

Exercise-Induced Muscle Damage Responses: Implications for Performance.

Ever had that stiff, dull aching feeling that lasts for a few days follows high intensity activity? What causes the pain? Is there any injury? How will it impact on my activities? Below explains the symptoms that contribute to this pain.

Muscle can be damaged by external factors such as contact in field and combat sports (Takarada, 2003). However, internal, mechanical and biological factors from unfamiliar exercise or movement ranges that involve eccentric muscle actions (figure 1) may contribute to temporary, but reparable muscle damage (Ebbeling & Clarkson, 1989). This is known as Exercise-Induced Muscle Damage (EIMD).

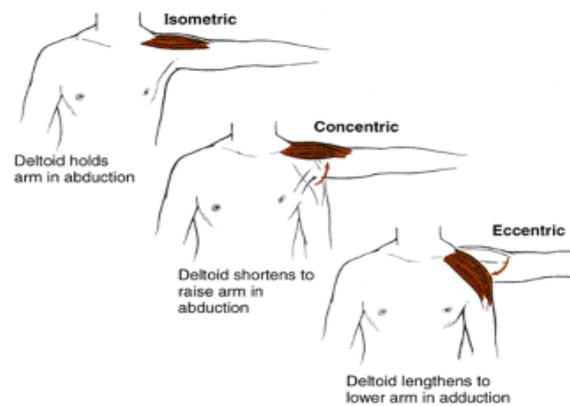


Figure 1. Highlighting the difference between an isometric, concentric and eccentric muscle action. Eccentric muscle action is a main contributor to EIMD

Proposed Mechanisms that Contribute to EIMD

Primary Damage: Metabolic

Restriction of energy supply capacity may contribute to EIMD. Ebbeling and Clarkson (1989) note that metabolic disturbances cause ischemia leading to muscle damage after exhaustive endurance exercise. Although endurance exercise is not normally associated with eccentric exercise, an activity such as downhill running would produce an over extension of hamstring muscles that would be classified as eccentric (Eston et al, 1995).

TERMONOLOGY:

Ischemia - The restriction of blood supply

Glycogen synthesis may also be a contributor to EIMD. Reproduction speed is severely reduced as the muscle fibres repair (Fournier et al, 2004) creating a deficiency in adenosine triphosphate (ATP) which is essential for continued activity or recovery.

Primary Damage: Mechanical

Eccentric contractions are stimulated by lower motor unit activation when compared to concentric and isometric contractions (Eston et al, 1995). Eccentric contractions produce larger forces leading to a greater torque imposed on the muscle (Nosaka & Cowen, 2011), exposing the contractile force mechanisms of a limited group of fast twitch muscle fibres to high stress (McHugh et al, 1999). This culminates in cellular disturbances of the thin filaments anchored by the Z-disk (Magaudda, et al, 2004), which may be observed on MRI scans (Clarkson & Hubal, 2002). Figure 2 illustrates sarcomere and Z-line positioning within skeletal muscle.

