Fish muscle physiology and plasticity

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Key points

- Locomotion is essential to the survival and fitness of fishes
- Locomotion is powered by skeletal muscle, which demonstrates a high degree of phenotypic plasticity in fishes
- Fishes can alter many of the structural and metabolic properties of their skeletal muscle in response to various stimuli, including temperature, oxygen availability, and exercise.
- Understanding how skeletal muscle remodeling alters the locomotor performance of fishes is challenging, but is of interest to fish physiologists

Glossary

Muscle plasticity The ability of an organism to express alternative muscle phenotypes in response to various environmental, mechanical, and/or physiological stimuli

Phenotypic flexibility Reversible and repeatable phenotypic changes that occur during juvenile or adult life stages over a period of several days or longer, typically in response to alterations in the environment

Developmental plasticity Phenotypic changes that occur during early development (embryonic or larval life stages) in response to an environmental stressor, and that tend to have persistent effects on the adult phenotype

Burst performance High intensity swimming that is powered by white muscle fibers and can only be maintained for short periods (<20 s) before muscular fatigue

Sustained performance Low intensity swimming that is powered by red muscle fibers and can be maintained for long periods of time (>200 min) without muscular fatigue

Prolonged performance Moderate intensity swimming that is the transitional mode between sustained and burst swimming. Prolonged swimming is powered by both red and white muscle fibers and can be maintained for intermediate intervals of time (20 s–200 min) before muscular fatigue

Steady swimming Swimming bouts in which speed and direction are nearly constant, such as long-distance migrations

Unsteady swimming Swimming bouts that include rapid changes in speed and direction, such as accelerations, fast-starts, and burst-coast movements

Electromyography (EMG) A technique for evaluating and recording the electrical activity produced by skeletal muscles

Kinematics The study of motion of objects (e.g., fishes) through space and time (e.g., position, velocity, acceleration), but excluding the forces that caused that motion

Swim tunnel (swim flume) A closed circuit where fish are obligated to swim against water flow, allowing for assessment of the physiology, behavior, and/or kinematics of swimming fish

Hypertrophy An increase in the size of a cell, typically identified by an increase in cell cross-sectional area

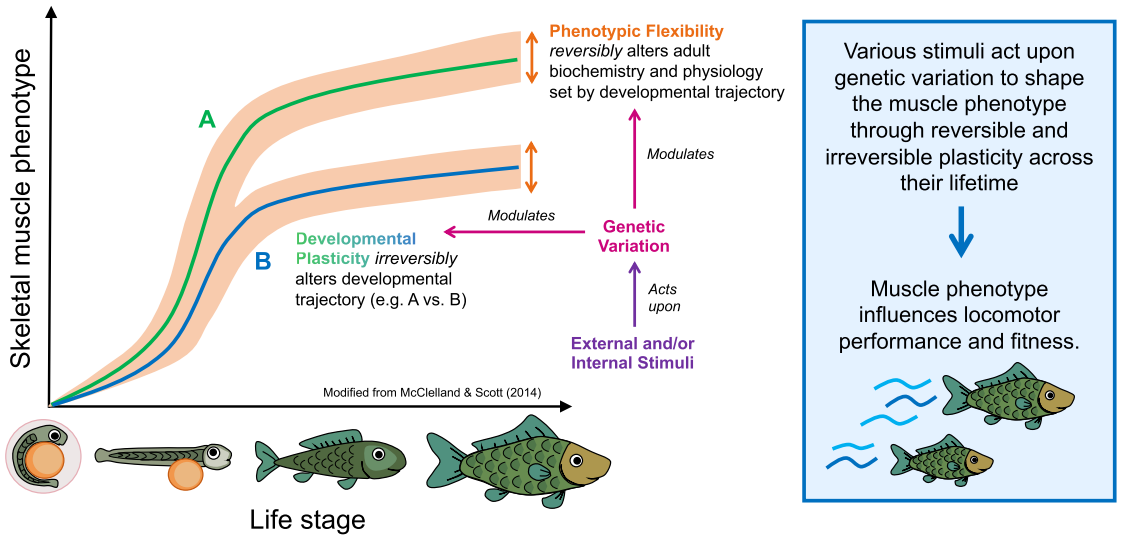
Hyperplasia An increase in the number of cells in a tissue generated via mitosis

Muscle atrophy Loss of muscle tissue due to net loss of proteins, organelles, and cytoplasm.
Thermal performance curve An experimental and analytical approach where the performance of an animal (e.g., swim speed or metabolic rate) is investigated at several discrete temperatures to characterize, the effects of a change in temperature on biological processes and fitness
Angiogenesis The growth of new blood vessels by extension or splitting of (e.g., branching from) existing vessels
Exercise training of fish Forced locomotion (e.g., swimming) at low-to moderate-intensities for long periods (e.g., hours, days), typically with a consistent routine
Oxygen cascade A complex and integrated pathway within an animal that involves the sensing, uptake, transport, and utilization of oxygen

Abstract

Skeletal muscle is one of the most plastic tissues in the body of fishes. Fishes can modify many of the structural and metabolic properties of their skeletal muscle in response to environmental, mechanical, and physiological stimuli. This chapter highlights the remarkable plasticity of fish skeletal muscle and discusses general patterns of muscle remodeling in response to stimuli such as exercise as well as alterations in environmental temperature or oxygen availability. This chapter also explores how changes in the skeletal muscle phenotype influence locomotor performance and therefore the ultimate survival and fitness of fishes.

