5-2015

The Neurology of Music for Post-Traumatic-Stress Disorder Treatment: A Theoretical Approach for Social Work Implications

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Recommended Citation
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The Clinical Research Project is a graduation requirement for MSW students at St. Catherine University/University of St. Thomas School of Social Work in St. Paul, Minnesota and is conducted within a nine-month time frame to demonstrate facility with basic social research methods. Students must independently conceptualize a research problem, formulate a research design that is approved by a research committee and the university Institutional Review Board, implement the project, and publicly present the findings of the study. This project is neither a Master’s thesis nor a dissertation.
Acknowledgements

A special thanks goes out to my chair, Lance Peterson, for being patient, supportive, and encouraging throughout this process. Lance takes pride in his student’s work, and it shows. Lance's flexibility allowed me to expand my knowledge and passion by researching the neurological implications of music and trauma. I also want to thank Loretta Steckelberg and Kerby Plante for being on my committee. This paper would be impossible without their willingness, constructive feedback, and support. I want to thank my program manager at my job for working with my schedule, caring about me as a person and not solely as an employee, and encouraging me professionally. My family also deserves gratitude for their encouragement, prayers, and love. Last, but not least, I want to thank my girlfriend, Jenna, for affirming my quest of knowledge, reminding me of the big picture, supporting me emotionally, and understanding of my hectic schedule.
Abstract

The purpose of this theoretical paper is to provide information on how trauma and music neurologically impact the brain, and how music can be used as a tool to neurologically aid in the treatment of post-traumatic stress disorder. Post-traumatic stress disorder impacts the hippocampus, the hypothalamus-pituitary-adrenal axis, amygdala, and prefrontal cortex. Music stimulates the hippocampus, amygdala, nucleus accumbens, the mesolimbic dopamine system, and impacts the HPA axis. Evidence also shows that music can stimulate neurogenesis and neuroplasticity. Music can be used as a tool to stimulate these areas to activate neurogenesis and/or neuroplasticity, thereby reconditioning the brain back to healthy functioning. Several implications for social work practice can be drawn from this theoretical work. Specifically, music can be utilized to build rapport, for grounding in reprocessing therapies (EMDR), for positive distraction for clients in crisis who have a hard time self-regulating, for provoking deliberate moods, for identifying and naming moods, for increasing socialization and group cohesion, for decreasing avoidant symptoms, for expressing one’s narrative nonverbally, for expressing rage through drumming, to reduce cortisol levels, to release dopamine, and to potentially quicken or ease the process of therapy.
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Post Traumatic Stress Disorder (PTSD) impacts 7.7 million adults (National Institute of Mental Health [NIMH], n.d., para. 16). According to the National Alliance on Mental Illness [NAMI], around 10% of women and 5% of men are diagnosed with PTSD in their lifetime (2014, p. 1). Additionally, PTSD is estimated to impact 6.8% of adults who have suffered from a crime, abuse, or other trauma (Duke University, 2012, p. 1).

The fundamental component of PTSD is the development of symptoms after exposure to single or multiple traumatic events. It can look differently for different people. Some people may predominately notice they do not enjoy the things they used to, or feel dissatisfied or indifferent about their life. Others may experience behavioral symptoms and/or a fear-based re-experiencing of the traumatic event. More symptoms include experiencing negative thoughts, avoidance, or becoming unnecessarily reactive to stimuli. There can also be a combination of symptoms (American Psychiatric Association, 2013, p. 274).

A problem people with PTSD encounter is not having other options when they do not respond well to traditional methods of treatment, such as eye-movement-desensitization-and-reprocessing (EMDR), cognitive behavioral therapy (CBT), or pharmaceutical treatment. (Carr et al., 2011). Brief explanations of traditional treatments, along with limitations, are included below.

**Traditional Treatments**

**EMDR.** Eye Movement Desensitization and Reprocessing (EMDR) was developed in the late 1980s by Francine Shapiro (Cornine, 2013), originally as a behavioral approach to PTSD, but now for a variety of disorders (panic disorder, major
depressive disorder, and substance abuse) (Cahill et al., 1999; Cornine, 2013). EMDR has a “multi-modal treatment strategy complete with its own principles, protocols and procedures” (Cornine, 2013, para. 1). The American Psychiatric Association lists it as an effective treatment for trauma. EMDR is based on the hypothesis that trauma causes the neurological excitatory and inhibitory balance to be damaged, causing neural pathology and symptoms of PTSD. Shapiro reports that bilateral stimulation (eye movements, auditory tones, or vibrating tactile stimulation) can balance the neurological pathways to normal functioning. The bilateral stimulation is believed to counter neurological pathology and helps the traumatic memory to be reprocessed and integrated (Cornine, 2013).

To summarize the EMDR process, the client thinks of a particular traumatic memory, and imaginal recall takes place (images come to his/her mind). While the client is thinking of a memory, the therapist guides 10 to 20 rhythmic lateral eye movements with his/her index finger. This is considered a set. After each set, the client reports his/her sensations, thoughts, and affect. The client also rates his or her distress on Wolpe’s (1973) Subjective Units of Distress scale (SUD). This cycle continues until the client reports a 0 or 1 on the 0 to 10 SUD scale. After desensitization, the client thinks of an alternative positive thought of the trauma (“I am now in control”), while visioning the trauma images. The therapist guides the client in the eye movements and the client rates his or her belief in the new thought. The new belief is rated on the Validity of Cognition (VOC) scale. The scale ranges from 1 (“completely false”) to 7 (“completely true”). The cycle is completed when the client reports a “high level of belief” in the new thought (an
exact numerical value of VOC was not given in the literature) (Cahill et al., 1999, para. 2).

**Criticism of EMDR.** A study by Davidson and Parker evaluated 34 EMDR studies in a meta-analysis. The studies had varying populations and measures. The meta-analysis investigated the following question: is EMDR overall effective, and is the mechanism (the eye movements) effective? It used the same process measures that previous EMDR studies used (ratings of subjective units of distress [SUD] scale when shown pictures to provoke fear, and validity of cognition [VOC] scale ratings to determine new thoughts about the same fear-provoking picture). The study used different outcome measures (psychometric or physiological assessments) than Shapiro’s studies (Shapiro only used process measures). The study revealed that EMDR therapy is better than no therapy and better than therapies not using anxiety-provoking stimuli. Both progress and outcome measures fared better for EMDR when compared to the group of no treatment and treatment that did not use anxiety provoking stimuli. However, EMDR, when compared to other anxiety-exposure techniques, showed to have no significant effect. Thus, according to this study, EMDR is not more effective than other exposure-based treatments (Davidson & Parker, 2001).

The study also revealed that the eye movements in EMDR may not be necessary. Two EMDR groups were compared: one group with the eye movements, and the other without. The results showed that there were no incremental effect of doing the eye movements. Thus, the eye movements are not necessary and do not impact outcome measures. Lohr (the author of *Science and Pseudoscience in Clinical Psychology* and who has 50 scholarly publications) completed many outcome based studies on the
mechanism (eye movements) of EMDR. They concluded that EMDR is just another
imaginal exposure technique that has a potential thought-stopping component. Critics
also scrutinized the measures used (SUD and VOC) for effectiveness in Shapiro’s
studies, stating that it lacked standardized outcome measures (Davidson & Parker, 2001).

