The concentration of ascorbic acid in the muscle declines significantly on starvation (Table). It is known that on starvation the animals live on the component tissues of their own body for energy purposes. The carbohydrate store of the liver and muscle of the scorpion *Palamneus bengalensis* has also been shown to decline on starvation (SINHA[5]). For ascorbic acid synthesis, the animal depends on the dietary source of hexoses. Due to rapid consumption of carbohydrates during starvation, the animal fails to get a sufficient amount of hexoses. Hence, the synthesis of ascorbic acid decreases on starvation as indicated by the low content of ascorbic acid in the starved scorpions.

Zusammenfassung. Bei 12 Tage hungernden Skorpionen (*Palamnaeus bengalensis*) wurde die Ascorbinsäure-Konzentration im Pedipalpen-Muskel mit 2,4-Dinitrophenyl nach ROE[4] bestimmt und dabei eine signifikante Abnahme der Ascorbinsäure festgestellt.

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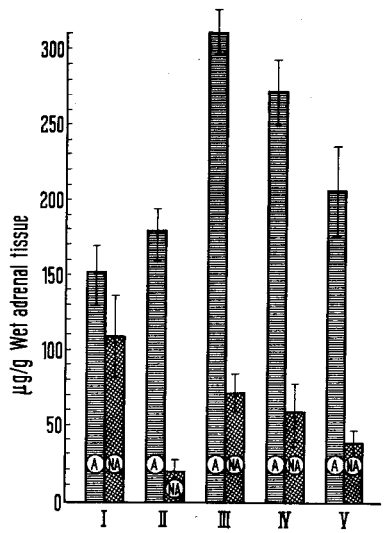
Effect of Aldosterone, Glucagon and Growth Hormone on the Catechol Amine Content and the Evolution of Chromaffin Hyperplasia in Guinea-Pigs

The neuro-humoral control and growth of chromaffin tissue are but little known[1]. There are some experiments concerning the release of catechol amines (adrenalin and noradrenalin) under the influence of hormones (insulin[2], cortisone and hydrocortisone[3], and glucagon[4]) or some drugs (reserpine and chloropromazine[5]) from the adrenal medulla of cats, rats and dogs. In our previous experiments[6,7], we noted the occurrence of chromaffin hyperplasia or pheochromocytoma after the administration of large doses of oestrogens and growth hormone in rats or guinea-pigs. The present paper studies the action of aldosterone, glucagon and growth hormone on the distribution of catechol amines and the evolution of medullary chromaffin hyperplasia induced by oestradiol administration in adult guinea-pigs.

Material and Method. The investigations were carried out in 50 adult guinea-pigs (males and females) which were divided into five experimental groups as follows: 1) 10 guinea-pigs served as controls. 2) 10 received concomitantly during about 4 months the following treatment: twice a week 300 μg α-oestradiol i.p. and 75 μg (75 γ) glucagon (Lilly) i.m., the total amount being 9500 μg oestradiol + 2400 μg glucagon for each guinea-pig. 3) 10 received simultaneously for about 4 months: twice-weekly doses 300 μg oestradiol i.p. and 16 μg d-Aldosterone (Aldocorten, CIBA) i.m., the total dose being 9500 μg oestradiol + 500 μg d-Aldosterone for each guinea-pig. 4) 10 received each week 300 μg oestradiol i.p. and 12 Evans units (EU) of growth hormone intramuscularly, the total dose being 9500 μg oestradiol + 400 EU growth hormone. 5) 10 received only twice a week 300 μg oestradiol by the intraperitoneal route, the end dose being 9500 μg oestradiol, to induce a medullo-chromaffin hyperplasia. After 4 months, all guinea-pigs were killed by light ether anaesthesia and the adrenal glands were collected: one adrenal gland for estimation of the adrenalin and noradrenalin content by an improved technique of VON EULER and LISHAJKO[8], and the other adrenal gland in order to evaluate the intensity degree of chromaffin hyperplasia after fixation in Bouin fluid and staining with PAS-hematoxyline eosin or the chromaffin reaction after fixation in chrome salt fixatives. Recent histochemical techniques[9] allow a good identification of adrenalin and noradrenalin-storing cells in adrenal medulla.

