# A study on the Effect of Dietary fiber from *Coriandrum sativum* in Malathion administered rats.

## Bijukumar, B.S<sup>1</sup>

Received 04/03/2018 Accepted 15/04/2018

# Abstract

Oral administration of malathion (500mg/kg Body weight–LD25) resulted in decreased levels of glutathione content (GSH) Glutathione Peroxidase (GPX) and Glutathione reductase (GR) in liver and kidney of rats. On the other hand significantly augmented levels of lipid peroxide content was observed in rats administered malathion. Feeding of Neutral Detergent fibre (NDF) from Coriandrum sativum (CS NDF) considerably ameliorated the toxicity induced by malathion. This effect was evidenced by the significant increase in the levels of GSH, GPX and GR, along with the low levels of lipid peroxide content.

Keywords: Coriandrum sativum, Malathion, Neutral detergent fibre

## Introduction

Malathion (mal) is a commonly used insecticide in India. Within the mammalian body mal is metabolized via three enzyme systems such as microsomal carboxyl esterase, microsomal cytochrome p-450 dependent monoxygenase system and cytosolic Glutathione S transferase (GST) (Buratti FM, Testai E 2005). As one of the major pathways of mal metabolism is via carboxyl esterase, in mammals, malathion is detoxified and excreted without much accumulation in the system. In addition to this pathway, mal is also detoxified through GST dependent dealkylation (Ketterman,A.J; Pond,S.M and Becker,C.E 1987). Here mal conjugates with GSH and thus get detoxified. Mal induces acute toxic effects in mammalian body (Inge M. Jensen, Paul Whatling 2010).

In India indiscriminate use of pesticides greatly contaminate both aquatic and terrestrial ecosystems, especially vegetable crops. Dietary fiber forms on of the major constituents of vegetables. The studies regarding the effect of dietary fiber on pesticide toxicity is meager. In south India especially in Kerala, Coriandrum is commonly used as an ingredient of many dishes.

In this work the effect of fiber isolated in the form of NDF from Coriandrum sativum in mal administered rats was studied.

## **Materials and Methods**

Male albino rats of Sprague - Dawley strain weighing 80-

120g bred and maintained in the animal house of the department of Biochemistry under standard laboratory conditions were used for the study. The rats were divided into 3 groups.

| Group I   | - Isocaloric fiber free diet (FF)        |  |
|-----------|--|--|
| Group II  | - Fiber free diet + malathion (FF + mal) |  |
| Group III | - 10% Coriandrum sativum NDF +           |  |
| -         | malathion (CSNDF + mal)                  |  |

The animals were fed with synthetic diet. 10g. of the NDF was added at the expense of CHO (CHO – equal parts of glucose, dextrin, sucrose and corn starch) in fiber diet fed groups. The caloric intake of all the groups was maintained unchanged by adjusting the food intake. The composition of diet is given below.

## **Composition of diet**

| _                    | Fiber free<br>(gm/100gm) | NDF<br>(gm/100gm) |
|----------------------|--------------------------|-------------------|
| *CHO                 | 65.00                    | 55.00             |
| Casein               | 20.00                    | 20.00             |
| (Vitamin & Fat free) |                          |                   |
| Ground nut oil       | 10.00                    | 10.00             |
| Fiber                |                          | 10.00             |
| Salt mixture         | 4.00                     | 4.00              |
| Vitamin mixture      | 1.00                     | 1.00              |
|                      |                          |                   |

\*CHO – Equal parts of glucose, dextrin, sucrose & Corn starch.

LD25 was selected as the dose by pilot tests. Accordingly mal was given 500mg/kg body weight. Pesticide was orally administered as suspension in ground nut oil. Duration of experiment was 30 days. After overnight fast, at the end of

<sup>&</sup>lt;sup>1</sup>Department of Zoology, Mahatma Gandhi

College, Thiruvananthapuram, Kerala, India

<sup>\*</sup>Corresponding Author email: bijukumarbsd@gmail.com



30th day, animals were sacrificed by cervical dislocation. Blood was collected from which the serum was separated. Tissues such as liver and kidney for the analysis were immediately collected and washed in 0.9% cold saline and stored in ice cold containers.

NDF was isolated from CS according to the procedure of Goering and Vansoest etal.189, Glutathione Content was measured by the method of Benke et al. The activity of Glutathione peroxidase (GPX) was determined by the method of Paglia and Valentine and Glutathione reductase (GR) was assayed by the method of Beutler. In the study of Lipid peroxidation, malondialdehyde was estimated by thio barbituric acid method of Nichans and Samuelsons . Statistical significance was calculated though one way ANOVA.

