TABLE I

Effect of castration and replacement therapy on sialic acid concentration of the genital organs of male rhesus monkey

Status	Sialic acid (Mg./100 gm.)					
	Testis	Caput epididymis	Corpus epididymis	Cauda epididymis	Seminal vesicle	Prostate
Control (4)*	13·4† (10·7–15·4)	51·0 (40·5–55·7)	48·6 (42·1–61·1)	37·1 (33·3–43·7)	20·5 (17·2–23·3)	24·3 (19·0–27·8)
Castrated (4)	49·9 (45·0–53·2)	50·1 (42·2–60·8)	39·9 (31·2–45·6)	19·72 (17·0–22·4)	26·5 (21·1–29·3)	
Castrated + testosterone propionate (4) ..	48·8 (42·3–55·7)	47·0 (42·1–49·8)	40·0 (33·9–45·7)	21·2 (15·2–23·4)	25·8 (21·0–32·1)	

* No. of animals. † Mean with range in parenthesis.

vesicles ($P < 0·05$). This was, however, not the case in the TP-treated group.

It thus appears that as in rats,[7,8] the epididymis of monkeys shows the highest concentration of sialic acid. However, in contrast to rats,[7,8] the level in the caput and the corpus portions is higher than that of cauda. The apparent non-responsiveness of sialic acid concentration of the genital organs to castration and TP therapy in both species, suggests that the level of this constituent is not under androgenic control of the testis.

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EFFECT OF ADRENERGIC BETA RECEPTOR BLOCKING DRUGS ON MONOAMINE OXIDASE ACTIVITY

ADRENERGIC beta receptor blocking drugs and their isomers produce varied effects on the central nervous system by mechanisms still unknown. *n*-isopropyl para-nitrophenyl ethanolamine; D(*l*) INPEA and its inert isomer L(*d*) INPEA have been observed to produce central excitatory effects.[5] As opposed to these, propranolol[1,5,6] and *n*-isopropyl-B (4-methanesulphonamidophenyl) ethanolamine (MJ 1999)[4] depress the central nervous system. Since monoamines as well as their degrading enzyme, monoamine oxidase play an important part in the mediation of brain functions it was considered worthwhile to study the effect of the above adrenergic beta receptor blocking drugs on monoamine oxidase activity.

Monoamine oxidase activity was estimated manometrically in Warburg's apparatus by the method of Creasey.[2] 0·2 ml. of 2 M tyramine was used as the substrate, and 2 ml. of a 1 : 10 homogenate of fresh rat liver in phosphate buffer (pH 7·4), was employed as the enzyme preparation. The drugs, D(*l*) INPEA, L(*d*) INPEA (Selvi and Co.), propranolol (I.C.I.) and MJ 1999 (Mead Johnson and Co.) were added to the homogenate in the flasks and incubated for 15 minutes prior to the addition of the substrate contained in the sidearm. The effect of these on MAO activity was compared with that of nialamide (Pfizer), a known inhibitor of MAO.

Table I reveals that D(*l*) INPEA has the most potent inhibitory effect on MAO which is comparable to that of nialamide. L(*d*) INPEA, in comparison, is a much weaker

inhibitor of MAO and also possesses lesser stimulant effect on the brain than the lævo rotatory optical isomer (unpublished observations). It can therefore be presumed that the property of isomers of INPEA to stimulate the central nervous system may be related to the inhibition of MAO. Propranolol also shows slight inhibition of MAO which is similar to the observation of Greeff and Wagner.[3] Paradoxically, this drug causes depression of the central nervous system, which may be due to some other mechanism of action.

TABLE I

Effect of adrenergic beta receptor blocking drugs on monoamine oxidase activity of rat liver, in vitro

Drugs	Dose (μg./ml.)	No. of observations	Units* of MAO activity during first 30 minutes ± S.E.	Per cent inhibition	'p' value
Control ..	..	18	4·2±0·31	..	..
Nialamide ..	37	8	2·6±0·25	38	<0·01
	71·5	8	2·2±0·2	47·6	<0·01
D-INPEA	37	12	2·8±0·61	33·33	<0·05
	71·5	10	2·4±0·18	42·8	<0·01
L+INPEA	37	10	3·4±0·351	19	<0·05
	71·5	10	3 ±0·41	28	<0·05
MJ 1999 ..	37	8	4·2±0·4	0	>0·9
	1	8	4 ±0·18	4·7	>0·6
Propranolol	37·5	8	3·9±0·26	5·2	>0·3
	71·5	8	3·2±0·17	23·8	<0·0.

* 1 unit activity = 1 μ litre of oxygen consumed per hour per gram of enzyme source.

It is interesting to observe that the alteration in the isomeric state of INPEA leading to an increase in beta receptor blocking property also confers to the compound a greater enzyme inhibitory potency. However, MJ 1999, a specific adrenergic beta receptor blocking drug,[4] does not cause any inhibition of MAO.

The present study reveals that although inhibition of MAO is not a common property which is shared by all adrenergic beta receptor blocking drugs, still it may be concerned with the central excitatory effect of INPEA.

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DIAGNOSIS OF SPIROCHAETOSIS OF POULTRY BY SLIDE AGGLUTINATION AND SPIROCHAETE IMMOBILIZATION TESTS

DIAGNOSIS of spirochætosis of poultry has been made by demonstrating *Borrelia anserina*, the causative organism of the disease in blood and tissues in the acute stage by ordinary microscopy, dark field microscopy (Nobrega and Reis, 1947, cited by Snoeyenbos, 1965) and fluorescent antibody technique (Gross and Ball, 1964). Immobilizing antibodies have been demonstrated in the treated and recovered birds (Levaditi *et al.*, 1952). In the present note the authors report quick plate agglutination and spirochæte immobilization tests which can be employed to detect the micro-organisms in the acute phase of the disease and the antibodies in poultry flocks where infection might have passed unnoticed.

The antigen was separated from the blood of artificially infected birds usually on fourth day of inoculation when the number of spirochætes was maximum in the blood circulation. The blood was collected in sterile citrated solution and its volume was increased two to three times with normal saline. Red blood suspension was thoroughly mixed and centrifuged at 500 rpm for 5 minutes to settle the blood cells. The supernatant containing most of the micro-organisms was collected in a separate test-tube and the sedimented red blood cells were resuspended in normal saline solution for centrifugation at the same speed and time for recollecting the supernatant. The pooled supernatant was centrifuged at 3,500 rpm for 15 minutes. The micro-organisms, settled at the bottom of the tube, were suspended in 0·5% carbol saline to match McFarland's opacity tube No. 4 which constituted the antigen for slide agglutination test. The antigen was coloured with 1% alcoholic solution if crystal violet at the rate of 0·03%.

The test was conducted by adding two loopful (4 mm. diameter) of the coloured antigen

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