Oral Median Lethal Dose (LD\(_{50}\)):

The oral median lethal dose (LD\(_{50}\)) value of malathion to male albino rats was found to be 893.36 mg / Kg b. wt.

3.2- Biochemical Assay:

Biochemical parameters are presented in Table (1) and Fig. (1) revealed that no significant changes were noted in the activity of hepatic AST after treatment with malathion comparing to control values at \( p \leq 0.05 \). However, GM group induced significant increase in hepatic AST activity comparing to M group. In addition, hepatic ALT, ALP activities and total protein level were not affected in M groups but ALP activity was decreased comparing to M group after supplementation with garlic. The significant inhibition of hepatic \( \gamma \) GT activity was recorded in G, M and GM groups versus control. Also, reductions in hepatic albumin levels were recorded in M group versus control. However, these reductions were improved after supplementation with garlic.

As regarded to kidney function, the recorded results in Table (1) and Fig. (2) indicated that, creatinine levels were elevated significantly in M group versus control and G (+ ve control), but urea level was decreased significantly in M group. However, supplementation with garlic improved effect of malathion on these parameters.

The data as seen in Table 2 and Fig. 3, the electrophoretic separation of liver protein showed a wide molecular weight range in different groups. The banding pattern showed 10 bands in malathion –intoxicated group but only 5 bands in GM group verse C and G groups (9 bands). There were five bands as 221, 107, 52, 15 and 6 KDa were appeared in M group then disappeared in GM group. Also, there were missing bands in malathion–intoxicated group verse control group.
Fig. (1): Effect of garlic supplement on some biochemical indicators of liver functions (AST, ALT, ALP, γ GT, T.P and Alb) in male rats treated with malathion.

Table (1): Effect of garlic supplementation (200 mg / Kg b. wt) on some biochemical parameters in liver and kidney of male rats treated with malathion (44.7 mg / Kg b.wt.) for 28 days.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Parameters</th>
<th>Control</th>
<th>Garlic</th>
<th>Malathion</th>
<th>Garlic + Malathion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AST (U / g)</td>
<td>146 ± 1.3</td>
<td>148.9 ± 4.5</td>
<td>142.2 ± 1.6</td>
<td>155.08 ± 5.2 c</td>
</tr>
<tr>
<td></td>
<td>ALT(U / g)</td>
<td>117.5 ± 3</td>
<td>115.1 ± 1.4</td>
<td>120.7 ± 2.5</td>
<td>120.9 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>ALP( U / g)</td>
<td>42.8 ± 0.3</td>
<td>54.1 ± 4.4 a</td>
<td>49.8 ± 2.8</td>
<td>39.6 ± 2.4 bc</td>
</tr>
<tr>
<td></td>
<td>γ GT(U / g)</td>
<td>2.8 ± 0.3</td>
<td>1.6 ± 0.12 a</td>
<td>0.9 ± 0.07 a</td>
<td>2.02 ± 0.3 ac</td>
</tr>
<tr>
<td></td>
<td>T.P(mg / g)</td>
<td>7.2 ± 0.3</td>
<td>7.4 ± 0.6</td>
<td>7.3 ± 0.2</td>
<td>7.3 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>Albumin (mg / g)</td>
<td>5.2 ± 0.2</td>
<td>4.05 ± 0.37</td>
<td>2.76 ± 0.3 a</td>
<td>4.2 ± 0.70 c</td>
</tr>
<tr>
<td></td>
<td>Urea (mg / g)</td>
<td>11.8 ± 2.3</td>
<td>9.84 ± 1.28</td>
<td>6.48 ± 0.8 a</td>
<td>10.1 ± 1.5</td>
</tr>
<tr>
<td></td>
<td>Creatinine (mg / g)</td>
<td>0.8 ± 0.03</td>
<td>1.05 ± 0.1 a</td>
<td>1.45± 0.02 ab</td>
<td>1.08 ± 0.03 ac</td>
</tr>
</tbody>
</table>

Values represent means ± SEM (five rats).

