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HOW MUSIC THERAPY EFFECTS THE TRAUMATIZED BRAIN: NEUROREHABILITATION FOR POSTTRAUMATIC STRESS DISORDER THROUGH MUSIC THERAPY

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Abstract

This review discusses the neurological components of posttraumatic stress disorder (PTSD) and how both structures and processes in the brain are altered in individuals with the disorder, specifically the neural network that includes the prefrontal cortex, the hippocampus, and the amygdala. This impacts awareness and responsiveness to stimuli. After examining these aspects, invasive and non-invasive treatment approaches are examined, with a specific emphasis on the treatment approach of music therapy. Musical stimuli are processed in many areas of the brain, so it has therapeutic potential for modulating neurological changes. Music therapy applies music clinically to address a variety of goals for clients with PTSD, including emotional, social, and cognitive objectives. Music therapists with an understanding of neuroplasticity and neurological impairments associated with PTSD can approach their practice with more specific goals and strategies for helping clients recover.

How Music Therapy Affects the Traumatized Brain:

Neurorehabilitation for Posttraumatic Stress Disorder Through Music Therapy

According to the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), posttraumatic stress disorder (PTSD) affects approximately 3.5% of adults in the United States each year (APA, 2013). Rates of the disorder are particularly high among certain populations who are exposed to traumatic events through their occupation, such as active military personnel and veterans, police, firefighters, and medical personnel. PTSD may occur in individuals who have been exposed to a traumatic event and experience significant distress or impairment due to the event. The diagnostic criteria for PTSD include exposure to a traumatic event through direct experience or witnessing, intrusive symptoms such as memories or dreams, altered reactivity towards stimuli associated with the event, avoidance behaviors of stimuli that are associated with the trauma, negative changes to cognition and mood, and clinically significant distress or impairment in areas of functioning (APA, 2013). In previous editions of the DSM, emotional reactions such as fear, helplessness, and horror were part of the diagnostic criteria, but it has become more apparent that individuals may present symptoms of PTSD very differently. While some people may present with fear responses to stimuli and re-experiencing, others present with dysphoric mood states or disassociation.

PTSD has an extreme psychological impact on individuals. Psychological distress occurs in response to cues that trigger these memories. Triggers can be actual events, or they can be physical sensations similar to the somatic sensations experienced with the trauma. PTSD alters cognition and behavior, so that many individuals lack positive emotions, display hypervigilance to stimuli, have a consistent negative outlook, express increased anger and aggression, or indulge

in more risk-taking behaviors. These alterations in cognition and behavior due to a traumatic event, as well as intrusive symptoms, suggest that changes have occurred in the brain to cause the disorder and lead to clinically significant distress or impairment (Boccia et al, 2016; Giustino & Maren, 2015).

Neuroplasticity is a term to describe how the brain can change and develop its structures and functions, from the synaptic level to remapping of entire areas (Nash, Galatzer-Levy, Krystal, Duman, & Neumeister, 2014; Stegemöller, 2014). Plasticity occurs in response to traumatic events and environmental conditions, but it can also occur to reverse adverse effects of a disorder. Treatments and therapies can provide the necessary conditions for neuroplastic changes to occur that help an individual recover from PTSD. Understanding the plasticity of the brain in persons with PTSD and neurological components of the disorder is crucial to gaining thorough knowledge about its effects and to providing future treatment.

To recognize how the brain is altered by PTSD, we can examine key structures in the brain that affect how memories are stored and how an individual experiences stimuli associated with emotion. These structures form a network in the brain in which messages about stimuli are sent to each other, and these messages regulate and influence an individual's responses (Flor & Nees, 2014). The circuitries are continually reinforced by habitual responses and more experiences of triggers. This network is altered in individuals experiencing PTSD. The brain is hyperactive in response to negative stimuli, which can result in impairment of day-to-day functioning. Fear conditioning occurs when an aversive stimulus becomes associated with a neutral stimulus, and the neutral stimulus becomes an elicitor of a fear response. Fear conditioning develops faster and more easily for people with PTSD (Flor & Nees, 2014). This suggests that there are specific aspects of the neural network that have been altered.

Key structures of this network in the brain include the medial prefrontal cortex (mPFC), the amygdala, and the hippocampus. Alterations to these areas of the brain are found across studies examining patients with PTSD (Boccia et al, 2016; Flor & Nees, 2014). These studies help us understand the behaviors and cognitive patterns of individuals with PTSD from both a neurological perspective and a clinical perspective. Other areas of the brain, such as the angulate cingulate cortex (ACC) and the dorsolateral prefrontal cortex, are also involved in this complex network, but our examination will specifically focus on the structures mentioned above. This is due to their prominence in the literature in examining neural alterations found in PTSD and their role in modulating responsiveness to stimuli.

The mPFC, the hippocampus, and the amygdala each have some specific functions that they control and modulate, which can be impaired due to PTSD. The mPFC is located within the frontal lobe of the brain. The frontal lobe is responsible for aspects of executive functioning, such as planning and personality expression. The prefrontal cortex is associated with decision-making and memory processes that are unconscious. Specifically, the mPFC is also involved in internal emotional processing and in regulating parts of the brain involved in emotion, like the amygdala, when information is being processed and responded to (Giustino & Maren, 2015; Legge, 2015). The mPFC plays an important role as individuals with PTSD experience fear conditioning and must regulate their responses.

