STUDIES IN TISSUE GLYCOGEN IN ACUTE STRESS

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Summary : The glycogen was estimated in liver, cardiac and skeletal muscles during the recovery period after electro-shock. The supercompensation in the level of glycogen was observed in cardiac and skeletal muscles at 1½ and 5 hrs respectively during the recovery period, after electro-shock. The liver glycogen level was lower than the control value after electro-shock at least upto 5 hrs of recovery period. Further, the glycogen level was observed to be minimum when the ventricular glycogen showed its supercompensation at 1½ hr of recovery period. The glycogen level of those three tissues returned to control level after 24 hrs of electro-shock.

Key words: electro-shock stress glycogen recovery period supercompensation

INTRODUCTION

The contradictory findings abound the literature on the depletion and recovery of glycogen in different tissues like liver, cardiac and skeletal muscles during and after the different types and degree of stress (4, 9, 10, 12, 14). The work has been undertaken to elucidate the recovery in glycogen following stress in different tissues with progress of time.

MATERIALS AND METHODS

Inbred albino rats (100-150 g) fasted for 24 hr have been selected for subjecting them to Acute Electro-shock (AES). This was done in a specially constructed grid box and the electro-shock was given at a 100 v for a period of 30 min at a frequency of 75/min. The duration of each shock was 1.2 msec. The animals were sacrificed in different time interval viz. '0' hr (immediately after AES), ½ hr, 1 hr, 1½ hr, 2 hr and 24 hr. Liver (right lobe), cardiac muscle (apex of the ventricle), skeletal muscle (gastrocnemous) were collected from these animals for the estimation of glycogen. Glycogen has been estimated by the method of Montgomery (11). The control group of animals were also fasted for 24 hr and the tissues collected for estimation of glycogen. The level of significance was determined by Student's 't' test.

RESULTS

The results are summerised in Table I.

The fall in the level of glycogen was observed in all the tissues at the beginning of the experiment after receiving the electric shock treatment.

The supercompensation in the level of glycogen in the ventricle was noted to commence after ½ hr of AES and attained its peak at 1½ hr ($P<0.01$) and returned to the control value within 24 hr.

Table I: Showing the glycogen level *mg/g* of wet tissue after Electro-shock for ½ hr.

Time in hr	*Ventricle Mean±S.E.*	*Liver Mean±S.E.*	*Skeletal muscle Mean±S.E.*
Control (28)	4.34±0.36	4.09±0.46	3.24±0.21
0(11)	2.21±0.32***	1.10±0.09***	1.68±0.25***
½(8)	3.90±0.42	1.85±0.23***	1.80±0.27***
1(14)	4.85±0.52	2.16±0.38***	1.90±0.22***
1½(8)	5.91±0.43**	1.06±0.21***	2.11±0.48*
2(8)	3.80±0.28	2.00±0.25***	2.18±0.53
5(13)	2.96±0.44**	2.23±0.39**	4.22±0.57
24 hr Control (7)	2.75±0.32	2.36±0.49	1.41±0.29
24 hr after Electro-shock (7)	2.65±0.51	2.13±0.53	1.14±0.20

*P=<0.05; **P=<0.01; ***P=<0.001
Number within parentheses represents the number of animals studied.

The supercompensation in the level of skeletal muscle glycogen attained its peak after 5 hr then resumed to its control level within 24 hr.

In the liver tissue, the glycogen level was observed to be lower than the control value at least upto 5 hr of recovery period of AES and returned to control level within 24 hr. Further, the glycogen level in the liver tissue was observed to be maximum when the ventricular glycogen level attained its maximum *e.g.* 1½ hr of the recovery period, but the reverse phenomenon was not noticed. As opposed to the other two tissues, the supercompensation was not noticed for the liver tissue within 24 hr of recovery period.