a: significant differences versus control at p ≤ 0.05.
b: significant differences versus garlic at p ≤ 0.05.
c: significant differences versus malathion at p ≤ 0.05.
Table (2): Effect of garlic supplementation (200 mg / Kg b. wt) on molecular weight (M.W) of liver proteins (KDa) of male rats treated with malathion (44.7 mg / Kg b. wt.) for 28 days.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Control</th>
<th>Garlic</th>
<th>Malathion</th>
<th>Garlic + Malathion</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.W (KDa)</td>
<td>M.W</td>
<td>M.W</td>
<td>M.W</td>
<td>M.W</td>
</tr>
<tr>
<td><strong>221</strong></td>
<td>0.3255</td>
<td>0.5664</td>
<td>0.6501</td>
<td>1.138</td>
</tr>
<tr>
<td><strong>200</strong></td>
<td>0.5664</td>
<td>0.5667</td>
<td>0.5657</td>
<td>0.5664</td>
</tr>
<tr>
<td><strong>162</strong></td>
<td>0.7173</td>
<td>1.457</td>
<td>1.457</td>
<td>1.457</td>
</tr>
<tr>
<td><strong>119</strong></td>
<td>1.18</td>
<td>1.121</td>
<td>1.121</td>
<td>1.121</td>
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<tr>
<td><strong>107</strong></td>
<td>1.419</td>
<td>2.815</td>
<td>2.815</td>
<td>2.815</td>
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<tr>
<td><strong>97</strong></td>
<td>1.18</td>
<td>1.121</td>
<td>1.121</td>
<td>1.121</td>
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<tr>
<td><strong>92</strong></td>
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<tr>
<td><strong>88</strong></td>
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<td>2.815</td>
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<td>2.815</td>
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<tr>
<td><strong>65</strong></td>
<td>1.525</td>
<td>0.947</td>
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</tr>
<tr>
<td><strong>52</strong></td>
<td>1.4572</td>
<td>0.49023</td>
<td>0.49023</td>
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<tr>
<td><strong>29</strong></td>
<td>4.882</td>
<td>5.533</td>
<td>5.533</td>
<td>5.533</td>
</tr>
<tr>
<td><strong>25</strong></td>
<td>4.837</td>
<td>7.166</td>
<td>7.166</td>
<td>7.166</td>
</tr>
<tr>
<td><strong>18</strong></td>
<td>2.384</td>
<td>2.317</td>
<td>2.317</td>
<td>2.317</td>
</tr>
<tr>
<td><strong>14</strong></td>
<td>2.141</td>
<td>1.645</td>
<td>1.645</td>
<td>1.645</td>
</tr>
<tr>
<td><strong>13</strong></td>
<td>1.473</td>
<td>2.921</td>
<td>2.921</td>
<td>2.921</td>
</tr>
<tr>
<td><strong>11</strong></td>
<td>0.7288</td>
<td>1.473</td>
<td>1.473</td>
<td>1.473</td>
</tr>
<tr>
<td><strong>8</strong></td>
<td>0.724</td>
<td>0.724</td>
<td>0.724</td>
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</tr>
<tr>
<td><strong>7</strong></td>
<td>3.169</td>
<td>3.022</td>
<td>1.695</td>
<td>1.695</td>
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<tr>
<td><strong>6</strong></td>
<td>1.695</td>
<td>2.921</td>
<td>2.921</td>
<td>2.921</td>
</tr>
<tr>
<td><strong>5</strong></td>
<td>2.125</td>
<td>1.298</td>
<td>1.298</td>
<td>1.298</td>
</tr>
<tr>
<td>Total No. Bands</td>
<td>9</td>
<td>9</td>
<td>10</td>
<td>5</td>
</tr>
</tbody>
</table>
* Appear in treatment
** Disappear in treatment
*** Appear in treatment and disappear in garlic + malathion
# Decrease

Fig. (3): Effect of malathion on electrophoretic protein pattern of liver male albino rats for 28 days. Lane 1 (PM) protein marker, Lane 2 (C) control group, Lane 3 (G) garlic group, Lane 4 (M) Malathion group, Lane 5 (GM) garlic + malathion group.

Fig. (4): Liver of rat (1A): from M group showing vacuolar degeneration (VD) of hepatocytes, (1B): from M group showing cholengitis (Ch), notice thickening in the wall of bile duct and inflammatory cells infiltration, (1C): from G group showing apparent normal hepatic parenchyma, and (1D): from GM group showing slight congestion (SG) of hepatic sinusoids (H & E X 200).
Fig. (5): Kidney of rat (2A): from M group showing congestion (C) of glomerular capillaries and intertubular blood vessels, (2B): from M group showing focal tubular necrosis (FTN) associated with leucocytic cells infiltration, (2C): from G group showing apparent normal parenchyma, and (2D): Kidney of rat from GM showing slight atrophy (SA) of glomerular tufts (H & E X 200).

3.3- Histopathological Changes:

As shown in Fig. (4), liver of rats from M group revealed vacuolar degeneration of hepatocytes (1A), cholengitis described by thickening in the wall of bile duct and inflammatory cells infiltration (1B). However examined sections from G group showed apparent normal hepatic parenchyma (1C). While, liver of rat from GM group revealed slight congestion of hepatic sinusoids (1D).

As shown in Fig. (5), kidneys of rats from M group revealed congestion of glomerular capillaries and intertubular blood vessels (2A), focal tubular necrosis associated with leucocytic cells infiltration (2B), as well as, capsular hemorrhage associated with vacuolation of epithelial lining subcapsuler renal tubules were noticed. while, other sections from G group revealed apparent normal renal parenchyma (2C), while kidney from GM group showed slight atrophy of glomerular tufts (2D) as well as focal renal hemorrhage.
3.4- Tissue Residual Levels:

The residual concentration of malathion was nondeductible in liver and kidney tissues.

