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¹ Perrott, P. E., *Trans. Brit. Mycol. Soc.*, **38**, 247 (1955).

² Raper, J. R., *Science*, **85**, 342 (1937).

³ Martin, J. P., *Soil Sci.*, **69**, 215 (1950).

Aldosterone and Experimental Hepatic Necrosis

THERE is some evidence that aldosterone, besides its potent effects on electrolyte metabolism, also possesses some cortisone-like activity. The data available, however, are rather scanty and somewhat contradictory. In recent years an effect of cortisone and of related hormones has been described which appears to be characteristic for the 'glucocorticoid' group of adrenocortical steroids. If given in a daily dose of 1 mgm. per 100 gm. body-weight, cortisone doubles or trebles the survival-time of rats fed a diet containing yeast as the only source of protein without, however, significantly altering the incidence of fatal liver necrosis caused by this deficient diet^{1,2}. Characteristically, this effect of cortisone still occurs if the administration of this hormone is started as late as the last third of the experimental period³. Of the hormones examined so far only cortisone, cortisol and adrenocorticotrophic hormone have significantly prolonged the survival time; progesterone, oestrogen, testosterone, oestrogen plus testosterone, and deoxycortone acetate have been without effect (Aterman, K., unpublished work). Corticosterone is now being investigated. In view of the lack of data on the biological actions of aldosterone it was considered of interest to see how its activity would compare with that of cortisone and of deoxycortone acetate in affecting the course of experimental hepatic necrosis.

14 pairs of male rats bred in the Department of Anatomy, Birmingham, weighing 31–41 gm., were weaned on the twenty-first day of life, the first day of the experiment, and were given the 'necrogenic yeast diet' of Lindan and Himsworth⁴. On the eighteenth day of the experiment the administration of D-X-aldosterone-21-monoacetate (Ciba) to one brother of each pair was started. It is apparent from Table 1 that the daily dose of 10 µgm. of aldosterone had

no effect on the survival-time, but a dose of 50 µgm. daily prolonged the survival-time in every animal, the effect being somewhat more pronounced if the dose of aldosterone was increased to 75 µgm. Of interest also is that the treated rat of group 14 died within 30 hr. of the last dose of aldosterone having been given when all the aldosterone available had been used up. The cause of death in every case was necrosis of the liver. Statistical analysis of the differences obtained shows a probability value of less than 0.02, if all the animals treated with aldosterone are considered, and a probability value of less than 0.01, if the four rats given the lowest dose of aldosterone are excluded on the assumption that the amount of aldosterone given was too small. This assumption appears justified if the increase in survival-time with increasing doses of aldosterone, which was statistically just significant ($P < 0.05$), is taken into consideration. These results, therefore, suggest that in the system examined here aldosterone has some 'glucocorticoid' activity and differs significantly from deoxycortone acetate which in doses of 0.5 and 2.5 mgm. daily has been completely ineffective. A more extensive study of this effect will, however, have to be undertaken when more aldosterone becomes available.

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¹ Schwarz, K., *Science*, **113**, 485 (1951).

² Aterman, K., *Endocrinol.*, **60**, 711 (1957).

³ Aterman, K., Meeting of Federation of American Societies for Experimental Biology, Chicago, Illinois (1957).

⁴ Lindan, O., and Himsworth, H. P., *Brit. J. Exp. Path.*, **31**, 651 (1950).

Atypical Segregation of Haptoglobin Types in Man

Smithies and Ford-Walker¹ have suggested that the three haptoglobin phenotypes, namely, 1-1, 2-1, and 2-2, detected by starch gel electrophoresis are determined by a pair of allelic genes *Hp*¹ and *Hp*². The findings in the eighteen families reported by Smithies and Ford-Walker and also in 106 families reported later by Galatius-Jensen² were fully consistent with this hypothesis. Furthermore, the frequencies of the phenotypes in different populations agree well with those expected assuming a Hardy-Weinberg equilibrium²⁻⁴.

We have recently investigated a further 107 families, and while our results for the most part support this simple genetical hypothesis a small number of exceptions to the expected pattern of segregation were encountered.

The electrophoresis was carried out on plasmas to which sufficient haemoglobin to saturate the haptoglobins had been added. The discontinuous buffer system described by Poulik⁵ was used. The haptoglobin-haemoglobin complexes were afterwards detected with a benzidine reagent. This technique reveals an even more complicated pattern of haptoglobin components than has previously been reported^{2,6}, but no ambiguity in classification was encountered. The kind of patterns which were obtained are shown diagrammatically in Fig. 1. While only a single *HbHp* complex can be detected

Table 1. THE EFFECT OF ALDOSTERONE ON THE SURVIVAL-TIME (IN DAYS) OF LITTERMATE RATS FED ON THE 'NECROGENIC YEAST DIET'

Pair No.	Control	Aldosterone		Difference in survival-time
	Days	Dose	Days	
1	32	10 µgm.	41	+ 9
2	31		26	- 5
3	32		26	- 6
4	30		36	+ 6
5	23	50 µgm.	31	+ 8
6	23		24	+ 1
7	23		30	+ 7
8	26		31	+ 5
9	22		25	+ 3
10	28		29	+ 1
11	26		32	+ 6
12	22	75 µgm.	40	+ 18
13	31		33	+ 2
14	28		42	+ 14