2.5 mg/kg also prevented implantation when applied on either of the day during 1-3% (table 2).

The present results reveal that 1,2-diethyl-1,3-*bis*-(*p*-methoxyphenyl)-1-propene is a potent antifertility agent as it inhibits the pregnancy in mice significantly. As the compound was shown to have significant anti-implantation activity in rats[1], there seems to be no difference in relation to species-specificity. It is thus concluded that the test compound possesses a potent anti-implantation effect in rats and mice which may be due to its estrogenic effect[1,2]. This effect may be due to the expulsion of ova from the tube[5-8] and by disturbing functional equilibrium between estrogen and progesterone[9,10]; thus creating a non-receptive stage in the uterus.

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VARIATIONS IN TOTAL PROTEIN CONTENTS OF THE ADENOHYPOPHYSIS, NEUROHYPOPHYSIS AND EIGHT BRAIN AREAS OF PHYTOHAEMAGGLUTININ—POISIONED RATS.

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SINCE the discovery of recin by Stillmark[1], considerable interest has been shown in the haemagglutinins from plants. Studies with these lectins have shown among several other toxic effects, the inhibition of protein synthesis[2], activation of protein catabolic enzymes[3] and the transaminases[4]; and quantitative increases in the blood values of urea, uric acid and serum protein[5]. Little is however known about the residual toxic effects of these lectins on cholinergic function, particularly as it relates to brain enzyme activity and total brain protein. This communication reports our investigation on the effects of cowpea and limabean haemagglutinins on the protein contents of the adenohypophysis, neurohypophysis and eight brain areas of the weanling rat.

Agglutinins were extracted and purified[6,7] from ground mature cowpea and limabean seeds. The mean lethal doses (MLD) as defined by Mohammed *et al*[8], were determined for albino rats of the Wistar strain (40–42 g) after intraperitoneal injection with different doses of the lectins dissolved in sterile 0.9% NaCl solution. Control rats were given sterile saline solution alone. Several preliminary experiments performed with different concentrations of haemagglutinins gave 2.5 mg/g of cowpea haemagglutinin and 2.0 mg/g of limabean haemagglutinin as the mean lethal doses. These concentrations caused death within 22 and 16 hr of injection in the cowpea haemagglutinin and limabean treated haemagglutinin-treated rats respectively. In the actual experiment, rats thus inoculated, were stunned and killed by decapitation 30 min before the expected onset of death. The heads of the moribund and control rats were removed and the whole brain excised immediately. The meninges were removed and the brain disected into pons, hippocampus, hypothalamus, amygdala, medula oblongata, cerebellum, midbrain and cerebral cortex. Each part was weighed and homogenized individually (1% w/v) in 0.1 M ice-cold phosphate buffer containing 0.1% Triton X-100 (Sigma), pH 7.4. Total protein was immediately determined by a colorimetric method[9], using bovine serum albumin (BSA) as standard protein. Results were analyzed by student's *t* test.

TABLE 1

Total brain proteins (g%)[1]

Parts	Control	Cowpea haemagglutinin (Mean lethal dose)	Limabean haemagglutinin (Mean lethal dose)
Adenohypophysis	0.67 ± 0.69	0.70 ± 0.72	0.73 ± 0.36
Neurohypophysis	0.86 ± 0.33	0.86 ± 0.39	0.88 ± 0.48
Pons	1.07 ± 0.35	0.84 ± 0.31	1.70 ± 0.52
Hippocampus	0.74 ± 0.32	0.96 ± 0.23	0.53 ± 0.35
Hypothalamus	0.71 ± 0.17	0.70 ± 0.30	0.75 ± 0.18
Amygdala	1.37 ± 0.70	3.76 ± 0.54*	3.68 ± 0.62*
Medulla Oblongata	0.87 ± 0.43	0.65 ± 0.51	1.31 ± 0.12
Cerebellum	1.10 ± 0.27	3.43 ± 0.44*	3.41 ± 0.35*
Midbrain	1.08 ± 0.51	3.01 ± 0.54	1.95 ± 0.27
Cerebral Cortex	0.80 ± 0.18	0.78 ± 0.33	0.94 ± 0.47

[1] Mean of six rats per treatment ± standard error of mean.
* Indicates significant difference from control at 5% level.

Total protein in control animals was highest in the amygdala, closely followed by the cerebellum while the lowest protein value was recorded in the adenohypophysis (table 1). In the haemagglutinin-treated rats, there was no significant change in total proteins in the brain areas, adenohypophysis or neurohypophysis when compared with controls except in the amygdala ($p<0.05$) and the cerebellum ($p<0.05$). These significant increases suggest a possible activation of the several enzyme systems in the poisoned rats. Because the brain is among the body organ that is most sensitive to metabolic deficiency, it is suspected that the increased brain total proteins may originate in the increased synthesis by protein catabolic enzymes. It is possible that a combined induction mechanism was influencing the activities of these enzymes. In one case, the synthesis of the catabolic enzymes might have been specifically activated and in the other, the total synthesis of organ protein enhanced. If this were so, more amino acids would arrive at the brain and undergo catabolic processes resulting in a higher total protein content.

It is however possible that the increase in brain protein was really a manifestation of some neurological endocrine alterations in the brain. That haemagglutinin has a definite effect on cholinergic function is not known and our results cannot be effectively discussed because similar studies have not been reported. Meanwhile, it would seem that the observations reported here would be of value in the demonstration, more directly than hitherto, of the influence of haemagglutinin on brain proteins. Also, if the increase obtained is an expression of the quantity of organ protein present, it could be considered that the protein catabolic enzymes in the amygdala and cerebellum have been spared with respect to the pons, medulla and midbrain of the cowpea-haemagglutinin poisoned rats, where slight increases in total contents were realized.

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