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Biochemical changes in enzymes, urea, bilirubin and glucose in serum of female albino rat treated with monocrotophos, an organophosphate

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Abstract
Daily oral administration of subacute concentration of monocrotophos for various durations in two groups i.e. TI (15 days) and TII (30 days) produced a significant inhibition of serum cholinesterase and significant rise in various serum enzymes (SGOT, SGPT, alkaline and acid phosphatases), glucose, bilirubin and urea as compared to control. The perturbation in enzymes was time dependent. Whereas a good deal of significant recovery was shown in R group (30 days treatment followed by 15 days recovery period) as compare to TII.

Keywords: Monocrotophos, organophosphates, acetyl cholinesterase, alkaline phosphatase, acid phosphatase, SGOT, SGPT glucose, bilirubin; urea

Introduction
Monocrotophos (3-hydroxy-N-methyl-cis-crotonamide dimethyl phosphate), an organo-phosphorous insecticide is extensively used for plant protection programmes. It is relatively a non-volatile, persistent, systemic insecticide which is commercially available as Nuvacron. This compound inhibits cholinesterase enzyme in tissues causing symptoms of cholinergic stimulation [1]. A few earlier workers have reported biochemical effects on serum enzymes, urea, bilirubin and glucose [1-4]. Hence this study was undertaken to investigate the biochemical changes exerted by monocrotophos on serum enzymes.

Materials and Methods
Monocrotophos was obtained as 70% pure technical grade. It was dissolved in distilled water. Three groups TI, TII & R, each of six female albino rats were administered 2.8 mg MCP per kg body weight (LD50 is 14 mg/kg body weight) orally for 15 days, 30 days, 30 days with recovery of 15 days respectively by intragastric intubation. The volume of oral dose was not more than 1 ml/100g body weight. Control animals received the vehicles only.
Blood samples were collected from the retro-orbital plexus of the eye from all rats after exposure period and were used to obtain serum for determination of serum GOT & GPT [5], glucose [6], bilirubin [7], urea [8], alkaline and acid phosphatases [9]. Serum cholinesterase was estimated according to the method of Ellman *et al.* [10]. All the rats were closely observed for toxic symptoms and occurrence of death.
The data were analysed statistically by using students t-test and significance assessed at $p<0.05$, <0.01 and <0.001 levels [11]. The significance of TI and TII groups was calculated with respect to control of R group with respect to TII.

Results
The growth retardation, lacrimation and salivation in monocrotophos treated female rats was observed in all the groups. The effect was maximum in group II. The mortality rate at the end of experimentation i.e. 45 days was 5%.
The biochemical mean ± SD values of serum compounds with their significant values have been given in Table.
The alkaline phosphatase significantly increased as compared to control i.e. 15.90% ($p<0.05$) in T, group and 20.72% ($p<0.01$) in T group whereas there was non-significant increase of 11.10% in R group. In TII group effect was more than TI group. Compared with control, acid phosphatase significantly increased, i.e. 10% ($p<0.05$) and 10.44% ($p<0.05$) in TI and TII groups respectively whereas there was non-significant increase of 3.03% in R group when compared with control. There was almost same effect in TI and TII groups (Fig. 1).

Serum analysis in I and II groups revealed a trend towards increase ie. 20.19% (p<0.01) and 33% (p<0.001) in SGOT and SGPT activity respectively (Fig 2). MCP in TI and III groups caused elevation i.e. 17.10% (p<0.001) and 19.73% (p<0.001) in bilirubin concentration, whereas significant inhibition i.e. 31.8% (p<0.001) and 43.64% (p<0.001) of AChE was observed in TI and III groups respectively. In R groups, though a high degree of change was still apparent in both bilirubin concentration and AChE activity, recovery period was able to significantly normalise the change in both (Fig 3). Blood urea levels raised 18.06% (p<0.05) and 26.58% (p<0.01) in TI and III groups respectively. Blood glucose level showed a significant increase ie. 32.47% (p<0.05) and 36.21% (p<.01) in TI and TII groups respectively. In R group both blood glucose and urea showed significant recovery (Fig. 4).

