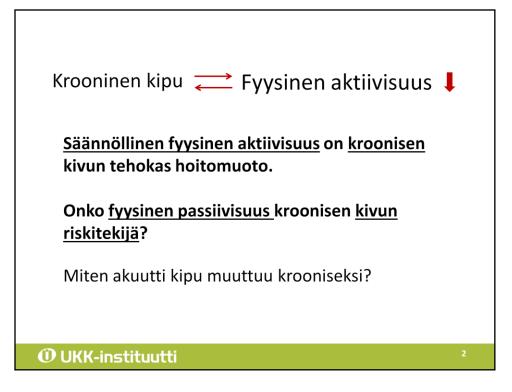
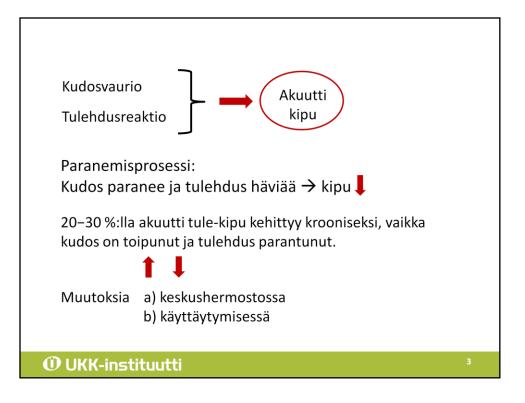
Miksi liike on lääke TULE-oireisellekin?

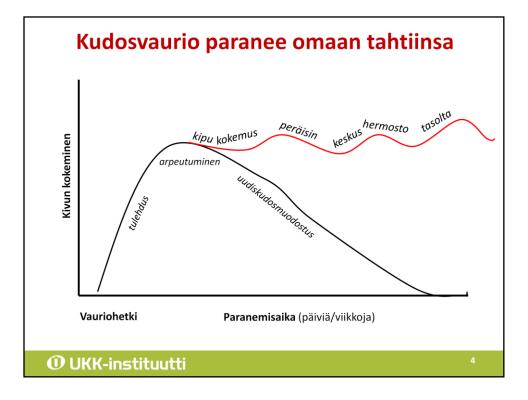
uunnittelija Annika Taulaniemi, THM, ft UKK-instituutti

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Kipu vähentää fyysistä aktiivisuutta. Vähäinen fyysinen aktiivisuus puolestaan voi johtaa kudosten rakenteiden heikkenemiseen, mikä puolestaan voi johtaa vaurioihin ja kipuun. Jotta ymmärtäisimme, onko fyysinen passiivisuus riski kivun kroonistumiselle, meidän tulisi ensin ymmärtää, miten kipu muuttuu krooniseksi.







<u>J Appl Physiol (1985).</u> 2013 Mar 15;114(6):725-33. doi: 10.1152/japplphysiol.01317.2012. Epub 2012 Dec 27.

Regular physical activity prevents development of chronic pain and activation of central neurons. <u>Sluka KA¹, O'Donnell JM</u>, <u>Danielson J</u>, <u>Rasmussen LA</u>.

Abstract

Chronic musculoskeletal pain is a significant health problem and is associated with increases in pain during acute physical activity. Regular physical activity is protective against many chronic diseases; however, it is unknown if it plays a role in development of chronic pain. The current study induced physical activity by placing running wheels in home cages of mice for 5 days or 8 wk and compared these to sedentary mice without running wheels in their home cages. Chronic muscle pain was induced by repeated intramuscular injection of pH 4.0 saline, exerciseenhanced pain was induced by combining a 2-h fatiguing exercise task with a lowdose muscle inflammation (0.03% carrageenan), and acute muscle inflammation was induced by 3% carrageenan. We tested the responses of the paw (response frequency) and muscle (withdrawal threshold) to nociceptive stimuli. Because the rostral ventromedial medulla (RVM) is involved in exercise-induced analgesia and chronic muscle pain, we tested for changes in phosphorylation of the NR1 subunit of the N-methyl-D-aspartate (NMDA) receptor in the RVM. We demonstrate that regular physical activity prevents the development of chronic muscle pain and exercise-induced muscle pain by reducing phosphorylation of the NR1 subunit of the NMDA receptor in the central nervous system. However, regular physical activity has no effect on development of acute pain. Thus physical inactivity is a risk factor for development of chronic pain and may set the nervous system to respond in an exaggerated way to low-intensity muscle insults.

