Of fish, fat, and fuel: fatty acid transport in trout muscle

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ABOUT 45 YEARS AGO, Edgar Black and coworkers (1–7) began examining the consequences of exhaustive exercise to fish muscle. These early pioneering studies laid the groundwork for the building of a fairly comprehensive model explaining how muscle fuels these bouts of high-intensity, exhaustive-type exercise (12). The early stage of exhaustive exercise (first 15–20 s) is fueled by hydrolysis of high-energy phosphates (e.g., ATP, PCr). There is a shift away from the initial phosphagen hydrolysis to activation of “anaerobic” glycolysis and glycolysis, as the rate of ATP demand exceeds which can be provided via oxidative metabolism (8, 9). Consequently, at exhaustion, there is a decline in muscle ATP, PCr, muscle glycogen, and accumulation of lactate (12). Regeneration of muscle ATP, PCr, and glycogen and clearance of the lactate load are necessary if fish are to regain their ability to sprint or burst perform. It is generally accepted that the accumulated lactate is not used as an oxidative substrate to fuel the recovery process; rather it is retained within the muscle as the main substrate for glycogen recovery (12). So, the question emerges: what is fueling the recovery processes? The obvious candidates are carbohydrate, protein, or lipid. The first two are unlikely given that fish (at least salmonid) muscle does not readily take up or use exogenous carbohydrate (e.g., glucose, lactate, pyruvate; Refs. 10, 11) and protein makes a negligible contribution to oxidative metabolism. That leaves lipid as the prime substrate to fuel recovery from exhaustive exercise. Because the white muscle of trout and other salmonids have considerable lipid reserves, they are considered “fatty fish.” However, relatively little is known about the contribution this fuel makes to muscle metabolism and how muscle lipid metabolism is regulated.

Recently, Richards and coworkers (15) examined the role of lipid as a fuel for trout white muscle and found that during recovery from exhaustive exercise, both intra- and extramuscular lipid is oxidized by the white muscle to fuel the regeneration of ATP, PCr, and glycogen (15). In a current study in this issue of the American Journal of Physiology-Regulatory, Integrative and Comparative Physiology (14), the question of how lipid traverses the muscle cell and the plasticity of the process is investigated. Richards et al. (14) test the hypotheses that 1) fatty acids traverse the muscle membrane via facilitated transport and 2) the rate of transport may be limiting to lipid oxidation, muscle lipid use was manipulated in two ways: fish were subjected to either 5 days of continuous “aerobic” swimming or 9 days of chronic cortisol elevation. Both chronic elevations in plasma cortisol and continuous aerobic swimming have been reported to stimulate lipid oxidation in trout. The hypothesis Richards et al. tested is that increased lipid use by muscle would be reflected in an increase in fatty acid uptake by the muscle. However, this hypothesis was not supported by the data, as palmitate uptake by vesicles from both red and white muscle was not altered by either treatment. This contrasts with what is seen in mammals where chronic muscle stimulation (akin to sustained swimming) results in an increase in both fatty acid uptake and oxidation, suggesting a link between rates of uptake and use. This was not the case in trout. It could be that either the treatment did not alter lipid oxidation as predicted or that processes unrelated to transmembrane transport regulate the rate of lipid oxidation.

In summary, this paper presents a novel and integrative approach to the study of fat transport and metabolism in fish, an important and poorly understood metabolic process in fish. Evidence is presented suggesting the existence of novel fatty acid transport proteins in red and white muscle of rainbow trout that likely represent another member of a family of fatty acid transport proteins.

REFERENCES


