Each treatment produced changes in the area of the nuclei, with confidence limits below 1 per cent, compared with nuclei from the resting mice. It is clear, therefore, that the different types of behaviour were reflected in changes in the dimensions of the nucleus, as assessed by the measurement of the area in transverse section. Although these changes probably reflect a change in volume, this is not certain in the absence of complementary measurements of the nuclei in sections cut longitudinally to the axis of the brain.

The standard deviation for each treatment group shows wide variation between treatments. It should be noted that whenever the activity includes periods of exhaustion ('Picrotoxin' induces intermittent seizures separated by periods of exhaustion), the variation is high. This could reflect the influence of recovery processes proceeding at different rates in each mouse, the different parts of the brain, or the separate nuclei. An analysis of variance revealed that only in the instance of swimming to exhaustion was there a significant difference between mice as well as a significant contribution to variance from parts of the brain (areas) and from area/mouse interaction. Significant differences between areas occurred in treatments 3 and 7, and area/mouse interactions were significant in 3 and 6.

The full details and analysis of results will be published elsewhere.

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Action of Ascorbic Acid on Corticotropin

Holzbauer and Walter[1] have recently reported that the adrenal ascorbic acid – depleting properties of corticotropin are destroyed by treatment with ascorbic acid at room temperature for 18–20 hr. The corticotropin preparation used by these workers had a potency of 3 I.U./mgm. In view of the potential importance of their observation, we have repeated their experiment using highly purified corticotropin. The preparation used was a sample of corticotropin A_1 (for nomenclature, see Dixon and Stack-Dunne[2]), made in this laboratory by ion-exchange chromatography of an oxycellulose concentrate of pig corticotropin. This preparation had a potency of 110 I.U./mgm. when assayed by the adrenal ascorbic acid – depletion method of Sayers, Sayers and Woodbury[3].

A solution of corticotropin A, containing 0·4 I.U./ml. in Ringer solution, was divided into two portions. One portion was kept as control while ascorbic acid was added to the other portion to give a final concentration of 0·084 mgm./ml. Both solutions were kept for 18 hr. at 25°. These conditions and concentrations are identical with those of Holzbauer and Walter[1]. Both solutions were diluted for bioassay by the adrenal ascorbic acid – depletion method. The solution treated with ascorbic acid assayed 147 per cent of the control (p = 0·95, 76–282 per cent).

Our results are thus in contradiction to those of Holzbauer and Walter. The increase in potency found is of doubtful significance, but could be explained by the protective action of ascorbic acid against autoxidation of the hormone[4].

The difference in the observations is most probably explained by the low potency of the preparation used by Holzbauer and Walter. The ascorbic acid may act by activation of proteolytic enzymes present in the impure preparation. The presence of such enzymes in crude pituitary extracts has already been demonstrated[5].

It must be concluded, therefore, that ascorbic acid has no effect on purified preparations of corticotropin.

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Prof. Morris's results are in good agreement with the observation of Holzbauer and Rigler[1] that the time necessary for the inactivation by ascorbic acid of the melanophore-expanding activity of pituitary extracts increased with increasing purity of the preparation. In these experiments, however, 88 per cent of the melanophore activity disappeared in 28 hr. even when the activity of the preparation was 150 I.U. of corticotropin per mgm. It is regrettable that a highly active preparation was not available to us when the experiments with K. Walter were carried out.

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Reactions between Amino-Acids, Organic Acids and Sugars in Freeze-dried Apricots

Haas and Stadtman[1] separated an apricot concentrate (30 per cent solids) into cationic, anionic and neutral fractions and showed that browning occurred when any two fractions were combined and stored at 57° C. The reactions of individual cations, anions and neutral compounds in freeze-dried apricot *purée* stored at 25° C. and 70 per cent relative humidity (79 per cent solids) have now been studied. After sixteen months, this material was a mid-brown colour. It contained 1-deoxy-1-(N-amino-acid)-fructoses[2] and traces of 2-deoxy-2-(N-amino-acid)-glucoses[3], together with sucrose, glucose and (traces of) fructose half-esters of malic and citric acids. These compounds replaced almost quantitatively the apparent losses of free amino-acids and malic and citric