

THESIS

THE EFFECT OF MUSICAL MOOD INDUCTION ON PAIN PERCEPTION IN
ADULT ONCOLOGY PATIENTS

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ABSTRACT

THE EFFECT OF MUSICAL MOOD INDUCTION ON PAIN PERCEPTION IN ADULT ONCOLOGY PATIENTS

The purpose of this pilot study was to examine the effect of musically induced mood on the pain perception of cancer patients. Enrolled participants (N=2) were both patients receiving radiation, chemotherapy, or follow up treatment at a local hospital where recruitment took place. During the experiment participants listened to a 20 minute recording of increasingly elative music, and completed questionnaires before and after that measured mood and pain perception. Measurement tools included the SF-McGill Pain Questionnaire, and the Positive and Negative Affect Schedule (PANAS). Due to low sample size, there were no statistically significant results. However, pain decreased in all tests scores for both participants, and negative affect measures also decreased. Suggestions for future research are discussed.

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CHAPTER I: INTRODUCTION

Purpose

The purpose of this study was to examine the effect of musical mood induction (MMI) on the pain perception of adults who are experiencing cancer pain. The mood state of participants were examined pre and post treatment through standardized self-evaluation measures to determine if MMI elicited positive mood change. The pain perception of participants was also measured before and after treatment in the same manner as affect. Analysis compared pretest and posttest affect and pain reports, and also examined pain scores in relation to mood induction scores.

Background

There is a currently a dearth of scientific, evidence-based research related to the effectiveness of music therapy in the hospice setting (Hilliard, 2005). As a growing specialization within music therapy, it is of great importance that clinicians, educators, and researchers continue to explore the reasons why music therapy works with this population and the frequently targeted goal areas addressed, such as pain reduction. The information learned could help distinguish effective protocols, provide a platform of better communication between therapists and other hospice care professionals, and validate the capacity of this specialization in general. Existing research tends to be

anecdotal or observational, and protocols based primarily on such literature are well known and accepted by most music therapy professionals possessing experience in end of life care.

Pain reduction is one of the most common goal areas music therapists address in hospice care (and oncology), where many patients experience pain that is both directly and indirectly related to cancer (Groen, 2007). Of interest is that many children and adults are under-medicated for pain, for reasons including poor assessment, misreporting by patients, and patient choice. According to the World Health Organization, only 50% of cancer pain control is ever achieved (International Association for the Study of Pain, 2005; International Association for the Study of Pain, 2008). The elderly population might suffer the most pain, due to concurrent pain complications and because older patients are more likely to not take prescribed medicine correctly (Chatwin, Closs, & Bennett, 2009). Poor pain management can lead to depression, anxiety, irritability, disturbances in sleep, and impaired physical function (International Association for the Study of Pain, 1994). This is concerning not only because it implies a decrease in quality of life for many patients, but also because recent pain research has shown that neuroplastic changes can occur when pain is under or untreated. These changes have been correlated with chronic pain in the form of hyperalgesia (hypersensitivity to a painful stimulus), neuropathic pain (originating from the nervous system), and allodynia (the sensation of pain in response to a benign stimulus). Patients who choose not to use pain medications, or who only take partial doses might actually put themselves at higher risk of these conditions. It is also important to note that similar neuroplastic changes might occur in concurrence with opioid use. For the reasons stated, there appears to be a

continued need for effective and financially feasible non-pharmacological pain treatment.

Cancer-related pain can be caused by either the disease itself (tumor secretions, pressure or damage by tumors to tissue, bone, viscera, or nerves) or by treatments such as radiation therapy, chemotherapy, and hormonal therapy. Malignant-related is not unlike non-malignant pain; however a combination of somatic, visceral, and neuropathic sources might be involved simultaneously (Levy, Chwistek, & Mehta, 2008). Cancer pain can be constant, intermittent, acute, or chronic, and it affects an estimated 50% of patients soon after diagnosis, increasing to about 75% of patients experiencing the advanced stages of the disease (International Association for the Study of Pain, 1994). Furthermore, a significantly higher percentage of cancer survivors experience chronic pain compared with the general population (Burton, Fanciullo, Beasley, & Fisch, 2007; Logan, Bartoshuk, Fillingim, Tomar, & Mendenhall, 2008; Peuckmann, et al., 2009). Whether or not a terminal diagnosis is made, cancer-related pain is also known to be exacerbated by social, spiritual, and psychological factors such as depression, anxiety, and catastrophizing. No matter the source or type of pain one experiences, pharmaceuticals are a commonly recommended treatment.

According to the International Association for the Study of Pain (2008), a large variety of pharmacological interventions are implemented for cancer-related pain, but the two used most frequently are non-steroidal anti-inflammatory drugs (NSAIDs) and a wide range of opioids. The former are prescribed for mild pain typically caused by tissue damage or inflammation, and the latter are used to address moderate to severe pain originating from diverse sources. NSAIDs carry a higher than average risk of gastrointestinal irritation or bleeding in cancer patients, and can also impact kidney or

heart function, especially amongst the elderly. Opioids, in addition to risks of tolerance and the already described link to chronic pain, can produce a number of significant side effects including nausea, vomiting, constipation, sleep disturbances, dysphoria, and sedation (International Association for the Study of Pain, (2008). These risks and side effects can make pharmacological management of cancer-related pain less than desirable, and unfortunately these approaches don't always succeed in controlling pain. Therefore, a multidisciplinary approach is often recommended (i.e. Levy et al. 2008, Turk, Swanson, & Tunks, 2008).

Cognitive-behavioral therapy is commonly used in conjunction with medication, and it both educates patients about pain and teaches self-coping skills such as relaxation, biofeedback, or imagery (Turk et al. 2008). Other commonly used non-pharmacological techniques include mindfulness-based practices, distraction, acupuncture, massage, transcutaneous electrical stimulation (TENS), and music listening. While all of these interventions are often used successfully, the hypothesized reasons for the effectiveness of each are many and diverse (eg establishing a sense of self-control or increasing alpha brainwave activity (Turk et al. 2008)).

This is equally true within the music modality, for which multiple hypotheses exist. One rationale, applied to multiple approaches, theorizes that positive changes in a person's affect subsequently lead to decreased pain perception. However, of the existing research investigating the palliative effects of music, only three studies have specifically targeted affect modification as a primary agent responsible for change in pain perception (Martin, Nathan, Milech, & van Keppel, 1988; Roy, Peretz, & Rainville, 2008; Tang et al. 2008).

The purpose of this pilot study was to examine the effect of musically induced mood on the pain perception of cancer patients.

Rationale

The definition of pain by the International Association for the Study of Pain, first drafted in 1979, has been very influential within most subsequent pain literature (Craig & Hadjistavropoulos, 2004). It describes pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (International Association for the Study of Pain, 1994, IASP Pain Terminology section, para. 1). The most current draft goes on to eventually add that “Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.” (para. 1). In congruence, much research over the last fifteen years has demonstrated that affect modulation and regulation play a highly significant role in both experimental and clinical pain perception (i.e. Strand et al., 2009; Tan, Jensen, Thornby, & Sloan, 2008; Zillmann, de Wied, King-Jablonski, & Jenzovsky, 1996). To date, the majority of studies using the modification of mood to decrease pain perception have used non-musical induction techniques. This is curious considering that music is often used casually and professionally as a way to manage both mood and pain.

Music has in truth been used to treat cancer-related and other types of pain throughout history, and it has been noted by several authors that multiple mechanisms are probably involved in pain responses to music (Magill-Levreault, 1993; Rider, 1985).

Within the literature however, there are two prominently cited rationales for its effectiveness. One, based on the gate control theory, postulates that music serves as a distraction. Such studies have used a variety of music induction techniques, have targeted both clinical and experimental populations, and have also noted that emotion in music might enhance the distraction effect (Mitchell & MacDonald, 2006; Tse, Chan, & Benzie, 2005). The other rationale involves relaxation via music-breath entrainment and consequent autonomic responses (i.e. Good, Anderson, Ahn, Cong, & Stanton-Hicks, 2005; Rider, 1985; Voss et al., 2004). Both explanations are logical and should not be dismissed, but the first does not necessarily implicate music therapy as preferable to other distraction techniques, and the second has consistently produced contradictory physiological outcomes. Furthermore, neither theory incorporates the above mentioned recent research that points toward the important role of affect modulation in pain perception.

Interestingly, there is a wealth of research supporting music as one of the most effective ways to alter affect. In fact, music has been shown to be one of the most successful modalities for mood induction, significantly more so than listening to audiobooks (Mitchell, MacDonald, & Brodie, 2006; Särkämö et al., 2008), watching film (Van der Does, 2002), or using the Velton technique (Clark, 1983; Sutherland, 1982; Västfjäll, 2001), which uses verbal self-statements and also may have methodological disadvantages compared with music (Pignatiello, Camp, Elder, & Rasar, 1989). Martin (1990) reviewed and compared sixteen mood induction techniques, and reported that music was one of the most successful and least biased in terms of demand characteristics. De l'Etoile (2002) asserts that MMI is one of the best induction techniques for creating

lasting mood change. Considering the demonstrated effectiveness of MMI to modify affect, it seems appropriate to further investigate its possible subsequent influence on pain perception. As noted earlier, only a few studies to date have used music to specifically target affect modification as a primary agent responsible for change in pain perception. Three of these will be briefly reviewed, following a closer examination of the MMI procedure itself:

In 1983 Clark reported that five studies had been conducted in which music was used to induce mood (all around that time, though only two were published). The experiments were all based on a similar procedure, first developed by Sutherland et al. (1982), in which subjects were given a directive to get into a specific mood state while listening to recorded music. Taking the findings from some pilot studies, Sutherland et al. chose to use music as an induction method because it had produced better results than other methods. Pre and post induction mood scores were measured in the experiment, and subjects were asked to choose music that they felt would best alter their mood from an assortment of preselected classical and modern recordings (Sutherland et al., 1982).

