



report: Kathryn Bistany

injury rehabilitation: WHY IS NUTRITION IGNORED?

every sport has its own list of most common injuries from strains, tears, dislocations, fractures and overuse. The common first aid protocol of rest, ice, compression and elevation with the use of anti-inflammatory medication is the usual approach to an injury. The nutritional protocol frequently calls for a reduction in caloric intake, especially if the athlete is unable to train. Yet nutrition plays a vital role in injury rehabilitation, from limiting the damage caused by chronic inflammation to actively promoting tissue healing and improving recovery times. As such, can any athlete ignore the role nutrition could play in his or her injury rehabilitation?▶



inflammation and NSAIDS

Non-steroidal anti-inflammatory medication is frequently used to deal with inflammation caused by injury. However, NSAIDs do have side effects, especially with long-term use.^{1,2,3} NSAIDs are used regularly by athletes, including adolescent athletes, with the majority of these users not recognising the possible toxicity or adverse effects of these drugs, especially on athletes subjected to intermittent dehydration.^{4,5} Considering the documented deleterious side effects of NSAIDs, the use of enzymes and diet could prove to be viable alternatives.

inflammation and enzymes

Enzymes have been reported to moderate the inflammatory cycle and up-regulate the healing process.⁶⁻¹² Their use is suggested for bruising, sprains, strains, fractures, low back pain, dental surgery, arthritis and post-surgical trauma and recovery. The anti-inflammatory action of these enzymes, which is thought to inhibit the arachidonic cascade, is also associated with increased tissue permeability, facilitating resorption of oedema and accelerated restructuring of the damaged tissue.^{6,13}

Enzymes are protein compounds capable of accelerating a change in its substrate by catalytic action. Proteolytic enzymes catalyse the hydrolysis of proteins and various protein end-products. In order for proteolytic enzymes to work, they must be absorbed in an active form from the gastrointestinal tract. However, the intestinal absorption of enzymes has been a matter of scientific controversy for many years.^{6,14,15} As enzymes are proteins, it is believed that if taken orally they are denatured by hydrochloric acid in the stomach.

The intestinal absorption of undegraded proteins, in particular proteolytic enzymes, has been noted for many years with research dating back to the 1960s.^{8,10-12} More recent studies have shown that the intestinal transport of undegraded, non-denatured proteins (often enterically coated) can take place to a small but significant extent and that these enzymes can decrease the recovery time of injuries.^{6,9,13,16-19} In addition, it has been suggested that because different enzymes have different targets of activity, different modes of action, and different absorption rates, the use of a combination of enzymes is thought to be more efficacious than singular use.^{13,14,18}

Moreover, proteolytic enzymes appear to be safe at high doses and when taken long-term.^{6,12} However, for best results, they require compliance as they have to be taken 2-4 times a day on

an empty stomach. The only known contraindications are for those on blood-thinning medication, and allergies to compounds in the enzyme combination preparation.¹⁸

inflammation and diet

fatty acids

Pro-inflammatory signals are mediated by metabolites of arachidonic acid (AA), an omega-6 polyunsaturated fatty acid (PUFA). To mitigate inflammation through dietary intervention requires a reduction of AA and increased intake of eicosapentaenoic acid (EPA), an omega-3 PUFA.²⁰⁻²²

Various studies on the effects of a diet low in AA in conjunction with the supplementation of fish oil have been shown to ameliorate the clinical signs of a number of inflammatory and autoimmune diseases in humans, and in particular rheumatoid arthritis.^{23,24} The beneficial effect of EPA, the active ingredient in fish oil, was found to augment by the reduction of dietary AA intake. EPA and DHA from fish oil are thought to be more biologically potent than omega-3 from flaxseed oil.^{22,25}

vitamins and minerals

Numerous vitamins and minerals are involved in wound healing, of which only a few will be reviewed.

Vitamin A plays a role in each stage of wound healing. It enhances the early inflammation phase, increasing the number of monocytes and macrophages. Vitamin A labilizes lysosomal membranes, promoting fusion of lysosomal and phagocytic vesicles and facilitating wound debridement. A deficiency results in decreased phagocytosis resulting in accumulation of pus and debris.^{26,27}

Vitamin A is available in dietary sources as preformed vitamin A or pro-vitamin A carotenoids. Preformed vitamin A is found in liver (beef, veal, chicken and turkey) and cod liver oil. Pro-vitamin A carotenoids are found in green leafy vegetables, carrots, sweet potatoes, butternut squash and mango.²⁸

Vitamin C is necessary for the hydroxylation of proline and consequently the synthesis of strong collagen.^{7,26,29} Vitamin C is also involved as an antioxidant in the scavenging of free radicals and it enhances iron absorption. Plasma levels of vitamin C decrease following a fracture, burn or major surgery but levels rise in healing tissue.^{26,27}

The dietary sources of vitamin C are fresh fruits and fresh fruit juices, raw red pepper, raw broccoli and tomatoes.²⁸

