# HEPATIC LIPOLYSIS DURING MUSCULAR ELECTRICAL STIMULATIONS

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### ABSTRACT

The hepatic lipid metabolism was modulated by localized muscular stimulations of short-term (SMS) and prolonged (PMS) durations. The hepatic tissue total lipids, triglycerides and cholesterol were significantly depleted, while phospholipid and glycerol contents were elevated with activated lipase in PMS animals. The possible applicability of PMS type of electrical stimulations to avert hepatic disorders is suggested.

## INTRODUCTION

WUSCULAR electrical stimulations or exercises were known to alter the metabolism of various tissues in the body<sup>1-8</sup>. The physical exercises and training programmes alter the hepatic metabolism<sup>9-11</sup>. However, studies on impact of localized muscular exercise on the hepatic metabolism are scanty. Our previous studies revealed that the muscular exercise modulates the hepatic carbohydrate<sup>8</sup> and nitrogen<sup>12</sup> metabolisms. Since deposition of fat components leads to hepatic disorders such as fatty livers and cirrhosis and physical exercises and localized muscular stimulations influence hepatic metabolism, an attempt has been made to elucidate the possible changes in hepatic lipid metabolism during short-term and prolonged muscular stimulations.

#### MATERIALS AND METHODS

The male frogs, Rana hexadactyla (Lesson)  $30 \pm 2$  g were used for the present study. The right gastrocnemius muscles of intact animals were stimulated by placing the two platinum electrodes at a distance of I cm apart to localize the stimulations using electronic stimulator (INCO/CSIO Research Stimulator -Ambala) as described by Reddanna et al.13, with a series of pulses (biphasic) of 5 V at a frequency of 2 c/sec for 30 min per day for one day in one batch of experimental animals (short-term muscular stimulations—SMS) and for 10 successive days in another batch (prolonged muscular stimulations—PMS). duration of each impulse was 100 ms and the interval was 400 ms. The liver was isolated from freshly pathed control and experimental animals and taken for biochemical assay. The total lipids (Folch et al. 11), lipase activity (Huggins and Lapides 16), free fatty acids, cholesterol, triglycerides (Natelson<sup>16</sup>), phospholipids (Bieri and Prival<sup>17</sup>) and glycerol (Burton<sup>18</sup>) were estimated in the tissue.

#### RESULTS AND DISCUSSION

The data presented in Table I reveal the modulations in hepatic tissue lipid metabolism, during in vivo muscular electrical stimulations. SMS showed non-significant changes in the levels of total lipids, triglycerides, and cholesterol of hepatic tissue. However, lipase activity was elevated suggesting the possible lipolytic activity in the tissue in response to muscular electrical stimulations. The levels of FFA and glycerol were depleted in spite of elevated lipase activity suggesting the possible mobilization of these components into tissue oxidations<sup>19</sup> and gluconeogenesis<sup>8</sup>.

In contrast, PMS resulted in significant depletion of hepatic lipid content which might be due to catecholamine mediated lipolysis<sup>20</sup> and for cortisol inhibited lipogenesis as reported in trained animals.

Consequent upon elevated lipase activity, triglyceride content was depleted and glycerol level was elevated. However, the FFA content was depleted suggesting its mobilization towards oxidative metabolism<sup>19, 21</sup> and ketogenesis<sup>22, 23</sup> as observed in the trained animal tissue. Since phospholipids form important requisite for mitochondrial membranes, elevated phospholipid content in the liver of the PMS animals supports the widely observed elevated mitochondrial content<sup>34</sup> in the tissues of trained animals. Since phospholipid synthesis gets stimulated under aerobic environment and hepatic tissue becomes more oxidative in PMS, it can be suggested that the hepatic tissue had congenial environment for phospholipid synthesis. Since cholesterol forms precursor for bile salts and bile salt production was elevated in trained animalo24,28, the depleted cholesterol content of tissue in the present study suggests the stepped-up bite formation in the liver. Since all these conditions of tissue metabolism were similar to trained animals, the PMS animals of present study can be considered as trained animals. In view of depleted total lipid content, triglycerides

TABLE I

The levels of total lipids, triglycerides, glycerol, phospholipids, cholesterol, free fatty acids, and lipase activity in liver of control (C), short-term muscular stimulated (SMS) and prolonged muscular stimulated (PMS) animals

(Values are mean of eight observations Mean  $\pm$  S D.; + and - indicate per cent increase and decrease over control, 'P' indicates the level of significance and NS non-significance