Clark (1983) notes that a key difference between the first two of the five studies using MMI and the later three (one being Clark & Teasdale's 1985 study, though Teasdale helped conduct all three), was the music itself. While the first two studies allowed subjects to choose between preselected tapes, the later experiments provided specific music meant to induce a depressed or happy mood. Clark & Teasdale's (1985) procedure used short excerpts that repeated to fill seven minutes. The excerpt used to induce a depressed mood was "Russia Under the Mongolian Yoke" by Prokofiev, from the film "Alexander Nevsky", and recorded at half speed. The music used to induce an elated

mood was an excerpt from “Coppelia” by Delibes. The researchers asked subjects to rate their mood honestly, and stressed the importance of this step (Clark & Teasdale, 1985).

Clark & Teasdale's (1985) publication has influenced many researchers to this day. Martin et al. (1988) used MMI to examine the relationship between headaches and mood. Using a method based on Clark & Teasdale's 1985 procedure, they attempted to induce a positive mood in one group, and a despondent mood in another. Mood and pain were measured before and after the intervention, using visual analogue and number scales. The mood measures proved to be statistically insignificant, but results showed an increase in headache intensity for the “despondent” condition, and a decrease in headache intensity for the “happy” condition.

Twenty years later, Tang et al. (2008) also followed Clark & Teasdale's 1985 procedure for MMI. Subjects were all chronic back pain patients, and various tools (number scales, the Short-Form McGill Pain Questionnaire, Brief Pain Inventory, Hospital anxiety and depression Scale, and Short Health Anxiety Inventory) were used to measure pain, depression, and cheerfulness. Subjects were asked to hold a heavy bag for 10 minutes or until it became too painful. Musical mood induction was then implemented, and the bag holding task repeated. Measures were taken before and after each task. Results demonstrated that subjects in the elated musical mood induction reported significantly less pain and increased tolerance than either the negative or neutral mood induction group.

That same year Roy et al.,(2008) used a different MMI procedure to compare the effects of pleasant versus unpleasant emotional valence on pain perception in a non-clinical population. The authors selected 5 minute excerpts of both pleasant and

unpleasant music matched for level of arousal, based on prior feedback from 20 independent participants. Participants were subjected to painful and non-painful thermal stimuli while listening to either one of the music conditions or to silence. They rated pain on a number scale as it was induced, and evaluated the level of musical arousal and valence on a number scale immediately following each procedure. Participants listened successively to all three conditions in a randomized order, and after total experimental completion their mood was measured using the Profile of Mood States (POMS) scale. Results showed that pleasant music caused larger reductions in pain intensity and unpleasantness than the other two conditions, and that lower arousal states enhanced the reduction.

Hypothesis

This study examined whether positive musical mood induction can produce statistically significant decreases in the pain perception of adult oncology patients.

The following null hypothesis was proposed: Positive musical mood induction will produce no differences in the pain perception of adult oncology patients.

CHAPTER II: RELATED LITERATURE

Pain processing and modulation is an extensive and complex system. Research over the last decade has increased quite a bit and not only found new evidence for pain processes, but has been a catalyst for new physiological, anatomical, chemical, and psychological theories. Some of these include the neuromatrix theory, the view that pain is a homeostatic emotion, the understanding that pain encompasses distinct sensory and affective domains with neurological correlates, the theory that chronic pain results from neuroplastic changes that occur soon after an initial insult, and the belief that chronic pain is sometimes correlated with both pharmacological pain management and non-treatment of pain.

Unfortunately, what becomes clear after considering these new ideas is that much is still not understood, and at this point the research remains abundant with contradictions, leaving little to certainty. All the same, enough is understood so that new methods of pain modulation can be knowledgeably explored. The following section will review psychological theories of pain, the physiology of pain as it is currently understood, neuroplasticity as it relates to chronic pain, and pain as it relates to affect. In addition a more in depth look at musical mood induction, mood and pain responses to musical structure, and the physiology of emotional responses to music will be examined.

Psychological theories of pain

There is much controversy and limited assurance over what physiological and psychological mechanisms constitute the experience of pain. Until the mid-twentieth century, most models of pain were based on an idea proposed by Descartes (1595-1650), which asserted that once a body part was injured, signals were sent to and processed in the brain. This did not explain instances of chronic pain, and someone experiencing persistent back pain would likely be labeled as insane (Melzack & Katz, 2004).

Psychodynamic models were proposed by Breuer & Freud (1893-1895/1957) and Engel (1959), the first of which proposed that chronic physical pain was an expression of emotional pain. Engel (1959) suggested that chronic pain may or may not stem from a physical source, and also that some people sense pain pathologically, as part of a self-protective function (likely based on learned associations from the reactions of others in childhood). Neither of these models have held up well against time or empirical testing, although the concept of psychogenic pain is credited to Engel (Asmundson & Wright, 2004).

Researchers continued to look for a more credible understanding of pain, and Descartes's 17th century biomedical theory began to evolve over the 1940's-60's until 1965 when Melzack and Wall published the *gate control theory*, which is still widely influential today. One of the most important contributions of this theory was the idea that mechanisms in the central nervous system (CNS) not only receive nociceptive input, but also modulate it via descending pathways that extend to the dorsal horn of the spinal cord. The namesake of the theory refers to proposed gates located in the substantia gelatinosa of the dorsal horn, where peripheral neurons first synapse with interneurons

(Melzack & Katz, 2004; Coderre et al., 2003).

At these gates, large sensory nerve fibers inhibit afferent spinal neurons that pass pain signals to the CNS, thereby “closing the gate”. At the same time, smaller nerve fibers act to excite the same spinal neurons, hence “opening the gate”. Also proposed was the idea that some large fibers project directly to the various regions in the CNS, bypassing the gate altogether and consequently modulating more directly (Coderre et al., 2003). One idea generated from the gate control theory is that attention toward pain increases the perception of it, and likewise, distraction from pain decreases it by recruiting large fibers to close the gate (competing sensations such as squeezing one's arm after bumping it similarly assist in closing the gate). This has been demonstrated mostly with short lived and mild pain (Padhi, 2005), although Hauck (2007) notes that some study outcomes have shown success decreasing chronic pain using distraction. He also cites evidence for enhanced synchronization to the sensorimotor cortex during focused attention to pain, and assuming a positive correlation between the two, this supports the idea of attention modulation (a decrease in attention to pain would cause a decrease in pain perception). While attention modulation has become a widely accepted phenomenon and the gate control theory is not often contested (though neither proven), it does not seem to offer a complete explanation for all known methods of pain modulation.

Recently, Melzack proposed a *neuromatrix theory*, which expands on the gate control theory. It describes pain as a multidimensional experience- the “body-self neuromatrix”, which involves multiple inputs from cognitive, sensory, and emotion related mechanisms in the brain and periphery as well as activity of the body's stress-regulation systems (Melzack & Katz, 2004). The output of this matrix includes pain

perception, but also encompasses action, stress regulation and overall awareness.

Melzack terms the output as a “neurosignature”, which is an always changing “stream of awareness”. The fundamental makeup of the neuromatrix is predetermined by genetics, but is ever changing in response to sensory input. This theory has been cited in both neurophysiological as well as psychological studies, and critical areas involved include the cingulate, insular, somatosensory, prefrontal, and cerebellar cortices (Hauck et al., 2008; Henderson, Gandevia, & Macefield, 2008; Khalsa, 2004). More recently the term “pain matrix” has come into use to specify that this network is at least somewhat pain specific, though there remains much debate over its definition and even its existence (Iannetti & Mouraux, 2010). The neuromatrix theory helps explain why change in affect may influence pain perception, though another recent theory by Craig (2003) does as well.

Craig (2003) proposes a biopsychological model in which pain is a homeostatic emotion. This theory differentiates the conventionally affective *aspect* of pain as being a pure, specific behavioral motivation (along with thirst, hunger, temperature etc.), unlike other “classical” emotions that are derived from interactions with one's external environment. This idea has since been referred to in numerous articles (i.e. Appelhans & Luecken, 2008; Connelly et al., 2007; Tse et al., 2005), and relates specific neuroanatomical structures to interactions between pain and emotion. In common with the gate control and neuromatrix theory, the idea of pain as a homeostatic emotion combines psychological and biological elements in an understanding of the pain experience. Some other models further integrate social factors in a biopsychosocial approach.

A few of the the more influential biopsychosocial models include the operant model, the Glasgow model, the biobehavioral model, and fear-avoidance models (Asmundson & Wright, 2004). Asmundson & Wright (2004) attempted to synthesize facets of all four mentioned biopsychosocial approaches into a comprehensive *diathesis-stress* model of chronic pain. This model recognizes the initial physiological elements of nociception, as well as later physiological responses in from the autonomic nervous system. It also suggests that individuals are predisposed to respond to nociception with or without anxious apprehension. Not only do pain behaviors tend to be communicated socially (verbally or not), but they are also influenced by the nature of social support offered so that too little or too much support can exacerbate pain perception (Asmundson & Wright, 2004).

Like the neuromatrix theory and the proposal of pain as a homeostatic emotion, biopsychosocial approaches such as the diathesis-stress model appear in both psychological and physiological articles published today. While these theories hold their own distinctions, they also complement each other in that they all encompass multiple dimensions. The inclusion of psychological contributions to our understanding of the pain experience is noteworthy not only because it has become so widely accepted, but because of its implications for treatment.

