

## **TWO MORE REASONS TO NOT TREAT ATHLETIC PAIN WITH NSAIDS**

Research Shows Common OTC Pain Relieving Drugs Not Only Cause GI, Hepatic, And Renal Stress, They Also Inhibit Healthy Muscle Growth & Recovery And Increase Oxidative Damage

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Not hard to imagine what is routine for readers of T&C -- you have an athlete out of commission with an injury. There is an obvious motivation to get him or her back into competition as soon as possible or at least to get them healthy enough to train and workout so that atrophy of conditioning and skills is kept to a minimum. Then imagine with the best, most technically and medically advanced treatment you can provide, your therapy is constantly taking two steps forward and one step back.

That's what is happening if the regimen for controlling the pain of their injury includes (or they are augmenting your treatment by self medicating with) popular NSAIDs like ibuprofen, aspirin, and other OTC pain relievers according to research out of Austria and from Appalachian State University.

It appears that even for acute pain, there are more reasons to look to other means of controlling discomfort as healing takes place – especially with athletes. The FDA has long warned of the liver, kidney, and stomach damage that can be caused by popular pain relievers such as acetaminophen, ibuprofen, and aspirin, and the medical community cautions that too many people take these drugs chronically. But now there is mounting evidence that real impedance of healing is occurring with the use of NSAIDs and that long-term health effects may include problems similar to those experienced by people with diets low in anti-oxidants.

### **MUSCLE, TENDON, BONE, and CARTILAGE REPAIR – beware of NSAIDs!**

At the 5<sup>th</sup> World Congress on Sports Trauma, Drs. Bjorn Dimmen and H. Alfredson reported findings that the drugs that inhibit or block cyclooxygenase pathways (COX-2) also interfere with tendon, bone, and cartilage repair. Alfredson presented that NSAIDs not only inhibit COX-2 which is responsible for the messaging system that creates the chemical cascade leading to prostaglandin release (and pain / inflammation) but they also have the negative impact of inhibiting tenocytes and virtually blocking protein synthesis in the muscle.

The detrimental effects of this are not hard to imagine – you are treating a muscle injury or tendon strain only to find that part of the treatment could possibly be counter-productive to muscle and tendon repair...?!?! Two steps forward, one step back.

Dr. Dimmen advised pointedly sports medicine colleagues to avoid NSAIDs or COX-2 inhibitors when treating fractures or even when working to repair cartilage damage noting the value of the COX biochemical interactions that are “responsible for ensuring balance between bone resorption and bone formation.” He went on to say, “The COX-2 enzyme is critical for fracture and bone healing and it [COX-2] is especially involved in repair during the first 3 weeks after a fracture. NSAIDs and COX-2 inhibitors impair fracture healing.”

Dimmen added, “Avoid NSAIDs and COX-2 inhibitors in all fractures or for cartilage damage.”

### **NSAIDs – the ANTI-ANTI-OXIDANT**

More research on NSAIDs have recently shown other disturbing effects on athletes. Dr. Steven McAnulty of Appalachian State University in Boone, North Carolina and colleagues found using NSAIDs could be bad for muscles even in cases where there was no damage initially. They noticed in studies that runners who took ibuprofen as a prophylaxis before strenuous exercise reported more soreness the following day than athletes that didn't take the anti-inflammatory drugs.

This observation causes one to wonder about the effects of common OTC pain relievers on recovery. Other testing supports the concerns that sports medicine officials should have with use of these drugs by athletes. Analysis of blood and urine revealed remarkable differences in the levels of protein carbonyls (PCs) of people who used NSAIDs vs. those who hadn't. PCs are key indicators of oxidative stress and they were "dramatically increased" after exercise in NSAID users only.

Oxidative stress can cause toxic effects through the production of peroxides and free radicals that damage all components of the cell, including proteins, lipids, and DNA. Long term oxidative stress is also understood to be involved in many diseases such as atherosclerosis, Parkinson's Disease, Alzheimer's Disease, and other neurodegenerative diseases.

### **SAFE / HEALTHY OPTIONS FOR PAIN FROM OVER-EXERTION**

Over the past 6 years, research has been conducted at major university medical centers on the effects of new compounds on controlling pain and effecting inflammation. Special consideration has been given to relief combined with safety.

