GLUTAMINE METABOLISM IN DOGFISH (SQUALUS ACANTHIAS) RED MUSCLE

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Elasmobranchs, unlike mammals and teleost fish, do not oxidize lipids in their muscles (Zammit and Newsholme, Biochem.J. 184:313-322, 1979; Singer and Ballantyne, J.Exp.Zool. 251: 355-365, 1989; Moyes et al., Am.J.Physiol. 258:R756-R762, 1990). Ketone bodies are oxidized by shark red muscle mitochondria and are thought to represent an important energy source for the red muscle (Moyes et al., Am.J.Physiol. 258:R756-R762, 1990). A recent abstract, however, indicates that glutamine is oxidized by skate red muscle mitochondria (Chamberlin and Ballantyne, The Physiologist 33:A110,1990) and therefore this amino acid may represent an important energy source in the elasmobranchs. In this study, glutamine metabolism in elasmobranch muscle was examined by isolating and characterizing mitochondria from the dogfish red muscle. The mitochondrial substrate oxidation as well as activities of several enzymes were measured.

Mitochondria were isolated from the lateral red muscle of the dogfish shark (Squalus acanthias) by differential centrifugation. Enzymes were assayed according to methods described by Chamberlin et al. (Am.J.Physiol. 160:R159-R166, 1991). The mitochondrial suspension was composed of well-coupled, intact mitochondria as indicated by respiratory control ratios exceeding 8 and negligible oxidation of NADH. Glutamine was oxidized at the same rate as β-hydroxybutyrate (n=5) and these two substrates were oxidized more rapidly than any other substrate tested. The isolated mitochondria had high activities of glutaminase and aspartate aminotransferase. Aminotransferases, however, do not appear to be involved in glutamine metabolism since aminooxyacetate (5 mM), an inhibitor of aminotransferases, failed to inhibit glutamine oxidation. Glutamate oxidation, on the other hand, was inhibited by 85% (n=3). This result indicates that intramitochondrial glutamate derived from glutamine is oxidized primarily via glutamate dehydrogenase, while oxidation of added glutamate is dependent upon transaminase activity. Complete oxidation of glutamine in isolated mitochondria requires the production of acetyl-CoA to maintain citric acid cycle activity. Malic enzyme, which is present in the mitochondria, could provide pyruvate which would then be oxidized to acetyl-CoA. The high activity of glutaminase and malic enzyme in the isolated mitochondria is similar to that found in cultured cells which use glutamine as an energy source (Kovacevic, Biochem.J. 125: 757-763, 1971.).

Glutamine synthetase could not be detected in dogfish red muscle while red muscle glutaminase activity was easily measured (3.3 μmol/min/g wet weight). This high glutaminase to glutamine synthetase ratio is similar to that measured in the red muscle of bony fishes (Chamberlin et al., Am.J.Physiol. 160:R159-R166, 1991), indicating that fish muscle, unlike mammalian muscle, is involved in glutamine oxidation, not synthesis. The rate of glutamine oxidation by dogfish mitochondria was 2.7- and 5.4-times greater than the respective rates observed in bowfin and lake char mitochondria (Chamberlin et al., Am.J.Physiol. 160:R159-R166,1991). In addition, the activity of glutaminase in the dogfish mitochondrial preparation was 14.7- and 14.0-times greater than that in bowfin and lake char, respectively (Chamberlin et al., Am.J.Physiol. 160:R159-R166, 1991). Although glutamine is oxidized in the muscles of fish representing widely divergent taxonomic groups, glutamine appears to be a particularly important energy source in elasmobranch red muscle.

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