4 - DISCUSSION

Pesticides include a variety of chemical compounds used mainly in the agriculture. Extensively used of pesticide has generated a series of toxicological and environmental problems, particularly, toxic effects in human and animals. Organophosphates pesticide, malathion may produce free radicals. Such products may increase lipid peroxidation, which can be harmful to different organs including liver and kidney. On the other hand, these free radicals, known to cause oxidative stress, can be prevented or reduced by dietary natural antioxidants through their capacity to scavenge these products (Aruoma, 1998). The present study was undertaken to determine whether garlic could prevent or reduce malathion-induced oxidative stress by examining biochemical parameters in the liver and the kidney of rats.

Our results clearly showed that no significant changes were noted in the activity of hepatic AST, ALT, ALP and total protein level after treatment with malathion comparing to control. These results were agreement with Srivastava and Raizada (1999) who reported that quinalphos at lower doses (0.5, 1.5, 2 mg / kg / day) did not alter liver enzyme profiles in rats. While, malathion caused inhibition of hepatic γ GT activity in liver tissue. This inhibition could be explained by the γ GT enzyme is found in highly concentration in the cellular membrane of liver cells. The depression in γ GT activity may be due to the formation of complex compounds with enzyme in the liver (Kalantari and Salehi, 2001). Reduction in hepatic albumin levels were recorded in animals treated with malathion. The reduction in hepatic albumin was produced by inhibition of albumin synthesis in liver (Wood, 1993). These alterations were prevented by garlic, perhaps due to its role in stabilizing the cell membrane and protect the liver from free radical-mediated liver cell toxicity (Hassan et. al., 2009).

Renal toxicity was evaluated by a significant increase in the creatinine levels after treatment with malathion and after supplementation with garlic. Significant elevation of creatinine levels may be resulted to the decreased of glomerular filtration rate of kidney and or tubular dysfunction in treated animals (Willard et. al., 1994). While, urea level recorded significant reduction in kidney animals treated with malathion. These abnormalities are suggesting an impairment of kidney functions, these effects could be attributed to the changes in the threshold of tubular
reabsorption, renal blood flow and glomerular filtration rate (Zurovsky and Haber, 1995). Garlic showed a clear improvement in creatinine and urea levels perhaps due to the antioxidant properties of garlic in scavenging free radicals leading to reduced levels of kidney dysfunction (Hassan et. al., 2009). The urea reduction may be attributed to impairment urea synthesis in Ornithin urea cycle; this could be due to stress-induced urea production minimal (Walmsley and White, 1994).

Our results conclude that oral administration of garlic a significant protection against liver and kidney damage induced by malathion these results agree with El-Shenawy and Hassan (2008) which showed that oral administration of either selenium or garlic produces a significant protection against liver and kidney damage induced by the HgCl, but garlic appears to be more protective.

The presented data has also reflected a wide range variation in protein pattern in liver tissue due to malathion treatment and/or Garlic supplementation, which indirectly reflect a parallel variation the process of gene expression and/or proteolytic activity. One of the most important mechanisms underlying the effect the oxidative stress and free radicals are their effect on DNA. Oxygen free radicals have shown to induced DNA breaks (Nakahara et. al., 1998). The appearance and disappearance of protein fractions in the present study may be attributed this damage or may be explained by Bedwell et. al. (1989) who claimed that the free radical promote sulfhydral mediated cross linking of the labile amino acids such as methionine, histidine, cysteine and lysine causing a fragmentation of polypeptide chains in the protein molecule. The present investigation supported by El-Zayat (2007 and 2008).

The above effects were confirmed by histopathological changes in liver and kidney of treated rats. Histopathological observations showed that garlic attenuated cholangitis, inflammatory cells and vacuolar degenerative changes significantly in liver and congestion of glomerular, necrosis and hemorrhage in kidney. Malathion produces cellular necrosis through the formation of lethal protein adducts with important structural and functional proteins. Prevention of adduct formation could also be one way through garlic effect. More so, garlic is known to inhibit the secretion of pro-inflammatory cytokines such as tumor necrosis. Finally, many steroidal saponins and sapogenins are present in garlic, and could play vital roles as anti-inflammatory agents, in the induction of protein synthesis, and in tissue regeneration and repair (Wei and Lau, 1998). These results agree with Choi et. al. (2002) which showed centrilobular
necrosis vacuolar degenerative changes significantly in liver. In addition fenitrothion showed damage of liver tissue, hemorrhagic spots and degeneration of hypatocytes as well as the kidney exhibited hemorrhage, swollen and degeneration in rats treated (Elhalwagy et. al., 2008).

The major part of malathion in mammals is excreted in the urine and faeces within 24 hours. The biotransformation of some organophosphate insecticides, particularly malathion, contain 5-alkyl phosphorothiolate esters is by oxidative desulfuration by liver microsomal enzymes, leading to the formation of malaoxon; malathion and malaoxon are hydrolysed and thus detoxified by carboxylesterases (Tomlin, 1994). Therefore, the tissue residual concentration of malathion was nondeductible in liver and kidney

**In conclusions:** Our results indicate that intoxication with malathion induced significant damage of the liver and kidney tissues leading to impairment in liver and kidney function as well as protein pattern. While, garlic supplementation attenuates the malathion induced hepatotoxicity and renal toxicity, through the prevention of the metabolic activation.

**5 - REFERENCES**


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