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Effect of Pain Medication on Pain Sensitivity of Pain-Related Words

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Abstract: Pain perception is influenced by cognitive and linguistic processes, in addition to sensory factors. Previous studies have shown that words that are related to pain cause both emotional and physical responses. This suggests that language processing and nociceptive pain share neural substrates. This study looks at how acetaminophen, a common painkiller, affects people's ability to understand language related to pain. It seeks to determine whether people's perceptions of semantic pain are impacted by pharmacological pain management. We hypothesize that embodiment theory underlies the processing of pain-related words. And we predict that acetaminophen will attenuate perceived pain intensity associated with such words. Alternatively, if painkillers do not influence linguistic pain perception, this would support disembodied theories, such as attention bias or memory-based mechanisms. We compare the pain-word sensitivity of three groups—a control group, a placebo group, and a Tylenol group—using a randomized controlled design. Participants rate the pain intensity of pain-related words on a Likert scale, allowing us to assess whether acetaminophen reduces the cognitive-emotional experience of linguistic pain. This study will help us understand how embodied cognition, pain processing, and language interact. This could have effects on how pain is treated in the clinic and on cognitive therapy.

Keywords: pain perception, embodiment theory, semantic pain, acetaminophen, placebo effect, language processing, cognitive-emotional pain.

1. Introduction

According to recent research, there are connections between language and pain perception, highlighting significant interactions between cognitive and sensory processes [1]. Studies demonstrate that pain-related vocabulary triggers both emotional and physiological responses. Researchers have discovered that individuals with increased pain sensitivity could process pain-related words more intensively [2]. The results indicate observable interactions between pain processing and language comprehension.

Semantic pain, the intersection of language and physical pain perception, shares neurological foundations with nociceptive pain from physical injury [3]. Neuroimaging studies by Borelli et al. and Kross et al. show that pain-related language activates brain regions linked with physical pain

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processing [3-4]. This neural convergence occurs within the brain's pain matrix, specifically in the somatosensory cortex and insula, which respond to both linguistic and physical pain stimuli [5-6]. Additional investigations confirm that pain-related words can activate nociceptive neural pathways, showing how language and physical pain share the same neural pathways [7].

Acetaminophen (Tylenol), traditionally used for physical pain relief, showcases broader psychological impacts. Research indicates that acetaminophen reduces emotional distress, including anxiety and psychological discomfort [8]. Additionally, studies demonstrate acetaminophen can reduce social rejection pain. Suggesting it shared neural mechanisms between physical and social pain experiences [9]. Placebo research further shows the mind's role in pain perception, revealing how expectation alone can activate endogenous opioid systems [10-11].

This study will investigate acetaminophen's and placebo's effects on pain-related language perception, exploring potential therapeutic applications. We will examine the pain matrix's role in processing physical pain and pain-related language, evaluating how the neural network is involved in verbal language and sensory pain interpretation.

We hypothesize that the embodiment theory is the mechanism that explains why we feel pain from pain-related words. In order to test that, we will utilize the acetaminophen drug, Tylenol, to see its effect on suppressing the transmission of pain signals in the central nervous system, reducing the individual's perception of pain. For the null hypothesis, the alternate theories are the mechanisms that explain why we feel pain from pain-related words, in which medication relieves physical pain, unrelated to the way people perceive pain-related language or cognition. Painkillers may only affect physical pain, not the perception of abstract concepts related to pain.

This research addresses two primary objectives: (i) examining embodiment theory's role in pain-related language processing and (ii) investigating acetaminophen's clinical potential for cognitive pain perception. We test whether embodied cognition extends to pain perception by examining acetaminophen's influence on both physical pain and pain-related language processing, given their shared neural substrates. Additionally, we evaluate whether conventional pain management pharmaceuticals can modulate cognitive and emotional pain responses, potentially informing clinical pain management protocols.

2. Implications

If pain medication could alter pain perception, it would offer significant causal evidence supporting the embodied stimulation hypothesis in the context of abstract domains. This would extend previous research, such as Pulvermüller, which explored embodied cognition in concrete domains [12]. Clinically, the findings could inspire new research into the use of pharmacological treatments for emotional pain management, potentially opening pathways for integrating cognitive therapies into pain relief strategies.

3. Proposed study

To address these questions, we will conduct an experiment where participants with high pain sensitivity are randomly assigned to one of three groups: (1) a control group receiving no treatment, (2) a placebo group, and (3) a Tylenol group. The Tylenol group takes acetaminophen before the experiment in contrast to the placebo group, who will receive a non-active pill. Using the established methods from studies on pain-related language processing conducted before [13], the study will rate participants' pain using a 5-point Likert scale, assessing how they closely connect each word with pain.

We will evaluate the changes in participants' pain word ratings among the three groups to test if acetaminophen helps reduce the perception of pain-related words in comparison with placebo and no

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intervention. The findings will help us understand neural and cognitive pain perception mechanisms for emotional and cognitive pain and explore their further pharmacological use.