Teaching slide



Introduction

Locomotor performance is linked to the survival and fitness of fishes. The ability to perform essential daily tasks (e.g., predator avoidance, prey capture) often requires bursts of speed or periods of prolonged activity. These propulsive movements are powered by the skeletal musculature, which is considered one of the most dynamic tissues in the body of fishes. Fishes can modify many of the structural and metabolic properties of their skeletal muscle to ensure that ecologically-important tasks can be performed in the face of changing environmental variables or physiological demands (for reviews, see Guderley, 1990; Guderley, 2004; Sänger, 1993; Davison, 1997; Watabe, 2002; Johnston, 2006; Kieffer, 2010; McClelland and Scott, 2014).

The ability of a fish to alter its muscle phenotype in response to various environmental (e.g., temperature), mechanical (e.g., exercise), and physiological (e.g., metabolic substrate supply) stimuli is termed **muscle plasticity**. Muscle plasticity often involves changes to muscle fibers and their organelles (e.g., Johnston and Maitland, 1980) or to supporting structures such as capillaries (e.g., Egginton and Sidell, 1989). In adult fishes, phenotypic changes to the muscle tend to be reversible (**phenotypic flexibility**) and can occur seasonally, with movement across environmental clines, or in response to extreme locomotory feats (e.g., migration). For example, chronic exposure to cold temperatures can lead to a reversible increase in muscle mitochondrial content to compensate for the decelerating effect of low temperature on biochemical reaction rates (e.g., Guderley and Gawlicka, 1992; McClelland et al.,

2006). The capacity for reversible muscle remodeling is thought to be advantageous when fishes occupy spatially and/or temporally variable habitats because it can enable phenotype–environment matching across a wider range of environmental conditions than could be achieved if traits were fixed.

In contrast, changes in the muscle phenotype that occur during early development (i.e., embryonic and larval stages) are often irreversible owing to the rapid pace of ontogenetic change, resulting in different developmental trajectories (**developmental plasticity**) (Johnston, 2006; McClelland and Scott, 2014). Developmental plasticity is thought to be advantageous when environments are relatively stable as, under these circumstances, phenotypic changes made during early life could prepare fishes for future environmental conditions. For example, zebrafish (*Danio rerio*) exposed to high temperatures as embryos exhibited improved swimming performance at similarly high temperatures during adulthood (Scott and Johnston, 2012). Although the importance of early life experiences have long been recognized, relatively few studies have explored the persistent effects of developmental exposures on the muscle phenotype and locomotor performance of adult fishes. In this chapter, we highlight the remarkable plasticity of skeletal muscle in fishes across various life stages and discuss general patterns of muscle remodeling in response to prevalent stimuli, including environmental temperature, oxygen (O_2) availability, and exercise. We also explore how changes in the skeletal muscle phenotype can influence the locomotor performance of fishes, which can have ultimate consequences for their survival and fitness.

Skeletal muscle anatomy

Skeletal muscle is the largest organ system in the body of fishes. Gram for gram, fish have more muscle than any other vertebrate, where it can account for more than 50% of an individual's body mass (Bone, 1978). The axial (swimming) muscle of fishes is generally composed of two functionally distinct muscle fiber types: white (fast-glycolytic) and red (slow-oxidative) muscle fibers, although some species also have pink (fast-oxidative) fibers that are intermediate between red and white fibers, in form and function (Fig. 1). Unlike mosaic muscles in tetrapods, the two fiber types in fish muscle are anatomically separated. White muscle fibers are arranged helically in hundreds of nested W-shaped blocks called myotomes. Each myotome is separated from adjacent myotomes with a thin sheet of connective tissue (myoseptum) that is firmly attached to the skin and skeleton. Red muscle fibers are typically situated directly beneath the lateral line, where they run parallel to the long body axis. In a minority of groups such as scombroids and lamnid sharks, red muscle tissue extends deeper into the body, and when combined with a vascular counter-current heater exchange mechanism, enables regional endothermy of the viscera and nearby tissues (Block and Finnerty, 1994).