The hippocampus is an inner structure of the brain that is chiefly responsible for memories. Long-term memories are stored in the hippocampus, and fear memories or memories with high emotional salience can have a particularly pronounced impact in an individual with PTSD. The hippocampus also alters stress responses through the hypothalamic-pituitary-adrenal

(HPA) axis. Information is sent to the pituitary and adrenal glands from the hippocampus to regulate the body's hormonal stress response (McNerney et al., 2018).

The last structure of this group is the amygdala. The amygdala is located in the temporal lobes, and it is part of the limbic system (Legge, 2014). The role of the amygdala is to regulate emotions and fundamental drives, such as eating and sex. The amygdala activates an individual's flight, fight, or freeze response to fear stimulants, and information about a stimulus is received from the sensory cortices of the brain such as the occipital lobe, which is responsible for vision. The amygdala is key to determining arousal levels based on stimuli. The amygdala is also involved in memory storage when memories are associated with strong negative emotions (Legge, 2015).

With this foundation of significant structures of the key neural network of PTSD, we can examine deviations found among persons with the disorder. Not all studies report the same findings about changes in the brain, due to the many variations that exist in individual participants. Differences in biological vulnerabilities, past experiences, developmental stages, as well as the type and duration of trauma, influence neuroplastic changes (Boccia et al., 2016). Studies also have limitations due to neuroimaging techniques that are still developing. This is true of studies on alterations in the mPFC. Boccia et al. (2016) reported hyperactivation of the mPFC, which may be linked to changes in emotional processing and autonomic activity for participants with PTSD. These results were found from a meta-analysis of 55 studies using fMRI scans to study neural processes of PTSD and observe commonalities between the studies. Xiong et al. (2013) conducted a study examining the emotional regulation of 20 subjects with PTSD by analyzing fMRI scans when participants were shown a neutral or negative image and asked to diminish, maintain, or enhance their responses. The PTSD group showed lower mPFC activation

in the enhancement setting, which would suggest that the mPFC becomes less active in regulating the amygdala (Giustino & Maren, 2015; Xiong et al., 2013). In addition, the control group of participants who did not have PTSD demonstrated greater ability to down-regulate to negative stimuli, suggesting impairment in neural mechanisms that regulate emotions in participants with PTSD. Kolassa and Elbert (2007) report findings from animal studies on tree shrews in which the mPFC showed atrophy due to stress, which would also suggest a diminishing of emotional processing and regulation.

From these different results, we can see that research on the complex networks of the brain is still developing in neuroscience, and further studies will continue to make clear how the mPFC is impacted. Some individuals show hyperactivation of the mPFC and heightened autonomic activity, while others show reduced activation of the mPFC, which could influence hyperactivity of the amygdala and emotional processing. The mPFC can be altered by trauma and PTSD, but it is not yet completely clear exactly how this occurs.

There is also research on how subregions of the mPFC are impacted by PTSD and how they exhibit neuroplasticity. Giustino and Maren (2015) examined subregions within the mPFC. These subregions are the prelimbic (PL) cortex and the infralimbic (IL) cortex, which were thought to act independently in fear expression and fear suppression, respectively. Both of these regions send information to the amygdala and play a part in the behavioral expression of fear. Giustino and Maren (2015) cite research arguing that these regions may actually overlap in their response to fear stimuli and their output to the amygdala. This would align with findings that the mPFC is hypoactive in individuals with PTSD and reduces its regulation of emotional processing in the amygdala (Xiong et al., 2013). Additionally, Jacques et al. (2019) demonstrated that the prelimbic cortex is activated when memories associated with fear learning are retrieved through

their study of phosphorylated mitogen-activated protein kinase (pMAPK), a molecular marker of neural plasticity. These studies continue to highlight the complexity of the interconnections between neural networks in the brain that are altered in PTSD.

A crucial aspect of the fear learning network is the deficiency that individuals with PTSD demonstrate in extinguishing fear learning and fear memories. In contrast with controls without PTSD, subjects with PTSD demonstrate enhanced fear conditioning, delayed extinction of fear responses, and memory dysfunction (Flor & Nees, 2016). A significant number of studies report a loss of hippocampal volume in participants with PTSD (Boccia et al., 2016; Flor & Nees, 2014; Kolassa & Elbert, 2007; McNerney et al., 2018). However, other studies have difficulty replicating this finding, and these differences may be due to the multitude of factors influencing neural processing, such as the type and duration of trauma, biological vulnerabilities, and comorbidity with other psychological disorders (Nash, Galatzer-Levy, Krystal, Duman, & Neumeister, 2014). Loss of hippocampal volume may be an effect of PTSD in some individuals, but not others.

Regardless of these findings on volume, the hippocampus is still important in the fear circuit because of its role in learning and memory. One way is through context conditioning, in which fear response are put into context for an individual to know when a fear response is necessary and when it is not. (Flor & Nees, 2016). Context conditioning could be impaired or altered by hyperactivation or loss of volume in the hippocampus because long-term memories store those associations and contexts of a memory. Another important role of the hippocampus is its influence on stress responses through the HPA axis. Since the hippocampus activates or inhibits the body's hormonal response to stress, hyperactivation of the hippocampus, observed in participants with PTSD, can enhance HPA axis activity (McNerney et al., 2018). This results in

increased and unnecessary stress due to hormonal activity and excess cortisol, the body's stress hormone. Boccia et al. (2016) found that the hippocampus was consistently hyperactivated among participants in studies among a meta-analysis examining PTSD through fMRI scans. Alterations to the structure and function of the hippocampus are important to consider for individuals with PTSD because the hippocampus affects how memories associated with fear are stored and responded to through hormonal activation.

