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Vitamin E, an Antioxidant, as a Possible Therapeutic Agent for Treating Pain

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LETTER TO EDITORS

I found a recent article written by Kim et al. [1] quite interesting. Reactive oxygen species (ROS), such as superoxides, hydrogen peroxides and hydroxyl radicals, can damage nucleic acids, proteins and lipids, especially at high concentrations. Notably, accumulating evidence indicates that ROS are involved in the development of exaggerated pain hypersensitivity during persistent pain. ROS production in the spinal cord increased after noxious hindpaw stimulation or nerve injury [2,3]. Vitamin E, a fat soluble vitamin, is one of the essential nutrients that functions as an antioxidant in the human body. It is the major chain-breaking antioxidant in the body and the first line of defense against lipid peroxidation, protecting cell membranes from free radical attack [4].

The antinociceptive effects of vitamin E have been reported in earlier studies. For example, dietary supplement vitamin E (12 g/kg per day, p.o. for 3 months) ameliorated nerve conduction deficits in streptozotocin induced diabetic neuropathy in rats [5]. It was reported that a single injection of vitamin E (0.1-5 g/kg i.p.) attenuated mechanical allodynia in rats with spinal nerve ligation [6]. Kim et al. [1] observed that i.p administration of vitamin E one hour before formalin injection diminished the nociceptive behavior in a dose-dependent manner during the early and late phases of the rat formalin test. However in this study, it is difficult to tell where the major site of analgesic action is for vitamin E. The pharmacokinetics of vitamin E may be important in understanding its analgesic mechanism.

The study by Kim et al. [1] suggested that vitamin E can exert its anti-nociceptive effects in the formalin test, primarily as a result of its antioxidant properties. Therefore, vitamin E could be a potential therapeutic agent in treating acute or chronic pain for a number of clinical conditions.

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