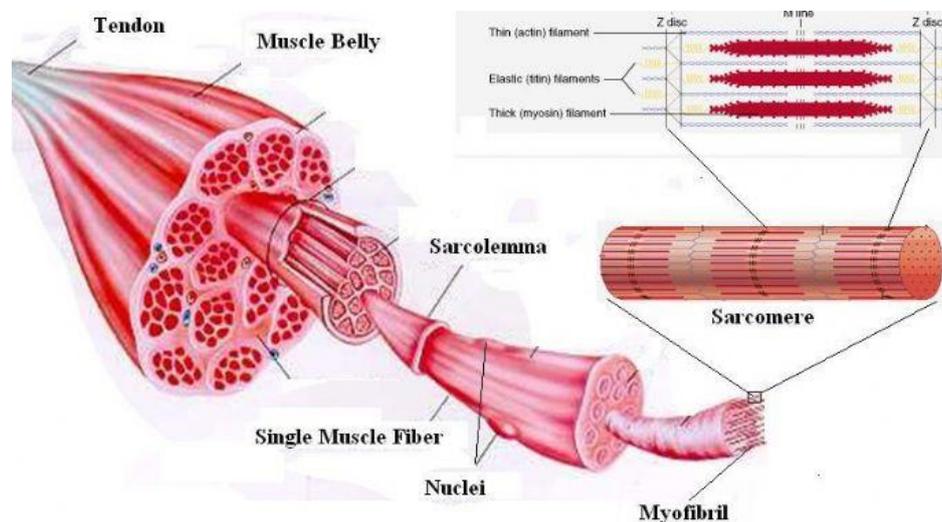


Figure 2. Muscle structure with Z-line (disc) location.

- Cellular Disturbances: Actin, Myosin & Cross-bridging

Cross-bridge attachment is where myosin and actin filaments in the sarcomere connect to cause a contraction. This theory is outlined in figure 3. Eston et al (1995) comment that under a concentric contraction, cross bridge detachment involves an energy release of ATP splitting, whereas an eccentric contraction cross-bridge detachment may be caused by a more forceful mechanical means. This may contribute towards inflammation and excitation-contraction (E-C) coupling alterations (Clarkson & Hubal, 2002).

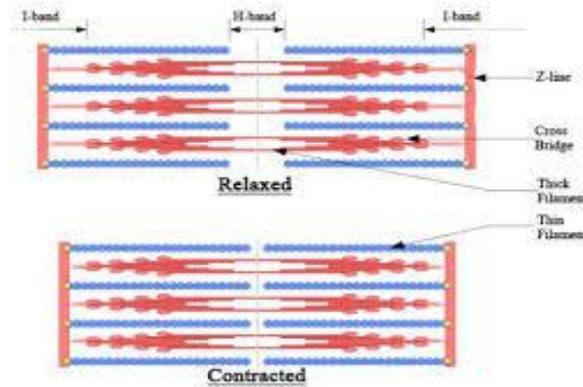


Figure 3. Sliding filament theory. Thick filament Myosin binds onto thin filament actin and ‘pulls’ the z-lines together, causing the muscle to contract.

Morgan and Proske (2004) provide a sarcomere ‘popping’ theory. They suggest that structural damage may occur during the lengthening process of an eccentric contraction. Sarcomeres may be stretched beyond an optimum functional length, creating instability thus rendering them weak. (Figure4).

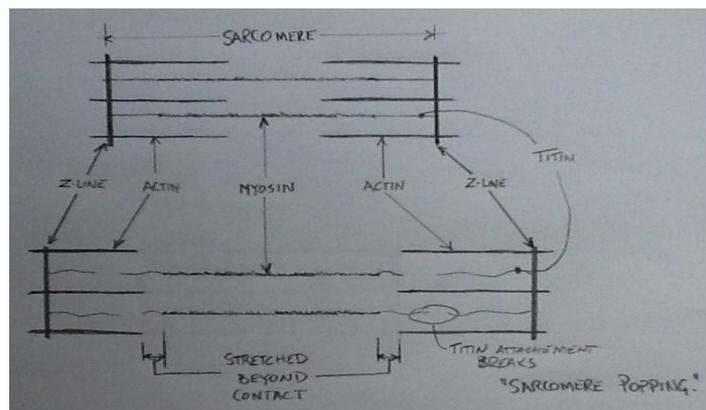


Figure 4. The bottom image shows an overstretch and a ‘snap’ of the connectin protein titin.

Secondary Damage

- Muscle Fibre Necrosis

Protein such as creatine kinase (CK) and myoglobin (Mb) may leak from damaged membranes into the blood circulation (Oakley et al, 2013) causing potential pain and fibre damage. Note: The quantity found in the system does not correlate with the amount of pain or damage (Nosaka & Cowen, 2011). If the sarcoplasmic reticulum is damaged calcium restoration will be delayed (Eston, et al, 1995). Eichner (2014) recently commented on the hospitalisation of athletes

TERMONOLOGY:

Necrosis - A rupture to a cell membrane.