White muscle fibers constitute about 90% of skeletal muscle in most fishes. These fibers are large in diameter and tightly packed with myofibrils that occupy about 75–95% of the fiber volume. They contain low mitochondrial densities and a sparse capillary network. Thus, white muscle fibers are heavily dependent on glycogen-fueled anaerobic metabolism for ATP production, which

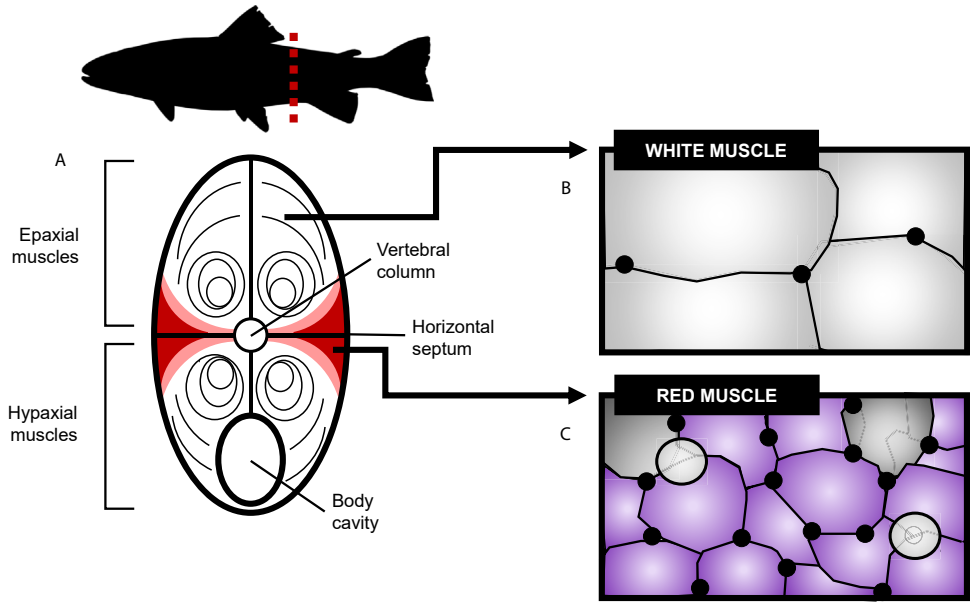


Fig. 1 Axial (swimming) muscle anatomy and physiology in fishes. (A) Red (slow-oxidative), white (fast-glycolytic) and, when present, pink (fast-oxidative) fibers are spatially segregated in fishes. (B) White muscle is composed of large diameter, fatigue-sensitive (glycolytic) fibers. Relative to other fiber types, white muscle has a low mitochondria abundance (as reflected by light staining for succinate dehydrogenase) and few capillaries (indicated by dark circles). (C) Red fibers are in the minority and cluster around the horizontal septum in a wedge-shaped region. Red muscle is composed of small diameter, fatigue-resistant (aerobic) fibers that are well-supplied by capillaries. It typically stains intensely for succinate dehydrogenase activity (indicating high mitochondrial content) and has high levels of the oxygen-binding protein myoglobin. Lipid droplets (indicated by larger yellow circles) may also be observed in red muscle.

is accompanied by the buildup of lactic acid. Since white fibers contract quickly and fatigue rapidly, they are generally recruited for burst activity, such as avoiding predators or to ambush prey.

Red fibers differ from white muscle fibers in fundamental ways and constitute about 10% of the skeletal muscle of fishes, although fishes with more active lifestyles tend to have a higher proportion of red muscle (Greer-Walker and Pull, 1973). Red fibers are small in diameter, contain high mitochondrial densities, and have a dense capillary network. They can contain high levels of myoglobin to facilitate mitochondrial O_2 supply, as well as increased lipid stores and a higher capacity for lipid oxidation compared to white muscle fibers. Red muscle fibers are recruited for sustained swimming activities, fueled by aerobic metabolism, such as long-distance migrations.

The selective recruitment of red, pink, and white muscle fibers allows the muscle to fulfill a diverse array of functional tasks, from burst escape responses to sustained endurance swimming. As swimming speed increases, red muscle fibers are initially recruited, followed by intermediate pink fibers, and then finally white muscle fibers at higher swimming intensities (Jayne and Lauder, 1994).

Assessing skeletal muscle function

The usual swimming mode of fishes involves movements powered by the axial musculature: undulations of the body and caudal fin. Thus, skeletal muscle function is routinely assessed by quantifying the swimming performance of fishes, which is broadly grouped into three categories: burst, prolonged, and sustained performance (Beamish, 1978). **Burst performance** involves high intensity swimming that relies almost exclusively on anaerobic metabolism within the white muscle for ATP production. Burst performance lasts for short periods of time (<20 s), can significantly reduce intracellular fuel stores, and leads to the accumulation of metabolites such as lactate. **Sustained performance**, on the other hand, involves low intensity swimming that is powered by the aerobic red muscle fibers and can be maintained for long periods of time (>200 min) without muscular fatigue. **Prolonged performance** involves moderate intensity swimming that is the transitional mode between sustained and burst swimming. Prolonged performance is powered by both aerobic and anaerobic metabolism and can be maintained for intermediate intervals of time (20 s–200 min) before muscular fatigue. In addition to burst, sustained and prolonged performance, swimming can be further categorized as **steady** or **unsteady**. During steady swimming, fish travel at a near constant speed and direction (e.g., sprints, migrations), whereas during unsteady swimming, fish make rapid changes in speed and direction (e.g., accelerations, fast-starts, burst-coast movements) (Langerhans and Reznick, 2010).