**Cognitive behavioral therapy.** Cognitive behavioral therapy (CBT) is a short-
term treatment approach that can be done in individual or group therapy, and can last up
to twenty sessions (Barlow & Durand, 2009; Truven Health Analytics Incorporated,
2014). During CBT, a client identifies negative thought patterns and tries to replace them
with more positive, balanced thought patterns. The idea is that if one has positive
thought patterns, it will change maladaptive behaviors (Barlow & Durand, 2009). It also
focuses on the distressing memories as well as the meaning derived from the event
(Butler, Chapman, Forman, & Beck, 2006).

However, there is debate on the effectiveness of CBT after treatment stops. Some
argue that cognitive and behavioral changes do not persist after treatment. Also, a meta-
analysis on the effectiveness of CBT concluded that CBT effectiveness has been
overstated in the literature (Butler et al., 2006). Another criticism is not all patients with
PTSD respond well to CBT treatment (Carr, 2011). “A small but significant number of
patients do not respond sufficiently, and still have significant symptoms after therapy”
(Carr et al., 2011, p. 180). Those who do not respond well to CBT are people who have
experienced prolonged, multiple traumas, or who have poor verbal memory
(remembering words). There needs to be another option for the small, but significant,
number of people who are not helped with CBT.
Pharmaceutical. The most common pharmaceutical treatment for PTSD are antidepressants and selective serotonin reuptake inhibitors (SSRIs), a specific type of antidepressant. It is suggested that clients try physiological approaches for treatment before pharmaceutical. At times, symptoms of PTSD are so severe that medication may be necessary to reduce symptoms (Cukor, Spitalnick, Difede, Rizzo, & Rothbaum, 2009); however, there are still criticisms of pharmaceutical treatment. The positive outcomes of psychopharmacological interventions typically do not extend past the active treatment time (Butler et al., 2006). Therefore, once one stops taking the medication, the symptoms persist. Additionally, a systematic review done on pharmaceutical treatment for PTSD showed the results did not meet the requirements to be clinically effective (Cukor et al., 2009). Besides the traditional treatments, a brief reference of nontraditional treatments will be acknowledged.

Nontraditional treatments. One nontraditional treatment for PTSD is thought field therapy (TFT). This therapy relies on the premise that emotions and biological activity can be accessed through “energy points of acupuncture” (Cukor et al., 2009, sec. 2.5.1.). The acupuncture points, or meridian points, are tapped on while the client is thinking of an anxiety-producing event. Another nontraditional treatment is virtual reality exposure therapy. This allows the client to process fear triggered by what he or she sees. It is also another option for clients who avoid the event or who are emotionally vacant when telling their story (Cukor et al., 2009). Other nontraditional therapies are art therapy and yoga (Pizarro, 2004).

As a social worker, it is important to know best practices for our clients. Competence is one of social work’s core values. It is the social worker’s responsibility to
be knowledgeable about treatment options. It is the social worker’s ethical responsibility to be committed to his/her clients, practice setting, and the profession (National Association of Social Work, 2008). Traditional methods of treating trauma, such as psychotherapy and psychopharmacology, can be helpful (Briere & Scott, 2013, p. 234). However, it is incumbent that social workers are aware of nontraditional methods, such as music, in treating trauma. This paper will show evidence of how trauma impacts the brain, how music evokes emotions, how music impacts the brain, and provide a theoretical framework for how music could be incorporated in social work practice to treat trauma.

**Literature Review**

**Trauma**

**Definition of trauma.** For the purpose of this paper, trauma will be defined by a diagnosis of PTSD. The American Psychiatric Association added the diagnosis of post-traumatic stress disorder to the Diagnostic and Statistical Manual of Mental Disorders III (DSM-III) in 1980 (Friedman, 2014). The DSM-5 (2013), has eight criteria individuals need to meet in order to be diagnosed with PTSD.

First, he/she has to experience one of the following four events: exposure to actual or threatened death, serious injury, or sexual violence by directly experiencing the traumatic event; witnessing an event that happened directly to someone else; learning that a traumatic event happened to a loved one (the event must have been actual or threatened death and must have been violent or accidental); or being repeatedly exposed to details of the traumatic events.
Secondly, the person also has to experience intrusion symptoms (distressing memories; distressing dreams; dissociative reactions; prolonged or intense psychological distress; or intense psychological reactions when exposed to cues that resemble an aspect of the event). Thirdly, there also has to be an avoidance of stimuli (avoiding memories or external reminders) that reminds individuals of the traumatic event. Fourth, there has to be negative changes in thoughts or moods that has one or more of the following: being unable to remember an important aspect of the event; having negative beliefs or expectations about oneself, others, or the world (“I am bad,” or “The whole world is completely dangerous”); experiencing distorted thoughts about the event, so that the individual inaccurately blame themselves or others; persistent feelings of fear, horror, anger, guilt, shame or another negative emotion; diminished interest in usual activities; feeling detached from others; and/or having the inability to experience positive emotions.

Fifth, he/she has to have marked changes in reactivity that consist of at least two of the following: angry outbursts or irritable behavior; self-destructive behavior; hypervigilance; an exaggerated startle response; difficulty with concentration; or disturbances in sleep. Sixth, the symptoms have had to last more than a month. Seventh, the symptoms have to impair or cause significant distress in occupational, social, or other areas of functioning. Lastly, the symptoms cannot be due to alcohol or drugs.

To summarize the broad criteria for PTSD, someone had to experience, directly or indirectly, a traumatic event; he/she has to experience intrusion symptoms; the individual avoids stimuli associated with the trauma; he/she experiences negative changes in moods or thoughts; the person has marked changes in arousal and reactive behavior; and the individual experiences distress or impairment in social, occupational, or other areas of
life (American Psychiatric Association, 2013, p. 274). The definition of trauma was provided; now a look into how trauma is different from common stress will be explored.

**Different than common stress.** Pierre Janet (1889), as cited in Van Der Kolk (1999), stated that common stress is very different from trauma (p. 199). Additionally, PTSD is physiologically different than depression or anxiety (Grace, 2003, p. 9). Trauma is shown to create biological changes within the body (Van Der Kolk & Saporta, 1999, p. 199). Lawrence Kolb (1987) proposed the symptoms of PTSD are due to neuroanatomical and neuroendocrine changes caused by stress. Since Kolb’s proposal, there has been evidence to support “neurological and neuro-anatomical stress-related changes” due to trauma (as cited by Grace, 2003, p. 1). In summary, there is evidence trauma can neurologically impact the brain.