Results. The biochemical methods show a significant change in the adrenalin and noradrenalin content of the adrenal medulla of guinea-pigs after administration of these hormones. Thus a striking increase of adrenalin was observed after aldosterone and growth hormone administration, a moderate increase after oestradiol, and



Variation of adrenalin (A) and noradrenalin (NA) content in the adrenal medulla of control guinea-pigs (I), treated with oestradiol and glucagon (II), with oestradiol and aldosterone (III), oestradiol and growth hormone (IV), and oestradiol only (V). The vertical lines at the top of the columns indicate the standard deviation [S.E. $= \pm \sqrt{\varepsilon d^2/n(n-1)}$].

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non-significant changes after glucagon; but the marked decrease of noradrenalin in comparison with the controls was observed after glucagon or oestradiol administration (Figure). About the same results were obtained by histochemical methods concerning the distribution of adrenalin and noradrenalin-storing cells.

The histological pattern shows the occurrence of an intense chromaffin hyperplasia after oestradiol administration. Simultaneous treatment with glucagon markedly reduced oestradiol-induced hyperplasia in the adrenal medulla.

Aldosterone administration induced a moderate stimulation of medullo-chromaffin hyperplasia. The most intense stimulation of the chromaffin hyperplasia was observed after concomitant administration of oestradiol and growth hormone (4); its appearance is that of a pheochromocytoma and hemorrhagic cyst.

Discussion. Our findings suggest that glucagon, aldosterone and growth hormone significantly influence the evolution of the oestradiol-induced chromaffin hyperplasia and the catechol amine content of the adrenal medulla of guinea-pigs. The administration of large doses of insulin induced in rats' and cats' adrenal medulla an exclusive depletion of the adrenalin; in dogs, i.v. administration of glucagon-free insulin induces a moderate increase in the catechol amine output. From our experiments, a progressive increase may be observed in the adrenalin content after glucagon, oestradiol, growth hormone and aldosterone administration in comparison with the controls, and a marked decrease in the noradrenalin content after glucagon, oestradiol, growth hormone and aldosterone administration. This variation runs parallel to the histological changes in the volume and structure of the adrenal medulla.

Résumé. La variation du taux des catécholamines (adrénaline et noradrénaline) et les modifications histologiques ont été étudiés après administration d'aldostérone, de glucagon et d'hormone de croissance sur l'hyperplasie médullo-chromaffine provoquée chez des cobayes par l'œstradiol.

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Association of Human Satellited Chromosomes

The ability for human satellited chromosomes to lie in close proximity to one another at metaphase has been observed by Harnden[1]. Shaw[2] further reported his observations in which chromosome No. 1 (Denver System) also formed a close association with the satellited chromosomes. Ferguson-Smith and Handmaker[3] defined this phenomenon, and later (1963)[4] showed the distribution between these chromosomes to be non-random, with a high frequency of associations between the short arms of the satellited chromosomes and specific regions of non-satellited chromosomes. Merrington and Penrose[5], by statistically analysing 62 cells, showed that the acrocentric chromosomes (which are usually satellited) tend to lie closer together than would be expected if the chromosomes were randomly arranged at metaphase. Frøland[6], in a study of 6458 cells, observed that the mean association per cell was 1.32 and that there was no difference between the age, sex, or karyotype of the subjects. Reitalu[7], in a more detailed study, showed that (a) only 6 satellited chromosomes out of the normal 10 took active part in satellite associations, (b) the chromosomes which exhibited this phenomenon were non-homologous chromosomes, and (c) the association frequency in the large and small acrocentric groups was the same.

Following this observation of Reitalu, the present study was undertaken to ascertain (1) whether or not all ten acrocentric chromosomes took part in associations, (2) whether or not homologous chromosomes took part in the association, and (3) to calculate the frequency with which individual and homologous chromosomes took part (if indeed they did) in satellite association.

The criterion used for the definition of satellite association is the same as that described by Ferguson-Smith and Handmaker[3].

Materials and methods. For this study, data were obtained from a total of 300 cells which were karyotyped. These cells were all obtained from blood leucocyte cultures set up by the methods described by Bishun et al.[8] and Robinson et al.[9], with some minor modifications. The metaphase cells selected were the best spread, with good morphology and with the modal chromosome complement of the patients. These were the cells usually

Table I. Frequencies of different chromosomes

Single chromosomes					
Chromosome No.	13	14	15	21	22
Frequencies (%)	11.2	10.9	11.2	14.7	9.6
Homologous chromosomes					
Frequencies (%)	6.02	8.9	5.7	15.8	6.3

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