A crucial structure within the neurocircuitry of PTSD is the amygdala. The amygdala is consistently overactivated in studies on responses to negative stimuli. The amygdala receives information from the sensory cortices and the thalamus to process stimuli (Nash et al., 2014). The amygdala is responsible for the body's fight, flight, or freeze response, so this overactivation explains why individuals have increased hyperawareness of both conditioned and unconditioned stimuli that are unrelated to the trauma experience. Flor and Nees (2014) reported both enlarged amygdala volumes and the enhancement of fear learning.

Subjects with PTSD more readily and quickly learned fear responses to cues and stimulation such as photographs. Sui et al. (2014) found that synaptic changes occurred in the amygdala and the cortical-amygdala pathway through auditory fear conditioning in rats. Jacques et al. (2019), through the analysis of pMAPK expression in marking neuroplasticity, observed activation in the amygdala during the storage of fear memories. As memories moved from recent to remote storage, they were reorganized in subregions of the amygdala. This suggests that salient, emotional memories of both trauma experiences and learned fear responses are even more solidified in memory through the storage in the amygdala. Supporting this finding is Kolassa and Elbert's (2007) analysis describing a building block effect in which increased sensitivity of neural networks continues to develop and build upon itself in response to stimuli in

individuals with PTSD due to hypertrophy of the amygdala. All of these studies discussed suggest that neuroplasticity of the amygdala in PTSD is crucial to understanding the impacts of trauma experiences. These findings also have important implications for providing effective treatment for PTSD, according to many of the researchers (Flor & Nees, 2014; Jacques et al., 2019; Sui et al., 2014).

In sum, in order to increase the efficacy of treating PTSD, researchers are examining the neurological alterations associated with it. These alterations are prominent in the neural network of the mPFC, the hippocampus, and the amygdala. Studies suggest changes in the volume of these structures and their activation in response to fear learning and salient emotional memories. However, an important consideration to be raised about neuroplasticity in PTSD is when these changes occur. Are these differences in the neurological structure and plasticity of subjects with the PTSD the result of trauma and disorder, or are they conditions that lead to the development of PTSD? This answer remains to be determined in further research.

These studies provide a foundation for understanding differences and deficiencies that individuals with PTSD may have so that these areas can be targeted in treatment of the disorder. An understanding of the neurological components of PTSD creates a foundation for clinicians seeking to provide the most effective treatment for clients. With the knowledge that a disorder leads to changes in the structure and function of the brain, treatment can focus on rehabilitating these areas to achieve full recovery and functioning. If forms of treatment, such as psychotherapy, do not address neurorehabilitation, the treatment may not be as successful when these areas continue to be impaired.

PTSD is a complex disorder that can occur at any age, and it is often comorbid with other psychological disorders such as depression, anxiety, or substance use disorder. Many individuals

with PTSD experience relapses of symptoms that have debilitating effects on daily functioning. As we have seen, these characteristic symptoms have a neurological basis that can be addressed through specific treatment focusing on rehabilitating areas of impairment. In some cases, strategies for neuromodulation may be more effective over other forms of treatment. In other cases, neuromodulation strategies can be used in conjunction with psychopharmacology and psychotherapy treatment forms. These types of treatments are enhanced by a foundation in neurocircuitry, because they will be more efficacious in targeting specific impairments.

Currently, there are a few treatment strategies utilizing electric stimulation for inducing neuromodulation. Researchers are examining these procedures in both animal studies and clinical trials. They include deep brain stimulation (DBS), transcranial direct current electrical stimulation (tDCS), and transcranial magnetic stimulation (TMS). Electric stimulation delivers seperate or continuous pulses to parts of the brain. When this stimulation is delivered at a high frequency of 100 Hz or more, it can inactivate cells and cell firing, altering the activity in various structures (Gouveia et al., 2019). This activity includes the retrieval of aversive memories and fear responses such as freezing.

DBS is the most common form of electric stimulation treatment, but it is considered invasive because it requires a surgical procedure to implant electrodes within the brain structure. TMS and tDCS are non-invasive forms of neuromodulation. TMS uses electromagnetic pulses, either at high or low frequencies and in constant or successive administration, to excite or suppress cell bodies. In tDCS, electrodes are applied directly to the head, and a constant direct current is administered. (Gouveia et al., 2019). These techniques can be applied to various structures in the brain that are key to the processes of fear conditioning and extinction, such as the amygdala, the hippocampus, and the mPFC. These forms of treatment have proven difficult

for researchers to study, due to the complexity of PTSD. Animal models often induce more short-term trauma symptoms, and these studies need significant support and replication before they are applied to clinical studies (Gouveia et al., 2019). However, these neuromodulation treatments have already been used in psychiatric settings for treating severe depression or obsessive-compulsive disorder. As neuroscience provides more insight into the neurocircuitry of PTSD, it is likely that these treatments will continue to develop. (Gouveia et al., 2019).

One approach to neuromodulatory treatment is DBS of the amygdala, because studies have found that individuals with PTSD experience hyperactivity of the amygdala. This leads to increased retention of aversive memories and reduced extinction of fear memories. Since DBS can modulate and suppress cell activity, this would suggest that DBS of the amygdala would be an effective approach to treating PTSD. DBS of the right amygdala in one rat model was shown to reduce the retention of fear memories (Sui et al., 2014). This was demonstrated by reduced freezing behaviors in the group of rats who received both auditory fear conditioning and DBS treatment. The researchers wanted to understand the mechanisms behind DBS that make it effective, so they studied the changes in the synapses between neurons that occur during the acquisition and consolidation of fear learning in the amygdala through auditory fear conditioning. These synaptic changes in the pathway between the sensory cortices and the amygdala that occurred due to fear conditioning were reversed after DBS was administered to the right amygdala. Another study by Hashtjini et al. (2017) confirmed the finding that DBS of the right amygdala reduced freezing behaviors in rats after contextual fear conditioning. Since the amygdala receives input from the sensory cortices and modulates the flight-or-fight response, these results help demonstrate how suppression of amygdala activity reduces the freeze response.

