

EFFECT OF LOCALIZED MUSCULAR EXERCISE AND TRAINING ON THE RENAL PROTEIN METABOLISM

P. REDDANNA, C. V. NARASIMHA MOORTHY AND S. GOVINDAPPA

Department of Zoology, S.V. University, Tirupati 517 502, Andhra Pradesh, India

ABSTRACT

The renal tissue proteolytic activity was modulated by exercise and training programme, resulting in the altered protein content of the tissue. The training programme exerted significant inhibition on the renal tissue protease activity suggesting its importance in averting kidney proteolysis. While exercise inhibited the amino acid oxidations, the training programme activated the same. Both exercise and training programmes enhanced the free ammonia content of the kidney, due perhaps to the buffering of the blood organic acids. The urea content of the kidney was significantly depleted during the training programme.

INTRODUCTION

ELECTRICAL stimulation of muscle tissue and heavy exercises were known to alter the tissue metabolism¹⁻⁹. *In vivo* muscular stimulations increased the levels of oxidative enzymes of all the tissues of the body¹⁰. Renal carbohydrate level was shown to be regulated by muscular exercise and training¹¹. The muscular training also enhanced the rate of citric acid cycle through the mobilization of lactic acid and free amino acids¹¹. Since the muscular exercise and training programmes induced alterations in the renal carbohydrate metabolism and the carbohydrate metabolism has a close link with the amino acid metabolism, an attempt has been made in the present study to analyse their impact on renal tissue protein metabolism.

MATERIAL AND METHODS

Frogs, *Rana hexadactyla* (Lesson), were employed for the present investigation. The right gastrocnemii of these animals were stimulated with electronic stimulator (INCO/CS10—Research Stimulator—Ambala, India) as described earlier¹⁰ with series of impulses (biphasic) of 5 V at a frequency of 120 PPM (100 ms duration and 400 ms delay) for 30 minutes for one day (exercised) and 10 successive days (trained) respectively.

The renal tissue was isolated from freshly pithed control and the experimental frogs and placed in amphibian Ringer solution to recover from shock effects. The protein contents in the supernatant (water soluble) and the residue (water insoluble—structural) obtained by the centrifugation of tissue homogenate (prepared in ice-cold double distilled water with mortar and pestle using acid washed sand) at 3000 rpm for 30 minutes, were estimated by the method of Lowry *et al.*¹². The levels of protease activity and free amino acids (Moore and Stein¹³), free ammonia (Bergmeyer¹⁴) urea (Nelson¹⁵), glutamine (Colowick and Kaplan¹⁶) and the activities of alanine amino transferase (AIAT—

EC 2.6.1.2) and aspartate amino transferase (AAT EC 2.6.1.1) (Reitman and Fraenkel¹⁷) were determined.

RESULTS AND DISCUSSION

The results in Table I reveal the extent of changes in the protein metabolism of the kidney of frog in response to localised muscular exercise and training. The soluble protein content of the tissue was elevated with a nonsignificant drop in the structural protein content in response to muscular exercise. The neutral protease activity was elevated in the kidney of exercised animal. Increased soluble protein content in the presence of elevated protease activity might suggest the possibility of induced changes in the solubility properties of proteins. The free amino acid content of the tissue was elevated which might be due to increased protease activity, or decreased amino acid oxidations as exemplified by GDH activity or due to uptake from the blood since blood free amino acid content was increasing on muscular exercise⁸. Both AIAT and AAT activities showed non-significant changes suggesting the probable non-involvement of amino acids in transaminations during muscular exercise. Free ammonia level increased in the kidney of exercised animal. This increased free ammonia during inhibited amino acid oxidations might suggest its origin either from glutamine or urea. However, glutamine level was elevated while urea showed non-significant change. Hence ammonia might be originating from some other source than the amino acids, probably through the deaminations of nucleotides. The free ammonia accumulation in renal tissue might be essential for neutralisation of acids of the blood, since organic acids of the blood increased during exercise⁸.

The situation of renal tissue metabolism in trained animals was different from that of exercised animal. The tissue proteolysis was significantly inhibited, leading to non-significant changes in the protein content. The free amino acid content decreased considerably which might be correlated towards the decreased

TABLE I

Levels of soluble and structural proteins, protease activity, free amino acids, ALAT, AAT, GDH, glutamine, ammonia and urea in kidney of normal and experimental animals

(Each value represent the mean of six observations. Mean \pm S.D., + and - indicate per cent increase and decrease over control)

