a 20" × 1·5" column of acid-washed alumina slurry-packed in benzene. The column was progressively eluted with benzene (400 ml.), benzene-ethanol (75%-25% ; 300 ml.), benzene-ethanol (50%-50% ; 300 ml. and ethanol (600 ml.) and 100 ml. fractions were collected. Fractions 2-4 gave 2-*o*-chloroanilinopropionitrile (2·3 g.) and fractions 11–14 gave 2·4 g. of the title product. It was crystallised from ethanol-benzene-hexane ; m.p. 69–70°.

TABLE I

$$R_1\text{-}NHCH_2CH_2\text{-}C(=N\text{-}O\text{-}R_2)(\text{-}NH_2)$$

No.	R_1	R_2	m.p. °C.*	Nitrogen % Found	Nitrogen % Calc.
1	Phenyl ..	H : 2 HCl†	180-82	16·45	16·67
2	Benzyl ..	H : 2 HCl†	181–83	15·95	15·80
3	Phenylethyl ..	H : 2 HCl	180-82	15·32	15·00
4	Cyclohexyl ..	H	114–15	22·79	22·71
5	*o*-Chlorophenyl	H	69–70	19·37	19·67
6	,, ..	H : 2 HCl	195–96	14·31	14·67
7	,, ..	$CONH_2$	59-60	21·62	21·83
8	*m*-Chlorophenyl	H	79-80	19·81	19·67
9	,, ..	H : 2 HCl	189–90	14·79	14·67
10	,, ..	$CONH_2$	84–85	21·63	21·83
11	*p*-Chlorophenyl	H : 2 HCl	170-72	14·74	14·67
12	,, ..	$CONH_2$	112–13	21·58	21·83
13	*o*-Tolyl ..	H : 2 HCl	188-90	15·64	15·80
14	,, ..	$CONH_2$	89–90	23·49	23·73
15	*m*-Tolyl ..	H : 2 HCl	193–94	15·45	15·86
16	*p* Tolyl ..	H	82–83	21·61	23·76
17	,, ..	H : 2 HCl	187–88	15·59	15·80
18	,, ..	$CONH_2$	68–70	23·54	23·73
19	*o*-Anisyl ..	H	91–92	20·34	20·09
20	,, ..	$CONH_2$	59–60	22·41	22·22
21	*m*-Anisyl ..	H : 2 HCl	182-84	14·63	14·89
22	,, ..	$CONH_2$	109–10	23·23	22·22
23	*p*-Anisyl ..	H	92-93	20·06	20·09
24	,, ..	$CONH_2$	123-24	22·53	22·22

* All melting points are uncorrected. † Halogen estimation corresponded to 2 HCl.

O-Aminocarbonyl-2- o *- chloroanilinopropion-amidoxime.*—To a cold solution of 2-*o*-chloroanilinopropionamidoxime (1·8 g. ; 0·01 mole) in dilute hydrochloric acid (10 ml. ; 2 N) was added a solution of potassium cyanate (0·9 g. ; 0·011 mole). After 10 minutes at 5°, the reaction solution was rendered neutral with ammonia. The resulting gummy mass gradually solidified ; it was crystallised from acetone-benzene-hexane ; m.p. 59–60°.

The rest of the compounds in Table I were similarly prepared.

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EFFECT OF PARAOXON ON THE SOMATOSENSORY EVOKED POTENTIALS IN THE RAT

THE transmitter function of acetylcholine in the cerebral cortex has been the subject of many investigations. Application to the cortex of drugs that influence cholinergic synapses gave results that were often contradictory and difficult to interpret.[3] Another approach to the cortical cholinergic mechanisms has been made by studying the effect of topically applied cholinomimetic drugs on the somatosensory evoked potentials. Bhargava[1] has shown that the cortical application of artificial cerebrospinal fluid (CSF) containing various concentrations of acetylcholine increases the amplitude of the repetitive after-discharges of the somatosensory cortex, whereas they do not influence the short latency positive-negative primary complex of the cortical evoked potentials. It appears that the short latency primary evoked potential is not cholinergic, as Mitchell[4] has shown that under chloralose anaesthesia acetylcholine output is greatly reduced while the initial positive-negative potential is increased. It seems that the activity of cholinoceptive units rather manifests itself in potentials of longer latency (*i.e.*, repetitive after-discharges).

The present experiments were undertaken to study the effect of cortical application of paraoxon (an irreversible inhibitor of cholinesterase) on the somatosensory evoked potentials, and to find out the cholinergic component of the cortical evoked potentials. The study may throw more light on the intra-cortical cholinergic mechanisms.

In rats (CFE strain from Carworth, Europe) lightly anæsthetised with pentobarbitone, the technique of Bhargava and Meldrum[2] was employed to record the averaged somatosensory evoked potentials. Computer derived averages of 32 consecutive somatosensory evoked potentials from both cortices, in response to the

stimulation of contralateral and ipsilateral forepaws, were recorded. Paraoxon dissolved in CSF was applied to the pial surface through especially designed cortical cups. In most experiments the drug was applied to only one cortex while the other cortex was bathed with normal CSF and served as control throughout the experimental period.

A continuous record of spontaneous electrocortical (EEG) activity and cortical and rectal temperatures was obtained on the different channels of the polygraph (Devices).

Paraoxon was applied in different concentrations (10^{-5} M ; 3×10^{-5} M ; 10^{-4} M and 10^{-3} M) to the cortex for a period of 30 minutes, and its effect on the somatosensory evoked potentials was observed. With each concentration at least 4 to 6 experiments were performed.