There is also a notable increase in the autonomic nervous system (ANS). This causes physiological changes in the body when stress is present. For example, people with PTSD often have higher blood pressure and pulse rates when compared to the general population. Cortisol production increases in the presence of acute stress (Grace, 2003, p. 9). Cortisol is a steroid hormone that is released in response to danger. It gives the person in danger the energy to fight or flee. Too much cortisol in the body can take a physical, mental, and emotional toll on the body. It also plays a significant role in the physiology behind PTSD symptoms (Ehlert, Gaab, & Heinrichs, 2001). Now that trauma and stress were differentiated, a review of the brain’s basic structures and function will follow.
The Brain

In order to understand the complexity of how trauma and music impacts the brain, it is good for one to know the basic parts and functions of the brain. The brain consists of nerve cells, or neurons, which communicate with the rest of the body through the spinal cord and nervous system. Nerve cells from the rest of the body bring information back to the brain, through the spinal cord, where it can be processed and an appropriate response can be computed. There are chemicals in the brain used to keep homeostasis. If the neurons are not firing properly, or if the chemicals are imbalanced, the brain will not be functioning at its highest capacity (MD Health, 2014).

The largest part of the brain is the cerebrum, which consists of four parts: the frontal lobe, the parietal lobe, the occipital lobe, and the temporal lobe. The brain is divided into two hemispheres, the left and right, which are connected by axons. Axons transmit messages to either side. The frontal lobe helps control judgment, behavior, attention, creative thought, problem solving, intellect, abstract thinking, muscle movements, physical reactions, smell and personality (MD Health, 2014). More specifically, the prefrontal cortex (the most forward part of the frontal lobe) is responsible for complex functioning, such as: attention, integrating information, reflecting on past behaviors and considering consequences before making a decision, emotional regulation, following social norms, personality, and planning for the future (Prefrontal Cortex, 2010). The parietal lobe controls movement; senses touch, pressure, pain; is responsible for language; and processes the senses. The temporal lobe interprets sounds and language. It also controls memory functions. The occipital lobe helps interpret vision input, such as color and word recognition (MD Health, 2014). Figure 1 illustrates the
four lobes and their basic function. Appendix A provides a table of the summary of brain structures, location, function, relationship to trauma, and the relationship to music.

Below the cerebrum is the cerebellum, which controls essential body functions such as posture, balance, and coordination. Another part of the brain is the brain stem, or the “reptilian brain,” because its basic function is to keep humans alive. It controls instinctive physiological responses such as heartbeat, breathing, and blood pressure. The brainstem consists of the midbrain, pons, and medulla (MD Health, 2014). Figure 2 displays the three basic parts of the brain. To summarize, the brainstem is responsible for basic living functions. More complex behaviors, such as emotion, are controlled by the limbic system.

The limbic system is composed of glands that help regulate hormones and emotions. It is also in charge of complex behaviors, such as reproducing, fighting, or fleeing. Additionally, the limbic system is how

![Lobe structures. This figure displays the four lobes in the cerebrum. Rajeev, L. (2012). Lobes of the brain and their functions. Retrieved from Buzzle.com/articles/lobes-of-the-brain-and-their-function.html](image1)

![Main structures of the brain. This figure illustrates the location of the cerebrum, cerebellum, and brain stem. Adapted from “The Nervous System: The Brain” by Boughey, 2012, retrieved from: http://msbougheygrade3.blogspot.com](image2)
humans assign truth and meaning to experience (Van Der Kolk & Saporta, 1999, p. 200). The limbic system contains the amygdala, hippocampus, hypothalamus, and thalamus. The amygdala helps the body to react to emotions such as fear, aggression, and anger. Emotion, reward, memory storage, and learning are linked to the hippocampus. The hypothalamus is used to control mood, hunger, temperature, thirst, and the pituitary gland. The thalamus monitors the sensations the body is experiencing (MD Health, 2014). Figure 3 illustrates the parts of the limbic system.

To summarize, the brain has two hemispheres, the left and right. It also has four lobes: the frontal, parietal, occipital, and temporal lobes. It has four basic parts, the cerebrum, cerebellum, the brain stem, and the limbic system. The latter has four main segments: the amygdala, the hypothalamus, the hippocampus, and the thalamus. The next section of the paper will focus on how the brain sends and receives information through neurons.

**Neurons.** Neurons are cells that receive and transmit information within the nervous system. The body of the neuron is called a soma, where the cell’s nucleus (the “brain” of the cell) is located. Neurons have branch-like structures called dendrites, which are used to receive information from other neurons. Once a dendrite receives the
signal, it travels to the soma, and leaves the soma through the axon. The axon is a long fiber that relays the signal from the soma to another neuron, muscle, or gland. At the end of the axon, there are tiny knobs that release neurotransmitters. The knobs are called terminal buttons. Neurotransmitters are chemicals that function as messengers and can activate other neurons. The specific location where neurons transmit information to each other is called a synapse. There is a microscopic gap between the two neurons in the synapse, called the synaptic cleft (synaptic gap). The presynaptic neuron, the neuron sending the signal, releases neurotransmitters into the synaptic cleft. The postsynaptic neuron, the neuron receiving the signal, has receptor cites on the cell membrane of the neuron that allows the neurotransmitters to bind to it. After the neurotransmitter fuses, it changes the postsynaptic neuron’s voltage. Once the postsynaptic neuron’s voltage reaches a positive charge, it will fire the positive charge through the neuron to the axon, and the process can start again (Weiten, 2007). The next section of this paper will explore how the adult brain is capable of renewal. Figure 4 displays the parts of a neuron and figure 5 shows a synapse sending and receiving signals.

![Diagram of neuron and synapse](image-url)

**Figure 4.** Parts of a neuron. This figure displays the dendrites, soma, nucleus, axon, and synapse of a neuron. It shows the presynaptic cell, the neuron that sends the signal, and the postsynaptic cell, the neuron that receives the signal. Adapted from “Types of Neurons”, 2010, retrieved from: [http://biomedicalengineering.yolasite.com/neurons.php](http://biomedicalengineering.yolasite.com/neurons.php).
Neuroplasticity. At one point, people believed the adult brain was a fixed entity and could not make new neural pathways. Neural pathways link regions of the brain to each other and connect information from the peripheral nervous system to the brain. The colloquialism of thought was that the brain was the most influential during childhood, and once someone reached adulthood, the individual could not form new pathways. However, studies show that the brain is much more malleable than originally thought.

During the late 1960’s, the word “neuroplasticity” was coined for describing structural changes in neurons (Fuchs & Flugge, 2014). Neuroplasticity is the neuron’s and neural networks’ ability to change connections and to reorganize the brain’s cellular and neural networks. The changes can be caused by sensory stimulation, new information, development, damage, or dysfunction. Neurons can develop and form synapses (Neuroplasticity, 2012). Synapses that are used more often are strengthened,
while synapses that are not used often are weakened. Eventually, synaptic pruning occurs: when idle synapses are completely eliminated (Neuroplasticity, 2012). Thus, synapses can be strengthened or pruned, making new networks or removing pathways that are not used. Next, the paper will show how stress impacts neuron structure.

