be reduced (females).† Occasionally the adrenals of rats in advanced stages of acute starvation do show enlargement. This is especially the case in moribund or dead animals. These glands are usually discolored (grey, maroon) and analysis indicates that the increased size is due largely to increased water content. The adrenals of moribund guinea pigs are heavier than glands of starved pigs with similar body weight loss. This "death hypertrophy" involves an increase in both water and solids with no significant alteration in the normal percentages of these constituents.

The administration of DOCA (10-15 mg daily) decreases the size of the adrenals in acutely starved and fed rats. In either instance, a significant diminution in solid content occurs. The color of these adrenals is usually pale yellow even in terminal stages of starvation. DOCA in the guinea pig is without effect on adrenal size in the fed animal, and fails to prevent adrenal hypertrophy in the starved guinea pig. Although only several analyses have been done in this series of animals, it appears that the solid and water content of the adrenals are not affected by DOCA injection.

A detailed histologic study is now being made of the adrenal and pituitary glands of the animals described in this report. These observations together with a more extensive discussion of the problem and consideration of the literature will be published at a future date.

Summary. The response of the adrenal gland to comparable degrees of acute starvation differs markedly in the rat and guinea pig. The guinea pig adrenal displays absolute hypertrophy with significantly increased solid content. In the rat, adrenal enlargement is a variable response and, when it occurs, is characterized by discoloration and percentage increase in water content. The solid content is unchanged or reduced. DOCA in high doses decreases the size and the solid content of the rat adrenal in the fed or acutely starved animal of either sex. DOCA in the guinea pig fails to affect adrenal size in the fed animal, and is ineffective in preventing the adrenal hypertrophy of starvation.

† The ratio of adrenal solid content to final body weight, however, is increased over the pre-starvation ratio. In this sense, the starved rat adrenal displays relative rather than absolute hypertrophy.

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Effect of Desoxycorticosterone upon Hypophyseal Corticotrophin Production.

FRANCISCO MOYA* AND HANS SELYE.

From the Institut de Médecine et de Chirurgie Experimentales, Université de Montréal, Montreal, Canada.

The atrophy of the adrenal cortex produced by chronic administration of cortical extract was shown to be counteracted by the simultaneous administration of adrenocorticotrophic hormone (ACTH);[1] presumably the corticoids merely inhibit hypophyseal corticotrophin production without rendering the cortex insensitive to the trophic hormone. A similar involution of the adrenal cortex in both the rat and the mouse was obtained with desoxycorticosterone acetate (DCA).[2] Sayers and Sayers[3] showed that the depletion of the adrenal ascorbic acid which follows the exposure of rats to stress can be prevented by the administration of

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1 Ingle, D. J., Higgins, G. M., Kendall, E. C., *Anat. Rec.*, 1938, **71**, 363.

2 Selye, H., *J. Am. Med. Assn.*, 1940, **115**, 2246.

3 Sayers, G., and Sayers, M. A., *Endocrinology*, 1947, **40**, 265.

TABLE I.

Exp.	Group	No. of rats	Treatment	Adrenal ascorbic acid: mg/100 g fresh tissue (mean ± stand. error)	Adrenal ascorbic acid: % decrease from normal control
1	I	9	DCA + cold	320 ± 12.0	12
	II	10	Cold	320 ± 13.6	12
	III	10	Normal control	362 ± 12.3	—
2	I	10	DCA + cold	259 ± 15.0	18
	II	10	Normal control	317 ± 14.4	—

DCA. They concluded that the production of ACTH by the pituitary is inversely proportional to the concentration of corticoids in the body fluids and corresponds to the cortical-steroid requirements of the peripheral tissues.

In the present communication we endeavor to show that this mechanism of pituitary regulation is not effective in the presence of very high concentrations of DCA in the body fluids.

The anesthetic effect of this steroid[4,5]—which is only obtained with excessive doses—served to establish that the amount administered was greatly in excess of the animals' requirements even under stress. The adrenal response to the treatment was estimated by determining the ascorbic acid concentration in the adrenals of all animals at the end of each experiment.[3]

Thirty male albino rats (80-100 g), fasted for 24 hours, were divided into 3 groups of 10 rats each. The first group received intraperitoneally 6 mg of DCA dissolved in warm corn oil and repeated doses of 2 mg were similarly administered to each animal to maintain it in a state of anesthesia during the 6 to 7 hours of the experiment. These rats were intermittently exposed to cold (10 ± 1°C) for one hour at a time and between exposures they were allowed to recuperate at room temperature for about half an hour. During the rest periods all animals about to awaken received an additional dose of 2 mg of DCA and those rats which were profoundly narcotized were allowed longer rest periods. Most of the animals were exposed for a total of 4 hours and none for less than 3; the total dosage of DCA administered ranged from 12 to 14 mg per animal. Group 2 received exactly the same cold treatment as group 1 but no DCA was administered to it. Group 3 received no treatment and thus served as an absolute control. At the end of the experimental period all animals were sacrificed and the adrenal ascorbic acid determined by Carruthers's method.[6] The results are summarized in Table I as Experiment 1.

