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SHORT COMMUNICATION

PLASMA ALDOSTERONE CONCENTRATION IN SODIUM DEPRIVED MICE OF TWO STRAINS

V. E. PAPAIOANNOU* and R. FRASER

Department of Genetics, Cambridge, England and MRC Blood Pressure Unit, Western Infirmary, Glasgow, Scotland

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SUMMARY

Plasma aldosterone concentrations were measured in male and female CBA/FaCam and Peru mice, fed a low sodium, high potassium diet. These concentrations were compared with previously reported normal diet concentrations. The experimental diet increased plasma aldosterone concentration in both strains and sexes but there were no significant differences between either strains or sexes in spite of genetic differences in the extent of the adrenal zona glomerulosa.

In a previous report [1] evidence was presented indicating that CBA/FaCam and Peru mice probably do not differ significantly in their rates of aldosterone production in spite of a genetic difference in the degree of development of the adrenal zona glomerulosa [2, 3]. This report is an extension of those findings.

Male and female mice from the strains CBA/FaCam (CBA) and Peru were raised under standardized conditions [3]. Plasma was collected and stored as previously described [1] from mice maintained for 25 days on a low sodium diet (Sodium Deficient Test Diet, Kodak Ltd.) with 3% KCl as a drinking fluid. This diet was sufficient to cause hypertrophy of the zona glomerulosa in CBA mice [2] and to cause an increase in *in vitro* aldosterone secretion in both strains [1]. Plasma was pooled from at least nine mice in each group; the age range was 8–40 weeks. In this study plasma aldosterone concentrations were measured by the radioimmunoassay technique of Mayes *et al.* [4]. Sensitivity of this technique was approx 0·05 ng/ml or 0·1 ng/sample.

The plasma aldosterone concentrations for male and female CBA and Peru mice fed a low sodium, high potassium diet are presented in Table 1 along with the previously reported plasma aldosterone concentrations in normally fed mice measured by a double isotope derivative technique [1]. The effect of the modified diet was to increase plasma aldosterone concentration in both strains and sexes by more than 10-fold in every case. Comparison of the results obtained with two different methods is justified in this case since the techniques have been compared by means of repeated assays of a plasma pool and also by analysis of a wide range of plasma samples. No significant difference was found in results obtained by these methods [5]. Analysis of variance with one observation per cell [6] was used on the present results converted to logs (Table 2). The effect of diet is significant, $P < 0{\cdot}05$ but not the effects of strain or sex, $P > 0{\cdot}05$.

* Present address for correspondence: Physiological Laboratory, Downing Street, Cambridge, CB2 3EG, England.

Table 1. Plasma concentrations of aldosterone in male and female mice from the strains CBA and Peru, fed either a control or low sodium, high potassium diet

Genotype	Number of mice pooled	Age range (days)	Mean age (days)	Plasma concentration of aldosterone (ng/ml)
Control diet				
CBA ♂	68	57–105	83·7	0·89
CBA ♀	101	57–237	83·5	0·40
Peru ♂	121	48–198	92·2	1·05
Peru ♀	159	54–196	111·7	0·74
Experimental				
CBA ♂	9	78– 84	79·8	10·42
CBA ♀	22	58– 74	65·5	11·67
Peru ♂	46	60–145	90·6	10·89
Peru ♀	133	58–290	123·3	10·09

Table 2. Analysis of variance of the plasma concentrations of aldosterone presented in Table 1.

Source of variation	d.f.	ssq	$F_{1,1}$
Total	7	2·8174	
Strains	1	0·0125	1·1
Diet	1	2·7139	240·0*
Sex	1	0·0259	2·3
Strain × diet	1	0·0206	1·8
Strain × sex	1	0·0024	< 1·0
Diet × sex	1	0·0308	2·7
Residual (strain × diet-× sex)	1	0·0113	

* $P < 0·05$.

These observations are consistent with *in vitro* aldosterone production rates for CBA and Peru mice fed either the deficient or the control diet [1] and are further evidence that the larger zona glomerulosa in CBA mice does not reflect a difference in maximal capability for aldosterone production.

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REFERENCES

1. Stewart J., Fraser R., Papaioannou V. and Tait A.: *Endocrinology* **90** (1972) 968–972.
2. Shire J. G. M.: *Endocrinology* **85** (1969) 415–422.
3. Shire J. G. M. and Spickett S. G.: *Endocrinology* **39** (1967) 277–284.
4. Mayes D. S., Furuyama S., Kem. D. C. and Nugent C. A.: *J. clin. Endocr. Metab.* **30** (1970) 682–687.
5. Fraser R., Guest Sheena and Young Jessie: *Clin. Sci. Mol. Med.* **45** (1973) 411–415.
6. Scheffe H. *The Analysis of Variance*. John Wiley & Sons, Inc. London, 1959, p. 98.