Table 1: Biochemical values in serum of female albino rat treated with monocrotophos (MCP)

Parameters	Control	Treated 1	Treated II	Recovery
Alkaline phosphatase (µnol min^{-1} L^{-1})	41.06±2.67	47.59±4.035*	49.57±12.715**	45.6±2.70
Acid phosphate (µmol min^{-1} 1^{-1})	25 38±1.54	27.92±1.24*	28.05±1.25*	26.15±1.32
SGOT (µmol min^{-1}' L^{-1})	48.37±3.22	58.14±2.36**	61.25±2.42***	52.13±2.35***
SGPT (µmol min' C^{l})	38.53±2.97	51.25±1.91***	54.24±2.01***	45.20±2.21***
Acetyl cholinesterase (µmol min^{-1} m1^{-1})	0.527±0.05	0.359±0.03***	0.297±0.04***	0.489±0.03***
Urea (µmol L^{-1})	6.92±0.70	8.17±0.35*	8.76±0.30**	7.15±0.56***
Glucose (µmol l^{-1})	4.28±0.21	5.67±1.14*	5.83±0.82**	4.86±0.51
Bilirubin (mg %)	0.76±0.04	0.89±0.02***	0.91±0.02***	0.80±0.02***

Results expressed as Mean ± S.D. (n=5)*, p<0.05; p<0.01;*** p<0.00l

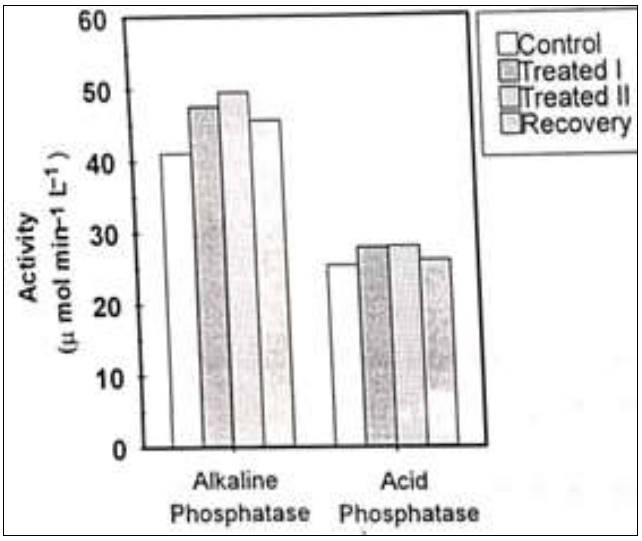

Fig 1: Effect of MCP on Alkaline Phosphatase and Acid Phosphatase in serum of female albino rat

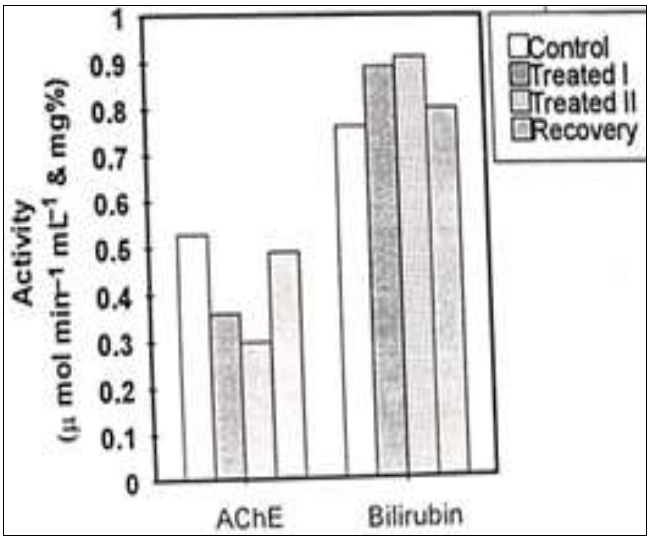

Fig 3: Effect of MCP on Acetyl cholinesterase and Bilirubin in serum of female albino rat

