

Is Postexercise Muscle Soreness a Valid Indicator of Muscular Adaptations?

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ABSTRACT

DELAYED ONSET MUSCLE SORENESS (DOMS) IS A COMMON SIDE EFFECT OF PHYSICAL ACTIVITY, PARTICULARLY OF A VIGOROUS NATURE. MANY EXERCISERS WHO REGULARLY PERFORM RESISTANCE TRAINING CONSIDER DOMS TO BE ONE OF THE BEST INDICATORS OF TRAINING EFFECTIVENESS, WITH SOME RELYING UPON THIS SOURCE AS A PRIMARY GAUGE. THIS ARTICLE DISCUSSES THE RELEVANCE OF USING DOMS TO ASSESS WORKOUT QUALITY.

INTRODUCTION

Delayed onset muscle soreness (DOMS) is a common occurrence in response to unfamiliar or vigorous physical activity. It has been noted observationally that many individuals who regularly perform resistance training consider DOMS to be one of the best indicators of training effectiveness, with some relying upon this source as a primary gauge (37). In fact, there is a long-held belief that DOMS is a necessary precursor to muscle remodeling (13).

Current theory suggests that DOMS is related to muscle damage from unfamiliar or unaccustomed exercise (48). Although the exact mechanisms are

not well understood, DOMS appears to be a product of inflammation caused by microscopic tears in the connective tissue elements that sensitize nociceptors and thereby heighten the sensation of pain (28,42). Histamines, bradykinins, prostaglandins, and other noxious chemicals are believed to mediate discomfort by acting on type III and type IV nerve afferents that transduce pain signals from muscle to the central nervous system (6). These substances increase vascular permeability and attract neutrophils to the site of insult. Neutrophils, in turn, generate reactive oxygen species (ROS), which can impose further damage to the sarcolemma (8). Biochemical changes resultant to a structural disruption of the extracellular matrix (ECM) also have been implicated to play a causative role (53). It has been proposed that damage to myofibers facilitates the escape and entrance of intracellular and extracellular proteins, whereas disturbance of the ECM promotes the inflammatory response (53). In combination, these factors are thought to magnify the extent of soreness. In addition, DOMS can be exacerbated by edema, whereby swelling exerts increased osmotic pressure within muscle fibers that serve to further sensitize nociceptors (6,28).

DOMS is most pronounced when exercise training provides a novel stimulus to the musculoskeletal system (4). Although both concentric and eccentric training can induce DOMS, studies show that lengthening actions have the most profound effect on its manifestation (7). As a general rule, soreness becomes evident about 6–8 hours after an intense exercise bout and peaks at approximately 48 hours postexercise (42). However, the precise time course and extent of DOMS is highly variable and can last for many days depending on factors, such as exercise intensity, training status, and genetics. The prevailing body of literature does not support sex-related differences in the expression of DOMS (11,45).

THEORETICAL BASIS FOR USING DOMS AS A GAUGE FOR MUSCULAR ADAPTATIONS

The first step in determining whether DOMS provides a valid gauge of muscle development is to establish whether the theory has biological plausibility. Plausibility can possibly be inferred from the correlation between DOMS and exercise-induced muscle damage (EIMD). It has been

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posited that structural changes associated with EIMD influence gene expression, resulting in a strengthening of the tissue that helps protect the muscle against further injury (1,44). When considering the mechanisms of muscle hypertrophy, there is a sound theoretical basis suggesting that such damage is in fact associated with the accretion of contractile proteins (48). What follows is an overview of the evidence supporting a hypertrophic role for EIMD. An in-depth discussion of the topic is beyond the scope of this article, and interested readers are referred to the recent review by Schoenfeld (48).

