agement, and to Prof. N. K. Adam for a gift of pure fatty acids.

Since submitting the above, our attention has been directed to a paper by Savary and Desnuelle[9], who employ a slightly different oxidation procedure for the estimation of oleic and linoleic acids in simpler mixtures. It is hoped to examine this method more fully in a later communication.

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Effect of Cortisone on the Neonatal Rat

Neonatal rats have been treated with cortisone acetate (Merck ; Roussel) in doses of 0·25–5 mgm./day subcutaneously, control litter mates being given the same dosage of suspending medium. The experiments were undertaken primarily to test the effect upon microglial immigration and transformation in the brain. Hortega's neonatal 'fountains' were found to be completely suppressed within 24–36 hr. of a single dose of 2·5 mgm. of cortisone, and in older animals myelination was retarded.

The treated animals showed a striking failure to grow, or in some cases a retarded growth followed by loss of body weight ending in death (Fig. 1). Such animals presented all the signs of pituitary failure (Fig. 2). They were small and shrivelled, with very little subcutaneous fat and a marked failure of hair growth. Without exception they showed a considerable degree of exophthalmos. In some cases their eyes actually opened before those of the control litter mates. Their appearance corresponded with that produced in chick embryos by Karnofsky *et al.*[1] by cortisone treatment. There was a general decrease

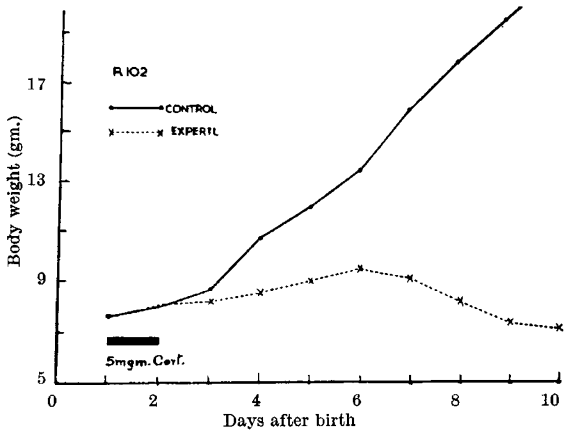

Fig. 1

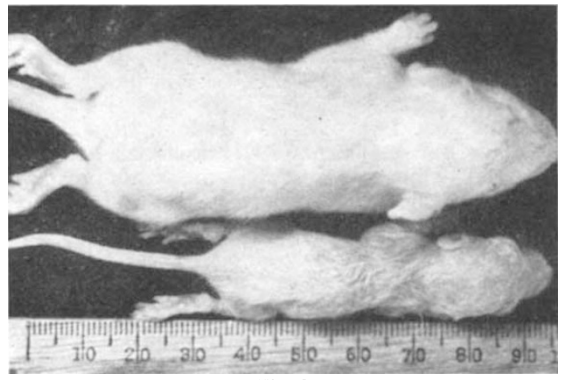

Fig. 2

in lymphoid tissue, and pituitaries were small and degenerate on histological examination. A detailed account of these changes will be published elsewhere.

Cortisone is known to depress adrenocorticotrophic activity of the anterior lobe of the pituitary gland[2]. It is also known to inhibit growth of the embryo chick after the eighth to tenth day of incubation[1]. On the other hand, it has been held not to diminish thyrotrophic secretion of the adenohypophysis but possibly, indeed, to increase it[3].

Exophthalmos has been produced in adult rats by prolonged exhibition of cortisone[4]. Such treatment may produce thyroid depression—"corticogenic hypothyroidism"—so that the exophthalmos might be attributable to thyrotrophic activity of the pituitary, since the thyrotrophic hormone is known to be capable of producing the condition in rats[5]. Aterman and Greenberg[4], however, are inclined to attribute the exophthalmos produced by cortisone in the rat to a direct alteration of the physico-chemical state of the tissues within the orbit, and there is then no necessity for assuming a stimulating effect of cortisone on thyrotrophic activity of the adenohypophysis in the present experiments.

Two points may be considered: (1) the simultaneous 'damping down' by cortisone of adrenotrophic activity and growth hormone of the anterior pituitary invites the speculation that the two principles may, under physiological conditions, be part of an articulated complex at the time of their elaboration[6]; (2) there may be a possibility of utilizing 'functionally hypophysectomized' neonatal rats as test animals for adrenocorticotrophic hormone activity—a modification of the method of Sayers, Sayers and Woodbury[7].

I wish to thank Dr. Max Reiss for helpful discussion of results. The cortisone used was supplied by the Medical Research Council, and the work was carried out while in receipt of financial aid from the Council.

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