HHS Public Access

Author manuscript

Pain. Author manuscript; available in PMC 2018 March 01.

Published in final edited form as:

Pain. 2017 March ; 158(3): 369–370. doi:10.1097/j.pain.0000000000000792.

How does physical activity modulate pain?

Laura Frey Law, PT, PhD and **Kathleen A. Sluka, PT, PhD, FAPTA**

Department of Physical Therapy and Rehabilitation Science, University of Iowa, Iowa City, IA, 52242, 319-335-9791

It has become increasingly clear that physical activity has numerous beneficial health effects. One of these effects is on the development of chronic pain. Large population studies show that individuals who are more physically active have a lower risk for development of chronic pain [10;16]. It is also well recognized that exercise is an effective clinical treatment to reduce pain and improve function in a variety of pain conditions [9]. On the other hand, a sedentary lifestyle enhances the risk for development of chronic pain [10], and those with chronic pain have lower levels of physical activity than healthy controls [4]. Further, as pointed out by Naugle et al., in this issue of PAIN [12], older adults have a higher incidence of chronic pain and are generally less physically active than younger adults. Despite the increasing body of evidence supporting the relationship between greater physical activity and pain reduction, the underlying mechanisms of how physical activity prevents pain and how exercise reduces chronic pain are only beginning to be unraveled.

The study by Naugle et al. in the current issue of PAIN [12], examined the relationship between physical activity and pain on central pain processing in pain-free (healthy) older adults. The current study showed that greater temporal summation to heat was associated with lower moderate to vigorous physical activity levels; while greater sedentary behavior and lower levels of light physical activity were associated with less conditioned pain modulation (CPM) to heat, but not to pressure following a cold water conditioning stimulus. These data would suggest that physical activity levels may modulate central excitability and central inhibition. These studies are similar to prior studies showing greater physical activity is associated with greater CPM and reduced temporal summation in younger healthy subjects [5;13], and thus, provide evidence that in older adults the same phenomena can occur. It is unclear, however if this association occurs in people with pain. It is also unclear if this association is causative, i.e. do physical activity levels alter the excitability and inhibition or do the differences in pain excitability and inhibition alter preferred activity levels.

Animal studies have directly addressed the causative nature of physical activity effects on inhibition and excitability in the central nervous system. Voluntary wheel running prior to induction of either chronic muscle pain or neuropathic pain prevented the development of hyperalgesia [6;14], suggesting a protective effect of physical activity. Further, regular physical activity and exercise reduces excitability of central neurons, measured by phosphorylation of the NR1 subunit of the NMDA receptor, alters neuroimmune signaling in the central nervous system, and increases release of endogenous opioids and serotonin in the brainstem pain inhibitory pathways [2;14;15]. Together these data support the notion that

regular physical activity can not only prevent the development of chronic pain, but also modulates physiological changes in the central nervous system.

In humans, a variety of quantitative sensory test (QST) measures are used to assess pain mechanisms. The two most commonly used are temporal summation of pain and conditioned pain modulation, believed to characterize the central excitability and central inhibitory state of an individual, respectively. But the story is more complicated than that. These two types of psychophysical measures can be assessed using a variety of noxious stimuli and at different anatomical locations that can alter the response, suggesting these centrally-mediated pain processing pathways may not be universal indicators as once thought. For example, the responses to deep tissue pressure or heat, two commonly used test stimuli, can show disparate responses as observed here by Naugle et al.[12]. Similarly, pain sensitivity is not uniform across modalities, where some individuals are more sensitive to noxious heat, others to ischemia, others to heat temporal summation [7]. Thus, it is not necessarily surprising that the relationships between physical activity and CPM using different test modalities were inconsistent in older adults in the Naugle et al study in this issue of PAIN.

While there is increasing evidence of a link between pain sensitivity and physical activity in healthy individuals, the findings are mixed. For example, triathletes demonstrated a stronger CPM response to heat than non-athletes, but no differences in TS of pain using a sustained cold water immersion or pain thresholds [5]. In another study, more vigorous physical activity was linked to lower TS of heat pain and greater CPM to heat, but no differences in heat pain thresholds, or heat or cold suprathreshold pain ratings [13]. Others have observed less pressure pain sensitivity [1] and lower pain unpleasantness and intensity to noxious heat stimuli [3] in those who engaged in more vigorous physical activity. Epidemiological investigations further support the direction of these findings. In a Dutch population-based survey, moderate physical activity was associated with less frequent back pain than physical inactivity [8]. However, this study also reported that those with the highest intensities of physical activity had a greater incidence of back pain than those with moderate physical activity [8]. Similarly, in a Norwegian population-based study, incidence of chronic musculoskeletal pain was from 10 – 38% lower in those who participated in regular physical activity (moderate exercise one – three times/week) than those not reporting any leisure-time exercise [10;11]. Collectively, many studies suggest a link between moderate physical activity and less pain incidence, intensity and/or pain sensitivity to select QST assessments; however, the findings are not always consistent.