Burst performance

Various protocols have been developed to assess swimming performance in fishes with relative ease and high repeatability. For example, burst performance can be assessed by eliciting a "fast-start" response in fish, a locomotor behavior that is often used during predator-prey interactions. Fast-starts are unsteady swimming bursts that rapidly propel fish away from a predator threat or toward a prey item. A fast-start is initiated by the contraction of muscle on one side of the body to form a C- or S-shaped posture, followed by the rapid contraction of muscle on the opposing (contralateral) side of the body to propel the fish forward (Domenici and Blake, 1997). The assessment of fast-starts typically involves the use of high-speed cameras to analyze swimming **kinematics** (e.g., velocity, acceleration, directional change of the body), thereby providing a non-invasive and quantitative measure of burst performance. Several studies have also used **electromyography (EMG)** to assess the burst performance of fishes in addition to other forms of swimming. EMG is a relatively invasive technique that involves the precise placement of electrodes within the skeletal musculature to measure and record the electrical activity of the muscle fibers as their membranes depolarize. EMGs provide insight into the timing and duration of signals inducing muscle contraction, as well as the fiber types or muscle groups activated during locomotion.

Prolonged performance

The most common method used to measure prolonged swimming performance in fishes is the **critical swimming speed (U_{crit})** test. In this test, a fish is placed in a **swim tunnel** and forced to swim against a particular water velocity for a set time interval. Time intervals as low as 2 min and as high as 75 min have been used in these trials. Water velocity (and therefore swimming speed) is then increased by a set increment until the fish fails to swim for the entire time interval. U_{crit} is calculated using the following equation and subsequently converted to body lengths per second (BL s^{-1}):

$$U_{crit}\left(\text{cm s}^{-1}\right) = V_f + \left[\left(T_1 / t\right) \times dv\right]$$

where V_f is the speed of the last completed interval (cm s^{-1}), T_1 is the time swum at the final velocity at which the fish fatigued (minutes), t is the time increment (e.g., 20 min) and dv is the velocity increment (e.g., 10 cm s^{-1}) (Brett, 1964). The time interval and velocity increment of U_{crit} tests can vary considerably across studies making inter- and intra-specific comparisons difficult. However, U_{crit} tests offer several advantages: (*i*) they are useful for evaluating the effects of various biotic and abiotic factors on swimming performance, (*ii*) they are often performed in swim tunnel respirometers that allow for the measurement of O_2 consumption rate during exercise, and (*iii*) they allow for the standardization of training intensity (as % U_{crit}) for submaximal exercise tests. Furthermore, U_{crit} tests can be readily modified for the assessment of **sprint swimming speed (U_{sprint})**, by shortening

time increments (e.g., 10 s). U_{sprint} tests are particularly useful for assessing the swimming performance of relatively inactive (e.g., benthic) species (Tierney et al., 2011).

Sustained performance

Sustained performance is often assessed using an endurance test, in which fish are exposed to a fixed water velocity until exhaustion. Although this procedure can be repeated for a number of sub-maximum swimming speeds, a major drawback is the test duration, as many species can swim at low water velocities for several hours or days. For example, Greer-Walker (1971) kept saithe (*Pollachius sirens*) swimming continuously between 2.1 and 3.0 BL s^{-1} for 42 days, after which the experiments were terminated. It is now widely accepted that if swimming is maintained at a fixed water velocity for more than 200 min, it is considered sustained performance, mainly for the purpose of convenience.

Alternative modes of locomotion

Swimming powered by body and caudal fin undulations is one of many locomotor modes exhibited by fishes. In some fishes, swimming is powered primarily by movement of the median and/or paired pectoral fins, which often results in slower speeds but greater maneuverability. Indeed, many fishes living in structurally complex habitats (e.g., coral reefs) rely on median and/or paired fin propulsion to power their locomotor movements (Thorsen and Westneat, 2005). Fin swimming can be assessed using similar methods to body- and caudal fin-powered locomotion, including kinematic analyses, EMG recordings, and U_{crit} tests. There are also several non-swimming modes of locomotion exhibited by species that spend time on land as part of their natural history. Diverse modes of terrestrial locomotion have been documented among amphibious fishes, including walking, crutching, and jumping (for a review, see Lutek et al., 2022). Terrestrial locomotion has been assessed using conventional methods, such as kinematic analyses and EMGs. For example, Foster et al. (2018) placed electrodes in the pectoral fin adductor and abductor muscles in *Polypterus senegalus* to assess fin muscle activity during walking movement on land. Other unique protocols have been developed for assessing terrestrial locomotion, from endurance jumping tests in killifishes (Brunt et al., 2016) to forced treadmill walking in mudskippers (Jew et al., 2013). Regardless of how a fish moves, the assessment of locomotor performance can serve as strong proxy for muscle performance and organismal fitness.

