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Latent Effects of Stress on Delayed Modulation of Chronic Low Back Pain: A Case Series

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INTRODUCTION

A consistent ten-day delayed response between stressful events and flares in chronic pain has been reported in studies on fibromyalgia, complex regional pain syndrome and phantom limb pain. Psychogenic release and latent activation of thyrroxin has been established as a delayed neuromodulation, upregulating nociceptive pathways and cognitive pain perception. For this study, low back pain was chosen as a pilot condition prefacing a larger research effort to determine which variants of chronic pain may manifest latent psychogenic modulation.

PURPOSE

This study’s purpose was to investigate the temporal relationship between psychogenic stress and perceived intensity of chronic low back pain (LBP).

METHODS

Over 12-15 weeks, five participants with chronic LBP completed daily visual analog pain, stress, and pain-related function scales. Temporal relationships between stress and perceived pain were analyzed using serial lag correlation coefficients up to a 10-day lag. Daily medication and quality of pain perception changes were reported. Females reported daily phase of their menstrual cycles, due to estrogen effects on thyrroxin binding globalins. The full survey was completed online via home computer or mobile device daily. Data automatically transferred to a secure central analysis database, a method translating easily to large-scale data collection for future studies.

PARTICIPANT 1

39 year-old male diagnosed with LBP 5 months prior to the study due to an MRI showing disc height or squaring and sitting for long periods of time provoked symptoms

Figure 2: Serial-lag correlations between perceived stress and pain.VAS showing peak correlation found on the same day as Participant 1 reported stress (10 day lag): r = 0.23, p = 0.06

PARTICIPANT 2

20 year-old female with LBP secondary to Ehlers-Danlos syndrome for the last 3 years. She experienced periodic down to bilateral feet chronically with stress, lack of sleep, and “having movements” as triggers for pain.

Figure 3: Serial-lag correlations between perceived stress and pain. VAS showing peak correlation found 2 days after Participant 2’s reported stress (2 day lag): r = 0.65, p = 0.0019

PARTICIPANT 3

19 year-old female diagnosed with LBP 3 years ago with converted seizures.

Figure 4: Serial-lag correlations between perceived stress and pain. VAS showing peak correlation found same day as Participant 3’s reported stress (0 day lag): r = 0.81, p = 0.0001

PARTICIPANT 4

24 year-old female with LBP originating 6-years ago secondary to a C 5-6 discopathy. Long periods of standing and walking and postural alignment while seated results in spasm along the thoracolumbar junction.

Figure 5: Serial-lag correlations between perceived stress and pain. VAS showing peak correlation found one day after Participant 4’s reported stress (1 day lag): r = 0.80, p = 0.0005

PARTICIPANT 5

52 year-old female diagnosed with LBP 2-years ago as a result of lumbar disc injury. Collapsing stress and sitting for long periods of time trigger symptoms.

Figure 6: Serial-lag correlation between perceived stress and pain. VAS showing peak correlation found 2 days after Participant 5’s reported stress (2 day lag): r = 0.60, p = 0.006

CORRELATION (r) COMPARISON FOR PARTICIPANTS 1-5

Figure 7: Stress and Pain Correlation (r) vs Serial-lag (days) for all 5 participants. Note correlation trend of diminishing correlations with the exception of Participant 1.

DISCUSSION

Serial lag correlations revealed variation between participants as to number of elapsed days between high stress and the strongest correlation with increased LBP intensity. Two participants showed the strongest correlation between pain and stress experienced the same day (r = 0.23 & 0.26). One participant had the strongest relationship between stress and pain experienced one day later (r = 0.25). For two other participants stress was best correlated with pain flares occurring 2 days later (r = 0.06 & 0.60). The exception to the overall diminishing correlation trend was Participant 1. Collapsing data across participants showed the strongest overall correlation at the 0-day time lag (r = 0.66, p = 0.00001), indicating spikes in pain the day of the stress episode. Unlike previous studies involving neuropathic pain flares there were no significant correlations between stress and LBP occurring ten days later (range, r = -0.21 to -0.21).

CONCLUSION

Chronic low back pain does not appear to be influenced by the same delayed psychoneuromodulation mechanism as chronic neuropathic pain conditions previously studied.

RELEVANCE

It has been beneficial for therapists and patients to know that delayed relationships exist between episodic stress and chronic pain. This helps explain and predict many flares in pain intensity. The current study’s findings do not support the hypothesis that chronic LBP perception is influenced by the same delayed psychoneuroendocrine modulation as neuropathic pain, suggesting that development of chronic LBP may not be due to neuromodulatory remodeling mechanisms used to understand pain syndromes of neuropathic origin, but rather nociceptive in origin.

This study supports the position that up not all chronic pain should be clinically approached in the same manner. A patient’s response to delayed neuromodulation may provide insight into whether the condition is primarily nociceptive or neuropathic and, therefore, treatments may be selected based on the mechanism of pain generation and modulation.

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References