## Results

A significant depletion in the level of hepatic and renal GSH was observed during mal administration in FF diet given groups. Feeding of NDF from CS along with mal caused significant elevation of GSH in liver and kidney (Table 1)

The activity of Glutathione peroxidase was significantly inhibited in the liver and kidney during the administration of mal to FF diet fed rats. Significant increase in the activity of Glutathione peroxidase was observed in the liver and kidney of CS NDF fed rats ingested with mal (Table 2).

Glutathione reductase activity in liver and kidney exhibited significant depression in FF diet fed group. Inclusion of 10% NDF from CS in the diet of mal administered groups resulted significant increase in the activity of Glutathione reductase (Table 3).

Lipid peroxide content in liver and kidney was enhanced by the administration of mal to FF diet fed rats as compared to fiber fed groups. Inclusion of CS NDF along with mal markedly decreased the lipid peroxide content in liver and kidney (Table 4).

#### Table 1. Glutathione content in liver and kidney

| Groups           | Liver<br>(micromoles/gm tissue) | Kidney<br>(micromoles/gm tissue) |
|------------------|---------------------------------|----------------------------------|
| 1.FF             | 7.00 + 0.021                    | 2.62 + 0.016                     |
| 2.FF+Mal         | 4.55 <sup>+</sup> 0.091         | 1.09 + 0.012                     |
| 3.CS NDF<br>+Mal | 5.80 + 0.052                    | 1.70 + 0.017                     |

Values are +- SEM from six rats in each group

Groups with common superscripts are not significantly different at P<0.05 Groups without superscripts are significantly different at P<005

### Table 2. Glutathione peroxidase in liver and kidney

| Groups           | Liver<br>(Nanomoles of NADPH<br>oxidized/min/mg<br>protein ) | Kidney<br>(Nanomoles of NADPH<br>oxidized/mi/mg protein) |
|------------------|--|--|
| 1.FF             | 359 <sup>+</sup> 1.06  | 300 + 7.50   |
| 2.FF+Mal         | 236 + 6.37   | 248 + 7.45   |
| 3.CS NDF<br>+Mal | 275 + 8.53   | 263 + 5.26   |

Values are +- SEM from six rats in each group

Groups with common superscripts are not significantly different at P<0.05 Groups without superscripts are significantly different at P<005

#### Table 3. Glutathione reductase in liver and kidney

| Groups           | Liver<br>(Nanomoles of NADPH<br>oxidized/min/mg<br>protein ) | Kidney<br>(Nanomoles of NADPH<br>oxidized/mi/mg protein) |
|------------------|--|--|
| 1.FF             | 71.50 + 2.14   | 187 <sup>+</sup> _ 5.61                                  |
| 2.FF+Mal         | 32.70 + 0.81   | 151 <sup>+</sup> _ 3.62                                  |
| 3.CS NDF<br>+Mal | 50.90 <sup>+</sup> 1.52                                      | 165 <sup>+</sup> 4.29                                    |

Values are +- SEM from six rats in each group

Groups with common superscripts are not significantly different at P<0.05 Groups without superscripts are significantly different at P<005

#### Table 4. Lipid peroxide content in liver and kidney

| Groups           | Liver<br>(Nanomoles of MDA*<br>formed /gm tissue) | Kidney<br>(Nanomoles of MDA<br>formed /gm tissue ) |
|------------------|---|--|
| 1.FF             | 19.38 <sup>+</sup> .591                           | 10.30 + 0.323                                      |
| 2.FF+Mal         | 28.05 + 0.630                                     | 14.92 <sup>+</sup> _ 3.61                          |
| 3.CS NDF<br>+Mal | 23.92 + 0.640                                     | 12.05 + 0.421                                      |

Values are +- SEM from six rats in each group

Groups with common superscripts are not significantly different at P<0.05 Groups without superscripts are significantly different at P<005 \*Malondialdehyde

#### Discussion

Glutathione (GSH) is a cofactor for enzymatic reduction of

peroxides and plays a critical role in protection against lipid peroxidation. A state of GSH deficiency is a threat to biological tissues. Depletion of cellular glutathione proceeds cell damage which in turn is associated with alkylation of various nucleophilic cell constituents (Ahmed T, Tripathi AK 2009). The synthesis of GSH and its conjugation to xenobiotics are essential for detoxification (Lu SC 2013). GSH together with glutathione peroxidase (GPX), GST, catalase and SOD efficiently scavenge toxic free radicals. The enzymes Glutathione peroxidase and glutathione reductase (GR) act in concert with Glucose -6-phosphatase (G-6 PD) and maintains Glutathione status (Zhang J, Zhou X, Wu W, Wang J, Xie H, Wu Z 2017).