DISCUSSION

The physiological variations like fasting, hypoxia and exercise cause the alteration in different tissues which are superimposed on the diurnal variations (2, 3, 7, 11, 16). A supercompensation in the level of myocardial and skeletal muscle glycogen had been reported in different stressed animals (4, 8, 15) as observed in this study. This might be due to single or combined effects of the factors like alteration of the adrenal function and of glucocorticoid levels (12), decreased sensitivity to insulin resulting in defective peripheral utilisation, decreased turnover and oxidation rates of glucose (5, 11) and an enhancing factor for glycogen synthesis localised in the tissue (1). However, the hyperglycaemic effect after stress could not be prevented using antiadrenergic drugs was suggestive of the involvement of the influencing factors other than glucocorticoid release (4, 9, 12).

The influence of catecholamine on skeletal muscle might be an important factor although to

a lesser degree for the late phenomenon of sopercompensation of glycogen in comparison to that of cardiac muscle (4, 5).

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REFERENCES

1. Bergstrom, J. and E. Hultman. Muscle glycogen synthesis after exercise: an enhancing factor localized to the muscle cells in man. *Nature*, **210** : 309-310, 1966.
2. Blount, D.H. and D.K. Meyer. Effects of cardiac work on glycogen fractions of the heart. *Am. J. Physiol.*, **197** : 1013-1016, 1959.
3. Bockman, E.L., D.K. Meyer and F.A. Purdy. Synthesis of cardiac glycogen in relation to its 24 hr rhythm in rats. *Am. J. Physiol.*, **221** : 383-387, 1971.
4. Choudhary, S. and D.P. Sadhu. Role of adrenergic system in thermoglycomic response in baby rabbits. *Am. J. Physiol.*, **199** : 727-728, 1960.
5. Christiande, S., J.M. Strythagen. I. Becsci and J. Lammerant. Effect of training on heart and skeletal muscle catecholamine concentration in rats. *Am. J. Physiol.*, **217** : 1589-1592, 1969.
6. Daw, J.C., A.M. Lefer and R.M. Berne. Time course of glucocorticoid dependent changes in cardiac glycogen concentration. *Proc. Soc. Exp. Biol. Med.*, **131** : 1042-1044, 1969.
7. Evans, G. The glycogen content of the rat heart. *J. Physiol.* (Lond.), **82** : 468-480, 1934.
8. Lamb, D.R., J.B. Peter R.N. Jeffress and H.A. Wallace. Glycogen, hexokinase and glycogen synthetase adaptations to exercise. *Am. J. Physiol.* **217** : 1628-1632, 1969.
9. Lauria, P., V.N. Sharma and S. Vanjani. Effect of prolonged stress of repeated electric shock on rat myocardium. *Ind. J. Physiol. Pharmac.*, **16** : 315-318, 1972.
10. Leenen, F.H. and P.A. Shapire. Effect of intermittent electric shock on plasma renin activity in rats. *Proc.* 208, *Exp. Biol. Med.*, **146** : 534-538, 1974.
11. Montgomery, R. Determination of glycogen. *Arch. Biochem. Biophysics*, **67** : 378-386, 1957.
12. Pal, A.K. and D.P. Sadhu. Utilization of glucose during acute thermal stress. *Indian J. Exp. Biol.*, **9** : 207-*Soc.* 1971.
13. Poland, J., D.T. Myers, R.J. Witorsch and R.B. Brandt. Plasma corticosterone and cardiac glycogen levels in rats after exercise. *Proc. Soc. Exp. Biol. Med.*, **150** : 148-150, 1975.
14. Poland, J. and D.A. Trauner. Effects of prior exercise on myocardial glycogenesis during a fast. *Proc. Soc. Exp. Biol. Med.*, **136** : 1100-1102, 1971.
15. Poland, J. and D.A. Trauner. Adrenal influence on the supercompensation of cardiac glycogen following exercise. *Am. J. Physiol.*, **224** : 540-542, 1973.
16. Russel, J.A. and W. Bloom. Hormonal control of glycogen in the heart and other tissues in rats. *Endocrinology*, **58** : 83-94, 1956