Skeletal muscle plasticity

The muscle phenotype of fishes is exceptionally dynamic and is capable of responding to various environmental (e.g., temperature), mechanical (e.g., exercise), and physiological (e.g., substrate supply) stimuli with a number of structural and metabolic changes. Among the most common changes reported in fish muscle is an increase in muscle mass through hyperplastic and/or hypertrophic growth. **Hyperplasic growth** refers to an increase in the number of muscle fibers, and is the primary mechanism for building muscle mass during development in early life (Johnston, 2006). During hyperplasia, precursor cells (myoblasts) fuse to form short, multinucleated myotubes. These myotubes then elongate by fusing with additional myoblasts to become mature muscle fibers. In adults, gains in muscle mass are primary driven by **hypertrophic growth**, an increase in the diameter (cross-sectional area) of existing muscle fibers (Johnston, 2006). During hypertrophy, myoblasts are absorbed into mature muscle fibers. The maximum fiber diameter achieved through hypertrophy is likely set by diffusional constraints that vary with body mass, activity patterns, and metabolic demand. Muscle fiber hypertrophy is typically accompanied by increases in myofibril content and other cellular components (e.g., mitochondria). In contrast to muscle hyperplasia and muscle hypertrophy, fish can also exhibit **muscle atrophy** due to disuse, aging, injury, or disease states. Muscle atrophy is the "wasting" of skeletal muscle that, on a gross level, manifests as a loss of muscle mass and a reduced cross-sectional area of muscle fibers. At the subcellular level, muscle atrophy is linked to a loss of protein, cytoplasm, and mitochondria (Reilly and Franklin, 2016).

Beyond large scale changes in muscle mass, other finer scale alterations in muscle structure and metabolism can occur in response to various stimuli. Changes to the supporting capillary network, intramuscular fuel stores, or the contractile properties of the muscle may alter muscle performance and/or optimize performance under specific conditions (for reviews, see Sänger, 1993; Davison, 1997; Guderley, 2004; Johnston, 2006; Kieffer, 2010; McClelland and Scott, 2014). Indeed, some fishes can alter the volume fraction of myofibrils or change myosin heavy or light chain (MyHC, MyLC) isoform expression in their muscle fibers to alter the overall contractile performance of the muscle (for a review, see Watabe, 2002). Many fishes can also reversibly change the volume fraction (percentage by volume, vol%) of mitochondria within red and white muscle fibers and/or the activity of mitochondrial enzymes, thereby altering the capacity for lipid or carbohydrate oxidation and ATP synthesis (e.g., Johnston and Bernard, 1982b; Egginton and Sidell, 1989; Guderley and Gawlicka, 1992). Overall, muscle plasticity in fishes is relatively well-characterized at several levels of biological organization. Below, we discuss common patterns of muscle remodeling in response to prevalent stimuli and how these changes can impact locomotor performance.