In most of the experiments paraoxon produced an increase in the amplitude of the repetitive after-discharges of the cortical evoked potentials. The short latency positive-negative complex was not affected after cortical application of paraoxon. This effect was more marked with less concentrations of paraoxon (up to 10^{-4} M); higher concentration had little effect and even caused the death of some of the animals. The best effect was however obtained with 3×10^{-5} M of paraoxon. The effect on the repetitive after-discharges usually started within 15–20 minutes of the application of the drug, and lasted for more than 6–8 hours after washing off the drug.

In the control cortex the evoked potentials following contralateral or ipsilateral stimulation were unchanged.

The present study shows that paraoxon only produces its effect on the long latency (repetitive after-dicharge) component of the cortical evoked potentials, whereas the primary complex remains unaffected. This effect of paraoxon is due to preservation of acetylcholine because of inhibition of the enzyme acetylcholinesterase. The result thus provides an electrophysiological evidence that the afferent pathways responsible for the repetitive after-discharges are cholinergic in nature while those for the primary complex probably do not involve cholinoceptive synapses. A more detailed study has to be done for further confirmation of this intra-cortical cholinergic mechanism.

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BIOSYNTHESIS OF PLANT STEROLS THE MECHANISM OF THE ALKYLATION OF α-SPINASTEROL AT C-24

IN the mechanism postulated for the alkylation of phytosterols, the hydrogen atom at C-24 migrates to C-25.[1,2] We reported earlier[3] that the incorporation of (3RS)-[2-^{14}C(4R)-4-$^{3}H_1$]-mevalonic acid into *Camellia sinensis* yielded α-spinasterol which had $^{3}H/^{14}C$ atomic ratio of 2·44 : 5 instead of 3 : 5. It was pointed out that the process of alkylation in this phytosterol may proceed simultaneously by two routes; one route involving the intermediate in which ^{3}H at C-24 is lost, possibly during the formation of 24-methylene intermediate and the other route in which ^{3}H is transferred to C-24 through the carbonium ion mechanism. In the present communication we report experimental evidence supporting the above possibility.

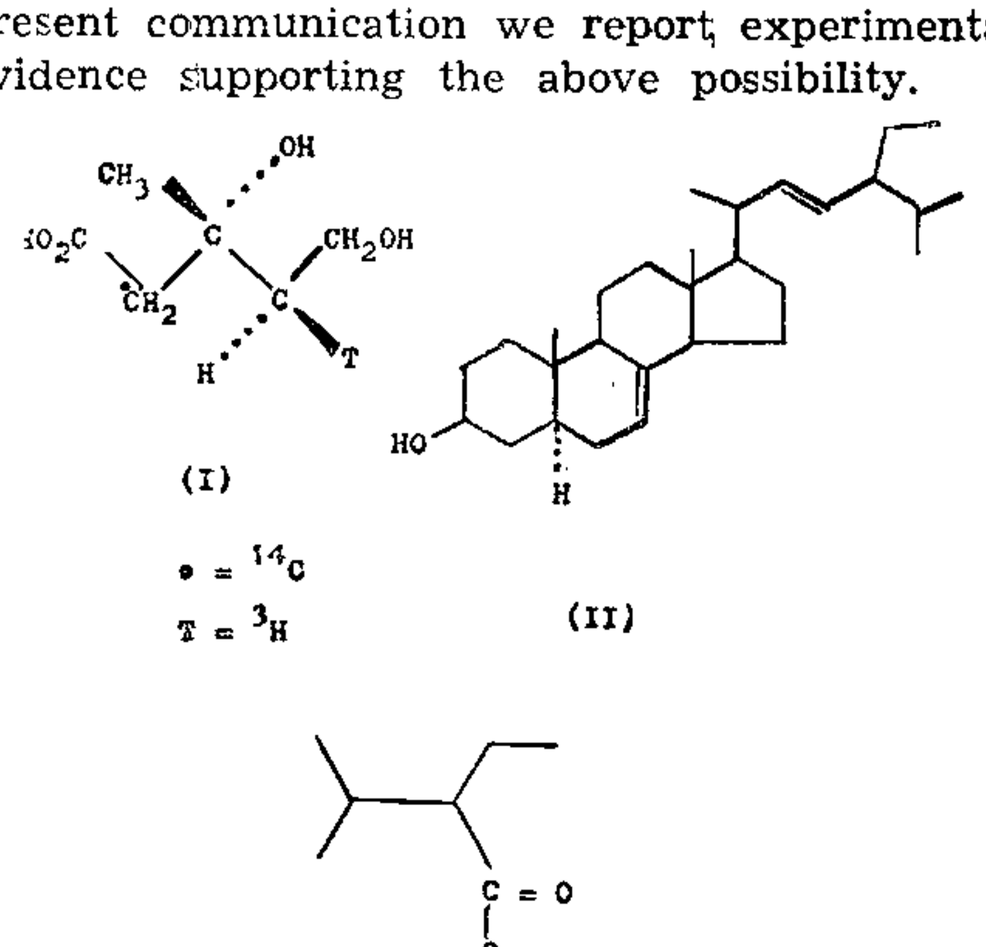

FIG. 1

(3RS)-[2-^{14}C-(4R)-4-$^{3}H_1$]-mevalonic acid (I) ($^{3}H/^{14}C$ ratio 5·37 ; 50 μc of ^{14}C) was