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CARTAS AL EDITOR

Effect of Testosterone Propionate on the Rate of Myofibrillar Protein Breakdown in Corticosterone-Treated Young Male Rats

The net catabolic effects of glucocorticoids on the rate of muscle protein turnover in the rat is well known (3, 6-9). It has been demonstrated that glucocorticoid-treated rats exhibited a marked reduction on the rate of muscle protein synthesis (9). However, the effect of these hormones on the rate of muscle protein breakdown has not yet been clearly shown, and the results reported in the literature are controversial (7, 8). On the other hand, androgens are steroid hormones that stimulate protein synthesis in many tissues, especially in skeletal muscle (2, 5), and the anabolic action of androgens is additive to that of growth hormone. Furthermore, the action of testosterone is not altered by adrenalectomy (2).

The goal of this communication is to report some preliminary results on the antagonistic effect of these two types of hormones by studying the rate of growth and myofibrillar protein breakdown in young rats. The rate of myofibrillar protein degradation will be measured by determining the urinary excretion of N^{τ}-methylhistidine (3-methylhistidine: 3-MH). The validity of such an approach has been widely demonstrated (1, 10).

Intact, adrenalectomized (AdX) and adrenalectomized-castrated (AdXC) young male rats (Charles River Breeding Laboratories, Wilmington, MA., USA), weighing about 100-120 g were divided into four groups of six animals each, as follows: intact, receiving vehicle injection (corn oil); AdX, receiving either the same vehicle or 10 mg of corticosterone (Calbiochem, San Diego, CA.) per 100 g of body weight per day; and, AdXC receiving both the same dose of the glucocorticoid and 2 mg of testosterone propionate (Sigma) per 100 g of body weight per day. Injections were given subcutaneously during five days. Animals were fed *ad libitum* a purified diet containing 18 % lactalbumin (6). Body weight changes were recorded daily. Urine collections and 3-MH determinations were carried out as explained in detail elsewhere (6).

As compared to the intact animals (table I), a significant reduction in the rate of growth along with a slight reduction in the output of 3-MH was shown by the AdX rats receiving vehicle injection. Treatment of AdX rats with 10 mg of corticosterone brought about a drastic reduction in the rate of growth, together with a highly significant elevation ($p < 0.05$) in the output of 3-MH, as compared to either intact or AdX rats receiving vehicle injection. These results agree with previously reported data from this labo-

Table I. *Body weight gain and urinary output of 3-methylhistidine (3-MH) of intact, adrenalectomized (AdX) and adrenalectomized-castrated (AdXC) rats receiving subcutaneously during five days either vehicle, corticosterone (Ctc) or testosterone propionate (TP).* Doses are mg/100 g body weight. Entries for weight changes are means ± S.E.M. of six rats in each group, and for 3-MH results are means ± S.E.M. of pooled samples taken daily within each group.

Group	Body weight gain g/day	3-MN (μmoles/100 g body weight)
Intact+veh.	9.4±0.5	0.97±0.01
AdX+veh.	6.0±1.2[a]	0.84±0.03
AdX+10 mg Ctc	—2.2±0.7[a]	1.28±0.14[a]
AdXC+10 mg Ctc +2 mg TP	3.0±0.7[a]	1.00±0.13
LSD[1]	1.7	0.23

[1] LSD, least significant difference (ANOVA) between means for $p < 0.05$. [a] $p < 0.05$, as compared to the intact group.

ratory (6, 8, 10) and others (4), and show once more the increase in the rate of myofibrillar protein degradation in rats treated with pharmacological doses of corticosterone given subcutaneously. However, when the glucocorticoid was administered simultaneously with testosterone propionate, there was a significant improvement in the rate of growth along with a significant reduction in the output of 3-MH, as compared to rats which received the glucocorticoid alone. These results suggest that testosterone may antagonize the catabolic effect caused by large doses of corticosterone on the rates of growth and myofibrillar protein degradation.

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