Lavano et al. (2018) and Gouveia et al. (2019) also report reduced activity in the amygdala and reduced freezing behaviors in their review of studies using DBS.

Fewer studies have examined the effects of DBS on the other key structures of this circuitry, the hippocampus and the mPFC, but Lavano et al. (2018) and Gouveia et al. (2019) discuss a few of these findings. Stimulation of some parts of the hippocampus have been shown to reduce extinction learning and reduce plasticity (Lavano et al., 2018, Gouveia et al., 2019). This finding is consistent with our understanding of the hippocampus' role in memory. Extinction memories need to be strong enough to override the recall and association of fear conditioning, but this would be impaired by DBS. Studies on DBS of the mPFC have found that the treatment can facilitate extinction and reduce freezing behaviors, along with other anxiety-related behaviors. However, Gouveia et al. (2019) also reported a study where DBS of the PL cortex within the mPFC actually inhibited extinction. This suggests that further research is needed in animal studies to determine which parts of the mPFC should be stimulated in order to successfully treat PTSD.

TMS as a form of non-invasive electric stimulation treatment has been explored. This treatment developed more recently than DBS, so there is less research about its effects. Some studies have reported that TMS during extinction reduces freezing and other anxiety-related behaviors in rat models (Gouveia et al., 2019). Another study examined the effect of TMS administration immediately after a trauma experience in a rat model as a preventative strategy and immediate treatment for the development of PTSD (Wang et al., 2015). They specifically looked at sensorimotor gating as an indicator of PTSD, which refers to an individual's ability to filter important stimuli that requires attention from other environmental stimuli. Individuals with PTSD have impaired sensorimotor gating and demonstrate hypersensitivity to stimuli or

overgeneralization of aversive fear responses to neutral stimuli. The study found that TMS administered to the right PFC at high frequencies reduced anxiety behaviors and prevented the impairment of sensorimotor gating, as demonstrated by a pre-pulse inhibition trial using sound recordings. No studies reported findings from tDCS on animal models.

As studies supporting the positive effects of DBS and TMS, more studies are able to use these techniques in clinical trials with subjects with PTSD. Currently, these studies are very limited, and typically these forms of treatment are solely used on subjects with extreme symptoms who have not responded to other forms of treatment. Gouveia et al. (2019) reports on the few that are available, showing that all studies but one using TMS administration to the PFC have reported a greater reduction of symptoms than control groups. These symptoms are measured using standardized scales such as the Clinician-Administered PTSD Scale (CAPS). A few studies reported side effects such as headaches and dizziness, and one study reported an extreme effect of a seizure occurring, so it is important to keep these effects in mind. Clinical studies on tDCS have paired tDCS with traditional psychotherapy to enhance treatment. In various studies, subjects receiving tDCS in the PFC had increased extinction recall. Gouveia et al. (2019) report only one study on DBS, where one subject received DBS of the amygdala and experienced a significant reduction in PTSD symptoms based on the CAPS assessment. These studies suggest promising results in clinical trials, but it is clear that continuing research is needed to verify these effects and understand potential risks.

Another area of neurological treatment for PTSD that is developing is the administration of glucocorticoids. Glucocorticoids are a form of the hormone cortisol, the stress hormone of the body that is also involved in memory formation and memory maintenance through the experience of arousing events and stimuli (De Quervain, D., Wolf, O. T., & Roozendaal, B.,

2019). The presence of glucocorticoids actually enhances the consolidation of extinction memories, but they also impair the retrieval of memories. Individuals with PTSD actually have lower levels of cortisol than control comparisons (De Quervain et al., 2019). This means that they lack the hormonal response to prevent them from constantly retrieving and reliving aversive memories of trauma. Thus, the administration of glucocorticoids has been found to be an effective form of treatment for PTSD because it helps diminish the retrieval of aversive memories and improves extinction memory consolidation. De Quervain et al. (2019) cite a study by Merz et al. (2018) describing the neurological processes glucocorticoids are involved with that underlie memory retrieval and extinction. The study found that cortisol reduced the activation of the amygdala-hippocampal neural network and increased activity in the ventromedial prefrontal cortex. Interestingly, Hashtjini et al. (2018) found that DBS of the amygdala in a rat model led to increased corticosterone levels (the cortisol hormone equivalent in rats) compared to control groups. This could occur due to neural network between the amygdala and other structures of the brain and fear learning that occurs. This study points to a link between DBS and cortisol that is important to analyze in future studies. Glucocorticoid administration appears to be a promising form of treatment for addressing neurological alterations in PTSD, but further study is necessary to understand cortisol's impact on complex memory processes that are central to the experience of PTSD, as well as the right timing and dosage of glucocorticoid administration.

In sum, electrostimulation procedures and glucocorticoid administration as viewed as forms of neuromodulation in PTSD treatment. These studies are important because they can directly examine the neurological impacts of the treatment.

