GENERAL CONCLUSIONS
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Methyl parathion administration in general, has resulted in significant alterations of the normal functioning of the developing central nervous system. This is well documented by changes found in the content of total RNA and total proteins in developing central nervous system on methyl parathion exposure.

A significant increase observed in the level of total RNA and total protein content clearly indicates a characteristic stimulation of protein synthetic machinery of the developing central nervous system on methyl parathion administration.

Studies on brain specific acidic protein S-100 revealed a decrease in its level indicating decreased neuronal functioning and disruption of molecular mechanism during methyl parathion induced toxicosis.

The decrease found in the level of regulatory protein, calmodulin and calmodulin dependent phosphodiesterase indicates the alterations in the general regulatory mechanism of the developing central nervous system due to the toxic impact of methyl parathion.

The decrease observed in the activity of AChE during methyl parathion exposure points to anticholinesterase
action of methyl parathion in developing central nervous system. Studies on the molecular heterogeneity of AChE indicated a general suppression in the activity levels of isozymal forms. Remarkable decrease in the activity levels of choline acetylase and acetylcholinesterase with profound accumulation of acetyl choline provide evidence of the impaired neuronal activity on methyl parathion exposure.

The general increase found in the amino acid transmitters viz. glutamic acid, aspartic acid and glycine is related to the potentiation of inhibitory transmission due to the toxic effect of the insecticide.

Considerable elevation in the levels of epinephrine and norepinephrine leading to stimulation of sympathetic system points to the altered neuronal activity imposed by methyl parathion exposure. The significant inhibition in the activity levels of monoamine oxidase also points to the rise in the levels of biogenic amines leading to neuronal disorder associated with the imbalance in the levels of biogenic amines.

The alterations in the activity of ATPases like \( \text{Na}^+\text{K}^+ \) ATPase, \( \text{Ca}^{2+} \) ATPase and \( \text{Mg}^{2+} \) ATPase indicates altered energy metabolism in all the compartments of central nervous system studied on methyl parathion exposure.
Exogenous addition of calmodulin extract (1-20µg) on the Ca\(^{2+}\) ATPase activity resulted in an elevation of the inhibited Ca\(^{2+}\) ATPase activity on methyl parathion exposure. The decrease in Ca\(^{2+}\) ATPase activity and Ca\(^{2+}\) pump could be reversed by the exogenous addition of calmodulin, suggesting that methyl parathion exerts its action by decreasing the level of calmodulin.

The general decrease found in the activity levels of dehydrogenases suggests the alterations in the oxidative metabolism on methyl parathion exposure. The decreased levels of SDH indicates a favour of anaerobic metabolism during methyl parathion toxicity. The decreased activity levels of GDH indicates a significant role in averting ammonia intoxication during methyl parathion induced toxicity. The decrease in the activity levels of PDH suggests decreased oxygen uptake and pyruvate mobilization during methyl parathion toxicosis.

The alterations occurring in the multiple forms of LDH provides an evidence for the profound disruption of the molecular mechanism due to methyl parathion exposure.

Thus, the inhibitory transmission gets stimulated in contrast to the cholinergic and adrenergic transmission system, which suffers remarkable inhibition on methyl
parathion exposure. Such alterations in the neurotransmission system and also in the regulatory protein calmodulin clearly indicate molecular disruptions which render the developing brain highly inefficient during methyl parathion induced toxicity.