Physiology of pain

More certainty exists in regard to the basic physiology of pain, but it should be clarified at this point that the occurrence of pain is distinct from nociception. Nociception is strictly mechanical and takes place in the peripheral nervous system. Pain on the other

hand takes place in the central nervous system, and involves a complex combination of psychological and sensory processes - hence the term neuromatrix (Hadjistavropoulos, & Craig, 2004; Willard, 2008). Within the peripheral nervous system, primary afferent nociceptive nerve fibers terminate at the skin, muscles, joints, viscera, and vasculature. These same fibers synapse at the dorsal horn of the spinal cord (or the trigeminal nuclei in the brainstem if the primary afferent is located from the neck up). The cell bodies of these neurons are in the dorsal root ganglia (Coderre et al., 2003; Willard, 2008). As noted above, nerve fibers vary in diameter. A β fibers are large, myelinated, have a low threshold, and have the capacity to change phenotype when injured. These are the fibers that are assumed to “close the gate” in the gate control theory. A δ fibers are smaller in diameter, and C fibers are even smaller as well as unmyelinated. Both of these fiber types have a high threshold and slow transmission speed compared with A β fibers. These are the fibers that “open the gate” in the gate control theory. At the dorsal horn, neurochemical changes take place some of which, in the case of injury, can lead to demyelization, cell death, and nerve sprouting (the pathological generation of new nerve endings that connect with nociceptive neurons) (Coderre et al., 2003). All these occurrences are thought to lead to chronic pain.

These neurochemical changes are complex, but are mentioned here to provide a better understanding of the many factors that contribute to pain processing, modulation, and dysfunction. When an injury occurs, chemicals including adenosine triphosphate and potassium are released from the injured tissue cells and likely produce pain. Nociceptors can be activated by mechanical, thermal, or chemical stimuli, and the chemoreceptive type fibers are activated by some of these substances, termed “alodyns”. Aside from

adenosine triphosphate, alodynogens include other neurotransmitters such as bradykinin, serotonin, tryptase, trypsin, prostaglandins, norepinephrine, excitatory amino acids, and histamine, which are released from blood, damaged tissue, and damaged mast cells. These are also believed to contribute to pain sensations. A number of neuropeptides are released from the dorsal root ganglion neurons, one of which is proinflammatory substance-P (Schaible, 2006; Willard, 2009). Likewise, immune cells release keratinocytes and cytokines, and Schwann cells and fibroblasts release nerve growth factor (Coderre et al., 2003).

Injured tissue becomes hyperalgesic, and peripheral/nociceptor sensitization (a decrease in nociceptor threshold, increase in firing frequency, and decrease in response time) occurs if the injury is severe enough. Spontaneous firing may also occur. This form of hyperalgesia, also called *primary* hyperalgesia, is temporary and evolutionarily necessary, as it causes one to behave in such a way that the localized injury is protected from further harm (If one's broken arm is hypersensitive to pain, then that person will typically use extra caution to protect it from further injury) (Coderre et al., 2003). *Secondary* hyperalgesia, a primary cause of chronic pain, will be discussed further in the next section. In short, it is a condition in which hypersensitivity is prolonged and not clearly related to the original injury. There are several possible (and debated) causes, one of which is that it is in part due to the expansion of a nociceptor's receptive field (Willard, 2009), and another being that it is caused primarily by central sensitization (also discussed in the next section) in the dorsal horn (Heinricher, 2008).

Moving on from the peripheral nervous system, the ascending pathways will be reviewed briefly. The spinothalamic tract acts as the primary spinal pathway for

nociceptor transmission. Primary afferent nociceptors terminate in laminae I, II, and V of the dorsal horn (Willard, 2009), and spinothalamic fibers project to the ventral posterior thalamic nucleus, which then projects to the somatosensory cortex. Other spinothalamic fibers project to the posterior thalamus on to the insular cortex. Nerve fibers in the spinoreticular pathway project to the brain stem reticular formation or medial thalamus, and are thought to play a role in arousal and the affective response to pain. Coderre et al. (2003) state that such diversity of projections might explain why emotion and motor responses are evoked so much in relation to pain. Other nociceptor pathways include the spinomesencephalic tract (primarily to superior colliculus and periaqueductal gray), and the spinohypothalamic tract, (to the hypothalamus) (Coderre et al., 2003). Ascending pathways can be modulated by descending pathways at any point (Schnitzler & Ploner, 2000). This mosaic of pain processing and response may be one reason why multiple approaches can be used in the treatment of pain, and taken a step further, may even support the use of more than one treatment modality at a time.

There are four structures in the cortex most often cited as being participatory in pain perception and modulation. These areas include the primary somatosensory cortex (SI), the secondary somatosensory cortex (SII), the anterior cingulate cortex (ACC), and the insular cortex. The SI is believed to process pain intensity as well as spatial, temporal, and intensity discriminatory information about the input. The SII is also thought to be involved with processing intensity as well as with pain recognition, learning, and memory of pain. Patients with lesioned SII have difficulty naming the nature of a painful stimulus (unable to state whether it is produced by temperature, or stabbing etc.). This area projects out to the temporal lobe limbic system via the insula, which is possibly most

affected by attention (Coderre et al., 2003; Duerden & Duncan, 2009). Of particular interest for this paper is the role of the ACC in pain processing. It deserves attention because it is also implicated in the emotional processing of music, to be discussed later.

Separate regions most likely process sensory pain (intensity, discrimination) versus affective (emotional) pain (Duerden & Duncan, 2009). The ACC probably processes pain affect (unpleasantness), cognition, modulation of motor/autonomic/response, and emotional memory. Considered part of the limbic system, the ACC has projections to and from the amygdala. It receives input from the medial thalamic nuclei, and is probably involved in the descending modulation of pain. The insular cortex on the other hand, possibly processes pain affect as it relates to learning and memory. It is believed to process a lot of visceral stimuli, and modulate autonomic reactions to pain. Both the insular cortex and anterior cingulate cortex have extensive connections to the limbic system, and are probably related to effects of attention, cognition, and mood on pain (Coderre et al., 2003). Also of note is the pre-frontal cortex, which has been tied to affective pain during f-MRI studies (Duerden & Duncan, 2009), and the amygdala, which demonstrates decreased activity during painful events. Similar to the ACC, the amygdala's role in pain is noteworthy due to its probable role in the emotional response to music listening. The amygdala possibly networks with the ACC (Coderre, 2003; Petrovic, 2004), and is thought to be involved with both the processing and modulation of pain. Duerden & Duncan (2009) note that the ACC is also likely involved in antinociceptive responses, since it contains many opiate receptors.

Inhibition of pain takes place mostly in descending pathways from the CNS. The descending pathways are modulated by serotonin and norepinephrine, which inhibit the release of substance P in the substantia gelatinosa via interneurons and opioids. As mentioned above, opioid receptors exist on dorsal root ganglion. These receptors open potassium channels and this decreases neuronal excitement (DeLeo, 2006). CNS mechanisms that are probably involved with pain modulation include cortical and limbic structures such as the amygdala. The ACC and prefrontal cortex, ventrolateral prefrontal cortex, and orbitofrontal prefrontal cortex might be important regions for *affective* modulation of pain (Coderre et al., 2003; Petrovic, 2004). Coderre et al. write that the inhibitory system is also influenced by learning and memory, which complements the neuromatrix theory.

Neuroplasticity and Chronic pain

Latremoliere & Woolf (2009) write that pain is a “dynamic reflection of central neuron plasticity” (p. 913). Chronic pain is defined as pain that persists after a primary injury has healed. It is distinct in that it is not thought to be caused by peripheral nerve input. Some examples of chronic pain include secondary hyperalgesia, allodynia, and neuropathic pain. About fifteen percent of peripheral nerve injury results in chronic pain, and patients with such injuries often show decreased cognitive processing ability (Coderre et al., 2003; Obermann et al., 2009). Chronic pain has many possible etiologies, none of which are considered certain.

It has been demonstrated that a decrease in gray matter correlates with chronic pain, and many believe that this decrease might be reversible, as seen in some

longitudinal studies (Obermann et al., 2009). Chronic pain might amount to less gray matter in the ACC, DLPFC, brainstem, SI cortex, and right thalamus. Increases in gray matter have also been observed in the putamen, cerebellum, and left posterior thalamus, though it has been suggested that these increases only exist during later stages of chronic pain, possibly playing a role in its termination (Obermann et al., 2009; Schmidt-Wilcke et al., 2006). The results of a few studies suggest that the decrease of gray matter within pain associated areas takes place not immediately following injury, but within the following few months (Obermann et al., 2009; Shyu & Vogt, 2009). Gray matter decreases in the ACC (which processes affective components of pain among other things) was suggested as a pivotal manifestation in the transition from acute to chronic pain. Although many of these studies target different types of pain, it has been suggested that the results are non-specific to pain type. It has also been suggested that chronic pain might be considered a neurodegenerative disorder especially affecting the DLPFC (Obermann et al., 2009). It is not known whether chronic pain causes cortical reorganization or vice versa, but more evidence seems to lean toward the latter (Schmidt-Wilke et al., 2006; Obermann et al., 2009). Among the researchers who have found evidence to support morphological changes being a result of pain, Flor (2003) reported that the amount of cortical reorganization increases as duration of pain increased.

Chronic pain is thought to be associated with reorganization in the SI cortex even in the absence of peripheral injury. Representation of painful areas in the homunculus shrink and increased shrinkage is correlated with increased pain intensity as well as increased pain unpleasantness. This is most clear in cases of neuropathic pain (Schmidt-Wilke et al.; 2006 Vartianen, et al., 2009). Pain inhibitory systems may also be altered in

chronic pain. The sympathetic nervous system is known to be involved with chronic pain, primarily via noradrenaline, which enhances primary afferents (Coderre et al., 2003). In addition, cortisol acts on the immune and endogenous opioid system by allowing high levels of glucose to break down protein in muscles, and inhibiting calcium replacement in bones. This is also thought to be a possible contributor to chronic pain (Melzack, 1999). Nuegebaur, Li, Bird, Bhave, & Gereau (2003) suggested that synaptic plasticity in the amygdala takes place when the presynaptic metabotropic glutamate receptor (mGluR1) changes expression and function. They found that the plasticity led to increased excitability of synaptic transmission in the central amygdala, which in turn could cause an increase in anxious responses to pain, as well as enhanced autonomic and endocrine responses to emotional pain.