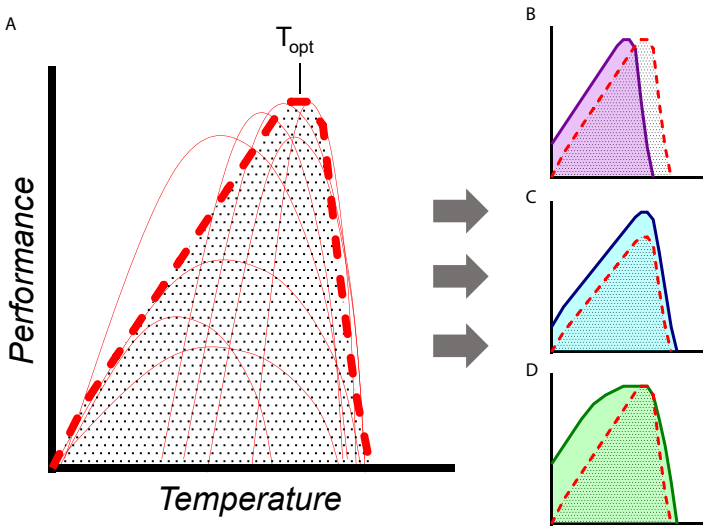


Fig. 2 Conceptual model of a thermal performance curve (TPC) and some potential effects of acclimation on TPCs. (A) A representative thermal performance curve describing how a biological process (i.e., performance) is altered by temperature. The temperature where performance is greatest (e.g., the peak of the TPC) is termed the optimal temperature (T_{opt}). Note that a typical TPC is the outcome of several overlapping TPCs (indicated by thin red lines), as most whole-animal or tissue-level performance metrics rely on several subordinate biological processes (e.g., for U_{crit}, underlying TPCs may include heart rate, muscle fiber power output, rate of fuel delivery to muscle cells, etc.). Many TPCs are plastic and can be altered by thermal acclimation. Some common alterations in TPC with acclimation include (B) shifts in T_{opt} (e.g., as may occur with alterations in enzyme isoforms and their respective environmental optima), (C) general alterations in performance (e.g., a general improvement in swimming performance due to muscle fiber hypertrophy), or (D) changes in the shape of the TPC (e.g., due to alterations in muscle fiber type ratios).

Temperature

Environmental temperature sets the pace of virtually all physiological processes in ectothermic fishes, and so systematically documenting the effects of temperature is of interest to many physiologists. The change in the rate of physiological process(es) across temperatures is routinely visualized using **thermal performance curves** (TPCs) (**Fig. 2**). TPCs are classically in the form of an inverted "U", where performance increases as temperature increases, peaks (reaches a maximum) at some intermediate temperature (T_{opt}), and then rapidly declines with further temperature increases (Schulte et al., 2011). Sharp declines in swimming performance above T_{opt} may be attributed to factors such as the breakdown of enzymes necessary for ATP production, a reduction in cardiac performance, or a reduction in O_2 availability. Interestingly, thermal acclimation can modify TPCs by altering their height (i.e., scope), the position of T_{opt}, and even the shape or breadth of curve. For instance, the temperature at which adult mosquitofish (*Gambusia holbrooki*) exhibited maximal U_{crit} performance (i.e., T_{opt}) increased by approximately 4–5 °C following acclimation to relatively warm water (Seebacher et al., 2015). A number of mechanisms may underpin changes in swimming performance driven by thermal acclimation, including the plasticity of skeletal muscle.

The influence of low temperature on muscle plasticity and function are also well-documented in fishes. Cold temperatures can immediately suppress high performance swimming by slowing critical biochemical processes involved in muscle contraction and O_2 movement through muscle fibers (Wardle, 1980; Sidell, 1998). During acute cold exposure, some fishes can temporarily offset the consequences of low temperature on swimming performance by altering patterns of muscle fiber recruitment. For example, Rome et al. (1984) showed that the threshold speed at which white muscle fibers were first recruited in common carp (*Cyprinus carpio*) decreased as the temperature was lowered. Thus, carp were able to achieve the same level of performance by recruiting faster-contracting muscle fibers, but at the expense of a more rapid onset of fatigue (Rome et al., 1984).

Chronic cold exposure can induce adaptive changes to the skeletal muscle that allow fishes to compensate for the effects of low temperature over the long-term. Although muscle responses can vary within and between species, cold acclimation tends to increase the aerobic capacity of fish skeletal muscle. For example, the cross-sectional area of aerobic fiber types in mosquitofish (*Gambusia holbrooki*) increased by 40% following cold acclimation, corresponding with a 20% higher U_{crit} than that of warm-acclimated fish when tested at a common low temperature (Hammill et al., 2004). Other studies have similarly reported increases in the proportion of red muscle with cold acclimation with concomitant improvements in cold-water swimming performance (e.g., Sisson and Sidell, 1987). Because cold acclimation increases the proportion of red muscle, the resulting decrease in white muscle could theoretically impair burst swimming performance. However, the opposite can also occur, as cold acclimation was found to improve the fast-start performance of goldfish (*Carassius auratus*) and mummichog (*Fundulus heteroclitus*) by increasing myosin ATPase activity in white fibers (Johnson and Bennett, 1995).

The aerobic capacity of fish muscle can also be enhanced via the proliferation of mitochondria in both red and white muscle fibers. Indeed, striped bass (*Morone saxatilis*) acclimated to 5 °C increased mitochondrial volume fraction in red muscle fibers by

50%, leading to the fibers being almost half-filled with mitochondria (Egginton and Sidell, 1989). The proliferation of mitochondria increases the overall content of mitochondrial enzymes in the muscle and reduces the diffusion distance for O_2 between the capillaries and mitochondria. Diffusion constraints may also be attenuated via **angiogenesis** in the muscle. For example, crucian carp (*Carassius carassius*) acclimated to 2 °C for two months more than doubled the capillarity of their red and white muscle fibers, thereby reducing maximum diffusion distances for O_2 by up to 30% (Johnston, 1982). Cold acclimation is also known to increase red muscle myoglobin content, and the activity of enzymes involved with the electron transport chain, the citric acid cycle, and lipid metabolism (e.g., Sidell, 1980; Guderley and Gawlicka, 1992). Interestingly, the activity of glycolytic enzymes tends to remain unchanged or decline slightly with cold acclimation (e.g., McClelland et al., 2006).