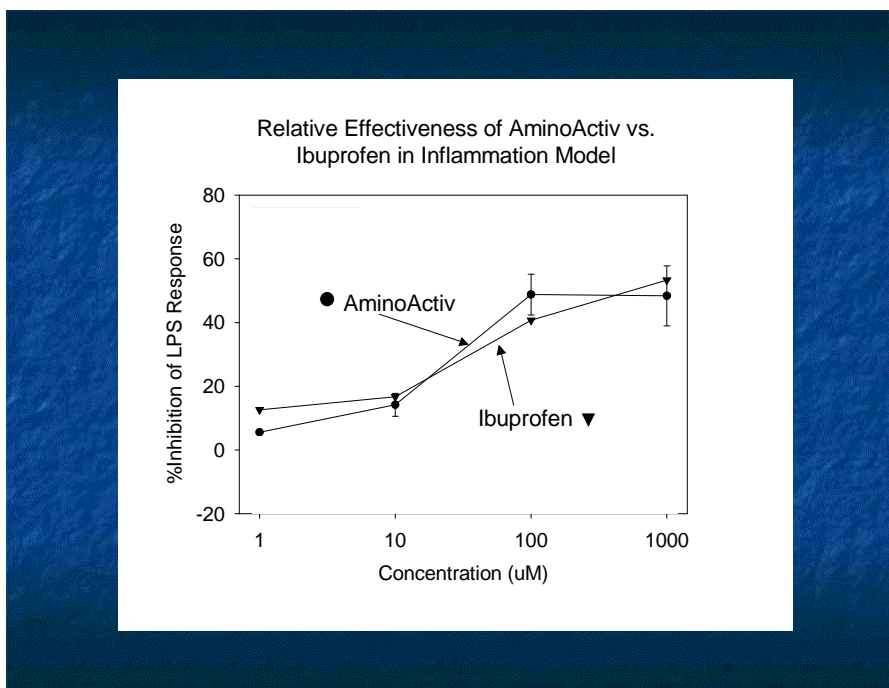
These new compounds (e.g. AminoActiv®) have dose related effects on controlling pain due to athletic over-exertion similar to NSAIDs but without the toxicity or impedance to healing. Necropsy studies showed that even super-

therapeutic doses had no effect on blood chemistry or organ profiles. (author's note: these new compounds also "pass" all doping control testing for banned substances by NCAA, NFL, MLB, IOC, etc.)

It is known that previous scientific literature has shown and documented the natural occurring compounds from which these new products are derived have chemical attributes that render them natural anti-inflammatory agents. However, because of their limited bioavailability and low uptake by cells, they have previously been limited in their applications. The research and development by these universities not only led to improved physical-chemical characteristics of the molecule, but also led to a dramatic improvement in the compound's anti-inflammatory activity.

When cultured cells are treated with bacterial toxins, they produce and release prostaglandins similar to injured tissue. The data below shows that this new class of pain relievers effectively retards the release of prostaglandins (PGE), a major cause of the pain response that occurs as a result of inflammation. The data shows that this inhibition is dose dependent with higher concentrations producing the greatest amount of inhibition of prostaglandin release.

It is important to note that these new compounds do not inhibit the COX-2 enzyme, but instead reduces prostaglandin release from cells by a different mechanism (mitochondrial membrane stabilization) which leads to improved mitochondrial function and less membrane damage that initiates prostaglandin synthesis and thus, less inflammation. This means that these new compounds do not have the negative side effects associated with non-steroidal anti-inflammatory drugs that are COX-2 inhibitors. And as an added benefit, they are known to have anti-oxidant properties.



The data show that these new compounds are equally effective as the leading NSAID (ibuprofen) in slowing the production and release of prostaglandin from cells treated with the bacterial toxin LPS (lipopolysaccharide). This ability to inhibit prostaglandin production as effectively as ibuprofen was observed at all concentrations tested.

An added benefit is that since these new compounds are classified as ATP Accelerators, they actually enhance the rate of recovery of tissue that has suffered over-exertion or common athletic damage. This benefit means that pain isn't just being masked, but that in addition to pain relief, recovery is actively being promoted rather than hindered as with other pain relievers.

Imagine, healthy pain relief and unimpeded recovery – that's a much improved prognosis compared to NSAID use.