It is hypothesized that the acute inflammatory response to damage is a primary mediator of hypertrophic adaptations. Macrophages, in particular, are believed to promote remodeling pursuant to damaging exercise (60), and some researchers have hypothesized that these phagocytic cells are required for muscle growth (23). Current theory suggests that macrophages mediate hypertrophy through the secretion of cytokines synthesized within skeletal muscle (also known as myokines). Myokines have been shown to possess anabolic properties, exerting their effects in an autocrine/paracrine fashion to bring about unique effects on skeletal muscle adaptation (36,43,51). It should be noted that some research has shown that myokine production may be largely independent of damage to muscle tissue (62). This may be a function of the specific myokine response because numerous myokines have been identified with each displaying unique responses to exercise training (40). Neutrophils, another phagocytic leukocyte, also may play a role in inflammatory-mediated post-exercise hypertrophy, conceivably by signaling other inflammatory cells necessary for muscle regeneration. One such possibility is ROS (65), which can function as key cellular signaling molecules in exercise-induced adaptive gene expression (14,20,21,59). Studies show that ROS promote growth in both smooth muscle and cardiac muscle (55), and it has been suggested that they

induce similar hypertrophic effects on skeletal muscle (56).

Muscle damage also may mediate hypertrophy by facilitating activation of satellite cells (i.e., muscle stem cells). When stimulated by mechanical stress, satellite cells generate precursor cells (myoblasts) that proliferate and ultimately fuse to existing cells, providing the necessary agents for remodeling of muscle tissue (63,67). In addition, under certain conditions, satellite cells are able to donate their nuclei to the existing muscle fibers, enhancing their capacity for protein synthesis (2,34). Evidence substantiates that satellite cell activity is upregulated in response to EIMD (12,46,49). This is consistent with the survival mechanisms of the muscle cell where damaged fibers must quickly obtain additional myonuclei to facilitate tissue repair. Activation of satellite cells provides these needed myonuclei and co-expressing various myogenic regulatory factors, such as Myf5, MyoD, myogenin, and MRF4, which are involved in muscle reparation and growth (10). To this end, studies indicate that a person's ability to expand the satellite cell pool is a critical factor in maximizing muscle growth (41). It should be noted, however, that satellite cells are responsive to both muscle damaging and nondamaging exercises (40), and it is not clear whether their activity is enhanced by EIMD in a manner that promotes meaningful differences in muscle hypertrophy.

Cell swelling is another potential mechanism by which muscle damage may promote hypertrophic adaptations. EIMD is accompanied by an accumulation of fluid and plasma proteins within the fiber, often to an extent whereby this buildup exceeds the capacity of lymphatic drainage (16,31,42). This results in tissue edema, with significant swelling persisting in trained subjects for at least 48 hours after an exercise bout (19). Cellular swelling is theorized to regulate cell function (17), stimulating anabolism via increasing protein synthesis and decreasing protein breakdown (15,33,54). Although the exact mechanisms remain poorly understood, it appears that

membrane-bound, integrin-associated volume sensors are involved in the process (27). These osmosensors activate intracellular protein kinase transduction pathways, possibly mediated by autocrine effects of growth factors (5). The effects of cell swelling subsequent to EIMD have not as yet been directly investigated, however, and it therefore remains unclear whether the associated edema promotes similar anabolic and anti-catabolic effects to those reported in the literature.

Despite the sound theoretical rationale, direct research showing a cause-effect relationship between EIMD and hypertrophy is currently lacking. It has been shown that muscle damage is not obligatory for hypertrophic adaptations (3,13,25). Thus, any anabolic effects resulting from damaging exercise would be additive rather than constitutive. Furthermore, it is important to note that excessive damage has a decidedly negative effect on exercise performance and recovery. By definition, severe EIMD decreases force-producing capacity by 50% or more (40). Such functional decrements will necessarily impair an individual's ability to train at a high level, which in turn would be detrimental to muscle growth. Moreover, although training in the early recovery phase of EIMD does not seem to exacerbate muscle damage, it may interfere with the recuperative process (24,38). Studies indicate that regeneration of muscle tissue in those with severe EIMD can exceed 3 weeks, with full recovery taking up to 47 days when force production deficits reach 70% (47). In extreme cases, EIMD can result in rhabdomyolysis (40), a potentially serious condition that may lead to acute renal failure (61).

When taking all factors into account, it can be postulated that EIMD may enhance hypertrophic adaptations, although this theory is far from conclusive. The hormesis theory states that biological systems' response to stressors follows an inverted U-shaped curve (44). This is consistent with Selye's (50) concept of the general adaptation syndrome and would

suggest that if EIMD does indeed promote muscle development, optimum benefits would be realized from mild to moderate damage. However, an optimal degree of damage for maximizing muscle growth, assuming one does in fact exist, remains to be determined.