Psychotherapy is considered a very common form of treatment for PTSD in clinical practice. Types of psychotherapy for PTSD include CBT, exposure therapy and eye movement desensitization and reprocessing (EDMR) (APA, 2013; Helpman, 2016; Taylor, 2017). Although readily used in for treatment, studies on specific neuromodulation due to psychotherapy treatment are less prevalent. Clinicians such as Taylor (2017) describe symptoms with a neurological basis, but do not study how therapy can induce neuroplastic changes. Taylor's (2017) work is addressed towards clinicians practicing treatment for clients with PTSD through cognitive-behavioral therapy (CBT). He discusses observable behavioral and cognitive differences that clinicians should be aware of and focus on in their treatment. These differences include deficits in attention and memory, hypervigilance toward trauma-related stimuli, enhanced fear conditioning, slower fear extinction, altered beliefs about the world and the self, and avoidance or suppression tendencies that increase PTSD symptoms. Each of these characteristics has a neurological basis, primarily in the fear neural circuitry, and these neurological components are crucial to our continued understanding and treatment of PTSD. However, Taylor (2017) does not address neuromodulation that occurs as a result of CBT.

Another study looked at prolonged exposure therapy, focusing on a part of the prefrontal cortex called the anterior cingulate cortex (ACC), which is involved in the executive control of emotions (Helpman et al., 2016). Although no differences in ACC volume or thickness were found between subjects with PTSD and trauma-exposed healthy controls pre-treatment, magnetic resonance imaging showed that participants with PTSD exhibited ACC volume reduction and thinning after 10 weeks of prolonged exposure treatment. The researchers suggest that the neurological components behind these changes could be that exposure therapy increases the extinction of fear memories because subjects are exposed to stimuli to decrease their aversive

responses. Thus, the strong neural connections and pathways underlying these associations are weakened and eventually pruned, while new pathways from extinction learning are formed. This study is in need of replication, but it shows the importance of examining the impact of psychotherapy on neurological processes, especially since this is a commonly practiced form of treatment for PTSD.

It is clear that research on neuromodulatory forms of treatment for PTSD is still developing. Animal models of electric stimulation for treating PTSD show promising results in the reduction of PTSD symptoms and aversive behaviors such as freezing. Specifically, DBS of the amygdala addresses these symptoms due to hyperactivity of the amygdala that occurs. However, more studies in clinical trials are needed in order for electrostimulation treatments, including TMC and tDCS, to become recommended for clients who are not responsive to other forms of treatment. Another potential for addressing not only PTSD symptoms, but memory processes themselves, is glucocorticoid administration, which can impair the retrieval of aversive memories and aid the consolidation of extinction memories. Future studies are needed to determine appropriate administration logistics like timing and dosage, as well as to create a greater understanding of the complex memory processes glucocorticoids impact. Finally, research is needed to continue to examine how psychotherapy can treat neurological alterations of PTSD. As psychotherapy is already common practice for treating PTSD, it is important that clinicians have a foundation in the neurological processes and alterations of PTSD so that they can provide more effective treatment addressing these alterations. All forms of psychotherapy have a neurological impact, so future studies should be dedicated to uncovering how psychotherapy can target neuromodulation for the treatment of PTSD.

Through examining neurorehabilitative treatment for individuals with PTSD using both invasive and non-invasive strategies, it is clear that there are many gaps in the research of this area. The need for providing effective treatment for these individuals is still very much present, so it is suitable to look at forms of alternative treatments that may be beneficial. One unique stimulus that could be applicable is music, because music involves many cognitive processes and engages multiple neurophysiological processes. If interacting with music has a cognitive and neurological foundation, then music may be a way to approach repairing multiple areas, networks, and functions of the brain that are impaired by PTSD. Studies on the neural processing of musical stimuli can help us understand how music could be beneficial in this population.

Auditory and neural processing of musical stimuli is a complex process. Music does not just travel to one area of the brain. Rather, musical stimuli travel neural networks associated with every functional domain - movement, cognition, communication, emotion, and social responses (Moore, 2018). As sound waves move through the outer ear to the middle ear, they cause the tympanic membrane, or the eardrum, to vibrate (Levitin, 2013; Moore, 2018). These vibrations also vibrate the small bones in the ear, the ossicles, and the sound wave becomes mechanical energy that moves into the fluid of the inner ear. In the cochlea, sensory receptor hair cells are pushed against the tectorial membrane because of the movement caused by the sound wave. Through a mechanical process, these hair cells stimulate an electrical signal that travel through the cochlear nerve to the brain. Before traveling through the thalamus to the primary auditory cortex, the electrical signal is already beginning to be processed in different nuclei of the brainstem, such as the cochlear nucleus. This initial processing explains how behavioral responses to music, like entrainment, visual-orientation towards a sound, and processing danger from a sound, occur subconsciously (Levitin, 2013; Moore, 2018). In the case of a sound that

may be dangerous, the signal is sent directly from the brainstem straight to the amygdala, which is involved in the fight-flight-or-freeze response.

While the electrical signals are initially processed in the brain stem, the signal also moves through the thalamus to the primary auditory cortex (Moore, 2018). Once the signal reaches the primary auditory cortex, it continues to disperse throughout many neural networks in the brain. Focusing on the areas of the brain impacted by PTSD, the cognitive and emotional domains are particularly important. The hippocampus, involved in memory and learning, is activated by music (Levitin, 2013; Moore, 2018). The PFC, involved in many areas of functioning including attention and executive functioning like planning and self-monitoring, is also activated through listening to music. The ventral striatum, involved in dopamine production and release within the reward system, are also activated by music, and research continues to analyze how these responses associated with emotion might differ for different kinds of music (Levitin, 2013; Moore, 2018). The amygdala, which is known to be hyperactive in individuals with PTSD, can respond differently to different types of music. For example, in music associated with negative emotions based on features like minor keys and lyrics, the amygdala becomes activated, while music associated with positive emotions can actually deactivate the amygdala (Legge, 2015). In addition to these crucial areas affected by PTSD, activation also occurs in areas throughout regions of the brain associated with motor responses, communication, and social responses that are located in the cortical, subcortical, and brainstem regions (Levitin, 2013; Moore, 2018).