Neuroplasticity related to chronic pain also takes place within the peripheral nervous system, and alterations have been observed in the dorsal root ganglion, dorsal horn, and brain stem. Even plasticity within the neural pathways might lead to chronic pain (Flor, 2003). Also in the dorsal horn, the smallest fibers sometimes experience apoptotic cell death when activated too much, and this could lead to enhanced excitation (Willard, 2009). In the spinal cord, evidence indicates that peripheral nociceptor input can cause neuroplasticity in connectivity, thereby causing sprouting of A δ fibers into the outer laminae of the dorsal horn, thereby making contact with nociceptive neurons, which would lead to increased pain sensations (Latremoliere & Woolf, 2009; Vartianen, et al., 2009; Willard, 2008). Such sprouting is thought to be one precursor to sensitization in the CNS, known as central sensitization.

While sensitization that occurs in the peripheral nervous system involves an enhanced reaction to noxious stimuli, central sensitization results from changes in the properties of CNS neurons, and is believed to contribute to inflammatory pain, neuropathic pain, and other pain types not usually associated with cancer (Latremoliere & Woolf, 2009). Unlike peripheral sensitization, normally unmatched inputs use nociceptive pathways and pain is processed without there ever being an external input. The idea of central sensitization fits well within the neuromatrix theory, in that it describes pain as a “sensory illusion” in the brain that is constantly changing.

Another possible cause of neuroplasticity related to pain is the use of opioids in pain treatment. Use of this type of medication is common with cancer and hospice patients, and has also become more common for chronic non-malignant pain over the last decade. Although usage is high, chronic neuropathic and radicular pain are usually resistant to both opioid treatment and NSAIDS (DeLeo, 2006). It has been documented as modifying the affective more than sensory aspects of pain (Kenner-Mabiala, Weyers, & Pauli 2007), but interestingly Padhi (2005) observed placebo opiates to be effective 30-49% of time. Opioid effectiveness in general is debatable, with some researchers claiming that its effect is not well evidenced.

There is a long list of side effects associated with opioid use including pruritus, nausea, sedation, respiratory depression, hyperalgesia, and hormonal and immune changes (Gardell, King, Ossipov, & Rice, 2005; Manchikanti, 2007; Silverman, 2009). Aside from abuse and antinociceptive tolerance building, a growing concern is that opioid use may cause allodynia and opioid induced hyperalgesia (OIH). Like secondary hyperalgesia, OIH takes place in the CNS without peripheral input. In the case of OIH,

increased sensitivity corresponds to increased opioid dose (Silverman, 2009; Gardell et al., 2005). With the exception of one study cited above (Schmidt-Wilke et al., 2006), other reviewed experiments did not report subjects' use of medication, which might be a significant oversight considering the possibility that certain types may contribute to neuroplastic changes.

Pain and Affect

Research over the last decade has increasingly examined the effect of mood on pain perception. Earlier investigations weighted interest toward pain and negative emotions which mutually impact each other, creating a downward cycle. Emotion regulation and resilience are also often cited as being influential in pain experiences (i.e. Hamilton, Zautra, & Reich 2007; Strand et al., 2009; Zautra, Johnson, & Davis, 2005), with better regulation evoking decreased pain. Following positive mood induction, pain decreases, although differences have been observed in regard to whether the affective (unpleasantness), sensory (intensity), or a combination of both dimensions respond. For instance, many researchers have noted that changes in attention (distraction) affect sensory attributes, whilst emotion influences affective pain (i.e. Villemure & Bushnell 2009; Loggia, Mogil, & Bushnell, 2008), possibly indicating separate circuits.

It is possible that some of the inconsistencies within the literature are related to differences in induction procedures and the subsequent strength of mood manipulations. Roy et al. (2008) and Kenntner-Mabiala, Andreatta, Wieser, Mühlberger, & Pauli, (2008) examined this, and observed that a potent mood manipulation affected both pain intensity and unpleasantness, although the effect on sensory pain was diminished. All the same,

there is evidence from multiple methodologies, including imaging, somatosensory evoked potentials, and lesion studies, that give credence to the existence of independent circuits. Neither of the cited investigations contends this, and instead presents neurophysiological evidence for attention and affect having distinct effects on pain response.

Another reason for the discrepancies between studies might be that it is difficult to distinguish between changes in mood and changes in attention. Villemure & Bushnell (2002) and Schön, Dahme, & Leupoldt (2008) also suggest separate pain modulatory systems for attention and emotion, citing the anterior cingulate, prefrontal, and insular cortices as possible modulating regions for pain affect. The ACC's role has been well documented across disciplines and over time, and most researchers agree that it encodes pain unpleasantness and modulates affective pain.

While pinpointing individual CNS structures and their role in the pain experience is important, it might be more beneficial for scientists to focus on the broader neural networks involved. Whittle, Allen, Lubman, & Yücel (2006) write that less is known about the neural circuitry involved in positive mood states compared with negative states, but suggests that the amygdala, nucleus accumbens (NAcc), ventral ACC, and left DLPFC all work together. They observed a left lateralized circuit linking subcortical limbic areas (primarily the NAcc via the amygdala) to the dorsal prefrontal cortex, and also downward projections in the opposite directions that regulate affective processing with additional output to the ventral ACC and hippocampus. Emotion processing, pain facilitation, and affective pain modulation all share many of the same brain regions and networks. Rhudy, Dubbert, Parker, Burke, & Williams, 2006, observed that the ascending

and descending pathways that activate pain facilitation and modulation have common neural mechanisms including the periaqueductal gray (PAG), amygdala, ACC, NAcc, and hippocampus. This overlap suggests that emotion modulates pain by activating the descending pain circuitry.

Musical mood induction

As mentioned earlier, the first study to use music for mood induction was published by Sutherland in 1982 (Martin, 1990). The purpose of the study was to learn whether undesirable intrusive thoughts were more difficult to remove when in a depressed mood than when happy, and the results supported this idea; subjects with an induced depressed mood found it more difficult to remove intrusive thoughts. Sutherland conducted two separate mood induction procedures, one using music and the other using the Velton method. He found the music induction procedure led to superior changes in mood, longer retention of mood, and that a greater number of subjects responded to it. Soon after, Clark & Teasdale (1985) authored an influential study that evaluated the effect of mood on word recall, and their induction procedure has been replicated numerous times since (i.e. Martin, 1990; Segal, Gemar, & Williams, 1999; Van der Does, 2002). A few years later, Pignatiello, Camp, & Rasar (1986) used a slightly different MMI procedure. Instead of using Clark & Teasdale's 1985 single seven-minute excerpts to induce mood, they applied a technique based on the iso principle, which is a clinical technique credited to I.M. Altschuler in 1948, and is comprised of introducing music to match a person's current mood and then gradually shifting the music qualities toward a desired mood state. Three twenty minute recordings were created that all began with the

same musical excerpt, but either became incrementally happy, sad, or remained neutral. This technique has also been replicated successfully in many other studies (i.e. Martin, 1990; Västfjäll, 2001), and more recently Mongrain (2007) used this same method with modifications to the music selections to make it more age relevant for young adults. A benefit to this technique over the one used by Clark & Teasdale (1985), is that a stronger change in mood might be induced (Västfjäll, 2001). This may not only be desired during experimental protocols, but also when used in clinical practice.

While these two MMI protocols have been replicated numerous times, the published variations in design that use different music or directives far outweigh them. This is in part due to disagreement over a best method, and although a standardized induction might benefit future research, one is not yet available. An example of this division is the considerable aggregate of researchers who have scrutinized whether demand effects bias subjects' responses during mood inductions. Martin (1990) and Pignatiello et al. (1989) argue that incremental music is independent of demand characteristics, because their subjects were unable to label the intended mood post treatment. Similarly, others have concluded that *any* music induction has low bias when compared to other induction modalities (i.e. Västfjäll 2001; Nyklicek, 1997); although Västfjäll goes on to write that a stronger modulation effect is produced with the addition of a demand. Contrasting observations include those made by Kenealy (1988), who noted that demand characteristics had no significant effect on induction of mood, and Gabrielsson (2001), who suggested that any mood induction procedure is unlikely to succeed without a concurrent demand.

One other contested element between various MMI investigations has to do with whether or not to use subject-preferred or experimenter-chosen music. A variety of outcomes have been reported, with several authors reporting no significant difference (i.e. Rider, 1985; Siedliecki & Good 2006; Thaut & Davis, 1993), others arguing that preferred music works better due to increased feelings of control and emotional engagement (i.e. Mitchell & MacDonald 2006; Perlini & Viita, 1996), and still others suggesting that using preferred music may bias post-treatment responses (Västfjäll, 2001). In one case, Rider (1985) reported that subjects least preferred music was also the most effective in reducing pain.

Looking beyond the method, it is interesting to consider the various clinical applications of MMI. Since the early 1980's, it has been used most commonly in conjunction with cognitive behavioral methods. It was at that time when Bower, (1981) first proposed the associative network theory of emotion and memory, which advanced the idea of mood-state dependent memory. Clark & Teasdale (1985) were first to publish an investigation into the effect of MMI on word recall, using the protocol described earlier on, and found that MMI successfully induced mood in both men and women. The purpose of their study was to research whether subjects recalled pleasant and unpleasant descriptive words better while in respective moods. Though only women showed a significant correlation between mood and recall, it was discovered upon further investigation that the gender difference was due to the fact that women more frequently used the chosen words in everyday conversation. Thaut (1989) and de l'Etoile (2002) also examined this effect, and suggested that since MMI has the ability to help individuals access positive memories, it might be used clinically in combination with cognitive

behavioral therapy to help break the cycle of depression. Mongraine (2007) even found that musical mood induction alone, without CBT, led to more adaptive beliefs on the results of a dysfunctional attitude scale. For the purpose of the current investigation, it is important to acknowledge that MMI can be used in conjunction with CBT because the latter is so often used to treat pain (International Association for the Study of Pain, 2008; Turk et al. 2008).

