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Muscle soreness, particularly the delayed-onset of muscle soreness (DOMS) typically occurs one to three days post-exercise, is best described as microscopic injury (microtrauma) to muscle tissue and fibers caused by muscle action and excessive mechanical force imposed upon the muscle and connective tissues (Powers & Howley, 2008). The occurrence of DOMS is described as a process that involves: 1.) Structural damage to muscle fibers; 2.) Damage to the membrane of the sarcoplasmic reticulum; 3.) Calcium leakage from the sarcoplasmic reticulum inhibiting ATP production; 4.) Protease activation resulting in the breakdown of cellular proteins; 5.) Inflammatory response; and 6.) Edema and pain (Powers & Howley, 2018). Notably, muscle soreness is not linked to the accumulation of lactic acid or muscle spasms (Fahey, 2019).

Muscle actions are classified as concentric, eccentric, or isometric (Haff & Triplett, 2016). A concentric muscle action is a shortening of the muscle whereby the force contracting the muscle is greater than the resistive force. An eccentric muscle action is a lengthening of the muscle whereby the force contracting the muscle is less than the resistive force. Isometric muscle actions occur when the muscle length remains fixed and the force contracting the muscle is equal to the resistive force. Eccentric muscle actions have been found to produce DOMS more than concentric muscle actions (Powers & Howley, 2018). Moreover, DOMS is exacerbated when performing unfamiliar exercises or using muscles/muscle groups that are less frequently used or that an individual is unaccustomed to engaging on a regular basis (Lewis et al., 2012).

McHugh et al. (1999) define a concept called the “subsequent bout effect” that describes a reduction of DOMS following the subsequent performance (bout) of the same exercise. The occurrence of the training bout effect is linked to three potential causal factors: 1.) Neural theory;

2.) Connective tissue theory; and 3.) Cellular theory. The neural theory points to the primary source of exercise-induced muscle injury as occurring in type II fibers. Additionally, training adaptations in the recruitment of muscle fibers and motor unit activation produce enhanced recruitment of motor units and, in turn, the involvement of a larger number of muscle fibers. As the number of muscle fibers engaged increases, individual fibers experience less contractile stress because the stress is distributed across a greater magnitude of fibers. The connective tissue theory supports the view that muscle fiber microtrauma that takes place during the initial exercise bout causes an increase in connective tissue so as to protect the muscle against the stress of further exercise. The cellular theory supports the concept that microtrauma caused by exercise instigates the creation of new proteins that enhance the structural integrity of the muscle (Powers & Howley, 2018).

Mitigation and treatment of pain and soreness associated with DOMS includes rest, ice, compression, and elevation, the use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin, Motrin, Aleve, ibuprofen, Ascriptin, or naproxen. Rest, ice, compression (to include compression garments), and elevation serve to reduce pain and inflammation and facilitate expedient healing of muscle tissue. Although the use of drugs to reduce pain and inflammation is a common treatment for DOMS, the most effective type, dosage and treatment pattern are less understood and may carry other physiological responses such as gastrointestinal issues (Powers & Howley, 2018). Moreover, the microtrauma caused to muscle tissue that occurs during exercise is a precursor and facilitative to muscle hypertrophy and strength development. As such, the use of NSAIDs can interfere with muscle protein synthesis, hypertrophic development of muscle tissue, and stop cyclooxygenase enzymes; an alternative to NSAIDs is acetaminophen (Tylenol) (Fahey, 2019).

In summary, muscle soreness related to exercise, known as DOMS, is due to muscle fiber microtrauma and is specifically related to calcium leakage from the sarcoplasmic reticulum into the muscle causing the breakdown of proteins triggering a pain response and muscle inflammation. An athlete can mitigate the acute soreness with rest, ice, compression, elevation, or medication. Chronically, an athlete's ability to engage in ongoing and consistent training (subsequent bouts) with progressive intensity and gradual introduction of overload overload will serve to enhance neural, connective tissue, and cellular level adaptations and minimize DOMS.

References

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