Arion ater uroporphyrin[10], 540 mμ of *Tivela stultorum* oxyhemoprotein[11], and 541.4 mμ of chiton myoglobin[12]. On the other hand, comparison with molluscan hemocyanin reveals no such resemblance. The spectra of mussel plasma do not show (i) the band at 560 mμ (characteristic of oxygenated hemocyanin[13]), or (ii) the peaks at 346 and 580 mμ (representing oxygenated Cu-complexes[14]), or (iii) the bands at 440, 570 and 700 mμ (similar to those of cupric proteins[15]), or (iv) the protein band of hemocyanin at 278 mμ[9].

The spectra of mussel plasma also show peaks (534–539 and 638 mμ; 1636 and 1690 cm^{-1}) which lie near those recorded for chlorophyll (535 and 640 mμ)[6] and its decomposition products viz phylloporphyrin (533, 536 and 537 mμ)[5] and pheophytin (1640 and 1650 cm^{-1})[8]. This resemblance could be meaningful because it is felt[16–18] that molluscan pigments, including vascular pigments, originate from chlorophyllous food.

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FATIGUE-INDUCED ALTERATIONS IN DEHYDROGENASE ACTIVITY PROFILES IN DIFFERENT SKELETAL MUSCLE TYPES OF ALBINO RAT

P. KRISHNA MOHAN, K. INDIRA and W. RAJENDRA

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

THE metabolism and functional efficiency of muscles depend on the delivery of substrates, intracellular enzyme activities, mitochondrial content, coenzyme, oxygen reserves as well as the milieu interior[1]. Temporary cessation of metabolic functions during prolonged contraction is due to inactivation of the enzymes of oxidative cycles[1,2]. Hence, in the present investigation, the alterations in oxidative metabolism of different muscle types were studied during exhaustion.

Healthy, adult, male wistar albino rats maintained under laboratory conditions (temp. 25±2°C; RH 75% and LP 12 hr) were subjected to prolonged swimming[3]. Suitable controls were also maintained. The gastrocnemius, GN (fast-twitch red, FR), red vastus, RV (fast-twitch mixed, FM) and white vastus, WV (fast-twitch glycolytic, FG) muscles[4,5] were excised in cold (0–4°C) and the lactate (LDH)[6], succinate (SDH)[6], malate (MDH)[6], glutamate (GDH)[7] dehydrogenase activities and proteins in the enzyme sources[8] were measured following the standard methods.

The decrement in the activity levels of all the dehydrogenases (table 1) of rat muscles during exhaustion suggests that the entire energetic pathway of muscle was put to strain. The degree of vitiation in the dehydrogenase profiles was greater in FG than in FM and FR fibres. Among the oxidoreductases, greater inhibition was observed in SDH followed by MDH, LDH and GDH. The relatively greater drop

Table 1 *Dehydrogenase activity profiles in different muscles of albino rat during fatigue*

Enzyme	Gastrocnemius Control	Gastrocnemius Fatigued	Red Vastus Control	Red Vastus Fatigued	White Vastus Control	White Vastus Fatigued
Lactate dehydrogenase	4.39 ±0.24	3 26 ±0.19 (−25.74)	4 46 ±0.31	3.09 ±0.23 (−30.72)	3.14 ±0 16	2.04 ±0 13 (−35 03)
Succinate dehydrogenase	1.91 ±0.12	1.15 ±0 09 (−40 15)	1.74 ±0.12	0 98 ±0 07 (−43 43)	0.84 ±0.09	0 45 ±0 06 (−46.31)
Malate dehydrogenase	1.86 ±0.13	1.28 ±0 11 (−31.18)	1.72 ±0.09	1.09 ±0 08 (−36 63)	0.94 ±0.10	0 51 ±0 04 (−45.75)
Glutamate dehydrogenase	0 19 ±0.01	0.17 ±0.02 (−10.19)	0.19 ±0.01	0.17 ±0 02 (−10 02)	0.18 ±0 01	0.15 ±0.01 (−14 84)

Values, expressed in μmol of formazon mg protein^{-1} hour^{-1} are mean and ± SD of eight observations. Values in parentheses are the % changes over control
All Values differ significantly from control values, $P<0.05$.

in LDH, MDH and SDH in WV reflect low operation of TCA cycle as evinced from high lactate and low pyruvate[9] and consequent insufficient energy budget in the muscle. Low activity profiles of oxidoreductases during exhaustion might be due to accumulation of metabolic products such as lactate, ammonia and organic acids, altered protein ionization and muscle fibre organization, leakage of enzymes into serum, vascular insufficiency and altered mitochondrial structure/properties[1,2,5,9]. Since the above mentioned changes are more prominent in FG rather than in FM or FR muscles[1–5,9], the glycolytic fibres are more vulnerable to fatigue effects.

The activity levels of dehydrogenases suggest that the SDH is more susceptible to fatigue-induced changes. Even among the mitochondrial enzymes (SDH, MDH and GDH), SDH is affected to a greater degree, which may be due to its localization in the inner membrane of mitochondria, whereas the MDH and GDH are localized in the matrix.

An overview of the activity profiles of oxidoreductases studied during intense muscular activity infer that the energy-producing systems become less efficient due to the depletion of energy substrates and low lactate, succinate, malate and glutamate oxidations during prolonged exhaustion. Further, GN and RV appear to be more resistant to fatigue than WV possibly due to predominant oxidative metabolism and special architectural dynamics which confer biochemical adaptability on the former muscles to resist fatigue than the latter one.

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