Pain and mood responses to music structure

A few researchers have attempted to dissect the music used in inductions in order to ascertain how internal structures determine the outcome. Of interest, an individual can typically discriminate a “happy” versus “sad” melody in 0.5 seconds (Bigand, Filipic, Lalitte, 2005; Peretz, Gagnon, & Bouchard, 1998). Whether this immediate recognition is due to one or a combination of several structural aspects such as mode, tempo, or rhythm (eg. sustained verses staccato), has not been ascertained, but it does appear to be effortless, immediate, and possibly unacquired. Dalla Bella, (2001) suggests that tempo is an innate way to discriminate the valence of music since, when presented with slow and fast excerpts, some one-year-old children can already distinguish the two, and most five-year-old children can. In this same study, only older participants (ages six through eight) were able to distinguish valence when presented with excerpts in major or minor melodies, suggesting that mode is not innate, but learned. Dalla Bella and many others (i.e. Nyklicekm 1997; Khalfa, Schon, Anton, & Liegeois-Chauvel, 2005) have concluded that tempo is the most important musical structure in determining and processing emotion. These authors are careful not to discount mode as an active agent however, and

Khalifa, Roy, Rainville, Dalla Bella, & Peretz, (2008) concluded that discrimination between “happy” and “sad” requires both pitch and tempo.

A number of investigations (i.e. Gomez, 2007; Kenntner-Mabiala, Gorges, Alpers, Lehmann, & Pauli 2007; Nyklicek, 1997) have described emotion in music as being dualistic, specifically meaning that mode determines valence, and tempo determines arousal. This is based on an understanding of emotion as a combination of the two dimensions. When one assumes that tempo is the most influential element of musical emotion and that its effect on arousal is most significant, there is a basis to measure physiological changes that occur in response to MMI. A few of the aforementioned authors have done this, and subsequently observed that rhythmic entrainment appeared to be responsible for changes in mood. Khalifa et al. (2008) compared subjects' ability to entrain to various musical excerpts using respiratory rate as a measure, and found that an isolated simple rhythm worked better than one embedded in music. This may or may not be of consequence to the music used during the experiment. Thaut (2005) notes that music played and composed with the intention to entrain can enhance rhythmic perception.

It appears that tempo likely does effect arousal, and subsequently emotion. This raises a question of whether tempo's effect on arousal may produce any other psychological changes, such as perception of pain. Similar studies have examined the effect of arousal on pain perception, resulting in inconsistent outcomes. Kenntner-Mabiala et al. (2007) and Schön, Dahme, & Leupoldt (2008) both observed tempo to be the most instrumental musical structure, and reported that increased arousal correlated with increased pain perception. Schön et al. (2008), who induced a negative mood,

observed a decrease in heart rate simultaneous with participants' increased arousal. In contrast, Rhudy et al., (2006) and Zillmann et al., (1996) both concluded that heightened arousal leads to a decrease in pain perception and sensitivity.

Considering the noted discrepancies that exist between research outcomes, depending solely on the internal structures of music when designing a MMI procedure would be short sighted. Mitchel & MacDonald (2006) suggest that extra-musical variables are more important than internal structure. They experimented with subject-preferred music, which was noted after examination to share few structural commonalities, and witnessed personal preference of music as being most influential in decreasing pain perception. Many other researchers (i.e. Peretz et al., 1998; Perlini & Viita, 1996; Thaut, 1989) have suggested that external factors play a large role in determining emotional response to music. Magill-Levreault (1993) states the reasons for music's ability to reduce pain as being a combination of affective, cognitive, and sensory processes, which ties in to the neuromatrix theory.

Physiology of emotional responses to music

Based on the incongruity between studies, using physiological markers to measure mood or pain responses do not appear to be a reliable method at this time. Etzel, Johnsen, Dickerson, Tranel, and Adolphs (2006) admit that the idea of unique physiological patterns for various emotions is controversial, and go on to say that the problem of separation between physiological response to tempo (entrainment), or response to musically induced mood may be impossible to solve. Still, many researchers have continued to focus on these responses, maybe because the thought of working with

more objective data is so attractive. Krumhansl (1997) posed that MMI responses may be measurable physiologically and argues that these changes increase over time (through longer inductions), but also acknowledged the results as being elusive. Meyer (as cited in Krumhansl, 1997, p. 338), an influential music theorist, rejected the “importance of physiological changes in response to music” in his well known 1956 book on emotion in music, which demonstrates that this question has been debated for well over fifty years.

Researchers who continue to investigate physiological responses as they relate to emotion in music often measure autonomic reactions. Nyklicek, (1997) and Etzel, et al., (2006) reported that the main physiological difference between positive and negative musically induced emotional states is respiratory rate, with higher arousal correlating with more positive feelings. Even these authors assert that physiological variables are poor discriminators, but add that the validity is better when describing arousal in comparison to valence.

Looking at neural networks and mechanisms, several researchers support the valence lateralization model that Whittle et al. (2006) adhered to and others, such as Khalifa, (2005) do not. Blood, Zatorre, Bermudez, & Evans, (1999) and Peretz et al., (1998) write that the structures involved with musical valence appraisal are distinct from ones involved with other musical appraisals (i.e. arousal, non-emotional judgements), but are the same as those used to determine valence of non-music stimuli. They suggest the right hemisphere is favored in the emotional processing of music, also supported by Outley (as cited in Gomez, 2007, p.383), who adds that the left hemisphere is more involved with musical mood induction. Brown (2004) writes that the left limbic and paralimbic areas are activated when processing positive emotions in music.

Dellacherie (2009) describes a circuit for emotional judgment of music citing first the orbitofrontal cortex, which projects to the amygdala, which then projects to the ACC. This is interesting because all three structures also have primary associations with pain inhibition. Similar parallels exist with a circuit proposed by Brown (2004) to process emotion in music, starting with the superior temporal pole, which projects to the orbital prefrontal cortex via the uncinate fasciculus. The temporal pole also projects to the subcallosal cingulate cortex, amygdala, and hippocampus. Brown also observed spontaneous limbic and paralimbic system activation in the ACC, retrosplenial cortex, hippocampus, anterior insula, nucleus accumbens, and subcallosal cingulate gyrus when subjects listened to unfamiliar but well liked music. Also congruent with Dellacherie, Khalifa et al., (2005) cites orbitofrontal and cingulate cortices in the emotional processing of music in addition to other stimuli. Because there is evidence that emotion and pain processing share mechanisms in the brain, a more certain physiological explanation of the effect of one on the other might eventually be in store.

Summary

There is a tremendous amount of elusiveness in the current understanding of pain modulation, emotional processing, and interactions between the two. Multiple theories from different disciplines overlap as well as contradict each other. Likewise, contrasting outcomes are common between MMI experiments, although a lack of replication or similarities in design might be responsible. This demonstrates a need for more replication and consistency of MMI design, as is echoed by many authors (i.e. de l'Etiole, 2002;

Mitchell & MacDonald, 2006). The few clear understandings that unite music processing, pain, and affect induction include the following: positive affect can decrease pain perception; MMI can successfully induce positive affect; and affective pain perception and modulation appear to share neural structures with musically induced positive affect.

Many people with cancer suffer from poor pain management, and both the under-treatment of pain and the use of opioids have been identified as risks for chronic pain, as well as decreased psychological, social, and physical function. Partially due to this, multidisciplinary treatment approaches are recommended by most experts. It appears likely that both internal and external musical attributes are involved in decreasing pain, and although the exact mechanisms for its success are vague, its use is clearly effective. Pain research within the last few years has used advanced methodologies and also provided more promising outcomes. As a pilot study, this investigation had limitations that diminished the probability of a significant outcome. However, the insights gained from recent clarifications should allow future research to be more convincing.

CHAPTER III: METHODOLOGY

Participants

Participants (N=2) were recruited from a local hospital and were receiving radiation, chemotherapy, or follow-up treatment with reports of experiencing pain or discomfort related to cancer. Participants were 51 and 60 years old, were female, and had early stage breast cancer. One participant had a history of fibromyalgia, but stated that her pain had always been kept under control with medication. The other patient had no history of chronic pain. To enroll, participants could have been of any ethnicity; must have had no significant hearing deficits or cognitive disabilities; rated their current pain, on a scale of 0-10, as a 2-6; had not taken analgesics within an hour before the experiment began; and described their pain as being sustained as opposed to getting noticeably better or worse. Each person signed an informed consent form approved by the C.S.U. and Banner Health institutional research review boards.

Materials

Materials included recorded music, a portable digital audio player, headphones, the short form of the Positive and Negative Affect Schedule (PANAS), which has been used in many pain and affect studies with high validity and reliability (i.e. Connelly et al., 2007; Strand et al., 2006; Zautra et al., 2005) and SF-McGill Pain Questionnaire, which

is designed to measure affective *and* sensory dimensions. The SF-McGill Pain Questionnaire consists of three sections: the first and main part contains 15 pain descriptors to be rated 0-3 in intensity, the second part is a 100mm Visual Analogue Scale (VAS), and the third part asks the participant to rate their overall discomfort on a scale of 0-4. This tool has also been shown to be a reliable measure (Dudgeon, 1993). The chosen musical mood induction recording was developed by Pignatiello et al., (1986) and intended to induce a positive mood state.

Procedure

The experiment took place in a quiet, low-lighted exam room after the participants received radiation or chemotherapy treatment. Participants sat in a chair and a small foot stool was available for use. Upon commencement, participants signed a consent form and complete the PANAS and SF McGill Pain Questionnaire. A verbal directive was given for participants to try to get into a positive mood state, after which they listened to a 20 minute recording of increasingly elating music (the same used by Pignatiello et al., 1986), made available on an mp3 player. Music preferences of participants were not examined for two reasons. First, the literature appears to better support the use of progressively elative music, and this would have been difficult using participant-chosen music. Second, by using the recording by Pignatiello et al (1986), higher reliability and external validity was thought to have been achieved since the recording is somewhat standardized. The choice to use this recording was also under the assumption that an increase in arousal would elicit beneficial results.

The investigator left the room for twenty minutes while the recording played. Participants listened to the music through headphones, and the mp3 player had adjustable volume which was demonstrated by the investigator. After twenty minutes, the investigator reentered the room and the participants once again filled out the assessments. It was stressed that upon repeating the subjective measures, participants should rate their mood and pain states honestly.