As the current study emphasizes, chronic pain rates increase in the older adult population and older adults are less physically active than younger adults. The relationship between higher levels of physical activity and reduced temporal summation of heat pain, coupled with the reduced inhibition to heat in individuals with greater sedentary time, provides evidence that physical activity may be an important factor influencing pain sensitivity. As such, future studies may benefit from characterizing physical activity levels in addition to the many other factors that have shown small, but significant, associations with pain perception, such as sex, gender, depression, anxiety, and pain catastrophizing to name a few.

In summary, research on physical activity and its influence on pain is just beginning. While there appear to be inconsistent relationships between pain sensitivity and activity levels, in general greater levels of activity are associated with less pain facilitation and more pain inhibition, with most studies done primarily in healthy controls using cross-sectional data. To move the field forward, future studies should examine these relationships in those with chronic pain, determine a causative role by examining if increasing physical activity levels alters QST or other pain-related outcomes, and determine the underlying mechanisms by which physical activity alters pain.

References

1. Andrzejewski W, Kassolik K, Brzozowski M, Cymer K. The influence of age and physical activity on the pressure sensitivity of soft tissues of the musculoskeletal system. J Bodyw Mov Ther. 2010; 14:382–390. [PubMed: 20850046]
2. Bobinski F, Ferreira TA, Cordova MM, Dombrowski PA, da CC, Santo CC, Poli A, Pires RG, Martins-Silva C, Sluka KA, Santos AR. Role of brainstem serotonin in analgesia produced by low-intensity exercise on neuropathic pain after sciatic nerve injury in mice. PAIN. 2015; 156:2595–2606. [PubMed: 26447701]
3. Ellingson LD, Colbert LH, Cook DB. Physical activity is related to pain sensitivity in healthy women. Med Sci Sports Exerc. 2012; 44:1401–1406. [PubMed: 22217571]
4. Ellingson LD, Shields MR, Stegner AJ, Cook DB. Physical activity, sustained sedentary behavior, and pain modulation in women with fibromyalgia. J Pain. 2012; 13:195–206. [PubMed: 22245361]
5. Geva N, Defrin R. Enhanced pain modulation among triathletes: a possible explanation for their exceptional capabilities. PAIN. 2013; 154:2317–2323. [PubMed: 23806655]
6. Grace PM, Fabisiak TJ, Green-Fulgham SM, Anderson ND, Strand KA, Kwilasz AJ, Galer EL, Walker FR, Greenwood BN, Maier SF, Fleshner M, Watkins LR. Prior voluntary wheel running attenuates neuropathic pain. PAIN. 2016; 157:2012–2023. [PubMed: 27355182]
7. Hastie BA, Riley JL III, Robinson ME, Glover T, Campbell CM, Staud R, Fillingim RB. Cluster analysis of multiple experimental pain modalities. PAIN. 2005; 116:227–237. [PubMed: 15964682]
8. Heneweer H, Vanhees L, Picavet HS. Physical activity and low back pain: a U-shaped relation? PAIN. 2009; 143:21–25. [PubMed: 19217208]
9. Hoeger Bement, MK., Sluka, KA. Exercise-induced hypoalgesia: An Evidence-based review. In: Sluka, KA., editor. Pain Mechanisms and Management for the Physical Therapist. Philadelphia: Wolters Kluwer; 2016. p. 177-202.
10. Landmark T, Romundstad P, Borchgrevink PC, Kaasa S, Dale O. Associations between recreational exercise and chronic pain in the general population: evidence from the HUNT 3 study. PAIN. 2011; 152:2241–2247. [PubMed: 21601986]
11. Landmark T, Romundstad PR, Borchgrevink PC, Kaasa S, Dale O. Longitudinal associations between exercise and pain in the general population–the HUNT pain study. PLoS One. 2013; 8:e65279. [PubMed: 23776464]
12. Naugle KM, Ohlman T, Naugle KE, Riley ZA, Keith NR. Physical activity behavior predicts endogenous pain modulation in older adults. PAIN. XXXX.
13. Naugle KM, Riley JL III. Self-reported physical activity predicts pain inhibitory and facilitatory function. Med Sci Sports Exerc. 2014; 46:622–629. [PubMed: 23899890]
14. Sluka KA, O'Donnell JM, Danielson J, Rasmussen LA. Regular physical activity prevents development of chronic pain and activation of central neurons. J Appl Physiol. 2013; 114:725–733. [PubMed: 23271699]
15. Stagg NJ, Mata HP, Ibrahim MM, Henriksen EJ, Porreca F, Vanderah TW, Philip MT Jr. Regular exercise reverses sensory hypersensitivity in a rat neuropathic pain model: role of endogenous opioids. Anesthesiol. 2011; 114:940–948.

16. Zhang R, Chomistek AK, Dimitrakoff JD, Giovannucci EL, Willett WC, Rosner BA, Wu K. Physical Activity and Chronic Prostatitis/Chronic Pelvic Pain Syndrome. Med Sci Sports Exerc. 2015; 47:757–764. [PubMed: 25116086]