If we know that musical stimuli are able to activate neural networks, then it is important to know if music can specifically target areas of functioning and modulate activation to induce neuroplastic changes. Stegemöller (2014) proposed a neuroplasticity model of music suggesting three ways that explain why music is effective neurologically. The first way music may influence

neuroplasticity is through increasing dopamine and acting as a reward stimulus. Dopamine reinforces learning processing by firing in the nucleus accumbens and vetregmental area, areas of the brain associated with reward, so that knowledge and skills are reinforced and solidified in memory. Dopamine also fires during music listening (Legge, 2015; Levitin, 2013; Stegemöller, 2014). Dopamine releasing while practicing a skill and listening to music will reinforce those skills, create stronger memories, and allow those skills to be more generalizable outside of a music setting. Even without a learning component, the release of dopamine is motivational for an individual. Just the act of music listening releases dopamine in the brain, so music therapy can be more enjoyable, motivational, and approachable as a form of treatment for clients.

Secondly, music can influence neuroplasticity by synchronizing neuronal firing. This is based on the Hebbian principle, which states that when neurons fire within milliseconds of each other, those neurons become linked together. Stegemöller (2014) applies this principle to music processes, proposing that music and rhythm help the body entrain. This can occur through movement, heart rate, respiration, or vocalization (Levitin, 2013; Stegemöller, 2014). Thus, the pairing of musical stimuli with non-musical skills and behaviors would strengthen the neural networks and synapses of those behaviors in the learning process. In addition to pairing, musical stimuli that is multisensory and in reference to something strengthens emotional processing in the prefrontal cortex and the limbic system (Legge, 2015).

The final part of Stegemöller's (2014) neuroplasticity model suggests that music may actually promote neuroplasticity because it is an organization of sound that is distinct from noise. Exposure to noise can adversely impact neuroplasticity by causing stress that influences cognition and memory. Reversely, music can actually promote neuroplasticity and learning by organizing sound into consonant and precise stimuli that is easier to process and remember.

From this model of neuroplasticity, we see that music does have a neurological impact through the release of dopamine, the synchronizing of neuronal firing, and the organization of sound.

Through this study, we can see that music is able to promote neuroplasticity and modulate changes in the brain that would be beneficial for individuals with PTSD. Music therapists can use these principles to provide treatment in physical, cognitive, emotional, and social domains. Music therapy, as defined by the American Music Therapy Association (AMTA), is "the clinical and evidence-based use of music interventions to accomplish individualized goals within a therapeutic relationship by a credentialed professional who has completed an approved music therapy program" (AMTA, 2019). While the healing value of music has been known in many historical cultures, music therapy really emerged as a field during and after World War I. Community musicians would play in hospitals for soldiers and veterans as they recovered from physical and emotional trauma. From this experience, doctors saw positive changes occurring in the patients as they listened to and played music (AMTA, 2019). The origins of music therapy can be found in improving quality of life for veterans with PTSD. Music therapy continues to be used with veterans and other individuals with PTSD (Gooding, 2018).

With this neuroplastic model of music therapy in mind, we can examine more closely how music therapy can address PTSD neurologically. What needs would be appropriate to address for clients with PTSD? Bronson, Vaudreuil, and Bradt (2018) report on the music therapy programs at the National Intrepid Center of Excellence (NICoE) at Walter Reed National Military Medical Center and Intrepid Spirit Center (ISFB) at Fort Belvoir. These centers work from a neurologic music therapy (NMT) model with veterans with PTSD, traumatic brain injury

(TBI), or both occurring comorbidly, to address autonomic functioning, cognition, social integration, and emotional regulation.

Based on the characteristic symptoms and neurologic impairments established for PTSD, each of these areas is important to focus on in music therapy treatment. Autonomic functioning can be regulated through music listening and entrainment. A steady tempo allows functioning like heart rate and respiration to regulate, functions of the parasympathetic system that is overactivated in individuals with PTSD (Bronson et al., 2018; Moore, 2018). Music therapy can impact cognition because the processing of musical stimuli is done through multiple areas of the brain involved in learning (Bronson et al., 2018; Moore, 2018). Much like neurological changes occur in people with music training, these processes are similar for clients in neurorehabilitation programs who need to address cognitive needs like executive functioning. NMT is used to work on social integration as well, because social relationships have an influence on psychological processes such as self-esteem and self-expression (Bronson et al., 2018; Moore, 2018). The polyvagal theory proposes that humans have a specific autonomic response to sounds at certain frequencies (Tomaino, 2015). The human voice occurs at a middle frequency that is associated with safety and comfort, suggesting that neural processes of socialization are important to consider, and that utilizing the human voice within music through singing is advantageous for addressing social needs (Tomaino, 2015). Finally, emotional regulation is appropriate to focus on in music therapy because music can modify activity in the brain that influences emotion like the amygdala, hippocampus, and other areas (Bronson et al., 2018; Moore, 2018). In addition, the limbic system is involved in releasing dopamine and acting as a reward in psychological processes (Bronson et al., 2018; Landis-Shack, Heinz, & Bonn-Miller, 2017; Moore, 2018; Stegemöller, 2014).

