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Plasma Corticosterone and Cardiac Glycogen Levels in Rats after Exercise (38992)

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Various physical conditions such as fasting (1–3), hypoxia (1), and exercise (2, 4) produce changes in myocardial glycogen concentrations that are superimposed upon the normal diurnal fluctuation of glycogen levels (5). The mechanisms by which these physiological conditions alter cardiac glycogen are not known. Although not all glycogen changes are dependent on the function of the adrenal glands (3, 6), glucocorticoids have been implicated as one regulatory substance of cardiac glycogen (6–8). The purpose of our study was to verify or dispel the proposed cause and effect relationship between glucocorticoid release and myocardial glycogen changes associated with physical exertion.

Methods. Male adult Wistar strain rats were used in all the experiments and two-thirds of the rats were bilaterally adrenalectomized under Brevital anesthesia. The completeness of the adrenalectomies was verified by comparing plasma glucocorticoid levels in the adrenalectomized rats with those of hypophysectomized rats. All rats were fed *ad lib.* and the adrenalectomized rats were maintained on 0.9% NaCl drinking solution. Sham operated animals were used as controls. Half of the adrenalectomized rats were given dexamethasone phosphate, a synthetic glucocorticoid, intraperitoneally 20 μg twice daily.

One week postoperation, rats were stressed by 15 min of swimming and then sacrificed at various times following the exercise. The animals were sacrificed with Brevital anesthesia since decapitation produces low glycogen levels (4). The thorax of each anesthetized rat was opened, the heart was removed rapidly (within 5–10 sec), and the resulting pool of blood in the thoracic cavity collected in a syringe. Total glycogen was extracted from whole hearts (9) and quantitatively determined by the anthrone method (10, 11). Plasma glucocorticoids were determined by the fluorescence procedure of Guillemin *et al.* (12). Data were analyzed by the analysis of variance and Duncan's multiple-range test under consultation with the department of Biometry.

Results. Figure 1 shows the myocardial glycogen levels before and at various times following 15 min of swimming in control, adrenalectomized, and dexamethasone treated-adrenalectomized rats. The degree of glycogen depletion was the same in sham operated and adrenalectomized rats; however, the glycogen recovery process was altered by adrenalectomy with glycogen values being significantly lower in the adrenalectomized rats 4 hr following the exercise. Dexamethasone treatment significantly elevated initial myocardial glycogen in adrenalectomized rats and enhanced the recovery of glycogen to levels above that observed in the controls. The magnitude of glycogen depletion during exercise in these rats was greater than in controls or adrenalectomized rats perhaps due to the availability of more endogenous glycogen.

Figure 2 shows plasma glucocorticoid levels for the same animals used in determining the glycogen data. Pre-exercise glucocorticoid levels were higher than reported for nonstressed levels in male rats (13) because of the difficulty in maintaining a completely tranquil environment in non-private animal facilities. However, 15 min of swimming significantly elevated these levels further. Four hours following the exercise, plasma glucocorticoids were at low levels similar to those of adrenalectomized and dexamethasone treated-adrenalectomized rats for which the values for the glucocorticoid determinations were always below 10 μg/100 ml.

The patterns of changes on myocardial glycogen and plasma corticosterone in con-

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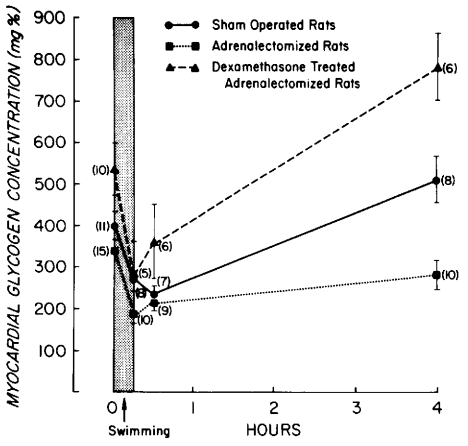

FIG. 1. Myocardial glycogen before and at various times after 15 min of swimming. Each symbol and accompanying bar represents the mean value ± SEM. Number of rats used to determine each point is given in parenthesis.

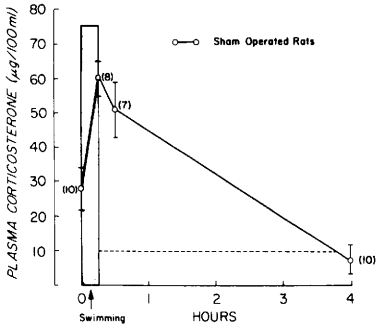

FIG. 2. Plasma glucocorticoid levels before and at various times following 15 min of swimming. Each symbol and accompanying bar represents the mean value ± SEM. Number of rats used to determine adrenalectomized and dexamethasone treated-adrenalectomized rats fell below the broken line.

trol rats following swimming are compared in Fig. 3. The elevated corticosterone levels occurred approximately 4 hr before the peak in the myocardial glycogen recovery.

Discussion. The extent of myocardial glycogen depletion during exercise is not influenced by the adrenal glands, since depletion occurs equally well in control and adrenalectomized rats (2), and consequently might be determined by local factors, as seems to be the case in skeletal muscle (14). The recovery of myocardial glycogen following exercise has been characterized as

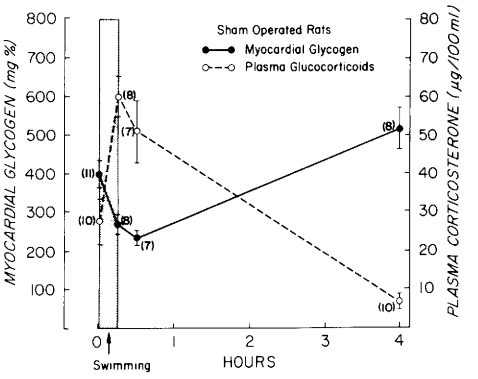

FIG. 3. Relationship between myocardial glycogen (Fig. 1) and plasma glucocorticoid levels (Fig. 2) in control rats before and at various times following 15 min of swimming. Each symbol and accompanying bar represents the mean value ± SEM. Number of rats used to determine each point is given in parenthesis.

having a period of supercompensation during which the heart's glycogen levels are significantly above control levels (4, 6). This supercompensation peaks around 4 hr after exercise and requires approximately 24 hr to return to its pre-exercise level (6). Also, this pattern of glycogen recovery is blocked by adrenalectomy but restored by dexamethasone treatment in adrenalectomized rats (6). The present study is in agreement with these findings. However, we questioned the association between glucocorticoid secretion and glycogen recovery following exercise since plasma steroid levels were not measured in the previous studies.

The data reported here show that there is an increase in both plasma corticosterone and myocardial glycogen following exercise of this severity although the peak in plasma glucocorticoids is relatively brief. Also, the 4-hr interval between the peaks in plasma glucocorticoid and myocardial glycogen levels appears appropriate for a cause and effect relationship since glucocorticoid administration takes approximately 4 hr to significantly increase either cardiac glycogen (7) or liver glycogen (15).

The association between steroids and myocardial glycogen recovery following exercise is still somewhat dubious; however, since chronic treatment with a potent glucocorticoid (dexamethasone) is as effective as a

burst of corticosterone (control rats) in permitting the observed pattern of cardiac glycogen supercompensation. This suggests that physical exertion and the presence of steroids (i.e., a permissive effect of glucocorticoid rather than a causative one) can produce changes, perhaps local, that are influential on cardiac glycogen for hours following the exercise.

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