Whereas cold acclimation tends to increase the aerobic capacity of skeletal muscle, warm acclimation tends to have the opposite effect. For example, rainbow trout (*Oncorhynchus mykiss*) acclimated to 18 °C showed 50% lower β-hydroxyacyl CoA dehydrogenase (HOAD) activity in the red and white muscle compared to fish acclimated to 4 °C, indicating a significantly reduced capacity for lipid oxidation (Guderley and Gawlicka, 1992). Nevertheless, warm acclimation has been reported to improve the aerobic swimming performance in several adult fishes (Seebacher et al., 2015; Parisi et al., 2020), suggesting that alternative mechanisms may underpin these performance changes (e.g., cardiac performance). During early development, elevated environmental temperatures can also have significant effects on muscle plasticity and performance. For example, the lifetime production of fast muscle fibers in zebrafish (*Danio rerio*) showed an optimum when fish were reared at 26 °C and was 19% and 14% lower at rearing temperatures of 22 °C and 31 °C, respectively (Johnston et al., 2009). Elevated rearing temperature was also associated with changes in muscle fiber composition and mitochondrial enzyme activities that persisted into adulthood, as well as improved U_{crit} performance at similarly high temperatures (Scott and Johnston, 2012; Schnurr et al., 2014). Understanding the link between muscle plasticity and performance in response to environmental temperature has become an important area of investigation, particularly in the face of rapid climate change.

Oxygen availability

Decreases in environmental O_2 availability (hypoxia) have considerable consequences for swimming performance due to short-term and long-term alterations in muscle phenotype. Acute hypoxia exposure typically limits aerobic swimming performance in fishes (e.g., reduced U_{crit}). This decline in U_{crit} has repeatedly been shown to be a direct consequence of limitation in maximum metabolic rate and aerobic scope (e.g., Petersen and Gamperl, 2010), presumably due to hypoxia-induced limitations in oxygen delivery or transport to red muscles fibers that are crucial for sustained swimming. In at least one case, hypoxia exposure also limits stamina at optimal swimming speed (in golden gray mullet, *Liza aurata*, see Vagner et al., 2008), likely due to an increased reliance on unsustainable anerobic metabolism to supplement muscle energetic demands. While the vast majority of species examined show a decline in U_{crit} in hypoxia, there is considerable interspecific and even intraspecific variation in the degree of performance impairment that is observed, emphasizing the complexity of this trait (Domenici et al., 2013).

Hypoxia acclimation induces a slew of molecular, biochemical, physiological, and behavioral alterations in fishes, many of which could foreseeably impact muscle phenotype and swimming performance. Spontaneous swimming activity may increase, decrease, or be unaffected by hypoxia exposure due to a variety of modulators including species lifestyle and the severity of the hypoxia exposure (Domenici et al., 2013). Many species initiate compensatory changes in their skeletal muscles to maintain aerobic performance in response to hypoxia exposure, including increased mitochondrial content, greater capillary density (Johnston and Bernard, 1984), and/or increased capacity for anaerobic performance (e.g., increased glycolytic enzyme activity) (Martinez et al., 2006). However, these responses are not universal. For example, when acclimated to hypoxia, the cyprinid *Tinca tinca* shows decreased mitochondrial volume in its red and white muscle, decreased cytochrome c oxidase activity (COX) (Johnston and Bernard, 1982b), and a decreased capillary:fiber area ratio (Johnston and Bernard, 1982a). Aside from alterations in the muscle tissue itself, fishes may also extensively modify other systems related to oxygen transport or metabolism in response to oxygen limitation, and so indirectly support exercise performance. For example, cyprinids extensively modify their gill structure and blood oxygen carrying capacity when acclimated to hypoxia, and this appears to also protect exercise performance in hypoxia in goldfish (Fu et al., 2011). Interestingly, prior hypoxia acclimation (and its associated compensatory responses) does not reliably prevent loss of swimming performance in hypoxia. Indeed, in some cases prior hypoxia exposure can lead to further loss of performance due to other associated negative effects such as loss of cardiac stroke volume (Petersen and Gamperl, 2010).

Compared to the effects of hypoxia, we know far less about skeletal muscle plasticity and performance in response to elevated environmental O_2 availability. Many fishes encounter oxygenation levels above normal partial pressures ($pO_2 > 21$ kPa; hyperoxia) due to aquaculture practices or algal photosynthesis, as well as elevated O_2 concentrations as a result of amphibious behavior. Although the skeletal muscle of some fishes is insensitive to high environmental O_2 availability, many others exhibit dramatic changes in their muscle phenotype and altered locomotor performance. For example, after 14 days on land, the amphibious mangrove rivulus (*Kryptolebias marmoratus*) reversibly remodeled skeletal muscle toward a more aerobic phenotype (Brunt et al., 2016) owing to the higher O_2 concentrations in air relative to water (Rossi et al., 2018). Fish were found to significantly increase the size of red muscle fibers, the capillary:red fiber ratio, as well as reduce lactate production after jumping on land, which collectively improved terrestrial locomotion (Brunt et al., 2016).