One important need to consider for using music therapy to treat clients with PTSD is how auditory stimuli may be triggering to them (Borczon, 2015; Bronson et al., 2018). This population is particularly vulnerable to being hypersensitive to stimuli that may be related or unrelated to the type of trauma they have experienced. Specifically, war veterans with PTSD may have a negative response to auditory stimuli that is associated with their experiences. (Bronson et al., 2018). In his description of various music interventions, Borczon (2015) notes that something like a drumming experience with loud and sharp sounds could have a triggering effect on clients. This can occur because individuals with PTSD have been shown to have increased fear conditioning and hypervigilance to emotional stimuli due to hyperactivation of the amygdala (Nash, Galatzer-Levy, Krystal, Duman, & Neumeister, 2014). Additionally, certain responses may be evoked consciously or subconsciously in response to certain songs, because memories are more accessible when information is stored in the organized form of a musical mode and the neural networks are strongly connected (Stegemöller, 2014; Tomaino, 2015). In response to this need, it may be appropriate or necessary for music therapy sessions to include music listening experiences that expose clients to various musical sounds and allow both the clients and the music therapist to understand a client's response. In one program, clients were able to use a device that provides biofeedback to them about their heart rate and respiration so that they can practice self-regulation in response to auditory stimuli (Bronson et al., 2018). Music therapists must have an awareness of how music may stimulate or trigger clients with PTSD so that they do not cause harm in their treatment.

To concentrate on these needs and goals of clients with PTSD, appropriate music therapy interventions are needed. Borczon (2015) proposes that the use of drumming and improvisation are ways to address many needs, including regulation, emotional expression, developing a sense

of social support, reducing anxiety, and increasing confidence. Specifically, improvisation is effective because that kind of musical exploration actually results in hypoactivation of the lateral PFC, allowing clients to be free of self-monitoring or executive decision-making that may inhibit them from playing because of self-consciousness or fear (Borczon, 2015; Tomaino, 2015). This is important for individuals with PTSD because the PFC has been shown to be hyperactivated in subjects with PTSD (Boccia et al., 2016; Nash et al., 2014). Interventions utilizing improvisation would allow clients to regulate and reduce the activation of the prefrontal cortex.

From Stegemöller's (2014) neuroplasticity model, music therapy interventions that pair a music activity or experience with a learning process such as emotional regulation or reducing fear to a stimulus would be beneficial because those neural networks are strengthened by the release of dopamine as a reward and by synchronized neuronal firing. In addition, when these skills are taught through the medium of music, clients may be equipped to learn more efficiently because the information is processed in an organized and pleasing way. With this in mind, it could be appropriate to engage in songwriting with a client to create lyrics that provide them with different ways of coping if they are experiencing fear or dysregulation due to a stimulus associated with trauma. Songwriting can be used to address coping skills and emotional regulation, building self-expression, increasing social connection, and cognitive skills (Borczon, 2015; Bronson et al., 2018).

Music listening using grounding and relaxation techniques can be used to address emotional regulation (Bronson et al., 2018; Landis-Shack et al., 2017). In addition to emotional regulation, regulation of autonomic processes like respiration and heart rate are important, and interventions such as guided relaxation with breathing, singing, or learning wind instruments could help clients learn to help control these processes through stressful environments and

triggers (Levitin, 2013). Other autonomic processes like blood pressure and levels of the hormone cortisol can also be regulated through the use of relaxing music, which is typically characterized by a slower tempo and softer instruments (Levitin, 2013). These are just some possible techniques for addressing the needs of clients with PTSD through music therapy treatment through a neurological foundation.

PTSD has debilitating symptoms with neurological impairments at the foundation. Treatments that address these impairments through neurorehabilitation are still developing, but music therapy is a unique treatment option due to music being the primary medium for targeting specific needs and goals. Music therapy is a research and evidence-based practice that has been used with veterans with PTSD since the early 1900s (AMTA, 2019). Music therapy can be used in neurorehabilitation because of music's complex influence on many areas and neural networks within the brain. These areas that are altered by music are involved in physical, cognitive, emotional, and social processes. These domains are commonly addressed through music therapy practice, but music therapists who understand the neurological basis for these treatments can more effectively address areas of need within PTSD. PTSD is known to impair functioning in areas of the brain including the hippocampus, the amygdala, and the mPFC. These structures impact how an individual experiences fear conditioning and is hypervigilant towards emotional or negative stimuli. These impairments can be addressed through forms of stimulation treatment like DBS and TMS, which have been shown to be effective in modulating the activity of the aforementioned brain structures. However, these treatments are still being developed. The use of music is effective because it stimulates many regions and networks of the brain. Music therapy can target impairments of PTSD through interventions with foundations in the neurological

effects of music. Future research on neuroscience and music therapy will continue to reveal how treatment can be more effective.

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Appendix

Relationship between Faith and Learning

My journey as a Christian and as a scholar has been varied and dynamic. I see my studies in music therapy as a convergence of my passions, skills, and efforts. My theological perspective brings immense value to my work, and it influences how I understand the principles of music therapy practice. My personal experiences growing up in a diverse community and playing music from a young age were crucial to developing my strong belief that every individual can benefit from music in their lives. While it took some time to understand how my passions could merge with scholarship in an academic setting, I can see how this relationship plays out in my work as a student and will play out in my work as a professional.