**Stress and neuron structure.** Chronic or repeated stress changes the structure of neurons in different parts of the brain. Neurons in the hippocampus (responsible for memory storage) and the prefrontal cortex are affected by stress in the same way; dendrites retract when chronic stress occurs. This is an important detail because when dendrites retract, neuronal surfaces are lowered, which reduces the number of synapses. Since synapses are how neurotransmitters communicate, it makes it more difficult for neurons to communicate with each other, thus, weakening the neuropathways and communication within the brain. During chronic stress, the hippocampus and prefrontal cortex do not communicate effectively (Fuchs & Flugge, 2014). The prefrontal cortex is responsible for complex functioning, such as: attention, integrating information, planning, reflecting on past behaviors to consider consequences before making a decision, emotional regulation, following social norms, personality, and planning for the future (Prefrontal Cortex, 2010). Deficits in communication involving the hippocampus and prefrontal cortex can cause difficulty in memory functioning, emotional regulation, planning, decision making, and changes in personality (Fuchs & Flugge, 2014). The hippocampus and prefrontal cortex are not the only brain structures impacted by stress; the amygdala is also impacted.

The amygdala has an opposite reaction to stress: dendrite communication is enhanced within the amygdala itself, more specifically the basolateral complex (Fuchs &
Flugge, 2014). The basolateral complex contains the lateral nuclei, which receives sensory information from the temporal lobe, basal nuclei, and accessory-basal nuclei of the amygdala (Baars & Gage, 2010). When the stress subsides, however, the synapses return to their original state. Being able to return to their original state shows how efficient neuroplasticity can be (Fuchs & Flugge, 2014).

To summarize, stress inhibits synaptic communication with the hippocampus and the prefrontal cortex, causing them not to function as efficiently. A hypoactive prefrontal cortex and hippocampus are one of the main contributors for the symptoms of PTSD. The other contributor is the amygdala having increased communication within itself. This leads to an overactive amygdala, which can explain a hyperactive startle response for people with PTSD (this will be explained in detail later). Neuroplasticity is not the only way the brain can renew itself. Studies found that the brain can generate new neural cells.

Neurogenesis. Neurogenesis is the brain’s ability to generate neurons. This opposes previous beliefs that there were a fixed number of neurons in the adult brain that could not be replaced after cells die. Prior researchers thought that neuronal regeneration could not happen because most brain regions did not have access to stem cells (Fuchs & Flugge, 2014). Stem cells are unspecific (unspecialized) cells that can grow into any one of the body’s 200 cell types. They can divide through their lifetime, and can take place of any cells that die (Stoppler, 2012).
**Process.** Changes in neuronal functions and structures are influenced by stress, hormones, neurotransmitters, specific drugs, growth factors, environmental stimulation, learning, and age (Fuchs & Flugge, 2014). Neurogenesis consists of cell division, migration, and differentiation (Gage & Van Praag, 2002). Figure 6 shows the steps of neurogenesis. One of the ways neurogenesis occurs, cell division, is when a singular cell divides into two or more daughter cells. Neuronal cells come from polarized progenitor cells that divide unequally. One daughter cell continues the division cycle, while the other cell goes through cellular differentiation. Cellular differentiation is when a cell zygote (daughter cell) turns into a specific cell type: in this case, a neuron or glia cell. A glia cell helps keep a neuron alive by removing pathogens, supplying nutrients, and giving it structure. Cell migration is when cells move from one location to another and then differentiates (Wodarz & Huttner, 2002). Figure 7 displays the process of neurogenesis.

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**Figure 6.** Neurogenesis steps and location. SVZ- subventricular zone; SGZ- subgranular zone; RMS- rostral migratory stream; OF- olfactory bulb. The first step in neurogenesis is cell division, which takes place in the SVZ and the SGZ. The second step is migration, which occurs along the RMS. Lastly, differentiation occurs in the OF, where neurons are formed.

**Figure 7.** Cell division process for neurogenesis. D.C. = daughter cell. Cell division start with a single cell dividing into two or more daughter cells. One D.C. continues the division cycle and the other D.C. goes through cellular differentiation and turns into a glia cell or neuron.
**Location.** According to studies completed up to now, there seem to be two areas where stem cells reside and multiply before migration and differentiation, the subventricular zone and the subgranular zones of the dentate gyrus. The dentate gyrus is a part of the hippocampal formation. The stem cells in the subventricular zone and the subgranular zone divide (the first step in neurogenesis). After initial cell division, one of the stem cells migrate along the rostral migratory stream to the olfactory bulb (located in the forebrain). Here, they differentiate further into neurons (Gage & Van Praag, 2002). The process and location of neurogenesis was explained. The next part of the paper will look into how trauma
psychologically and neurologically impacts someone. Figure 5 also displays the locations of each step. Figure 8 and 9 show the parts of the brain involved in neurogenesis.

**Neurological impacts of trauma.** There are multiple biological systems and pathways involved in PTSD, and not one model is solely sufficient in explaining the complex pathophysiology involved. The biology behind PTSD is complex and results can be contradictory and difficult to interpret. “PTSD, as it is currently described, may not represent a single disorder, but rather a collection of outcomes that vary depending on individual differences in genetics, underlying neurophysiology, stress response, and exposure to traumatic events” (Briere & Scott, 2013, p. 226). Since there are so many factors when considering PTSD, two people may experience the same event, but only one develops PTSD.

However, this paper will examine the main findings in how trauma impacts the brain. Studies show that an over-active hypothalamus-pituitary-adrenal (HPA) area and the amygdala can cause symptoms of PTSD (NIMH, n.d., para. 5-6). Additionally, the hippocampus and prefrontal cortex are inhibited (Arnsten, 2009; NIMH, n.d., para. 5-6). The following paragraphs will explain this in detail.

**Hypothalamus-pituitary-adrenal axis.** The autonomic nervous system (ANS) is how the brain communicates with the rest of the body. Typical functions of the ANS include regulating breathing, blood pressure and heartbeat. The ANS is divided into two components. The first is the sympathetic nervous system, which triggers the fight or flight response. It is also known as “the gas pedal” because it releases adrenalin (epinephrine), which causes physiological changes in the body. Some of these changes
include an increased heart rate, blood pressure, and breathing, which prepares the body to fight or flee danger. The second component of the ANS is the parasympathetic nervous system. It is called the “brake” because it prompts the calming response after danger has passed. Now that a basic understanding of the autonomic nervous system is understood, a look at how the hypothalamus-pituitary-adrenal axis (HPA axis) is triggered will be helpful (Nickel, 2011; Harvard Health Publications, 2011).

The HPA consists of the hypothalamus, the pituitary gland, and the adrenal gland. The HPA axis includes the interactions between those three entities. Glands, hormones, and parts of the midbrain use the HPA axis to communicate (Hans, 1974).

As mentioned earlier, the hypothalamus is used to control mood, hunger, temperature, thirst, and the pituitary gland (MD Health, 2014). For PTSD, the most important function of the hypothalamus is how it controls mood and impacts the pituitary gland. The HPA axis is physiologically activated when psychological or physiological homeostasis does not occur, or when someone thinks he or she is in danger. When someone is in perceived danger, the HPA axis is activated so that he or she can survive (Ehlert et al., 2001, p. 142).

