

EXERCISE AND SPORT SCIENCES REVIEWS



ESSR Journal Club

Covered Article: “[Myofilament Protein Alterations Promote Physical Disability in Aging and Disease](#)” by Mark S. Miller and Michael J. Toth.

Exercise and Sport Sciences Reviews. 41(2): 93-99, April 2013.

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1. Why is a reductionist approach necessary to identify the mechanisms that contribute to age- and disease-associated declines in muscle power?
2. Why is the force generated by a half-sarcomere equivalent to that produced by the myofibril in which it is located?
3. How are the numbers of strongly bound cross-bridges and the force exerted by each cross-bridge calculated?
4. Why is contraction velocity related to myosin attachment rate?
5. How does altering the number of strongly bound cross-bridges, the attachment time of each cross-bridge, and the force exerted by each cross-bridge influence the force generated by a half-sarcomere (Fig. 2)?
6. How do myosin ATPase and the myosin light chains influence cross-bridge kinetics?
7. Why do older adults produce less muscle power than young adults when there is no difference between the two groups in the peak force during a maximal isometric contraction?
8. Why does an age-associated decrease in the phosphorylation of the fast isoform of the regulatory MLC reduce the rate of force production?
9. Does myofilaments protein content or isoform expression change with advancing age?
10. How can heart-failure patients exhibit no reduction in isometric force when they experience a decrease in single-fiber myosin content?
11. Contrast the adaptations experienced in aging and with heart failure that both decrease muscle power (Fig. 3).
12. Does physical inactivity produce a shift in MHC isoform expression in heart-failure patients?
13. What is the main conclusion of the paper?