The results indicated that malathion induced impairernent in GSH status and has led to the inhibition of the activity of related enzymes like GPX and GR. This could result in higher production of hydroperoxides as indicated by higher lipid peroxide content. Low activity of Glutathione per oxidase, glutathione reductase and increased level of lipid peroxide content in malathion exposed rats fed with low protein diet was reported by Sakuntala Prabhakaraan and Devi K.S( 1993).

GSH is the substrate for GPX. The inhibition in the activity of GPX in the present study may be due to the low level of its substrate, GSH. The enzyme GR catalyze the reconversion of GSH from the oxidized glutathione (GSSG) The observed inhibition of GR in the liver and kidney of malathion administered rats is due to the reduced level of GSSG which in turn forms the substrate of GR.

Feeding of NDF from CS significantly enhanced GSH level. Restoration of the activity of redox cycle together with increase of GSH in CS NDF group would protect the tissues against the oxidative damage from malathion and their metabolites. A significant decrease in lipid peroxide content also showed that the deleterious effect of mal exposure was reduced by the inclusion of NDF for CS in the diet. Since GSH is involved in the conjugation and excretion of malathion or other metabolites, it is conceivable that low depletion of GSH results in low toxicity of malathion in CS NDF fed rats. The observed increase in GSH level may be resulted due to the decreased accumulation of malathion in fiber fed groups. The little amount of mal that was absorbed from the intestine was detoxified and excreted in fibre fed rats.

## Conclusion

The study indicates that NDF from CS brings about remarkable protection from the toxic effects induced by malathion. This is evidenced by the changes in GSH status, lipid peroxidation and antioxidant enzyme system. This may be due to the binding and consequent elimination of mal by the dietary fibre. Dietary fibre also enhances intestinal motility. The chemical components and physicochemical properties of fiber are involved in the absorption and excretion of the pesticide from the intestine.

## References

- Ahmed T, Tripathi AK, Suke SG, Kumar V, Ahmed RS, Das S, Banerjee BD. (1997) Role of HSP27 and reduced glutathione in modulating malathion-induced apoptosis of human peripheral blood mononuclear cells: ameliorating effect of N-acetylcysteine and curcumin: Aquatic Toxicology, Volume 39, Issue 2, 93-110
- Aurélie Doyotte, MarcBabut, PauleVasseur (1997) Antioxidant enzymes, glutathione and lipid peroxidation as relevant biomarkers of experimental or field exposure in the gills and the digestive gland of the freshwater bivalve Unio tumidus. Aquatic Toxicology 39(2):93-110
- 3. Benke,G.M, Cheerer,K.L, Mirer,F.E and Murphy,S.P (1974) Toxicol. Appl.Pharmacol,28:97
- Beutler, E (1974) Glutathione in Glutathione reductase proceedings of 16 th conference of the German society of Biological chemistry. Acdemic press New York P 109
- Buratti FM, D'Aniello A, Volpe MT, Meneguz A, Testai E. (2005) Malathion bioactivation in the human liver: the contribution of different cytochrome p450 isoforms. Drug Metab Dispos.;33(3):295-302.
- Buratti FM, Testai E.(2005)Malathion detoxification by human hepatic carboxylesterases and its inhibition by isomalathion and other pesticides. J Biochem Mol Toxicol.;19(6):406-14.
- Goering,H.K and Van Soest,P.J. (1970) In Forage Fiber analysis, Agricultural Hand Book no.379,Agricultural Research Service, United States Department of Agriculture.
- Inge M. Jensen , Paul Whatling, (2010) Malathion- A review of Toxicology in Hayes' Handbook of Pesticide Toxicology (Third Edition), 1527– 1542
- Ketterman, A.J; Pond, S.M and Becker, C.E (1987) Toxicol Appl. Pharmacol;87:389-392.
- Lu SC (2013). "Glutathione synthesis". Biochimica et Biophysica Acta. 1830(5): 3143–53.
- 11. Nichans Jr,W.J and samuelson,B (1968) Eur .J.Biochem, 6:126
- 12. Paglia, D.E and Valentine W.N (1967) J.Lab.Clin.Med, 70:158
- Shakuntala Prabhakaran, Shameem, F. and Devi, K.S. (1993) Vet. Human Toxicol., 35(5): 429. 129.
- Zhang J, Zhou X, Wu W, Wang J, Xie H, Wu Z (2017). "Regeneration of glutathione by α-lipoic acid via Nrf2/ARE signaling pathway alleviates cadmium-induced HepG2 cell toxicity". Environ Toxicol Pharmacol. 51: 30–37.