Sarcoplasmic Reticulum – Releases calcium ions during a muscle contraction & absorbs them in relaxation.

from rhabdomyolysis where muscle cells, such as Mb found its way into their kidneys after high intensity push-up drills. Therefore, caution must be taken when designing the intensity of sessions.

EIMD Signs and Symptoms

Prominent symptoms of EIMD are pain, swelling, stiffness, reduced strength and fatigue. (Nosaka et al, 2011). Most of these symptoms likely indicate the *delayed-onset of muscle soreness* DOMS.

Smith, et al (1994), hypothesis that post exercise regeneration of muscle fibres, my lead to morphological adaptations. The advancement of the alteration changes should coincide with the DOMS symptoms of swelling and stiffness. (Eston et al, 2003). Common symptoms of DOMS, and a timeline of when they are in effect, are seen in Table 1.

<u>Symptom / Indicator</u>	<u>Timeline after exercise</u>		
	<u>Starts</u>	<u>Peaks</u>	<u>Ends</u>
Pain	Hours	1-3 days	7-10 days
Swelling	Hours	4-6 day	10 days
CK & Mb	1 Day	5-6 days	10 days
Glycogen depletion	Hours	9 Days	18 Days
Ultra-structural Damage	Immediate	8-20 Days	21 Days+
Weakness	Immediate	1-2 Days	14 Days

Table1 DOMS symptoms & timeline. (Adapted from Wilmore and Costill, 2004 p103 & Nosaka et al, 2011 pp 179-184).

EIMD Effects on Performance

Most sport actions require a stretch-shortening cycle (SSC). Therefore, an eccentric muscle action would seldom be performed in isolation. Re-examination of the symptoms within Table 1 should highlight the impact EIMD will have on sports performance.

TERMONOLOGY:
SSC – An eccentric contraction immediately followed by a concentric contraction.

Power and strength output reductions were noted by Eston et al (2003) when observing EIMD effects on vertical jumps, maximal cycling and intermittent high intensity activities. This was a result of eccentric actions causing damage to type II muscle fibre which inhibits neuromuscular control and reflex ability. Wilmore and Costill (2004) note inform that E-C coupling failure can contribute a 25-37% strength reduction within the first 5 days of exercise.

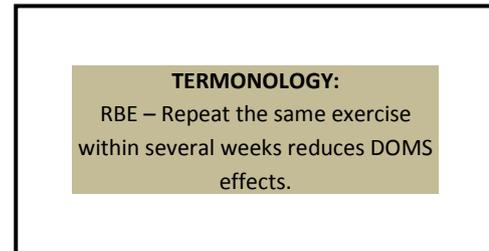
Naturally, range of motion is affected. Muscle starts to become harder around 3 days post eccentric exercise (Murayama et al, 2000). Naturally, this stiffness will lead to swelling.

Glycogen depletion has already been discussed, however, Casey et al (1995) noted Type I muscle fibres was 25% more efficient in re-synthesising than type II muscle fibres that are commonly associated with high-intensity eccentric actions.

If Eccentric Exercises Cause Such Damage, Why Do Them?

- Repeated Bout Effect (RBE)

RBE may improve the response to inflammation by series laying additional sarcomers. This could guard against future sarcomere strain (Smith et al, 1994). Other theories suggest RBE may: remodel compromised myofibrillar integrity; increase motor unit recruitment (Clarkson & Hubal, 2002); and improve the coordination and use of intramuscular connective tissue (tendons for example) (McHugh et al, 1999). However, the full extent is still unknown.



Other adaptations from subsequent eccentric exercise include reduction in plasma CK levels (Ebbing and Clarkson, 1989; Nosaka & Cowen, 2011).

Eccentric exercise results in degeneration of muscle protein, however, it also creates greater stimulation to synthesis muscle protein when compared against concentric training. This may lead to improved hypertrophy (Nosaka & Cowen, 2011).

Conclude

It is important to limit the extent of EIMD. The adaptation and protection that may be provided by eccentric exercises should suggest that eccentric exercises are essential to a well structured strength training programme.

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