The HPA axis activation process begins when someone perceives a threat. In an instant, the amygdala processes the trigger and sends a message to the hypothalamus (Nickel, 2011). The sympathetic nervous system is activated by the hypothalamus, and the physiological changes (breathing, blood pressure, and heart rate) happen within the body. The hypothalamus also triggers a corticotropin-releasing-hormone (CRH) to be produced and released into the blood stream. The CRH travels to the anterior pituitary gland, connected to the hypothalamus by nerve fibers and located at the base of the brain,
and triggers the release of the adrenocorticotropic hormone (ACTH). The stimulation of ACTH activates the adrenal gland, located on top of the kidneys, to release cortisol, a steroid hormone (specifically a glucocorticoid). The HPA axis has a negative feedback loop. This means that when cortisol is released, it tells the hypothalamus and anterior pituitary gland to stop producing cortisol, because it is already being produced (Ehlert et al., 2001, p.1)

![Diagram of the HPA axis process](image-url)

Figure 10. HPA axis process. CRT = Corticotropin-releasing-hormone. ACTH = Adrenocorticotropic hormone. The amygdala processes the threat and sends a signal to the hypothalamus (HPT), which produces CRT and releases it into the bloodstream. The CRT stimulates the anterior pituitary gland (APG), which releases ACTH. This activates the adrenal gland and produces cortisol. The cortisol has a negative feedback cycle, which prohibits the HPT and APG from producing cortisol.
To summarize, someone perceives he/she is in danger; the amygdala tells the hypothalamus that he/she is in danger; the hypothalamus releases CRH; the CRH goes to the pituitary gland and triggers the release of ACTH; ACTH goes to the adrenal glands and prompts the release of cortisol (Harvard Health Publications, 2011). The HPA axis is able to stop because cortisol creates a negative feedback loop that inhibits the pituitary gland and hypothalamus, which reduces the secretion of CRH and ACTH (Ehlert et al., 2001, p. 142). People with PTSD can have an overactive and prolonged stress response in the HPA axis (Sherin & Nemeroff, 2011). The biology of the HPA axis is known, but what does the HPA axis have to do with trauma? (42; Healthline, 2014a, para 1). Figure 10 illustrates the HPA-axis process.

**HPA axis and trauma.** The behavioral and physiological effect of the cortisol depends on the ability of the cortisol to bind to glucocorticoid receptors. Cortisol is a glucocorticoid, so when it binds to a glucocorticoid receptor, it tells the hypothalamus to stop making cortisol because it is already being produced. The functioning of the HPA axis is controlled by the effectiveness and number of glucocorticoid receptors, and by the negative feedback cycle of the HPA axis (Ehlert et al., 2001, p. 142). Thus, if the negative feedback cycle of the HPA axis is prohibited (due to the number and effectiveness of the glucocorticoid receptors), it will keep on producing cortisol, which can increase stress-like symptoms. Also, if one perceives to be in constant danger, one can experience this cycle dozens of times a day and it takes longer for the HPA axis to return to baseline (Nickel, 2011). The HPA axis contains an important chemical called cortisol. A detailed look at cortisol can give insight into the how it impacts PTSD.
Cortisol is released in response to stress or when someone has low blood sugar. The increase in blood sugar provides someone in the flight-or-fight response to have energy needed to get out of harm’s way (Ehlert et al., 2001, p. 142; Healthline, 2014b, para. 1; Nickel, 2011). Cortisol and adrenaline work together to create flashbulb memories (Kennedy, 2014). Flashbulb memories are vivid snapshot moments when emotionally charged information was heard or an event was experienced (Brown & Kulik, 1977). The purpose of flashbulb memories are to help people remember what to avoid in the future (Kennedy, 2014). However, continued exposure to cortisol damages the hippocampus. This can inhibit learning and the recall of previously stored memories (McAuley et al., 2009). The inability to recall memories can hinder learning and can contribute to repressed memories. Now that the hypothalamus-pituitary-adrenal axis is understood, a look at how trauma affects the amygdala will be identified.

**The amygdala.** The amygdala is associated with how fear is processed. A study conducted by Duke University on returning war veterans who have PTSD showed that a smaller amygdala volume is associated with PTSD (Morey, 2012). The study displayed the amygdala volume does not shrink due to the frequency, severity, or duration of trauma. This indicates that the amygdala does not shrink due to traumatic exposure and that people are born with a certain size amygdala that does not change. Thus, it appears that people with a smaller amygdala are more prone to develop PTSD. However, more studies need to be done in order to conclude this (Morey, 2012, p. 1).

Additionally, Rauch et al. suggests that an exaggerated amygdala response, and a deficit in frontal cortical functioning, can accentuate PTSD symptoms by not being able to regulate the fear response (as cited by McFarlane, 2010, p. 5). This was briefly
explained in the neuroplasticity section stating that stress enhances the synaptic communication between the amygdala itself (Fuchs & Flugge, 2014). Therefore, stress increases the amygdala’s response because it enhances the communication within the amygdala (Fuchs & Flugge, 2014, McFarlane, 2010). Therefore, the amygdala is more efficient, and responds faster to perceived danger (Fuchs & Flugge, 2014, McFarlane, 2010). The amygdala is not the only part of the brain that is impacted by trauma; the hippocampus is also affected.

**The hippocampus.** People with PTSD tend to have a smaller hippocampus (Sherin & Nemeroff, 2011). There is considerable debate on whether reduced size is due to trauma, or if people with PTSD have a smaller hippocampus to begin with, and are, therefore, more prone to develop PTSD. Gilbertson and colleagues studied 40 identical twins (as cited in Sherin & Nemeroff, 2011). One of the twins was a Vietnam Veteran, who was exposed to combat trauma, and the other twin was not (Sherin & Nemeroff, 2011). The results showed that the Vietnam Veterans had smaller hippocampal regions than those without PTSD. However, the non-PTSD twins also had abnormally smaller hippocampal regions. The results indicate that the smaller hippocampal regions could be pre-existing, and at least shows the multifactorial vulnerability for developing PTSD. Despite the cause, there is evidence that people with PTSD have a smaller hippocampus than those who do not have PTSD, and that the difference can prompt PTSD symptoms (Sherin & Nemeroff, 2011).

The hippocampus is involved with encoding new memories, retrieving old memories, and distinguishing past and present memories (Wlassoff, 2015). As mentioned earlier, cortisol decreases the hippocampus’s ability to communicate, because
it causes dendrites to retract. This reduces the neuropathways within the hippocampus, causing it to ineffectively communicate within itself and with other brain structures (Fuchs & Flugge, 2014).

The abnormal functioning of the hippocampus can be why people with PTSD experience flashback, and have a difficult time distinguishing between the present moment and past memories (Wlassoff, 2015). It can also explain the declarative memory deficiencies (not being able to consciously recall memories, facts or knowledge) for those who have PTSD (McFarlane, 2010; Perlman, 1999).

Additionally, the hippocampus is vital in contextual fear conditioning (McFarlane, 2010). A hippocampus that is not functioning properly can result in generalizing the fear from the traumatic event to learned-fear in situations that are not related to the traumatic event. Thus, the person with PTSD has a difficult time distinguishing between safe and unsafe stimuli, and re-experiences the physiological and emotional toll similar to the traumatic event. The results may include hypervigilance, an exaggerated stress response, increased behavioral activation, paranoia, and not being able to prevent other fear associations (McFarlane, 2010, p. 5; Sherin & Nemeroff, 2011).