Vitamin E, as a lipid-soluble vitamin, accumulates in cell membranes where it protects polyunsaturated fatty acids from peroxidation by free radicals. Vitamin E has an anti-inflammatory action by inhibiting phospholipase-A2 activity and consequently the production of prostaglandins, which are chemical mediators of the inflammatory response. Studies on vitamin E are equivocal with some showing a detrimental effect on wound healing. It is thought that excessive concentrations of vitamin E may inhibit collagen synthesis.^{26,29}

Dietary sources of vitamin E are wheat germ oil, vegetable and nut oils, sunflower seeds, brazil nuts, almonds and pistachios.²⁸

Zinc: Marginal deficiencies of zinc are common due to our soil being zinc depleted.²⁶ Moreover, there is growing evidence that zinc deficiency occurs even when micronutrient intake appears to be adequate.^{30,31} The reason for this is unclear but may be linked with the ingestion of inhibitors of zinc absorption such as phytates.³⁰ Zinc deficiency results in delayed closure of wounds, a decreased tensile strength of collagen and increased susceptibility to recurring infection.³² Zinc deficiency also decreases taste acuity which may lead to decreased food intake.³³ There is an apparent

decrease in serum zinc during wound healing with a greater concentration in wound tissue, in surgical patients.^{26,34}

Dietary sources of zinc are oysters, beef, liver (beef, veal, chicken and turkey), the dark meat of turkey, pumpkin seeds, almonds, pecans, brazil nuts and clams.²⁸

Iron: Ribonucleotide reductase, the enzyme that produces deoxyribonucleotides for DNA synthesis, requires iron as a cofactor. Iron deficiency impairs proliferation of all cells involved in wound debridement and healing.²⁶ Iron deficiency leads to anaemia, which can be a contributory factor in tissue hypoxia.^{29,32} Iron is required for the hydroxylation of proline.^{26,29} Without this step, the triple helix formation of collagen is unstable, resulting in weaker collagen. Iron deficiency has been noted in endurance athletes and in particular female athletes.³⁵

There are two types of iron: non-haem (ferric) iron, which is found in eggs, plants and vegetables, and haem (ferrous) iron, which is found in meat, fish and poultry. The dietary sources of iron are clams, oysters, offal, pumpkin seeds, tofu, meat, sweet potato, pinto beans, macadamia nuts, walnuts, artichoke, spinach and tomato juice.²⁸

conclusion

The body's metabolic rate increases during times of repair: the increases correlating to the severity of the injury.^{26,35} As such, the nutritional protocol during injury should be for increased, and not decreased, calorific intake. If activity level has dropped

considerably due to the injury, the increase in BMR from injury is often cancelled by the decrease in activity level.³⁶ Calorific intake should therefore remain more or less the same as when active. The USRDA for protein increases from 0.8g/kg to 2-4g/kg to support wound healing.²⁶ However, the increased calorific requirement is not simply for quantity but for a particular quality of nutrients that assist and promote repair and recovery. To ensure optimum repair and reduced recovery time, comply with the following:

1. Eat regularly, especially if the injury is severe; basal metabolic rate can increase by up to 20%.
2. Eliminate all junk food.
3. Eliminate all refined and processed foods and especially all refined sugar.
4. Avoid all saturated fats; ie, pro-inflammatory foods found in pork products (ham, bacon, salami, sausages, pâté) and fatty red meats such as lamb. Instead, have fish, chicken (without the skin) and extra lean cuts of red meat (limit red meat intake to twice a week).
5. Increase fruit, vegetable, legume and pulse intake.
6. Use an omega-3 oil (never heat the oil as it is prone to oxidation).
7. Ensure that protein intake remains high; ie, 1.0-1.5g/kg
8. The use of proteolytic enzymes is best administered under the supervision of a qualified nutritionist. **En**