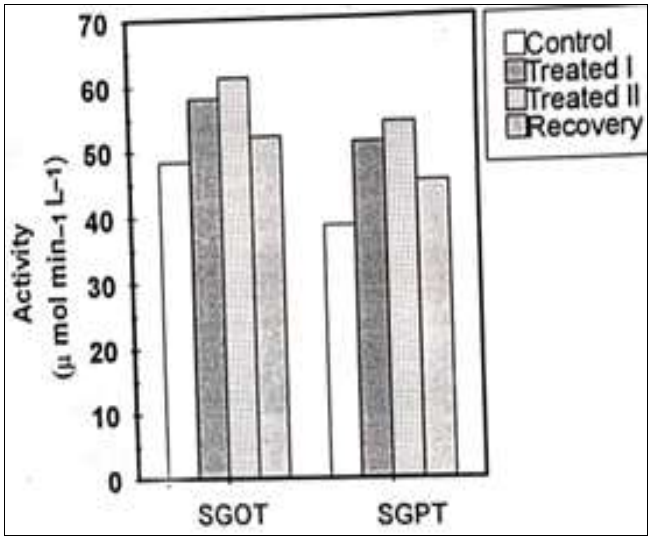

Fig 2: Effect of MCP on SGOT & SGPT in serum of female albino rat

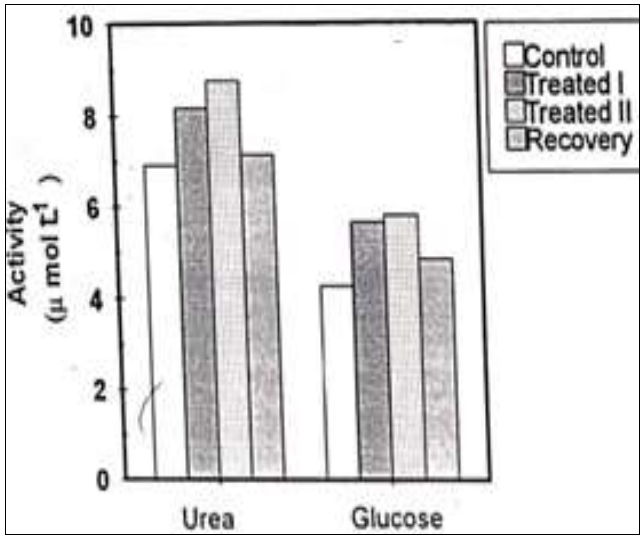

Fig 4: Effect of MCP on Urea and Glucose in serum of female albino rat

Discussion

The serum hydrolases, alkaline and acid phosphatases showed an increase after monocrotophos treatment. Sandhu and Malik [12, 3] reported similar observations of these enzymes in buffalo calves after oral treatment with MCP respectively. Janardhan and Sisodia [1] observed increase in alkaline phosphatase in rats. The rise in alkaline phosphatase may be the result of toxic chemical stress on the cellular membrane [13] and whereas increased acid phosphatase may be due to lysosomal damage in various tissues [14].

The serum acetyl cholinesterase showed a decrease after MCP treatment. The inhibition of AChE after organophosphate treatment has been reported by many workers as reviewed by Koelle [15]. According to them, the inactivation of AChE by an OP agent results in the accumulation of ACh at all sites of cholinergic transmission.

Serum transaminases, SGOT and SGPT showed an increase after MCP treatment. Similar changes were observed by Janardhan and Sisodia [1] in rats after MCP treatment and by Hanafy *et al.* [2] in rats after Tamaron treatment. This increase may lead to faster decrease of glutathione in liver. According to Gillette *et al*. [16] glutathione is a key compound in biochemical defence and its depletion increases vulnerability of the animal to many poisons.

The elevation of serum urea after treatment with MCP has been observed. The same observations have also been reported by Janardhan and Sisodia [1] in rats after MCP treatment and by Hanafy *et al.* [2] in rats after treatment with Tamaron. This increase seems to be due to nephrotoxic effect.

The elevation serum glucose after treatment with MCP has been reported. Similar observations have been reported by Janardhan and Sisodia [1] in rats after MCP treatment and Hanafy *et al*. [2] alter Tamaron treatment. According to Neumann and grosdanoff [7] it is one of the criteria for evaluation of diabetogenic compound. This may be due to damage to liver and islets of Langerhans. The histopathological necrosis due to MCP has been reported by Janardhan and Sisodia [1].

The elevation of serum bilirubin after treatment may be due to damage to liver. Similar observations have been reported by Janardhan and Sisodia [1] in serum and liver.

In R groups, significant recovery has been observed in SGPT, AChE and bilirubin. However, recovery was also observed in alkaline and acid phosphatases, SGOT, urea and glucose. These results were not of significant value. These observations need confirmation by histopathological studies of various tissues like liver and kidney which are under study by the present workers and will be published shortly.

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