Etiology of myofascial pain and dysfunction syndrome leading to active trigger-points with Traumeel's treatment intersection

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Medical professionals involved with sport-related injuries agree that the most common mechanism responsible for the development of myofascial trigger points over time is overuse and abuse. Although the mechanism is universally common, the eventual symptomatic expression varies e.g., sacro-illitis, iliotibial band syndrome, achilles tendonitis and patellofemoral-tracking abnormalities, to name but a few. Once the normal biomechanical chain has been altered, the active muscle syndrome is only meters away.

The most common complaint that we deal with in practice during the "training-hour-glass-almost-empty-pre-comrades-phase" is chronic muscle fatigue (before a major competition when time for preparation is almost over but there has been insufficient or perceived to be insufficient training, the athlete over extends himself in an effort to catch-up). The mechanism behind muscle overuse and abuse is very rarely singular and we normally find a combination of unscientific training protocols, poor road surface and camber, incorrect running shoes and biomechanical imbalances to be responsible for the start of fatigue syndromes.

To understand muscle fatigue, we need to understand what happens to a muscle at cellular level when we abuse it continuously. A number of pathways within the muscle will contribute to sustained muscle contraction and fatigue over time.

In short the following happens at cellular level:

- Due to the continued stress of overuse, straining of the muscle fibers occur which leads to initial micro-trauma.
- Due to physical tearing of muscle membranes, disruption of the sarcoplasmic reticulum causes the release of stored calcium.
- The disrupted sarcoplasmic reticulum cannot remove all of the calcium from the injury site.
- Extra calcium plus normal levels of ATP to the myofibrils results in sustained contraction of the sarcomere, **even at rest.**
- Sustained contraction leads to fatigue and further trauma.
- Fatigue and trauma disrupts the small blood vessels causing platelet release.
- This leads to leaked metabolites from platelet storage organelles like lysosomes, alpha granules and dense granules, causing the release of certain substances like serotonin and arachidonic acid. Arachidonic acid is converted by an enzyme called cyclo-oxygenase and thromboxane synthetase to thromboxane B2 which stimulates further platelet aggregation and prostaglandin which in turn, will sensitize class 3 and 4 nerve endings. Type A delta and type C fibers, both of which mediate the protopathic pathway, lead to poorly localized aching pain.
- As we know, muscle tissue is almost devoid of mechano- and thermo-receptors, but large quantities of type A delta and type C nerve fibers are present. A large group of the type A delta fibers act as ergoceptors (metaboreceptors) that respond to the local chemical environment and would be primarily responsible for the pain impulse due to the presence of irritants like bradykinin, histamine, serotonin and potassium chloride.
- Connective tissue damage leads to degranulation of mast cells.
- Histamine sensitizes and stimulates pain nerve endings.
- A trigger point in muscle is thereby characterized by an area of high metabolism, decreased blood supply, palpable band and a high concentration and accumulation of metabolites irritating certain sensory nerve ending leading to pain.

HOW CAN WE MANAGE THIS SYNDROME?

A number of treatment modalities have proven themselves to be extremely effective. Before any direct treatment protocol can start, it is essential to correct all biomechanical imbalances, movement abnormalities and to identify, isolate and remove possible mechanisms responsible for the etiology behind the development of the myofascial disorder, *e.g.*:

1. Re-evaluate the patient's training program and methodology.
2. Ensure normal biomechanical movement of all joints but specifically the sacro-iliac joints. Maintain healthy structure and function relationships, thereby limiting mechanical strain and overload on the muscle.
3. Follow a strict stretching protocol.

   By adopting a regular stretching routine, muscle tension is reduced and an increased range of motion is established. Effective stretches minimize arterial compression due to abnormal muscle tone and normalize blood flow. Regular stretching reduces pain by normalizing muscle physiology and reducing chemical imbalances that may irritate sensory and motor nerve endings, thereby causing pain referral patterns.
4. Dry needling of the involved trigger points can be extremely effective, especially if combined with heat and stretch protocols.
5. Biopuncture techniques using Traumeel have proven to be the most effective treatment protocol by far. If one understands the physiology behind the effect of Traumeel at cellular level, one cannot but agree to its efficacy.

