Nutritional Influences on Illness
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The Effect of Minerals on Pain

Copper

Copper deficiency has been shown under experimental conditions to lower enkephalin levels, thus affecting the endogenous control of pain perception. Conversely, intracerebral injection of cupric ion caused naloxone-reversible analgesia in mice and showed potency close to that of morphine, and intracerebral injection of copper sulfate in mice potentiated morphine analgesia.

For a group of children and adolescents with recurrent abdominal pain, copper intake was only 0.82 mg daily compared to the RDA of 1 to 2.5 mg daily. However, studies have yet to be performed to evaluate whether copper supplementation is of any value in treating pain.

Magnesium

A deficiency of magnesium may be associated with a decrease in the pain threshold as well as cramping of both striated and smooth muscle, causing a wide variety of pain syndromes including headache, muscle cramps, angina pectoris, and epigastric cramps. In such a case, the symptoms can be expected to respond to magnesium repletion.

Evidence from animal work suggests that magnesium deficiency causes hyperalgesia through its effect on the NMDA (N-methyl-D-aspartate) receptors. These are located, not only centrally, but also peripherally in the skin, muscles, and knee joints and play a role in central nociceptive transmission, modulation, and sensitization of acute pain states as well as in the sensory transmission of noxious signals. Magnesium has been shown to be a physiological blocker of NMDA receptors, suggesting that supplementation may have broad applications in reducing the experience of pain.

Compared to oral codeine and diclofenac (a non-steroidal anti-inflammatory drug), intravenous magnesium sulfate (2 g/70 kg), started during surgery and continued three days postoperatively, was more effective in reducing pain after coronary artery bypass grafting, resulting in a reduced need for morphine and thus less respiratory depression. Similar results have been found for intravenous magnesium
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