Sl. No.	Component	Kidney		
		Control	Experimental	
			Exercised	Trained
1.	Soluble proteins (mg/gm wt)	97.67 ± 8.48	135.11 ± 7.14	92.83 ± 12.0
		+38.33 P < 0.001	-4.96 NS	
2.	Structural proteins (mg/gm wt)	62.32 ± 9.04	56.28 ± 8.66	58.14 ± 7.87
		-9.70 NS	-6.71 NS	
3.	Protease activity (μ M/mg protein/hr)	0.107 ± 0.001	0.148 ± 0.003	0.082 ± 0.003
		+38.3 P < 0.001	-23.36 P < 0.001	
4.	Free amino acids (μ M/gm wt)	20.24 ± 2.59	33.45 ± 5.3	10.71 ± 2.37
		+65.27 P < 0.001	-47.08 P < 0.001	
5.	ALAT (μ M pyruvate/mg protein/hr)	0.552 ± 0.1	0.554 ± 0.08	0.539 ± 0.06
		+0.36 NS	-2.35 NS	
6.	AAT (μ M pyruvate/mg protein/hr)	0.21 ± 0.009	0.203 ± 0.003	0.184 ± 0.01
		-2.87 NS	-11.96 P < 0.001	
7.	GDH (μ M formazan/gm/hr)	23.36 ± 2.16	20.42 ± 3.46	41.43 ± 6.14
		-12.59 P < 0.05	+77.35 P < 0.001	
8.	Glutamine (μ M ammonia/ gm wt)	10.3 ± 1.21	13.1 ± 0.83	12.7 ± 1.17
		+27.18 P < 0.001	+23.3 P < 0.001	
9.	Ammonia (mg/gm wt)	0.086 ± 0.007	0.116 ± 0.003	0.196 ± 0.023
		+34.88 P < 0.001	+127.91 P < 0.001	
10.	Urea (mg/gm wt)	0.602 ± 0.029	0.586 ± 0.031	0.505 ± 0.045
		-2.58 NS	-16.15 P < 0.01	

tissue proteolysis. Moreover the amino acid oxidations exemplified by GDH activity were largely elevated which also may be responsible for the decreased amino acid level of the tissue. Both the activities of amino transferases were decreased indicating inhibited level of transamination reactions in the tissue. In response to increased amino acid oxidations and depleted level of urea tissue free ammonia content was largely elevated, probably an essential requirement for buffering the blood organic acids. Glutamine content was slightly elevated, probably aimed to decrease the ammonia content.

Since the training programme effectively inhibited tissue protease activity, its importance in averting kidney proteolysis can be envisaged.

ACKNOWLEDGEMENTS

The authors (PR and CVNM) are thankful to CSIR, New Delhi, for the award of Senior and Junior, Research Fellowships during the tenure of which this work was carried out.

1. Gutmann, E., *The Denervated Muscle*, Publishing House of Czechoslovak Academy of Sciences, 1962.
2. Dieter, M. P., Altland, P. D. and Highman, B., *Canad. J. Physiol. Pharmacol.*, 1969, 48, 723.
3. Novosadova, J., *Biochemistry of Exercise* (ed. J. R. Poortmans), Karger, Basel, New York, 1969, 3, 254.
4. Harri, M. N. E. and Valtola, J., *Acta Physiol. Scand.*, 1975, 95, 391.
5. Rennie, M. J., Winder, W. W. and Holloszy, J. O., *Biochem. J.*, 1976, 156, 647.
6. Harri, M. N. E., Tirri, R. and Karki, A., *Experientia*, 1977, 33, 620.
7. Siltovuori, A., Jirri, R. and Harri, M. N. E., *Acta Physiol. Scand.*, 1977, 99, 457.
8. Reddanna, P. and Govindappa, S., *Curr. Sci.*, 1978 a, 47 (15), 531.
9. — and —, *Ibid.*, 1978 b, 47 (20), 753.
10. —, Haranath, V. B., Ramachandra Rao, M. and Govindappa, S., *Ind. J. Exp. Biol.*, 1978, 16 (3), 366.
11. — and Govindappa, S., *Ibid.*, 1979 (In press).
12. Lowry, O. H., Rosebrough, N. J., Farr, A. L. and Randall, R. J., *J. Biol. Chem.*, 1951, 193, 265.
13. Moore, S. and Stein, W. H., *Ibid.*, 1968, 243, 6281.
14. Bergmeyer, H. U., *Methods of Biochemical Analysis*, Academic Press, New York and London, 1974.
15. Natelson, S., *Techniques of Clinical Chemistry*, Charles, C. Thomas Publishers, USA, 1971, p. 728.
16. Colowick, S. P. and Kaplan, N. O., *Methods in Enzymology*, Academic Press, New York, 1967.
17. Reitman, S. and Fraenkel, S., *Am. J. Clin. Pathol.*, 1957, 28, 56.

THE INSTITUTE OF PHYSICS, LONDON

The 10th European Solid State Device Research Conference, in conjunction with the 5th Symposium on Solid State Device Technology, will be held at the University of York, UK, from 15th to 18th September 1980. The aim is to bring together scientists and engineers working in the broad field of Solid State Devices and to provide a European forum for the presentation and discussion of the latest research and technology. A particular feature of this series of conferences has been the range of invited papers reviewing progress in a number of key subjects. It is hoped to maintain this tradition for 1980 and it is anticipated that the following topics will be covered. ESSDERC: The Influence of Electron Beam Lithography on Device Performance; Future Developments in VHSIC; Amorphous Silicon Devices; New CMOS Technologies;

Recent Progress on Flat Panel Solid State Displays; Mos Power Devices—Trends and Results; Modelling of Sub-Micron Devices. Symposium; Vapour Growth of III-V Compound Semiconductor Layers; Reliability Problems of Metal Conductor Lines in Integrated Circuits; Process Induced Defects in Silicon. Offers of Contributions (together with 600 word abstracts—2 × A4 pages) should be sent as soon as possible, but not later than 9th May 1980, to the Conference Secretary, Dr K. H. Nicholas, Philips Research Laboratories, Cross Oak Lane, Redhill, Surrey RH1 5HA, U.K. Abstracts must be submitted in a form suitable for Offset Photolitho reproduction. Instructions may be obtained from the Meetings Officer, The Institute of Physics, 47 Belgrave Square, London SW1X 8QX.