references

1. Baker J, Cotter JD, Gerrard DF, Bell ML, Walker RJ (2005) Effects of Indomethacin and Celecoxib on Renal Function in Athletes. *Medicine & Science in Sports & Exercise* 37(5): 712-717.
2. Shoor S (2002) Athletes, nonsteroidal anti-inflammatory drugs, coxibs, and the gastrointestinal tract. *Current Sports Medicine Reports* 1(2): 107-115.
3. James DS (1999) The multisystem adverse effects of NSAID therapy. *The Journal of the American Osteopathic Association* 99(11S): S1-7.
4. Gorsline RT, Kaeding CC (2005) The use of NSAIDs and nutritional supplements in athletes with osteoarthritis: prevalence, benefits, and consequences. *Clinics in Sport Medicine* 24(1): 71-82.
5. Warner DC, Schnepf G, Barrett MS, Dian D, Swigonski NL (2002) Prevalence, attitudes, and behaviours related to the use of nonsteroidal anti-inflammatory drugs (NSAIDs) in student athletes. *Journal of Adolescent Health* 30(3): 150-3.
6. Miller PC, Bailey SP, Barnes ME, Janelle Derr S, Hall EE (2004) The effects of protease supplementation of skeletal muscle function and DOMS following downhill running. *Journal of Sports Sciences* 22(4): 365-372.
7. Burke ER (1997) Nutrients that accelerate healing. *Strength and Conditioning* 19(5): 19-23.
8. Buck JE, Phillips N (1970) Trial of chymoral in professional footballers. *The British Journal of Clinical Practice* 24(9): 375-377.
9. Brown SA, Coimbra M, Coberly DM, Chao JJ, Rohrich RJ, Nancy L, Bass P (2004) Oral nutritional supplementation accelerates skin wound healing: a randomized, placebo-controlled, double-arm, crossover study. *Plastic and Reconstructive Surgery* 114(1): 237-44.
10. Stevens MA, McCown IA, Campbell EA (1962) Control of swelling in sports injuries – a key to prompt and complete recovery. *New York State Journal of Medicine* 62(11): 1817-21.
11. Trickett P (1964) Proteolytic enzymes in treatment of athletic injuries. *Applied Therapeutics* 6(8): 647-652.
12. Cirelli MG (1967) Five years of clinical experience with bromelains in therapy of edema and inflammation in postoperative tissue reaction, skin infections and trauma. *Clinical Medicine* 74(6): 55-59.
13. Stauder G (1995) Pharmacological effects of oral enzyme combinations. *Časopis lékařů Českých* 134(19): 620-4.
14. Cichoke AJ (1995) Absorption of enzymes. *Townsend Letter for Doctors & Patients*. Oct: 24-26.
15. Coleman E (2000) Oral proteases for sports injuries? *Sports Medicine Digest* 22(1): 5-6.
16. Castell JV, Friedrich G, Kuhn CS, Poppe GE (1997) Intestinal absorption of undegraded proteins in men: presence of bromelain in plasma after oral intake. *American Journal of Physiology* 273 1(1): G139-G146.
17. Kleine MW (1990) Introduction to systemic enzyme therapy and results of experimental trials. *Proceedings of the XXIV World Congress of Sports Medicine*, Elsevier Science Publishers, Amsterdam: 1131-1133.
18. Kleine MW (1997) Introduction to oral enzyme therapy. *International Journal of Immunotherapy* 13(3/4): 59-65.
19. Baumüller M (1990) Therapy of ankle joint distortions with hydrolytic enzymes—results from a double blind clinical trial. *Proceedings of the XXIV World Congress of Sports Medicine*, Elsevier Science Publishers, Amsterdam: 1137-1140.
20. Adam O (2003) Dietary fatty acids and immune reaction in synovial tissue. *European Journal of Medical Research* 8(8): 381-387.
21. Calder PC (2003) Immunonutrition. *BMJ* 327(7407): 117-118.
22. Plat J, Mensink RP (2005) Food components and immune function. *Current Opinion in Lipidology* 16: 31-37.
23. Adam O, Beringer C, Kless T, Lemmen C, Adam A, Wiseman M, Adam P, Klimme KR, Forth W (2003) Anti-inflammatory effects of a low arachidonic acid diet and fish oil in patients with rheumatoid arthritis. *Rheumatology International* 23(1): 27-36.
24. Calder PC (2003) N-3 polyunsaturated fatty acids and inflammation. *Lipids* 38(4): 343-52.
25. Simopoulos AP (2002) Omega-3 fatty acids in inflammation and autoimmune diseases. *Journal of the American College of Nutrition* 21(6): 495-505.
26. Mazzotta MY (1994) Nutrition and wound healing. *Journal of the American Podiatric Medical Association* 84(9): 456-462.
27. Scholl D, Langkamp-Henken B (2001) Nutrient recommendations for wound healing. *Journal of Intravenous Nursing* 24(2): 124-132.
28. Hands ES (2000) Nutrients in Food. Lippincott Williams & Wilkins, Maryland USA.
29. Reynolds TM (2001) The future of nutrition and wound healing. *Journal of Tissue Viability* 11(1): 5-13.
30. Lönnerdal B (2000) Dietary factors influencing zinc absorption. *The Journal of Nutrition* 130(5S): 1378S-1383S.
31. Hambidge M (2000) Human zinc deficiency. *The Journal of Nutrition* 130(5S): 1344S-1349S.
32. McIlwaine C (2003) Importance of holistic nutritional assessment in wound healing. *Journal of Wound Care* 12(8): 285-8.
33. King JC, Shames DM, Woodhouse LR (2000) Zinc homeostasis in humans. *The Journal of Nutrition* 130(5S): 1360S-1366S.
34. Lansdown ABG (1996) Zinc in the healing wound. *Lancet* 347(9003): 706-7.
35. Manore MM (1999) Nutrition needs of the female athlete. *Clinics in Sports Nutrition* 18(3): 549-563.
36. Bucci LR (1995) *Nutrition Applied to Injury Rehabilitation and Sports Medicine*. CRC Press, Inc, Florida, USA.