Design/Analysis

A single-system design was used with 1 independent variable and two phases- pretest and post test. Change scores and one-sided paired t-tests were conducted to analyze the results of the pain and affect scores.

Results

SF-McGill Pain Questionnaire

Change scores and one-sided paired t-tests were conducted to identify significant changes between pretest and posttest pain scores. No significant changes were found for any of the pain scores. Data for mean change score, standard deviation, p-value, and power for each score are as follows: Sensory Pain Rating Index (S-PRI): M = .275, SD = .134, p-val = .106, power = .349; Total Pain Rating Index (T-PRI): M = .24, SD = .99, p-val = .09, power = .408; Visual Analog Scale (VAS): M = 2.35, SD = 1.909, p-val = 1.66, power = .217. Data for the Affective Pain Rating Index (A-PRI) and the Present Pain Intensity scale (PPI) were not calculable due to the standard deviation being zero.

Descriptive data for these scores and all others are represented in Figures 1-5. Change scores for all parts of the McGill Questionnaire are combined in a bar graph in figure 6. All pain data is summarized in Figure 9.

Positive and Negative Affect Schedule (PANAS)

Change scores and one-sided paired t-tests were conducted to identify significant changes between pretest and posttest mood scores. No significant changes were found for any of the mood scores. Data for mean change score, standard deviation, p-value, and power for each score are as follows: Positive Affect: $M = .04$, $SD = .141$, $p\text{-val} = .379$, $\text{power} = .079$; Negative Affect: $M = .14$, $SD = .113$, $p\text{-val} = .165$, $\text{power} = .218$.

Descriptive data for these scores are represented in Figures 7-8. All pain data is summarized in Figure 10.

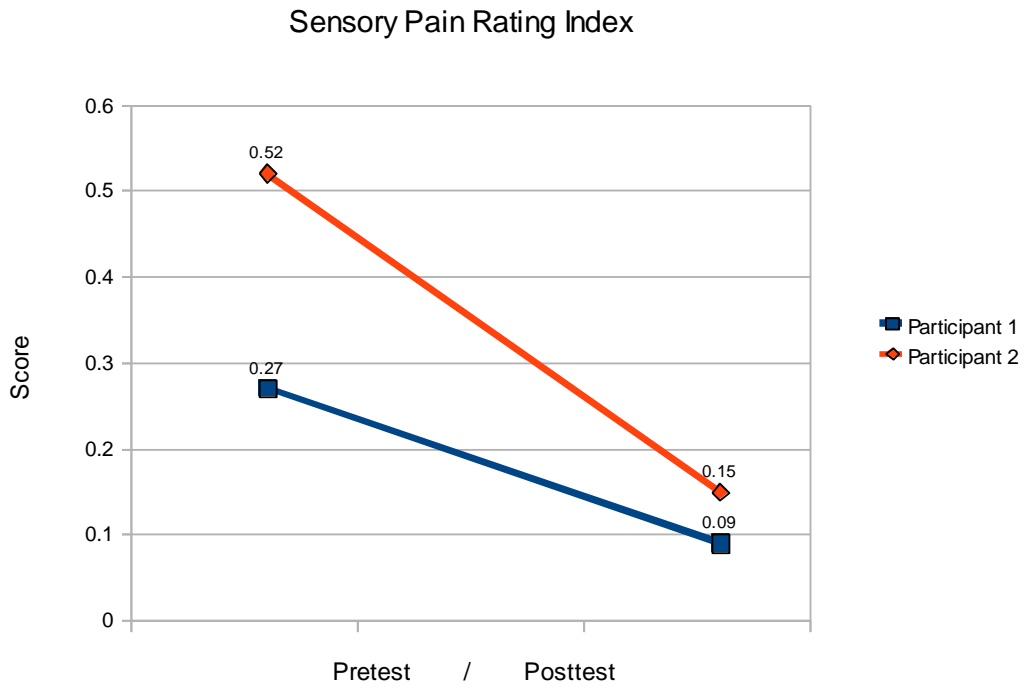


Figure 1.

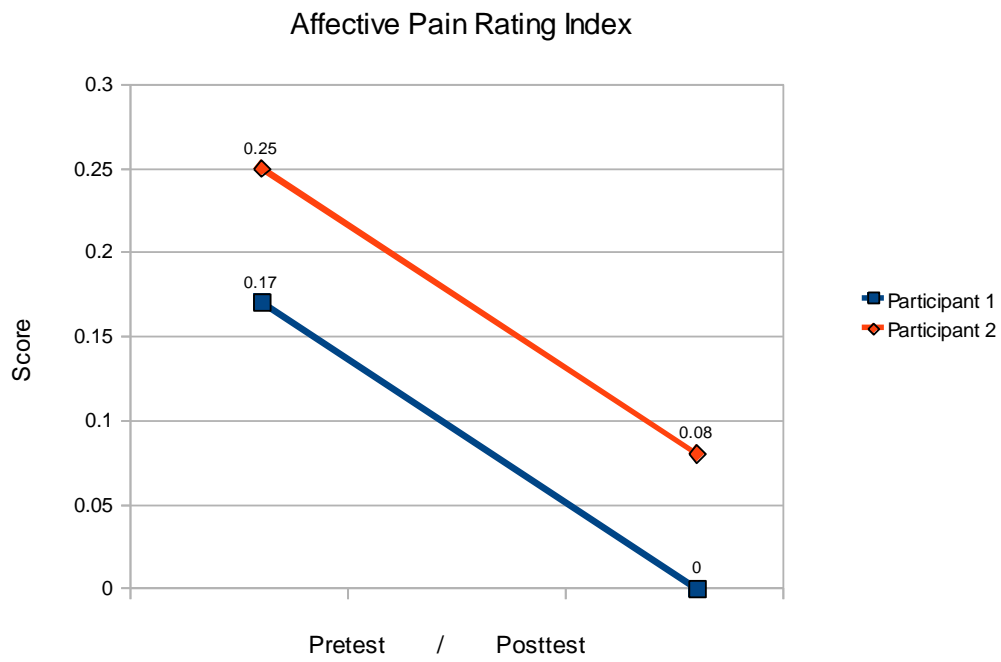


Figure 2.

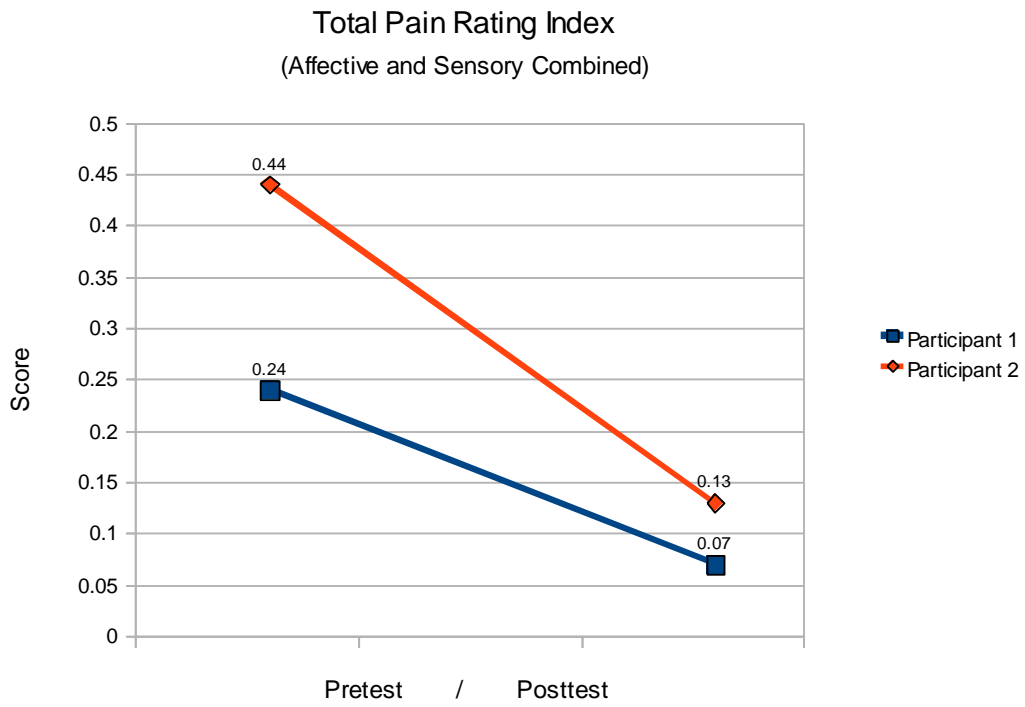


Figure 3.

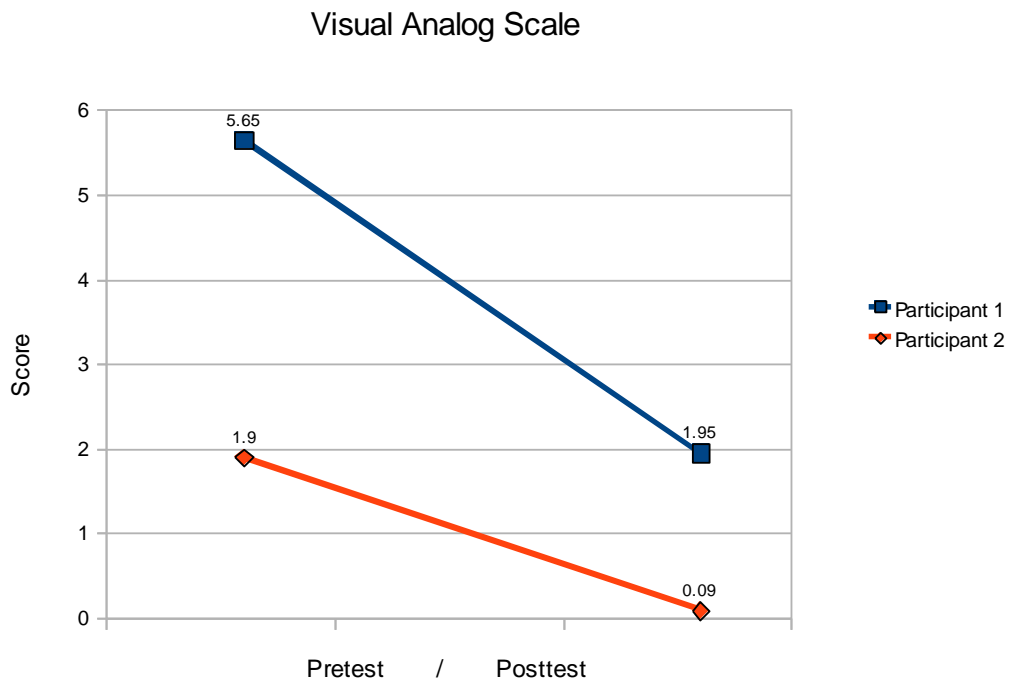


Figure 4.