My own academic faith began at a young age. My dad, as a librarian, encouraged a love for reading and learning about new people and places through books. My mom began homeschooling my sisters and I in elementary school, so we had the freedom to learn in an individualized way that incorporated faith into study. My life completely changed in sixth grade when my family moved to Jos, Nigeria as missionaries. In a city and country completely different from my home in Washington, I was exposed to a diverse community, both in nationality and religious beliefs. The international school that I attended for middle school and high school had students and teachers from all over the world, with families who were Hindu, Muslim, and many different Christian denominations. I think that my experience in high school helped me to incorporate my beliefs into other areas of my learning, because I was able to discuss my faith perspective on other subjects like English and Psychology. This was on a surface level, however, and I never had a full understanding of how my individual scholarship was really a part of worship or contributing to a broader community good.

In my secondary school experience, separate from any form of academics or scholarship, was my enjoyment of music. Because my dad was a musician, he helped me get involved with the church worship team and sound crew early on. Playing music, both classical piano and in a worship band, was an important part of my life and how I most often understood God speaking. Through music, I saw both my individual gifts and my collaboration with a group as crucial to my Christian walk. I loved having the opportunity to be a part of other styles of worship, like dancing to Nigerian praise songs or singing a call-and-response traditional melody over a multitude of percussive beats. These were new experiences for me that I am so thankful for now because they helped me have a broader perspective of what the kingdom of God looks like and how Christian practices differ around the world.

Choosing music therapy as my field of study was easy because it combined my passions and important values about God's kingdom that I learned growing up. I knew music's influence from my own personal experience, and I knew that music gave me a better understanding of beliefs and cultural differences. What changed as I began studying music therapy was understanding music's role in an academic setting and evidence-based field. Scholarship became much more personal to me because I finally had the opportunity to pursue what I was passionate about in a collegiate environment. I had never understood scholarship in this way before, but Douglas and Rhonda Jacobsen (2004) articulate my new experience well in *Scholarship and Christian Faith: Enlarging the Conversation* when they write, "Scholarship necessarily mixes sustained effort with creative insight. Take away the hard work and all we have is effluent self-expression; take away the creativity and all that is left is the cataloging or repetition of what others already know" (p. 123). Music therapy was a way of merging my creative endeavors with persistent study in a way that intrigued me and forced me to ask new questions about the world.

Music therapy, as an evidence-based field, is a clinical approach to using music to address goals in physical, emotional, cognitive, social, and spiritual domains. Music therapy values the individual abilities, preferences, and needs of each person, and music therapists form therapeutic relationships with clients to best address those needs and goals. Seeing the priceless worth every person as a person made in the image of God, no matter what background they are from, is a crucial commitment I have from growing up getting glimpses of people's lives all over the world. From a humanistic approach to music therapy practice, it is important to recognize the capacity of every individual to change, grow, and improve their overall wellness (Unkefer & Thaut, 2005). I see music therapy as immensely valuable because music is a unifying part of every culture that can act as a bridge to cross barriers and divides. These characteristics of music and music therapy practice perfectly aligned with my own core beliefs and experiences.

Scholarship in music therapy looks for evidence from experimental studies and practical methods to assess the effect of music on people and how music can be used to help people address their individual needs. Using Ernest Boyer's models of scholarship in *Scholarship Reconsidered* (2015), I see music therapy as a synthesis of discovery and application. Research plays a crucial role in the music therapy field, particularly to understand the specificity of music's influence on the human body, like in brain development and muscle strengthening. However, music therapy emerged as a field in the twentieth century due to the practical application of music, and music therapy continues to develop as a field through the practical service of music therapists who find new methods and techniques to address the unique needs of individuals and communities in the world. Boyer accurately describes how the scholarship of application is done in music therapy as he writes, "theory and practice vitally interact, and one renews the other" (p. 85). This plays out in a multitude of ways because no single clinical use of

music will always have the same impact on someone. A music therapist must understand a client's background and preferences through a therapeutic relationship that allows them to give the best care possible to a client. More applications of music therapy continue to develop as research demonstrates how music has a wider scope of influence. These possibilities are exciting to me as I start my professional career.

Another important aspect of music therapy treatment that is important to my personal beliefs is the importance of self-reflection. Self-reflection allows a music therapist to be aware of themselves, including their own background, experiences, preferences, and biases that influence how they provide treatment for a client. This helps them ethically offer the best possible practice and understand when their own experiences may hinder them from these opportunities. Self-reflection is key to my own personal journey of faith as I constantly seek to learn from my experiences and grow in my relationship with God. I see self-awareness in music therapy as extremely valuable because it is a way for me to grow professionally and to understand how I can uphold personal and professional values as I provide care and healing through music.

My practical experience as a music therapy student at various sites in the broader Seattle community has allowed me to reflect on the role that my personal faith has in my work. While I do not have the opportunity to explicitly share these beliefs, they do inform why I provide care for clients and how I provide care for clients in a way that elevates the image of God in all people and works towards healing. George M. Marsden, in his book *The Outrageous Idea of Christian Scholarship* (1997), encourages Christian scholars to be aware of the theological perspective they bring to their discipline, even if those perspectives act as a background to their academic work. He writes, "it is fair for Christian scholars to ask the question conditionally: "Suppose someone believed in God, how would the assumptions or conclusions of our discipline

look different?"" (p. 84). Marsden's example of the doctrine of the incarnation is particularly relevant to me. My assumptions about music therapy may differ from my colleagues because I believe that the supernatural and natural realms interact in the world, and I am open to God's divine work in the world. God could work through music or a music therapy session in someone's lives in ways that I do not understand. This theological perspective does not directly change the work that I do, but it does inform how I understand my role in the world and my role as a music therapist.