**Prefrontal Cortex.** Tasks that require complex thinking (problem solving or working-memory tasks) require the function of the prefrontal cortex. Working memory is one’s ability to use current memories or previous memories to guide behavior, thoughts, and emotions. When someone is under acute stress, and feels he/she has little control over the stress, it impairs prefrontal cortex functioning, and improves the functioning of the amygdala. Stress activates the amygdala, which switches the brain’s processing from the prefrontal cortex’s flexible, thoughtful pathway, to the amygdala’s
rapid, emotional, reactive pathway (Arnsten, 2009).

Chronic stress leads to architectural changes in the prefrontal cortex by decreasing the dendrite’s length and number. This decreases the prefrontal cortex’s ability to communicate. Chronic stress disrupts the swift connection between the prefrontal cortex and the hippocampus, which is needed for working memory and decision-making. Changes can occur in the prefrontal cortex after one week of stress, or even after one stressful event. Chronic stress reduces the structures in the brain (prefrontal cortex) that can respond healthily to the stress, and strengthens the structures (amygdala) that foster the stress response. This creates a cycle of dysfunction. The hypoactivity of the prefrontal cortex, (not being able to effectively problem solve, not being able to guide behaviors, thoughts, and emotions by utilizing working memory, and not being able to focus), can explain some of the symptoms of PTSD (Arnsten, 2009).

A near-infrared spectroscopy study by Matsuo and his colleagues examined the relationship between hemodynamic responses of the prefrontal cortex during a cognitive task (2003). The results showed people with PTSD had a decrease in attentional capacity (Matsuo et al., 2003). Matsuo and his colleagues suggest the reduced ability to focus, one of the symptoms of PTSD (APA, 2013), may be the cause of prefrontal dysfunction (Matsuo et al., 2003).

*Cortical thickness.* Geuze et al. (2008) completed a study using computational analysis to measure cortical thickness in the prefrontal cortex. They studied 25 males with PTSD and 25 males without PTSD. The results showed that the cortical thickness in the prefrontal cortex is reduced for people with PTSD. The cortical thickness correlated with memory scores. The thinner the prefrontal cortex, the worse memory outcomes.
Geuze and his colleagues concluded that prefrontal thinning may correspond to functional difficulties for people with PTSD (Geuze et al., 2008).

To summarize, someone perceives he/she is in danger, which is processed by the amygdala. The amygdala informs the HPA axis to release cortisol so that he/she can either fight or run from the danger. If the event was traumatic, cortisol could have made the dendrites retract in the hippocampus and prefrontal cortex, causing communication to be inhibited. Meanwhile, cortisol increases the amygdala’s ability to communicate within itself and with other brain structures, causing the amygdala to be overactive. The overactive amygdala, and an inhibited prefrontal and hippocampus, strengthen the cycle of the startle response. This contributes to the manifestation of the symptoms of PTSD. Thus far, the paper defined trauma and provided information on how trauma impacts parts of the brain, now the paper will discuss how an overactive startle response causes other symptoms of PTSD through conditioning.

**The Psychology Behind the Symptoms of PTSD.**

**Contextual fear conditioning.** Contextual fear conditioning uses the philosophy of classical conditioning. This is when a neutral stimulus (something that produces no reaction) is paired with an unconditioned stimulus (which naturally, physiologically produces an unconditioned response). Conditioning is when the neutral stimulus (NS) no longer needs the unconditioned stimulus (US) to produce the unconditioned response (UR). Thus, the presence of the neutral stimulus produces the unconditioned response on its own. An example can help explain the concept (Alvarez, Biggs, Chen, Pine, & Grillon, 2008).
Ivan Pavlov discovered classical conditioning by observing his dog. He noticed that his dog would start to salivate whenever an assistant entered the room. Salivation is a reflexive response that cannot be consciously controlled. He hypothesized that his dog did this because the dog associated the white lab coat of the assistant with receiving food. He decided to complete a study that used a ticking metronome as the neutral stimulus (NS), food as the unconditioned stimulus (US), and salivating as the unconditioned response (UR). He first presented the ticking metronome (NS) and then the food (US). After several pairings, the dogs eventually associated the ticking metronome (NS) with getting food (US), which produced the unconditioned response (UR), salivating. Thus, eventually, the dog would hear the ticking metronome and start to salivate. The neutral stimulus produced the unconditioned response (Alvarex et al., 2008).

In contextual fear conditioning, the neutral stimulus (or neutral context) can be anything (a particular person, location, smell, situation, sound, etc.), and the unconditioned stimulus is the traumatic event that produced feelings of danger, the unconditioned response. Eventually, the person associates the neutral stimulus (a smell, location, sound, etc.) with fear and danger. For example, if someone is driving on a spring day and he/she has a car accident, he/she might unconsciously associate the smell of spring (flowers) with the danger felt when in the accident. Thus, next year, smelling the spring air again, he/she might uncontrollably feel panic because the brain associated the smell of spring with the car accident, danger (Alvarax et al., 2008).

However, there is another aspect to contextual fear conditioning that magnifies its effect. Contextual fear conditioning is more effective when the neutral stimulus (the smell, room, person, situation) is not always paired with the unconditioned stimulus (the
traumatic event). The unconditioned stimulus is perceived as being unpredictable. Studies show that both humans and animals avoid contexts (environments, situations) when they perceive the unconditioned stimulus as unpredictable. An example of this is when someone is physically abused by his/her significant other in locations or settings that seem unpredictable (the location of abuse can be anywhere: in the privacy of his/her home, a restaurant, with friends, or any other public setting). Therefore, the person associates and generalizes all or most settings as dangerous (Alvarax et al., 2008).

A study by Alvarax and colleges used functional magnetic resonance imaging and virtual reality contexts to study contextual fear conditioning in humans. The results supported evidence that other studies found on animals: contextual fear conditioning activates the hippocampus and amygdala. The neurological process of contextual fear conditioning is when context information (external stimuli, the environment) is processed by the hippocampus (which stores it as flashbulb memories) and joins with information about the unconditioned stimulus (the traumatic event) in the amygdala. Neural plasticity strengthens this fear response through contextual fear conditioning. Thus, the learned fear pathways are strengthened in the brain through the association of neutral stimulus and the unconditioned stimulus (Alvarax et al., 2008).

Another component of contextual fear conditioning includes traumatic memories. Traumatic memories play an important role in the manifestations of symptoms of PTSD. Studies have shown traumatic memory accounts for the gap between the exposure to the traumatic event and the symptoms of hyperarousal and avoidance (McFarlane, 2010). The triggering of memories sustains these symptoms because of contextual fear conditioning. When someone is exposed to trauma, the body physiologically responds to
stress. Reliving or re-experiencing the traumatic event is a prime symptom of PTSD, which has parts of psychophysiological reactivation and psychological distress. Some argue, “PTSD is the failure of the retention and extinction of conditioned fear that is an acquired deficit in the condition” (McFarlane, 2010, p. 5). One way PTSD can be explained is that the stress response is overactive and fires when normally it would not (McFarlane, 2010). Thus, PTSD is when someone cannot control his/her fear response. Besides fear conditioning, sensitization is another way trauma impacts the behavior of someone with PTSD.

