

LOWERED REACTIVITY OF RATS TO ANDROGEN IN ALLOXAN DIABETES

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The reactivity of the accessory sex glands of castrated rats to testosterone propionate is considerably lower in animals with alloxan diabetes than in animals with an intact pancreas.

KEY WORDS: alloxan diabetes; androgens; accessory sex glands; castration.

It has been shown in vitro that the prostate gland taken from castrated rats reacts to androgen only in the presence of insulin [5]. The question arises, to what extent is insulin necessary for the androgen effect in vivo.

To study this problem experiments were carried out on castrated rats, some of them with alloxan diabetes.

EXPERIMENTAL METHOD

An aqueous solution of alloxan in a dose of 18 mg/100 g body weight was injected subcutaneously into male albino rats (mean weight 300 g). Two weeks later the testes were removed from these animals and also from intact rats and an oily solution of testosterone propionate was injected intramuscularly in a dose of 100 μ g daily for 7 days. Control animals received the same number of injections of oil. The diuresis and sugar concentration in the urine of the rats were determined in the course of the experiment [3]. At the end of the experiment the blood-sugar concentration was determined [2].

EXPERIMENTAL RESULTS

The rats receiving alloxan developed severe diabetes, reflected in a fivefold increase in the diuresis, and the appearance of glucosuria (up to 8.4 %) and hyperglycemia (316 mg%), and they also showed a decrease in body weight.

TABLE 1. Response of Rats of Various Experimental Groups to Androgen ($M \pm m$)

Animals	No. of animals	final body weight (mg)	Weight of seminal vesicles				Weight of prostate			
			absolute (in mg)	%	relative (in mg/100 g body weight)	%	absolute (in mg)	%	relative (in mg/100 g body weight)	%
Intact	9	344	1264,44 \pm 114	100	367,45 \pm 26,88		479,89 \pm 55,68	100	139,46 \pm 12,62	
Castrated	14	313,5	417,36 \pm 31,84	33,1	133,13 \pm 10,65	100	135,93 \pm 15,39	28,32	43,36 \pm 3,27	100
Castrated and receiving androgen	14	316	1547,86 \pm 58,32	122,41	489,94 \pm 13,28	368,72	471,21 \pm 37,02	98,19	149,15 \pm 6,48	343,98
With alloxan diabetes and castrated	8	249	370 \pm 88,44	29,26	148,22 \pm 28,85	100	132,12 \pm 8,58	27,53	52,93 \pm 3,87	100
With alloxan diabetes, castrated, and receiving androgen	14	243	985 \pm 74,52	77,9	405,35 \pm 25,83	273,48	290,7 \pm 27,78	60,58	119,63 \pm 9,53	226,01

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Castration of rats with an intact pancreas caused a significant decrease in weight of the seminal vesicles and prostate ($P < 0.001$). Subsequent injection of the androgen into these animals increased the weight of the seminal vesicles by 3.7 times and that of the prostate by 3.5 times ($P < 0.001$). As a result the prostate regained its normal weight and the seminal vesicles exceeded their normal weight ($P < 0.05$) (Table 1).

In rats with alloxan diabetes the weight of the accessory sex glands after castration fell by the same degree as in rats with normal pancreatic function. Under the influence of the androgen the weight of the seminal vesicles in the castrated rats with diabetes increased by 2.7 times and the weight of the prostate by 2.3 times ($P < 0.001$); the absolute weight of the accessory sex glands in these animals did not, however, reach normal.

The response of animals with diabetes to androgen was thus clearly weaker than that of the healthy animals. Thus, reactivity of the body to androgen was reduced in vivo in animals with insulin insufficiency.

However, rats with diabetes nevertheless still responded to androgen, although less strongly than the healthy rats, whereas in vitro no androgen response at all appeared without insulin. The possibility cannot be ruled out that in alloxan diabetes the β -cells of the islet tissue remain capable of secreting a small amount of insulin, not enough to prevent the disturbances of carbohydrate metabolism but enough to permit a weak response to androgen.

In alloxan diabetes in male rats atrophy of the accessory sex glands is observed. This effect is usually explained by weakening of the gonadotrophic function of the pituitary and a secondary disturbance of testicular function [1, 4]. The results of the present experiment suggest that atrophy of the accessory sex glands in alloxan diabetes may also be due to lowering of the reactivity to androgen.

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