4. Method

4.1. Participants

A total of 150 native English speakers (aged 18-40) with neither any comprehension disorder nor other language-related issues will be recruited for the experiment. These participants will be divided into three groups: (1) 50 participants with no prior history of analgesic consumption will not be instructed to take any medication during the experiment; (2) 50 participants will be instructed to take Tylenol during the experiment; (3) 50 participants will be instructed to take placebo pills during the experiment.

4.2. Stimuli

We assembled a list of 100 English pain words. All words are concrete nouns (e.g., hammer, tank, snake) and selected from a standard English dictionary to ensure the clear definitions. A survey consisting of these 100 words will be presented to the participants. Abstract nouns will not be selected for the experiment, as Rueter et al. illustrated that no significant result could be obtained for people's ratings on abstract nouns.

4.3. Procedure

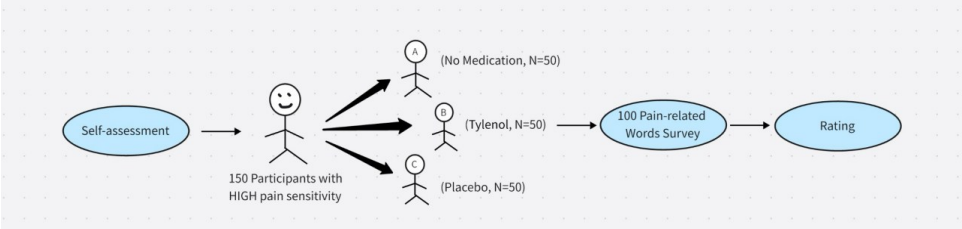

Figure 1: Visual Experimental Procedure for Assessing Pain Perception of Pain-Related Words

Figure 1 shows the visual experimental procedure for assessing pain perception of pain-related words. In the preliminary test, participants will be asked to self-assess their pain sensitivity on a 3-point Likert scale (1=low, 2=moderate, 3=high) before the survey. This test will be conducted to select participants with only *high* pain sensitivity levels. This is done to minimize the impact of individual differences in pain sensitivity on pain perception [14]. The reason for choosing *a high* pain sensitivity level as a control variable lies in the fact that Reuter et al. have demonstrated a significant difference between the *high* and *moderate* groups as well as between the *high* and *low* groups, but an insignificant difference between the *moderate* and *low* [14].

Responses will be collected to obtain 150 participants with "high" pain sensitivity. The participants involved in the formal experiment will be the selected 150 individuals. They will be randomly assigned to each group, and corresponding medications will be distributed according to groups.

Before the survey, we will follow the procedure conducted by Reuter et al. The participants will be informed that they will be presented with a list of words and required to rate the pain level of reading each word. They will be given 1000 milliseconds for reading each word on the screen. After reading the word "x," they will be asked to rate how strongly they associate the word "x" with pain on a 5-point Likert scale (1=not at all, 2=slightly, 3=medium, 4=strongly, 5=very strongly) by pressing the matching button [14].

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4.4. Predictions

From this experiment, we propose two predictions, in accordance with the two hypotheses.

Our experimental hypothesis predicts that (P1) pain medication will reduce the perceived intensity of pain-related words in individuals with moderate pain sensitivity. This will show alignment between embodiment theories and perception of pain with pain-related words.

Our alternate/null hypothesis predicts that (P2/P0) participants under the pain medication group and placebo group will report similar perceived intensity of pain-related words with high pain sensitivity. This will raise questions about which alternate mechanism this study is achieved through.

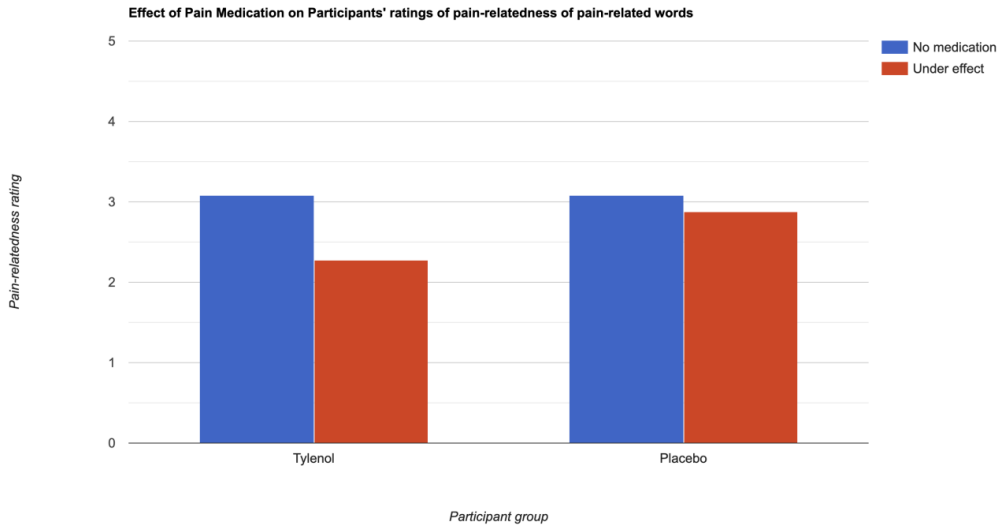

Figure 2: An example of the expected pain ratings from P1 of the 3 participant groups

Figure 2 is an example of the expected pain ratings from P1 of the 3 participant groups: no medication, Tylenol, and placebo. Participants under Tylenol will report lower pain ratings than no medication and placebo.