IS THERE A CAUSAL LINK BETWEEN DOMS AND MUSCLE HYPERTROPHY?

Given that DOMS is related to EIMD and assuming EIMD is indeed a mediator of hypertrophy, the question then becomes whether these events can be linked to conclude that DOMS is a valid indicator of growth. Although it is tempting to draw such a relationship, evidence suggests reason for skepticism. First, it remains debatable as to whether DOMS is an accurate gauge of muscle damage. There is little doubt that DOMS is a by-product of EIMD (6,40). However, studies show that soreness, as reported on a visual analog scale, is poorly correlated with both the time course and the magnitude of accepted markers of EIMD, including maximal isometric strength, range of motion, upper arm circumference, and plasma creatine kinase levels (39). Magnetic resonance imaging changes consistent with edema also do not correlate well with the time course of DOMS, with soreness peaking long before swelling manifests (6). So although DOMS may provide a general indication that some degree of damage to muscle tissue has occurred, it cannot be used as a definitive measure of the phenomenon.

What is more, humans can experience DOMS without presenting local signs of inflammation (40). In a study of subjects who performed different forms of unaccustomed eccentric exercise (including downhill treadmill running, eccentric cycling, downstairs running), Yu et al. (66) found no significant evidence of inflammatory markers postexercise despite the presence of severe DOMS. Other studies have reported similar findings after the performance of submaximal, eccentrically based exercise (29,30). These results provide reason for caution when attempting to

use DOMS as a gauge of muscular adaptations given the theorized role of the acute inflammatory response in tissue remodeling subsequent to EIMD. It also deserves mention that noneccentric aerobic endurance exercise can cause extensive muscle soreness. Studies show the presence of DOMS after marathon running and long-duration cycling (57). These types of exercise are not generally associated with significant hypertrophic adaptations, indicating that soreness alone is not necessarily suggestive of growth.

Moreover, DOMS displays a great deal of interindividual variability (58). This variability persists even in highly experienced lifters, with some consistently reporting perceived soreness after a workout, whereas others experiencing little, if any, postexercise muscular tenderness. Anecdotally, many bodybuilders claim that certain muscles are more prone to soreness than others. They report that some muscles almost never experience DOMS, whereas other muscles almost always experience DOMS after training. Recent research supports these assertions (52). Because the bodybuilders possess marked hypertrophy of the muscles that are and are not prone to DOMS, it casts doubt on the supposition that soreness is mandatory for muscle development. Moreover, genetic differences in central and peripheral adjustments and variations in receptor types and in the ability to modulate pain at multiple levels in the nervous system have been proposed to explain these discrepant responses (35). Yet, there is no evidence that muscle development is attenuated in those who fail to get sore postexercise.

Resistance exercises and activities that place peak tension at longer muscle lengths have been shown to produce more soreness than exercises that place peak tension at shorter muscle lengths (22). Whether these alterations affect the magnitude of hypertrophic adaptations has yet to be studied, but it has been postulated that torque-angle curves in resistance training might augment hypertrophy through varying mechanisms (9). It is therefore conceivable that

exercises that stress a muscle maximally at a short muscle length can promote hypertrophic gains without inducing much, if any, soreness.

Training status has an effect on the extent of DOMS. Soreness tends to dissipate when a muscle group is subjected to subsequent bouts of the same exercise stimulus. This is consistent with the "repeated bout effect," where regimented exercise training attenuates the extent of muscle damage (32). Even lighter loads protect muscles from experiencing DOMS during subsequent bouts of exercise (26). Therefore, training a muscle group on a frequent basis would reduce soreness, yet could still deliver impressive hypertrophic results. A number of explanations have been provided to explain the repeated bout effect, including a strengthening of connective tissue, increased efficiency in the recruitment of motor units, greater motor unit synchronization, a more even distribution of the workload among fibers, and/or a greater contribution of synergistic muscles (3,57).

