Hypothesized theories about the aetiology of Delayed Onset Muscle Soreness

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The phenomenon of Delayed Onset Muscle Soreness (DOMS) was first described in 1902 by Theodore Hough. DOMS consists in a sensation of discomfort and pain after taking part into an unaccustomed physical activity. It is characterized by decreasing of maximal force generation and soreness that increase intensity in first 24 hours after exercise, peaks from 24 to 72 hours, and then subsides within 5 to 7 days post exercise. The aim of this review is to briefly describe the most supported theories based on experimental evidences. Moreover, the lactic acid theory would be discussed.

Muscle damage

Muscle damage or microtrauma theory is based on little ruptures and misalignment of sarcomeres. Produced during exercise.

The presence in blood flow of enzymes normally localized in muscle fibres has been taken as evidence of myocytes disruption.

Connective tissue damage

Connective tissue damage theory explains that little tears are produced during exercise. In connective tissue. Soreness in tendon regions and analysis in urine of hydroxyproline, an amino acid very present in collagen, reiterate this explanation.

Enzyme efflux

Enzyme efflux theory postulates that calcium from interstitial liquid accumulates in injured cells inhibiting cellular respiration in mitochondria and activating proteases and phospholipases, which, finally, cause further injury because of degradation of contractile proteins.

Inflammatory response

Inflammatory response theory is based on the finding that aspects of the inflammatory response are evident following exercise, mainly produced by macrophages and neutrophiles infiltration in site of muscular injury.

The current belief of most researchers is that a single theory cannot explain the onset of DOMS. Therefore , consensus about the aetiology of that phenomenon includes part of each theory. Taking into account the strong evidences, a hypothetical sequence of events has been proposed:

During exercise, high tensile forces produced during contraction disrupts structural proteins in muscle fibres. Then, released calcium induce more damage in muscle cells, which increases the physical exigency on connective tissue because of higher tension producing little tears. Within a few hours, there is a significant infiltration of immune cells due to the harmful effect. Immune response is then produced mainly by macrophages and neutrophiles in order to repair damage in cells, producing delayed pain.

Breaking the dogma?

Lactic acid has been long believed to be the direct cause of delayed soreness in muscle, but why? First lactate was seen as a waste substance of anaerobic metabolism produced during anoxic exercises that waits until the recovery period to be returned to glucose. It was commonly thought to be the cause of pain and fatigue, and that lactic acid accumulated in muscle cells forming crystals that tears muscle fibres, producing, then, delayed soreness. However, there are some evidences to refuse that theory.

Relationship with DOMS

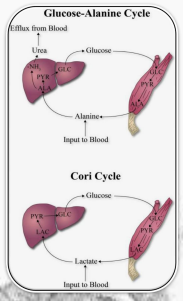
First studies in the 50's trying to get with the relationship between lactic acid and DOMS did not found a direct correlation between lactic acid and delayed soreness, suggesting that lactic acid could not be the main cause of DOMS. Moreover, lactate crystals have never been found in observations *in situ* or in muscle biopsies.

McArdle disease

McArdle disease, or glycogen storage disease type V, is a metabolic disorder caused by the deficiency of myophosphorylase, which causes the non ability to metabolize glycogen. For that reason, patients are called "exercise intolerants" because they suffer extremely from early fatigue. As they cannot practise sport, they normally do not produce lactic acid although they suffer from DOMS equally.

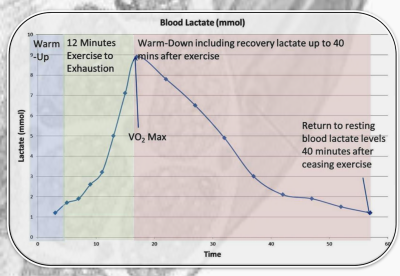
Cori and alanine cycles

Lactate accumulates in cell at low concentrations: 20% of produced lactate is released to blood flow directly or being previously metabolized to alanine. In both pathways, the liver assimilates the fasting metabolite and converts it into glucose. The remaining 80% is metabolized within the muscle cell to recycler it. For this reason, lactate do not accumulate in cell and concentration of its quickly decrease after exercise ceasing.



Lactate Shuttle

The old view of lactate as a waste product has changed. Apart from determining that is constantly converted to be reused, is has been postulated that it is also related with redox signalling in peroxisome and mitochondria, gene expression and lipolytic control. Therefore, it has given to the term "lactormone", as it was a signalling hormone.



Conclusions

1. **DOMS is defined as a complex phenomenon in which many factors intervene: muscle and connective tissue damage, second messengers action and inflammatory response by immune cells.**
2. **There is no single mechanism to explain the aetiology of DOMS. It is thought that independently postulated theories work in concert. The sequence of events showed is currently the accepted theory of DOMS.**
3. **Lactic acid is not the direct cause of DOMS as it has been thought for many years. There are some evidences to refuse that theory although it could also be involved in that complex process.**

References

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