Exercise

Fish show significant muscle remodeling in response to **exercise training**, which is typically described as forced locomotion (e.g., swimming) at low-to moderate-intensities for long periods. Numerous studies have demonstrated that exercise training can lead to an increase in muscle mass by stimulating the hypertrophy of muscle fibers (e.g., Johnston and Moon, 1980). It is not well understood whether exercise training also affects muscle fiber recruitment (hyperplasia). From an applied perspective, exercise-induced hypertrophy is of particular interest to fish physiologists because it has applicability to the aquaculture industry by mediating flesh quality. Indeed, the stimulatory effects of exercise training on muscle growth in economically-important species (e.g., salmonids) were initially reported more than 30 years ago, when East and Magnan (1987) discovered that moderate-intensity swim training caused brook trout (*Salvelinus fontinalis*) to increase in both length (3.5%) and weight (34%). From an ecological perspective, exercise-induced muscle growth can have significant implications for locomotor performance and, therefore, a fish's ability to perform ecologically-important tasks. For example, McFarlane et al. (2019) recently showed that (terrestrial) exercise training in the amphibious mangrove rivulus (*Kryptolebias marmoratus*) led to significant red muscle hypertrophy, resulting in enhanced terrestrial locomotor performance (i.e., jumping). For the mangrove rivulus, effective terrestrial locomotion is critical for traversing terrestrial landscapes to forage, disperse to new aquatic environments, seek moist terrestrial habitats during the dry season, avoid predation, and deposit embryos out of water.

Beyond changes in muscle mass, fish also tend to respond to exercise training by developing a more aerobic muscle phenotype, much like mammals respond to endurance training (for reviews, see Davison, 1997; Kieffer, 2010; Palstra and Planas, 2011; McClelland, 2012; McClelland and Scott, 2014). In several species, exercise training enhances the aerobic capacity of both red and white muscle fibers via the proliferation of mitochondria and/or the elevated activity of enzymes involved in aerobic ATP production (e.g., HOAD, COX, citrate synthase [CS], phosphofructokinase [PFK]) (Farrell et al., 1991; McClelland et al., 2006). In many cases, fishes also exhibit improved U_{crit} swimming performance following training (e.g., Young and Cech, 1993). Interestingly, exercise-induced changes in fish muscle and performance are often accompanied by modifications at other levels of the **oxygen cascade**, which is the series of physiological 'steps' that facilitates the transport of O_2 from the environment to the mitochondria where it is ultimately for ATP production (Scott, 2011). For example, crucian carp (*Carassius carassius*) exercised at 70% of their U_{crit} for 7–8 h exhibited a significant increase in gill surface area that improved their capacity for O_2 uptake from the environment (Brauner et al., 2011). Several studies have also demonstrated that cardiac performance can improve with exercise training (e.g., greater stroke volume; Farrell et al., 1991), which can enhance a fish's capacity to deliver oxygenated blood to the muscle. Another way in which fishes can enhance O_2 delivery to the muscle during exercise is to proliferate the capillary network in the muscle. Although **angiogenesis** can be regulated through numerous pathways, increased blood flow and, therefore, shear stress through the capillaries during exercise can result in the upregulation genes that promote **angiogenesis**, such as vascular endothelial growth factor (VEGF) (Fraisl et al., 2009). Increased muscle capillarity can serve to match O_2 delivery with O_2 utilization during exercise and maintains appropriate diffusion distances in muscle fibers experiencing exercise-induced hypertrophy.

Overall, exercise training influences multiple tissues in the body of fishes from the gills to the heart to the skeletal musculature. Work by Palstra and Planas (2011) even suggests that exercise training can be an important mediator of neuroplasticity, cognitive performance, immune function, and overall fish health. Thus, continuing to explore how exercise mediates plasticity and performance among fishes will be an important and fruitful avenue for future work.

Conclusions

Locomotion is essential for the survival and fitness of fishes and it is powered by their skeletal muscles, a complex and highly integrated system for metabolism and movement. Variations in skeletal muscle composition, placement, and activation have wide-ranging implications for how a fish behaves, performs, and interacts with its habitat. At the same time, skeletal muscle is highly plastic and capable of remodeling at the molecular, biochemical, cellular, and physiological level over development and in response to environmental challenges. Understanding how plastic changes in the skeletal muscle phenotype directly alter the locomotor performance of fishes is challenging and has become a major area of interest for fish physiologists working at the interface of organisms and their environment.

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