SI. No.	Component (mg/g wet wt.)	Control (1)	SMS (2)	PMS (3)	% of change (3) over (2)
1.	Total lipids (TL)	61·31 ± 3·41	60·32 ± 2·89 − 1·61 NS	51·20 ± 3·97 -16·49 P < 0·001	-15.12
2.	Triglycerides (TG)	3·43 ± 0·25	3·31 ± 0·28 - 3·61 NS	$ \begin{array}{r} 2.67 \\ \pm 0.01 \\ -22.03 \\ P < 0.001 \end{array} $	-19.11
3.	Glyceroi	1·20 ± 0·02	$ \begin{array}{r} 1.107 \\ \pm 0.02 \\ -7.98 \\ P < 0.001 \end{array} $	$3.278$ $\pm 0.034$ $+172.48$ $P < 0.001$	+196-11
4.	Phospholipids (PL)	15·19 ± 0·75	16·13 ± 0·78 + 6·22 NS	$ \begin{array}{r} 25.87 \\ \pm 1.50 \\ +70.37 \\ P < 0.001 \end{array} $	+60.60
5,	Cholesterol	7·21 ± 0·72	6·69 ± 0·56 - 7·21 NS	$2.55$ $\pm 0.27$ $-64.60$ $P < 0.001$	-61.85
б.	TG/TL	0.056	0 055	0.052	
7.	PLITL	9.25	0-27	0.51	
8,	Cholesterol/TL	0-12	0-11	0.05	
9.	Free fatty acids	18·57 土 1·67	$14.85$ $\pm 1.51$ $-20.03$ $P < 0.001$	$8.837$ $\pm 1.23$ $-52.41$ $P < 0.001$	-40.49
<b>0</b> .	Lipase activity (µ moles of P-nitrophenol formed/mg protein/hr)	0·22 ± 0·003	$0.27$ $\pm 0.004$ $+19.11$ $P < 0.001$	$0.30$ $\pm 0.05$ $+35.11$ $P < 0.001$	+13.43

and cholesterol, the PMS can be looked upon as a means of decreasing the fat content of the liver and the applicability of muscular electrical stimulations to avert fatty liver conditions and hepatic cirrhosis should be examined.

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- 1. Dieter, M. P., Altland, P. D. and Highman, B., Can. J. Physiol. Pharmacol., 1969, 48, 723.
- 2. Novesadova, J., Biochemistry of Exercise (ed.) J. R. Poortmans Karger Basel, New York, 1969, p. 254.
- 3. Herbison, G. J., Teng, C. and Gordon, C. E., Arch. Phys. Med., 1973, 54, 156.
- 4. Pette, D., Smith, M. E., Staudte, H. W. and 1973, 338, 257.
- 5. Hickson, R. C., Bomze, H. A. and Holloszy, J. O., J. Appl. Physiol., 1977, 42, 372.
- 6. Reddanna, P., and Govindappa, S., Curr. Sci., 1978a, 47, 531.
- 7. and —, Ibid., 1978b, 47 753.
- 8. and —, J. Anim. Morphol. Physiol., 1979, 26. 156.
- 9. Gollnick, P. D., Am. J. Physiol., 1963, 205, 453.
- 10. Richard, M. L., Merles, F., Water, D. B. and Jairum, D. F., Metabolism, 1976, 25, 160.
- 11. Simko, V., Ondreicka, R., Chorvathova, V. and Bobek, P., J. Nutr., 1970, 100, 1331.

- 12. Reddanna, P., Narasimha Moorthy, C. V., Krishna Murthy, V., Vemananda Reddy, G. and Govindappa, S., Proc. Indian Natl. Sci. Acad. (in press).
- 13. —, Bhaskara Haranath, V., Ramachandra Rao, M. and Govindappa, S., Indian J. Exp. Biol., 1978, **16**, 366.
- 14. Folch, J., Less, N. and Stanley, G., J. Biol. Chem., 1957, 226, 497.
- 15. Huggins, C. and Lapides, J., Methods Enzymol., 1955, 1, 627.
- 16. Natelson, S., Techniques of Clinical Chemistry, (ed.) C. T. Charles, Thames Publishers, Illinois. 1965.
- 17. Bieri, J. G. and Prival, E. L., Comp. Biochen. Physiol., 1965, 15, 275.
- 18. Burton, R. M., Methods Enzymol., 1957, 3, 246.
- 19. Paul, P., Metabolic Adaptation to Prolonged Physical Exercises, (eds.) H. Howlad, and J. R. Poortmans, Biskhauser Verlag Basel, 1973, p. 156.
- Vibova, G., Pflugers Arch. Eur. J. Physiol., 20. Karki, N. T., Acta Physiol. Scand., 1956, 39, Suppl. 132.
  - 21. Paul, P. and Issekutz, B. Jr., J. Appl. Physiol., 1967, **211**, 1313.
  - 22. Hagenfeldt, L. and Wahren, J., Scand. J. Clin. Lab. Invest., 1968, 21, 314.
  - 23. Drury, R. R., Wick, A. N. and Mackay, M. E., Am. J. Physiol., 1941, 134, 761.
  - 24. Kiersling, K. H., Pilstrom, L., Karlsson, J. and Pichl, K., Clin. Sci., 1973, 44, 547.
  - 25. Malinow, M. R., McLaughlin and Peerhey, P., Science, 1968, 160, 1239.
  - 26. Simko, V., Ondrecicka, R., Chorvathova, V. and Bobek, P., J. Nutr., 1970, 100, 1337.