Experiment 2 represents the results obtained with a similar experimental arrangement in which the cold-exposure period was increased to 4½ hours and the DCA dosage to 16 mg per animal.

Both in Experiment 1 ($P<0.05$) and in Experiment 2 ($P<0.02$) the difference in adrenal ascorbic acid between the DCA-treated, cold-exposed group and the normal controls is significant.

The results reported above show that in the presence of a high tissue concentration of DCA, the pituitary of animals exposed to stress still elaborates sufficient ACTH to induce a significant decrease in adrenal ascorbic acid. In Experiment 1 the exposure to cold alone (Group II) brought about a decrease of the same order as in the animals (Group 1) which simultaneously with the cold treatment received the DCA overdosage. These findings are incompatible with the assumption that pituitary adrenocorticotrophin discharge is regulated solely by the corticoid concentration in the body fluids. Our observations point to the possibility that in the presence of ex-

[4] Selye, H., PROC. SOC. EXP. BIOL. AND MED., 1941, **46**, 116.

[5] Selye, H., *Anesth. a. Analg.*, 1942, **21**, 42.

[6] Carruthers, C., *Ind. Eng. Chem. Ana. Ed.*, 1942, **14**, 826.

cessively high corticoid concentrations—as at times of very acute stress—the regulation of the corticotrophin discharge may become relatively independent of circulating cortical hormone(s).

Summary. In the rat large doses of desoxycorticosterone acetate do not prevent the decrease in the concentration of adrenal ascorbic acid which accompanies exposure to cold. It is suggested that during severe stress, corticotrophin discharge is conditioned by factors other than mere lack of corticoids.

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Adenochrome-Like Pigment of the *Polyzoa bugula neritina* (L).

GILBERTO G. VILLELA.

From the Division of Chemistry and Pharmacology, Instituto Oswaldo Cruz, Rio de Janeiro, Brazil.

Bugula neritina (L), a *Polyzoa* (Bryozoa) of the order *Cheilostomata,* presents a dark purple pigment not commonly found in other animals of the same order.[1] As far as we know no study of the pigments of Polyzoa has yet been reported.[2,3] Our specimens were collected in the bay of Rio de Janeiro near the Hydrobiological Station of the Institute.

The pigment of *Bugula* appears morphologically in the granular corpuscles of the distal part of the *zooecium* (Fig. 1) and is easily extracted with distilled water. The extract is wine-red in color and shows a Tyndall effect and a pH of 6.8.

The physical and chemical properties investigated showed that the pigment closely resembles the adenochrome isolated by Fox and Updegraff from the branchial hearts of the *Octopus*.[4] The absorption spectrum, however, exhibits a different maximum (545 mμ as compared with 505 mμ for the adenochrome).

Fox and Updegraff suggested that adenochrome is probably a waste product, as shown by its localization in the excretory tissues of the *Octopus* and the apparent storage of most of its nitrogen in the amide state, releasable as free ammonia in the presence of KOH.[4] In *Bugula* the pigment is accumulated in the distal part of the *zooecium* and around the *brown body* which is known to be an excretory waste mass. However, until now we have been unable to establish any relation between the pigment and the excretory function of *Bugula*. Other water-soluble pigments behaving as indicators of pH have been reported by Crozier in two sponges and a nudibranch but unfortunately no detailed chemical and spectroscopical data were published.[5]

Isolation and properties of the pigment. Animals from a colony (about 30 g) were washed with filtered sea water to free them of associated organisms, then weighed and extracted with boiling distilled water for 5 minutes. The residue was reextracted with boiling water until the solution was only slightly yellow. The extract was filtered and washed with petroleum ether (b.p.40-60°). The aqueous phase was made alkaline (pH 11.0) with KOH and the flocculent purple pigment was separated by centrifugation. The precipitate was dissolved in 10% acetic acid, then diluted with a few ml of

[1] Marcus, E., *Bol. Fac. Fil., São Paulo,* 1937, **1**, 67.

[2] Lederer, E., *Biol. Rev.,* 1940, **15**, 273.

[3] Fox, D. L., *Ann. Rev. Bioch.,* 1947, **16**, 443.

[4] Fox, D. L., and Updegraff, D. M., *Arch. Biochem.,* 1942-43, **1**, 339.

[5] Crozier, W. J., *J. Biol. Chem.,* 1918, **35**, 455.