PMID: 23271699 [PubMed - indexed for MEDLINE] PMCID: PMC3615604 Free PMC Article

Miten fyysinen aktiivisuus voi vähentää kipua?

- liikunta stimuloi aivojen alueita, jotka tuottavat kivun lievitystä
- kemiallisten, kipua lievittävien välittäjäaineiden eritys
- käyttäytymisen muutokset
- positiiviset vaikutukset kipuun, "tuoretavaraa", fyysisen aktiivisuuden säännöllisyys.

Keskushermoston "normaaliin" tilaan liittyy fyysinen aktiivisuus. Liikkumattomuus voi saattaa keskushermoston "tautitilaan", jolloin reagointi ja toiminta muuttuvat tavalla, jonka seurauksena kipu kroonistuu.

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Pain Physician. 2012 Jul;15(3 Suppl):ES205-13.

Dysfunctional endogenous analgesia during exercise in patients with chronic pain: to exercise or not to exercise?

<u>Nijs J¹, Kosek E, Van Oosterwijck J, Meeus M</u>.

Abstract

BACKGROUND: Exercise is an effective treatment for various chronic pain disorders, including fibromyalgia, chronic neck pain, osteoarthritis, rheumatoid arthritis, and chronic low back pain. Although the clinical benefits of exercise therapy in these populations are well established (i.e. evidence based), it is currently unclear whether exercise has positive effects on the processes involved in chronic pain (e.g. central pain modulation).

OBJECTIVES: Reviewing the available evidence addressing the effects of exercise on central pain modulation in patients with chronic pain.

METHODS: Narrative review.

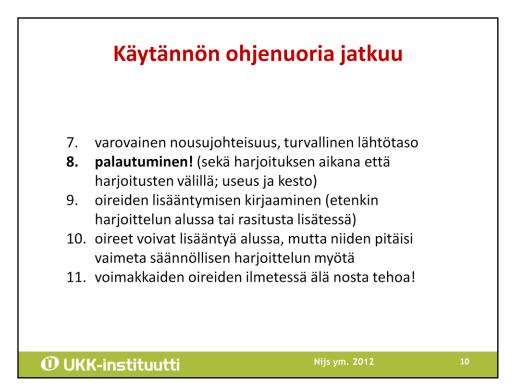
RESULTS: Exercise activates endogenous analgesia in healthy individuals. The increased pain threshold following exercise is due to the release of endogenous opioids and activation of (supra)spinal nociceptive inhibitory mechanisms orchestrated by the brain. Exercise triggers the release of beta-endorphins from the pituitary (peripherally) and the hypothalamus (centrally), which in turn enables analgesic effects by activating µ-opioid receptors peripherally and centrally, respectively. The hypothalamus, through its projections on the periaqueductal grey, has the capacity to activate descending nociceptive inhibitory mechanisms. However, several groups have shown dysfunctioning of endogenous analgesia in response to exercise in patients with chronic pain. Muscle contractions activate generalized endogenous analgesia in healthy, pain-free humans and patients with either osteoarthritis or rheumatoid arthritis, but result in increased generalised pain sensitivity in fibromyalgia patients. In patients having local muscular pain (e.g. shoulder myalgia), exercising non-painful muscles activates generalized endogenous analgesia. However, exercising painful muscles does not change pain sensitivity either in the exercising muscle or at distant locations.

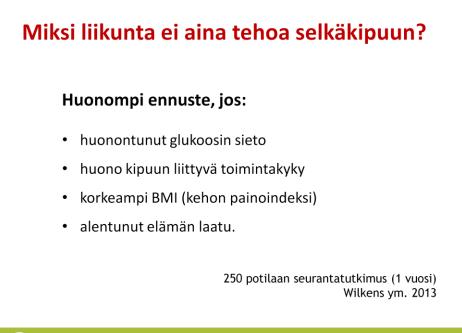
LIMITATIONS: The reviewed studies examined acute effects of exercise rather than long-term effects of exercise therapy.

CONCLUSIONS: A dysfunctional response of patients with chronic pain and aberrations in central pain modulation to exercise has been shown, indicating that exercise therapy should be individually tailored with emphasis on prevention of symptom flares. The paper discusses the translation of these findings to rehabilitation practice together with future research avenues.









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