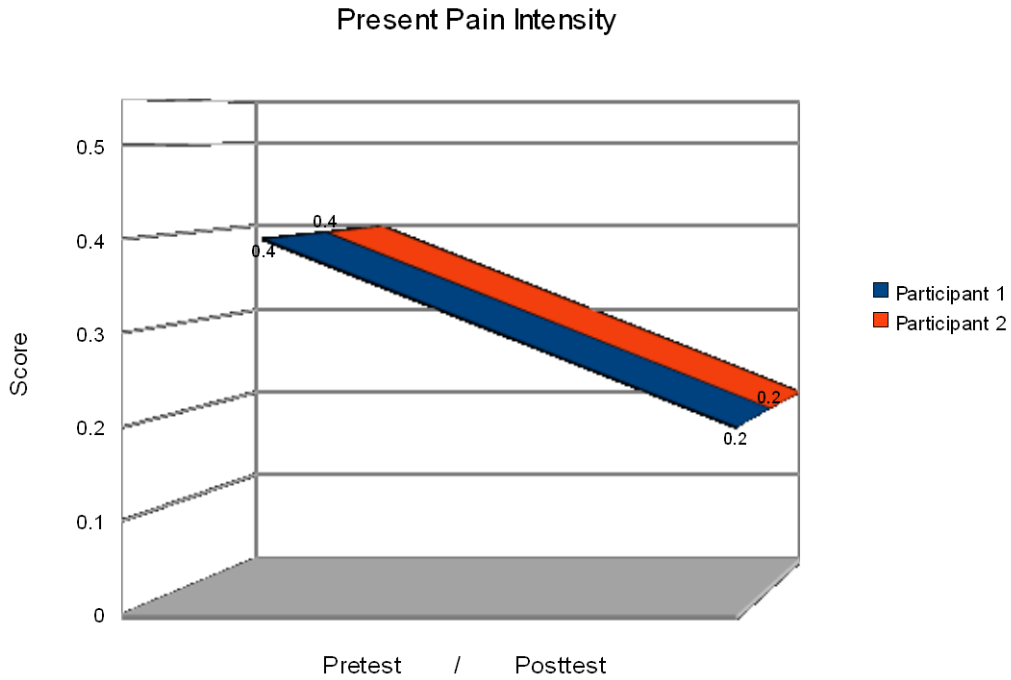


Figure 5.

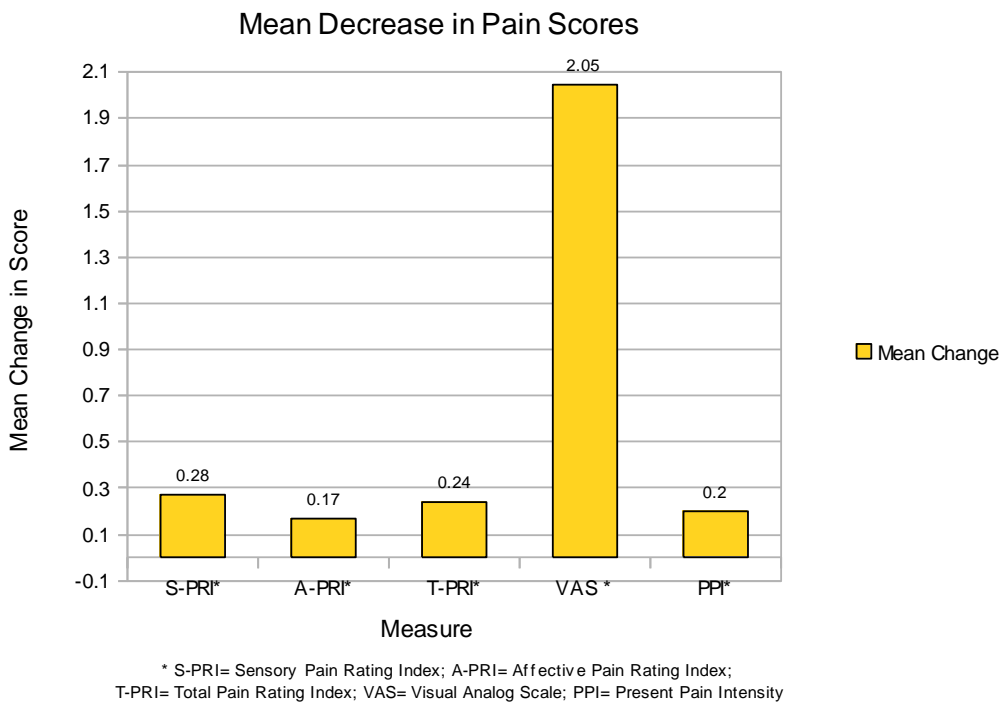


Figure 6.

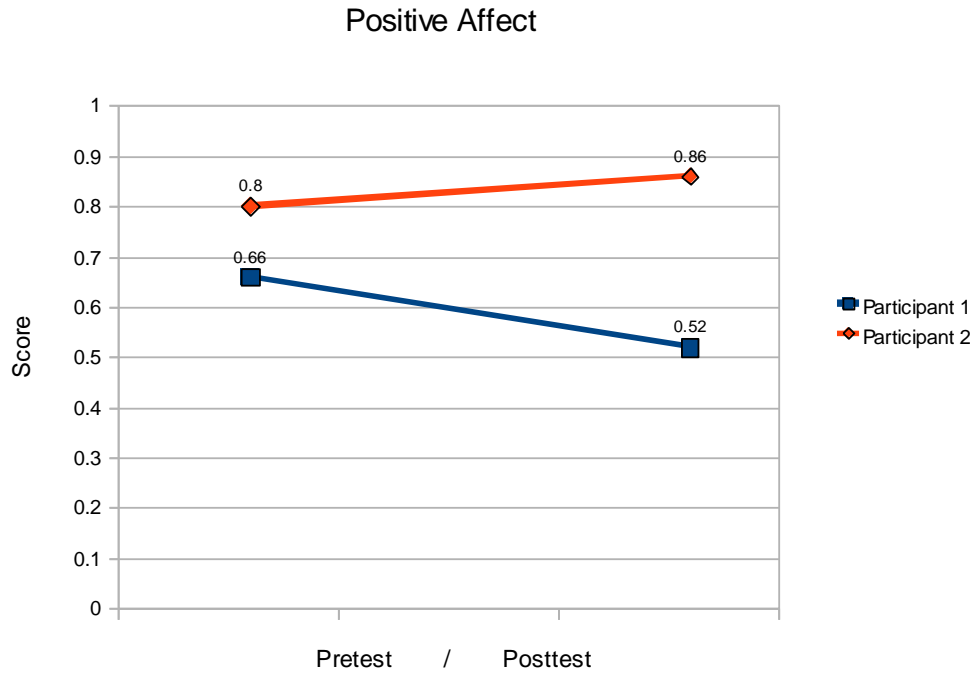


Figure 7.

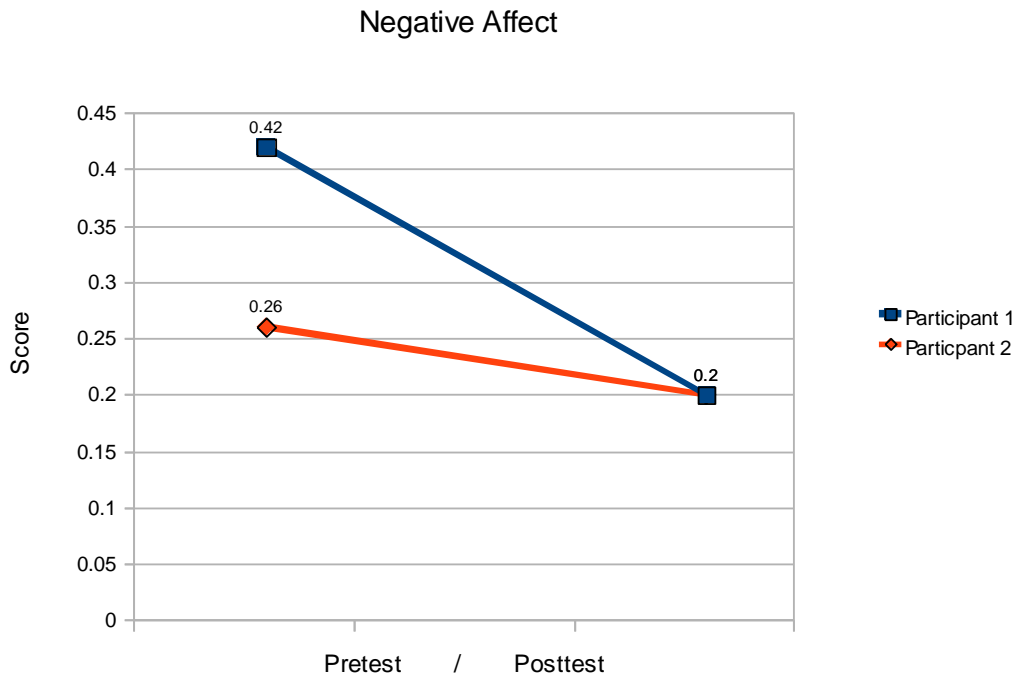


Figure 8.