**Sensitization.** Sensitization is a consequence of contextual fear conditioning. The number one function of the brain is to keep the body alive (Packer & Gensheimer, 2014). People with PTSD feel like they are in a constant state of survival, and the brain responds to all stimuli as if it is a matter of life or death. For example, if someone believes there is a tiger in a bush, and there is not a tiger, the result is unnecessary stress and anxiety. On the other hand, if someone does not believe there is a tiger in the bush, and there is, the result is death. A brain impacted by PTSD is unable to rationalize or use reason to determine if there really is a tiger, so it automatically wants to survive by assuming there is one (Packer & Gensheimer, 2014). The process of responding to a broader range of stimuli, and also having an increased response to the stimuli, is called sensitization.

Sensitization is the process in which the individual becomes reactive to minor cues, which normally would not have triggered a reaction. The individual may not recognize the cues that cause the psychophysiological, neurohormonal, and emotional distress. People with PTSD react to the potential presence of threat with increased
intensity, which ultimately develops into a pattern of generalized over-reactivity to stimuli. Since their range of triggers has increased, and their reaction to those stimuli is magnified, it cements the cycle of reactivity (McFarlane, 2010; Van Der Kolk et al., 1996). A look into how sensitization happens physiologically can help explain this phenomenon.

_The physiology of sensitization._ The neuronal circuits in the limbic system (hippocampus, amygdala, hypothalamus, thalamus) can become sensitized after repeated stimulation, so the firing thresholds are lowered and produce a behavior which would not have been previously produced. An example of this is when someone who has PTSD gets tapped on the shoulder. Before the trauma, he/she would have simply just turned around. Now, this person automatically throws his or her arm back in self-defense. The fight-or-flight-response threshold was lowered and produced a behavior (throwing the arm back) that had not previously existed. Hence, the amygdala being repeatedly stimulated causes prolonged alterations in neuronal excitability (Van Der Kolk & Saporta, 1991). Sensitization is not the only way trauma can impact someone with PTSD; memory can also be impacted.

_Memory._ One intrusion symptom of PTSD is memory deficits. Learning and extinction do not function properly with those who have PTSD. One reason may be due to altered norepinephrine activity. Norepinephrine heightens the encoding of fear memories. Thus, increased norepinephrine activity can lead to enhanced encoding of traumatic memories (Sherin & Nemeroff, 2011).

According to Pierre Janet (as cited by Van Der Kolk & Saporta, 1991), trauma can disrupt information processing, and hyperarousal can cause memory disturbances by...
interrupting information processing. When trauma is severe, it can cause memories to be split from consciousness. According to Janet, these memories are stored in bodily sensation or visual images. The result can be manifested in nightmares, flashbacks, behavioral reenactments, physiological reactions, and emotional states (Van Der Kolk & Saporta, 1991). As mentioned earlier, trauma can also cause declarative memory deficiencies (Sherin & Nemeroff, 2011). Related to memory is dissociation, a severe symptom of PTSD.

**Dissociation.** Some researchers believe dissociation can be explained by how memories are encoded, stored, and retrieved. They also argue that there are three types of memories (Perlman, 1999). First are declarative memories, which are integrated and can be articulated verbally. Declarative memory is found in the hippocampus, and typically starts to function around three or four years of age. Second are procedural memories, which are encoded kinesthetically and through bodily experiences. These memories are present at birth. Theoretically, memories before age three are encoded and stored in an unconscious, kinesthetic-oriented memory system because verbal communication had not developed. The last memory is traumatic memory, which includes unprocessed memories typically stored without being integrated into overall memory (Perlman, 1999,). As mentioned by Janet (1889), the unprocessed and non-integrated traumatic memories can manifest symptoms of PTSD (Van Der Kolk & Saporta, 1991). The definition of trauma, the parts of the brain impacted by trauma, and the neurological and physiological causes for PTSD symptoms were provided. Now, the paper will focus on music, and how music is heard.
The Biology of Hearing

The parts of the ear. In order to understand the process of how music is heard, one needs to understand the parts of the ear. The auditory system has three parts, the outer, middle, and inner ear (Kostek, 2005). The outer ear consists of the ear you can see and the ear canal (Critchley & Henson, 1977). The middle ear includes the tympanic membrane (the eardrum) and the tympanic cavity, which is the air below and around the three bones in the middle cavity. These bones are called the malleus (hammer), the incus (anvil), and the stapes (stirrup). Together, they are called the ossicular chain. The malleus is attached to the inner surface of the eardrum. The incus is connected to the malleus, and the stapes to the incus. The final bone in the ossicular chain, the stapes, is connected to the oval window. The oval window is the beginning of the inner ear (Critchley & Henson, 1977). The inner ear has three parts, the vestibule, the semicircular canals, and the cochlea. Only the cochlea aids in hearing; the
other two are used for balance (Critchley & Henson, 1977). Next, the process of physically hearing music will be explained to understand how sound waves are changed and computed in the brain. Figure 11 shows the parts of the outer, middle, and inner ear.

The process of hearing.

The purpose of the outer ear is to collect and channel sound to the middle ear through the auditory canal (Kostek, 2005). Sound in the outer ear is in the form of a pressure wave (sound wave) that has interchanging high and low pressure regions. Figure 12 displays a pressure wave. In a sound wave, the high air pressure regions are called compressions, and the low-pressure regions are called rarefaction. The ear canal is structured so it can amplify sounds with frequencies of approximately 3000 Hz. Sound waves cause the tympanic membrane (the eardrum) to vibrate, making a compression wave. The eardrum and ossicular chain are what changes the sound wave into a compression wave. Compression (the high-pressure part of a compression wave) pushes the eardrum inward, and rarefaction (the low-pressure part of a compression wave) forces it outward, causing the eardrum and ossicular chain to vibrate at the frequency of the sound wave.

The purpose of the middle ear is to transform the sound wave into vibrations that are sent through the ossicular chain to the oval window of the inner ear. To summarize the process thus far, a sound wave travels through the auditory canal, which hits the
eardrum, causing it and the ossicular chain to vibrate. This new vibration changes it from a sound wave to a compression wave (Critchley & Henson, 1977; Kostek, 2005).

The inner ear contains the cochlea, which is what converts the compression wave into audible sound. The cochlea is filled with fluid and the inner surface is lined with over 17,000 hair-like nerve cells. There are three rows of outer hair cells, and one row of inner hair cells, all varying at different lengths. The vibrating stapes connects to the oval window, causing the nerve cells of the cochlea to move. Each nerve cell corresponds to a specific frequency. When a nerve cell frequency matches the frequency of the compression wave, the nerve cell moves and produces larger vibration amplitude. When this happens, it prompts the nerve cell to elicit an electrical impulse to the auditory nerve. From there, the electrical impulse travels to the brain, where it is interpreted and decoded (Kostek, 2005). The next part of the paper will show the validity of utilizing music in treatment by providing information on a profession dedicated to it, music therapy.

**Music Therapy**

**History of music therapy.** The National Association for Music Therapy was founded in 1959 (Davies & Booth, 1978). The first documented reference to music therapy was found in the *Columbian Magazine* titled “Music Physically Considered” in 1789. In the early 1800s, two medical dissertations were completed: The first dissertation recorded music therapy intervention in an institutional setting; and the second recorded a systematic experiment for music therapy (American Music Therapy Association, [AMTA], 2014b).