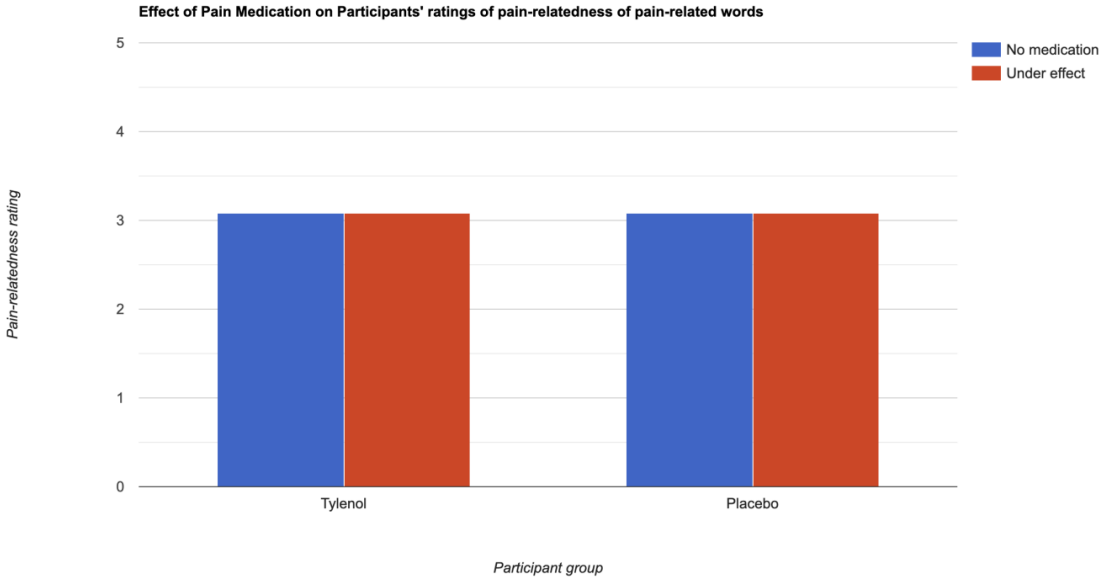

Figure 3: An example of the expected pain ratings from P2 of the 3 participant groups

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Figure 3 is an example of the expected pain ratings from P2 of the 3 participant groups: no medication, Tylenol, and placebo. All three participant groups will report relatively the same pain ratings.

Embodiment theories will account for P1 because participants will perform simulations of the physical and emotional pain associated with pain-related words. For example, upon hearing the word "knife," participants will imagine the pain of a knife cut. This causes the subsequent pain rating. Once analgesics are taken, self-reported pain sensitivity in participants will be nullified, resulting in a feeling of "less pain" upon hearing the same words. The addition of a no-medication control group will account for the psychological effects associated with taking pain medication (e.g.., Believing you are in less pain, rather than feeling it) that participants under placebo might experience. This will narrow down inflicted pain derived from pain-related words to exclusively physical and affective pain.

Alternate theories, aside from embodiment, will account for P2 because participants will feel no effect from the analgesics. This includes memory bias, attention bias, and prototype analysis.

5. Conclusion

If participants in the control and experiment groups do not show significant differences when rating the pain-relatedness of words, this outcome would suggest a disembodied explanation for pain ratings of pain-related words, like attention and memory bias or prototype analysis theories. This result would also challenge the embodied theory on abstract words in general.

Alternatively, if participants randomly assigned to the Tylenol group rate the pain relatedness of words as lower compared to those assigned to placebo, then this would provide strong evidence for the embodied stimulation explanation for pain. Specifically, our study would support the embodied hypothesis as one of the proposed mechanisms in the studies [13,15]. More broadly, this result would provide causal evidence for the embodied stimulation hypothesis, specifically in the abstract domain, expanding the causal evidence of concrete domains as studied using TMS on action word perception [12]. On the clinical side, this result would also encourage studies on emotional pain management with pharmacological treatment.

Our study investigates pharmacological intervention on non-physical stimuli. Future research could study the relationship in reverse. That is, can non-physical stimuli like words affect physical pain? Such research could bring cognitive therapy into the domain of pharmacological pain management.

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