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EDITORIAL

Human Skeletal Muscle Fiber Type Switching Revisited

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Skeletal muscles are composed of several fiber types that differ in their structure, molecular composition, metabolic activity, and functional properties. The four major myosin heavy chain (MyHC) isoforms, slow-twitch oxidative (type I), fast-twitch oxidative (type IIa), fast-twitch oxidative glycolytic (type IId/x), and fast-twitch glycolytic (type IIb), are heterogeneously distributed in the skeletal muscles of mammals, and are useful markers for the muscle fiber types [1]. In humans, MyHC IIb is not detectable in skeletal muscles, but *MYH4*, the gene encoding this protein, is present in our genome. Muscle performance is, at least partly, dictated by the composition of muscle fiber types, and precise understanding of the fiber type has attracted much attention from sports scientists. In addition, increased interest in the role of skeletal muscle in metabolic diseases has extended the awareness of muscle fiber types to a wide audience in clinical medicine [2].

There is considerable inter-individual variability in the composition of skeletal muscle fiber types among humans. For example, the proportion of type I fibers in the vastus lateralis muscle ranged from 15% to 85% in a large cohort of sedentary and physically active individuals of both sexes, and type I fibers accounted for less than 35% or more than 65% of the fibers in 25% of the individuals [3]. Many studies published in the 1960s to 1980s of muscle biopsies from athletes also showed marked variability in the fiber type profile because type I fibers tended to be more prominent in endurance athletes and type II fibers were more predominant in sprinters [4]. It was long thought that the human muscle fiber type was genetically determined [5-8] because most studies that compared the composition of muscle fiber types before and after training showed that neither endurance training nor strength training altered the muscle fiber composition [9-30]. Only a few studies have reported that endurance training causes a shift from type IIx to type IIa fibers [31-34] or from type II to type I fibers [35,36]. However, since the 1980s, researchers in this field have hypothesized that a switch in fiber types may also occur in human skeletal muscles based on the results of mouse and rat studies and the changes in oxidative/glycolytic enzymes following endurance/sprint exercise training in humans [37-38]. In addition, most of studies above employed myofibrillar actomyosin ATPase histochemistry for determination of fiber type composition. This analysis appears to be significantly different from antibody-based analyses. Since myofibrillar actomyosin ATPase histochemistry is vulnerable to subtle changes in pH and requires staining of serial sections, caution should be taken when interpreting the data, particularly with regard to changes in type I fibers [39]. It should be emphasized that there is still a lack of evidence from longitudinal exercise training studies showing a switch between type I and type II fibers. Therefore, the extent to which these variations are elicited by training or simply reflect genetic factors remains unknown [40].

However, a complete switch from type I to type II fibers in human skeletal muscle can occur after long-term (over several years) spinal cord injury [41]. This finding indicates human skeletal muscle may possess the capacity to undergo a shift in muscle fiber type in response to physiological/pathological stimuli. Therefore, more evidence for an exercise-induced switch in muscle fiber types should be accumulated from controlled human studies. The abundance of mitochondria and oxidative enzymes is greatest in type I fibers and lowest in type IId/x fibers in human muscles, whereas the oxidative potential is highest in IIa fibers and lowest in IIb fibers in mouse and rat muscles. Considering these species differences, mouse skeletal muscle does not seem to be the best model of human muscle, and it is important to consider these differences when one tries to extrapolate conclusions derived from studies of genetic models to human conditions [2].

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CONFLICT OF INTEREST

The authors declare that they do not have conflict of interest.

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