SF-McGill Pain Questionnaire Pre and Post Scores

	Pre S-PRI	Pre A-PRI	Pre T-PRI	Pre VAS	Pre PPI	Post S-PRI	Post A-PRI	Post T-PRI	Post VAS	Post PPI
Participant 1	0.27	0.17	0.24	5.65	0.4	0.09	0	0.07	1.95	0.2
Participant 2	0.52	0.25	0.44	1.9	0.4	0.15	0.08	0.13	0.9	0.2

* S-PRI= Sensory Pain Rating Index; A-PRI= Affective Pain Rating Index;
T-PRI= Total Pain Rating Index; VAS= Visual Analog Scale; PPI= Present Pain Intensity

Figure 9.

Positive and Negative Affect Schedule Pre and Post Scores

	Pre PA	Pre NA	Post PA	Post NA
Participant 1	0.66	0.42	0.52	0.2
Participant 2	0.8	0.26	0.86	0.2

*PA=Positive Affect; NA=Negative Affect

Figure 10.

CHAPTER IV: DISCUSSION

The discussion that follows is based on the descriptive statistics available from the two participants, and it needs to be stressed that no assumptions about trends, variance, or other aspects of the results can be realistically made. Nonetheless, there are things to consider based on the available data. For instance, all pain scores decreased, which could possibly show the beginning of a trend. Similarly, the data show an overall decrease in negative affect. This effect does not appear to be as obvious with positive affect, which was hypothesized to increase after mood induction. Calculation of correlation was not statistically possible due to sample size but the beginnings of a positive correlation trend might exist between the decrease in negative affect and the decrease in pain. As is, the data fail to reject the null hypothesis.

Another consideration that arises from the results is that variability was higher for some scores - substantially so for the VAS scores and to a lesser extent for both PANAS scores. Again, without a larger sample size, it's impossible to tell whether this variability signifies the absence of a trend, but it's interesting because these three measures all differ from the other scores. The VAS is distinct in that it was the only measure used that did not involve checking a number, and instead is more of a visual task. However, none of the reviewed studies that incorporated the SF-McGill tool reported such a variance. The PANAS questionnaire is simply different due to its measurement of a different event - mood as opposed to pain. Both the VAS and PANAS are tools that are used independently

of the McGill Pain Questionnaire.

Limitations

An obvious limitation of this study was the low enrollment, which was due to two main factors. First, the primary investigator did not have direct access to potential participants at the hospital. People were initially contacted by staff, who then notified the investigator if someone expressed interest in learning more about the study. It's possible that a more direct recruitment technique would have resulted in a higher number of eligible candidates being contacted.

Second, eligible candidates who agreed to take part in the study become ineligible during the few days between verbally consenting and the scheduled trial. Possibly due to the nature of cancer pain, patients' pain either subsided below “2” (on a scale of 0-10) upon receiving a new pain medication, or increased above “6” when not managed well. Maybe because patients do not often experience pain early on from radiation treatment, there was a short window of time between when their pain level made them eligible to participate, and when their treatment was completed. With an overall short time frame to contact and enroll participants, successfully scheduling a trial while their described pain remained between 2 and 6 proved difficult. Given this dilemma, it's possible that the choice of population (outpatient cancer patients) itself was a limitation.

Another limitation involved the recording used for the MMI induction, which has not been updated since its inception in 1986. Some of the audio features of the excerpts could be improved with the use of more modern technology, using source files that are electronic and cleaning up some of the abrupt transitions between excerpts. Today's

listeners are not accustomed to hearing white noise, and it's possible this decreased the effectiveness of the induction.

In addition to improving the MMI recording, it would also be interesting to add a second music group that allowed participants to select the music they listened to, and compare the results between music groups. The fact that music preferences of participants was not examined and all participants listened to the same recording is considered a limitation of the current study, since it is known that responses to music are individualized and based on preferences. In a clinical study using music therapy as a variable, MMI and music preference could easily be combined. It would be interesting to examine whether this would create a more effective mood induction, and such a study might also eventually lead to a more standardized MMI technique.

Lastly, the single systems design was limiting because it did not allow for comparisons between groups. Even if the sample size had been larger, and/or if the result proved to be statistically significant, the fact that no control or alternative treatment group existed would have made the internal validity weak, and it would be impossible to conclude anything beyond whether or not MMI impacts pain perception. As a pilot study the design was sufficient, but any future studies should include multiple treatment groups. By comparing MMI with other similar treatments, one could gain some insight into whether music is any more effective than other alternative modalities. Depending on the outcome, this information could then be built on in the clinical context, testing the effectiveness of a MMI over time. Conversely if MMI was not supported as a significantly more effective approach, and one could achieve an equitable effect by watching television or conversing with a friend, it would give music therapists cause to

reassess the use of that technique to treat pain.

While not considered a limitation, working with multiple internal review boards was an unforeseen difficulty. The protocol process at the hospital, being part of a large system, demands considerable time and attention. This paired with having to coordinate between the IRBs at Colorado State University and the hospital took significant amounts of time, especially when there were differences of opinion between the two boards.

Conclusions

A future study with a larger sample size might use a randomized control trial design with 4 independent variables: pre-recorded MMI, preferred music, audiobooks, or silence. Then, a one-way analysis of covariance (ANCOVA) could be implemented, with the pre-test affect and pain scores as covariates, and the post-treatment measurements as dependent variables will be performed to compare changes from pre- to post-test between groups. One-way ANCOVA could also be used to analyze the effect of mood change on pain, with change in mood as the covariate, and change in pain as the dependent variable. In addition, separate one-way analysis of variance (ANOVA) could be performed for examining average pre and post scores in order to test for variability within groups as well as on the correlation.

It is recommended that the MMI recording used for the current study be revised to provide improved sound quality. An alternative would be to pre-record several different MMI recordings of various genres that participants could then choose between. This would still allow for a non-MMI music group to be added as a separate variable.

An ideal study would also target a more specific population, possibly with similar stages of disease, ages, or gender. Somewhat homogenous pain intensity within the sample might also be desired as well. The decision to conduct this study with oncology patients was initially made due to its possible relevance to patients in hospice care. This might not be ideal though, as the choice of target population for the current study likely affected the sample size. Multiple research candidates who initially agreed to take part in this study became ineligible during the few days between initial contact with the investigator and the scheduled experiment. This might be due to the nature of cancer pain and current treatment options, as candidates either began a new medication regime that decreased pain below eligible levels or their pain increased to a level that was too high.

To summarize, no significant results were generated from this study, and the data fail to reject the null hypothesis that positive musical mood induction would produce no differences in the pain perception of adult oncology patients. In addition, little is understood about the pain experience at this time, and researchers continue to present conflicting conclusions. The topic of pain management is important however, because current pharmaceutical treatments clearly carry high risks and by themselves don't often provide adequate analgesic effects. Knowing that under-treated pain can lead to chronic pain, and that a high percentage of cancer survivors experience chronic pain, only adds to the importance of research to find better treatments for this population. Hospice patients who are not expected to survive also need better ways to manage pain so that they are more comfortable, and so that they can avoid some of the medication side effects. Music therapy, along with complementary medicine of all sorts appears to make a difference by

lowering risks and providing increased palliative effects. It's up to future research to explain why these methods work, and therapists will be able to use that understanding to provide more effective treatments.

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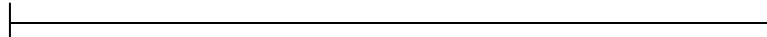
APPENDIX I

**SHORT-FORM MCGILL PAIN QUESTIONNAIRE
RONALD MELZACK**

The words below describe average pain. Place a check mark (X) in the column that represents the degree to which you feel that type of pain.

	<u>NONE</u>	<u>MILD</u>	<u>MODERATE</u>	<u>SEVERE</u>
THROBBING	0 _____	1 _____	2 _____	3 _____
SHOOTING	0 _____	1 _____	2 _____	3 _____
STABBING	0 _____	1 _____	2 _____	3 _____
SHARP	0 _____	1 _____	2 _____	3 _____
CRAMPING	0 _____	1 _____	2 _____	3 _____
GNAWING	0 _____	1 _____	2 _____	3 _____
HOT-BURNING	0 _____	1 _____	2 _____	3 _____
ACHING	0 _____	1 _____	2 _____	3 _____
HEAVY	0 _____	1 _____	2 _____	3 _____
TENDER	0 _____	1 _____	2 _____	3 _____
SPLITTING	0 _____	1 _____	2 _____	3 _____
TIRING-EXHAUSTING	0 _____	1 _____	2 _____	3 _____
SICKENING	0 _____	1 _____	2 _____	3 _____
FEARFUL	0 _____	1 _____	2 _____	3 _____
PUNISHING-CRUEL	0 _____	1 _____	2 _____	3 _____

NO
PAIN



WORST
POSSIBLE
PAIN

Overall intensity of total pain experience. Place a check mark (X) in the appropriate column:

- 0 NO PAIN _____
- 1 MILD _____
- 2 DISCOMFORTING _____
- 3 HORRIBLE _____
- 4 EXCRUCIATING _____

APPENDIX II

The PANAS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel this way right now, that is, at the present moment. Use the following scale to record your answers.

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely

_____ interested

_____ irritable

_____ distressed

_____ alert

_____ excited

_____ ashamed

_____ upset

_____ inspired

_____ strong

_____ nervous

_____ guilty

_____ determined

_____ scared

_____ attentive

_____ hostile

_____ jittery

_____ enthusiastic

_____ active

_____ proud

_____ afraid

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APPENDIX III

Data Summary Spreadsheet

Participant Number	
Treatment Condition	
Gender	
Age	
Pre-Treatment Pain Level (0-10)	
History of Chronic Pain (Y/N)	
Year of Diagnosis	
Cancer Type	
Cancer Stage	
Medications	
Pre-PANAS Score	
Post-PANAS Score	
Pre-McGill Score	
Post-McGill Score	