In addition to reducing joint torque and muscle force, DOMS may negatively affect subsequent workouts in other ways and therefore impede strength and hypertrophic gains. Pain associated with DOMS has been shown to impair movement patterns, albeit in individuals with high pain-related fear (64). Altered exercise kinematics arising from DOMS-related discomfort can reduce activation of the target musculature and potentially lead to injury. Moreover, some researchers have speculated that DOMS could reduce the motivation levels involved in subsequent training, reducing exercise adherence (18). Therefore, excessive DOMS should not be actively pursued because it ultimately interferes with progress.

PRACTICAL APPLICATIONS

In conclusion, there are several takeaway points for the strength coach or personal trainer as to the validity of using DOMS as a measure of workout quality. Because muscle damage is theorized to mediate hypertrophic adaptations (48), there is

some justification to actively seek muscle damage during a training session if maximal hypertrophy is the desired goal. Given that DOMS is a gross indicator of EIMD, soreness can provide a modicum of insight as to whether damage has taken place postexercise. So although common strategies to minimize DOMS, such as increasing training frequency, adhering to the same exercise selection, performing concentric-only exercises, and performing solely exercises that stress short muscle lengths, can help maintain short-term athletic performance, they may ultimately compromise hypertrophic adaptations by blunting EIMD.

On the other hand, caution must be used in drawing qualitative conclusions given the poor correlation between DOMS and the time course and extent of EIMD. Some muscles appear to be more prone to DOMS than others, and there seems to be a genetic component that causes certain individuals to experience persistent soreness, whereas others rarely get sore at all. In addition, high levels of soreness should be regarded as detrimental because it is a sign that the lifter has exceeded the capacity for the muscle to efficiently repair itself. Moreover, excessive soreness can impede the ability to train optimally and decrease motivation to train. Thus, the applicability of DOMS in assessing workout quality is inherently limited, and it therefore should not be used as a definitive gauge of results.

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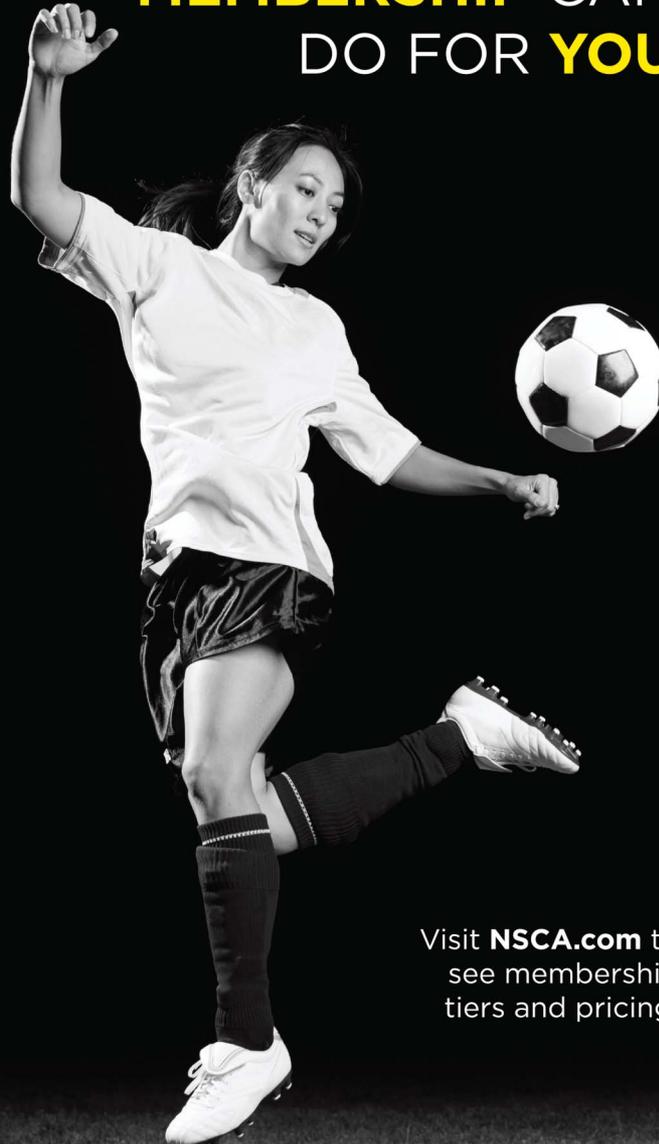
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