SHORT COMMUNICATION

THE EFFECTS OF HYPOPHYSECTOMY AND TESTOSTERONE ON THE ACID PHOSPHATASE ACTIVITY OF THE SEMINAL VESICLE AND VENTRAL PROSTATE OF CASTRATED RATS

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Summary: Rats were hypophysectomized-castrated on day 70 of age and were treated with testosterone propionate (TP) during 80-90 days of age. Acid phosphatase activity of seminal vesicle and ventral prostate was studied both in presence and absence of pituitary gland. The growth and acid phosphatase activity of the organs were significantly increased in non-hypophysectomized-castrated animals when chalanged with TP; this action was inhibited in absence of hypophysis.

Key words: hypophysectomy seminal vesicle acid phosphatase activity castrated rats testosterone

It has been reported that a rise in the activity of acid-phosphatase of the adenohypophysis occurs concurrently with an increase in the secretion of gonadotrophins in birds and rat (2 and 5). Further in rats the highest acid phosphatase activity was seen in the anterior lobe in PAS positive gonadotrophs (5). It seems likely that acid-phosphatase of the adenohypophysis has a bearing on the reproductive organs.

A question arises here as to whether the acid phosphatase activity of the accessory organs is in some way controlled by the adenohypophysis. The present experiments were concerned with the examination of the responsiveness of seminal vesicle and ventral prostate gland acid-phosphatase activity of castrated rats treated with testosterone-propionate both in presence and absence of pituitary.

Forty seven wistar rats from the randomly mated closed colony maintained in the Anatomy Department were used. Rats were hypophysectomized by a parapharyngeal approach. Completeness of the hypophysectomy was judged from continuing loss of body weight and microscopic examination of sella turcica at post mortem. Hypophysectomized rats were castrated on day 3 of post-operation under ether anaesthesia and were treated as outlined in Table I.

At the time of autopsy, the rats were killed by decapitation, seminal vesicle and ventral prostate were taken out weighed and assayed for acid phosphatase activity. The assay method used was based on that of Berthet and de Duve (1) using final concentrations of 3 mM-P-

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TABLE I: Effects of testosterone* on the acid phosphtase activity of castrated and hypophysectomized-castrated rats (means ± S.E.).

Group No.	Treatment	Body wt. g	Seminal vesicle weight mg/100 g body wt.	Ventral prostate weight mg/100 g body wt.	Acid Phosphate activity (μg protein/hr/mg wet weight tissue) Seminal vesicle	Ventral prostate
1.	Control (10)	287 ± 13	3.281 ± 0.29	1.536 ± 0.32	265.7 ± 20.6	303.7 ± 9.1
2.	Castration (10)	224 ± 12	2.114 ± 0.23§	0.225 ± 0.05§	159.7 ± 0.6§	171.7 ± 5.9§
3.	Castration + TP (10)	246 ± 12	2.859 ± 0.42**	1.999 ± 0.03**	253.0 ± 2.5**	341.3 ± 5.7**
4.	Hypophysectomy (7)	189 ± 10	1.387 ± 0.34§	0.571 ± 0.01§	129.4 ± 4.4§	126.7 ± 8.2§
5.	Hypophy. + Castration (5)	175 ± 6	1.289 ± 0.47§#	0.604 ± 0.21§#	137.1 ± 5.9§#	143.9 ± 2.7§#
6.	Hypophy. + TP (5) + Castration + TP (5)	218 ± 11	1.846 ± 0.57≠	0.408 ± 0.01≠	169.7 ± 11.3***	189.1 ± 5.7***

§ $P < 0.001$ compared with control
** $P < V0.01$ compared with group 2
# Not significant compared with group 4, 6
≠ Not significant compared with group 4,5
*** Not significant compared with group 5

Figures in parentheses represents the number of rats.
Acid phosphatase activity: means of six determinations.
All figures ± S.E.M.
*1 *mg*/day for 10 days begining on day 10 post-operation.

nitrophenyl phosphate adjusted to pH 5.6, as substrate, and sodium acetic acid buffer (0.01M; pH 5.0). An incubation period of 1 hr at 37°C was used. Sonication for 15 seconds was made to release the lysosomal acid-phosphatase enzyme. The reactions were terminated with 1M sodium hydroxide solution.

Protein present was determined by the method of Lowry *et al.* (3). The total acid phosphatase activity was calculated and expressed in absolute units of *μg* protein/hr/*mg* tissue.

Table I summarizes the changes in the accessory sex organ weights. It would appear that 1. Testosterone propionate brings about a significant increase in the weight of seminal vesicle, and ventral prostate of castrated-non-hypophysectomized rats. 2. Testosterone-propionate failed to increase the weight of the seminal vesicle & ventral-prostate in absence of the hypophysis.

The changes in the total acid phosphatase activity were as follows:—

1. The concentration of acid phosphatase in the seminal vesicle and ventral prostate were significantly raised in those animals which received testosterone propionate (group 3 Table I).
2. However, testosterone propionate did not bring about any significant increase in the acid phosphatase activity in these organs in the absence of hypophysis (Table I group 6).

Acid phosphatase activity in rat seminal vesicle and ventral-prostate is a quantitative measure of lysosomal function (4). After castration the enzyme activity is low. Testosterone administration restores it. Enzyme activity was further low after hypophysectomy.

Testosterone failed to influence the enzyme activity in hypophysectomized castrated

rats. It seems therefore, that the enzyme activity of seminal vesicle and ventral prostate is controlled by a pituitary gland factor.

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