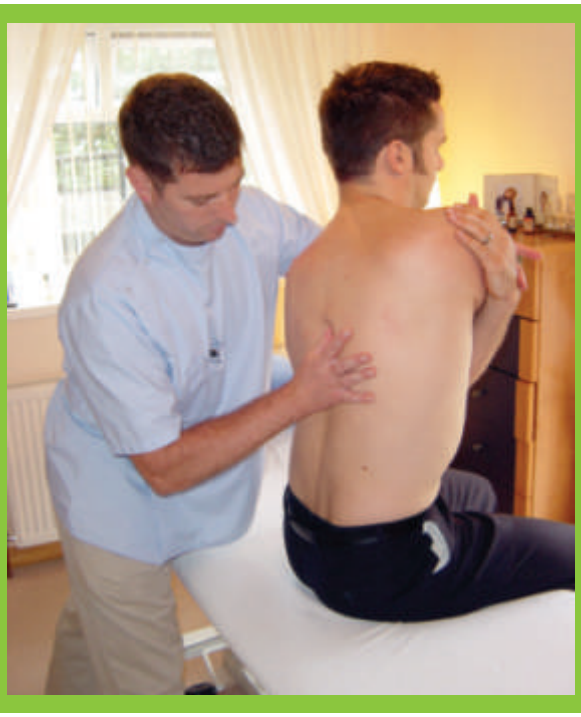
Traumeel is a biotherapeutic anti-inflammatory that contains many low potentized proteins and complies completely with the conditions for arousing an immunological bystander reaction.

By stimulating the formation of Th3-cells, the inflammation will be restrained. It is important to note that this form of therapy is not suppressive, but regulating. The self-regulating control of the inflammatory process will not be touched. NSAIDs, although effective in the short-term, suppress the inflammatory reaction by intervention at the cyclo-oxygenase level, thereby limiting the formation of prostaglandins. Patients will however complain that pain will return as soon as the effect of NSAIDs diminishes within four to eight hours.

When an antihomotoxic agent with low potentized proteins is introduced into the GRS (Ground Regulation System), patrolling macrophages will digest it almost completely. It does not matter whether the agents enter the body via the mucous membrane (sublingual) or directly in the bloodstream or the GRS (injection). The residues are transported back to the macrophage surface in the form of short amino acid chain motifs. There, they act like an antenna on the cell surface. The motifs are recognized by passing T-lymphocytes, taken away from the macrophages and bound to receptors of their own. This is the

signal for transformation into Th3-cells (regulatory lymphocytes). The Th3-cells are then transported to the lymph nodes (homing) where they will be multiplied (cloning). The activated Th3-cells search for inflammation-promoting lymphocytes (Th1, Th2, T4, ….) from the inflammation area and for which motifs are dependent on the foreign substances that caused the inflammation. The Th3-cell will look for lymphocytes with a similar motif (not equal, but following the simile principle). As soon as the similarity is confirmed, the Th3-cells immediately start with the synthesis of the highly active TGF-ß (Transforming Growth Factor ß), which will decrease the activity of the Th1 and Th2 lymphocytes thereby modulating the inflammatory reaction allowing tissues to heal.

Traumeel works by modulating the generation of reactive oxygen species by activated neutrophils and by inhibiting the release of inflammatory mediators and neuropeptides.



The advantages of using a product like Traumeel are:

- No gastrointestinal toxicity
- Does not inhibit platelet aggregation
- No sodium and fluid retention
- No adverse renal, hepatic, cardiovascular or CNS side effects
- Enhances cellular recovery and therefore healing

To conclude, train smarter, rest more, stretch more and modulate the adverse cellular and biochemical pathways in the muscle with products like Traumeel.