In the United States, the need for music therapy appeared after World War I and World War II. Community musicians, both professional and amateur, played music at
the Veterans hospitals. The physical and emotional responses to music lead to nurses and doctors requesting musicians to be hired by hospitals. It became apparent the need to train musicians before entering the hospital. Thus, a new curriculum was developed (AMTA, 2014b).

In the 1940’s, there were three main founders in making music therapy an organized, clinical profession. Ira Altshuler was a psychiatrist and music therapist who advocated for music therapy for thirty years; Willem van de Wall wrote the first “how to” music therapy text and initiated the use of music therapy in state-funded facilities; and E. Thayer Gaston, “The father of music therapy,” was vital in having music therapy become an organizational and educational entity. In 1944, Michigan State University “established the first academic program” for music therapy (AMTA, 2014b).

**Definition of music therapy.** According to the AMTA, “Music Therapy 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” (2014a). It is a recognized health profession that uses a therapeutic relationship, along with music, to address the emotional, social, cognitive, and physical needs of patients. Like social workers, music therapists assess the needs and strengths of the client. Music therapists determine what intervention is appropriate: singing, making music, listening to music, or moving to music. Music therapy strengthens the client’s capabilities. It also allows people to express themselves nonverbally (AMTA, 2014a).

A brief explanation of music therapy was provided in order to understand a profession that utilizes music for healing. The next section will explain the scientific
procedure that allows researchers to know humans anticipate musical conclusions. Knowing that humans subconsciously make musical prediction and anticipate musical conclusions is important because it is the foundation on why humans feel emotions during music.

**Music**

“Modern neuroscience has revealed that music is a highly complex and precisely organized stimulus that interacts with the human brain and modulates synaptic plasticity and neuronal learning/readjustment in the brain” (Shuai-Ting et al., 2011, p. 34).

In order to survive and evolve, the brain tries to accurately predict future events. Predicting future events also occurs with music, and is what produces emotion (Hallman, Cross, & Thaut, 2009). Emotions from music are due to expectation and anticipation from what will come next in a sequence of musical factors (Koelsch, 2014). The impact of lyrics on emotion was not included in the study. The scientific evidence behind emotions being produced by prediction is captured through an electroencephalogram (EEG).

**EEG studies.** An electroencephalogram (EEG) is used to reveal how the auditory system makes musical predictions. When a neuron fires, an electrical field potential is created by the flow of sodium and potassium ions (Hallman et al., 2009). When a lot of neurons fire at the same time, the sodium and potassium ions create a field potential, which is measured by an EEG. The field potential shows that neurons were fired. Sensors are placed on the scalp to measure the field potential. An event-related potential (ERP) is the measured brain response from the EEG. It is shown in wave form with milliseconds being the x-axis and the field potential being the y-axis. (Hallman et al.,
The EEG is the scientific procedure; the ERP are the results from the EEG, which are shown in waveform. An ERP is important because the activation of a field potential can show what prompts neuronal activation in music. Figure 13 illustrates the components of an ERP. The y-axis is reversed: the positive voltages are plotted below the x-axis and the negative voltages are plotted above the x-axis. This is done so the field potential is easier to read, because it creates an upward spike (Luck, 2005).

In the study, the same sound was repeatedly played, and then an unexpected sound was played. The EEG measured the ERP of the stimulus (sound) change. Researchers wanted to measure how the brain responds when an unexpected sound occurs, to see if humans anticipate musical conclusions.

When the sound changed, the ERP had a negative, frontal waveform. This is significant because field potentials are present (meaning enough neurons were fired to be measured) when a negative waveform is present.

![Figure 13. Components of an event-related potential (ERP) in an EEG. The potential is measured in volts ranging from -300 to 300. P1- the first positive waveform; N1- the first negative waveform; P2- the second positive waveform; N2- the second negative waveform; and P3- the third positive waveform. N1 is the ERP because it has a negative waveform of -300 volts. The ERP shows that neurons were fired. Adapted from “Components of ERP”, 2008, Mononomic (Talk) 16(12), from http://en.wikipedia.org/wiki/File:ComponentsofERP.svg.](image-url)
in an ERP (the spike in figure 13). In this study, when the ERP peaked, it is called a mismatch negativity (MMN). The peak only occurred when a mismatch between the expected and unexpected stimulus happened. A MMN shows neurons were fired when an unexpected sound occurred (Hallman et al., 2009). The MMN showed what occurs in the brain when an unexpected sound is presented instead of an expected sound.

The study also tested what would happen if other aspects of music were changed from what was expected. The researchers tested frequency or pitch change in a tone; timbre changes (example, a piano playing a C chord at the same pitch and loudness verses a guitar playing a C chord at the same pitch and loudness); variations in duration of tones (even for a few milliseconds); the spatial location of sounds (where sounds are located); an omission of an expected sound; and increases and decreases in intensity. All of the tested changes produced an MMN, which shows that a field potential was present during the unexpected changes. Thus, every time there was a change of an unexpected sound, neurons were fired and only fired when the change occurred. This supports the theory that the brain tries to predict what will come next in music (Hallman et al., 2009).

Some may argue someone could be thinking of something else instead of the sounds while he/she participated in the study. This may be true. However, the brain’s reaction to the different sound stimulus is subconscious and cannot be changed by cognition (Hallman et al., 2009). Therefore, the brain subconsciously predicts what will come next. When the prediction is not accurate, the brain is stimulated. The results from the ERP are significant because it:

Reflects a process in the auditory system for predicting future sound events on the basis of the recent past, and the brain’s reaction when these predictions are not fulfilled. We propose here that music fundamentally relies on expectation-realization processes, and that these processes are reflected in the MMN, whose
main generators are in the secondary auditory cortex (Hallman et al., 2009, p. 172).

This last section showed the science behind the brain making musical predictions. These predictions and anticipation are subconscious. They are automatic and cannot be controlled by thoughts. This is important because the next part of the paper will focus in detail on how emotion is evoked through music.

**Music and emotion.** Music theory is based on Western music principles (Davies & Booth, 1978). The music studied focused on instrumental music, not music with lyrics. Principles of musical tension, musical expectancy, and emotional cogtagion (expressing emotion through music) are due to music itself, and not due to the memories associated with them (Koelsch, 2014). This is because music is highly structured through time, space, and intensity in the manifestation through single tones, chords, and sequential sounds (such as melodies). The unconscious perception of musical structure (time signature, phrasing, and organization of musical phrasing) arouses emotions that are not due to memories. The different emotions that surface (for example, pleasant or unpleasant) are due to musical tension (Koelsch, 2014).

**Musical tension.**

**Acoustic factors.** One factor of musical tension includes acoustic factors, such as loudness, timbre (the distinct sounds produced by different instruments or voices), sensory dissonance (when notes sound like they are in disagreement), and sensory consonance (when notes are in agreement). Sensory consonance and dissonance are subconsciously processed in the auditory brainstem and regulates changes in the laterobasal amygdala (see table 1 for a summary of brain structures and their relation to music). The combination of acoustic information and musical structure leads to low-
level musical predictions and inferences. The subconscious interest in musical structure (for example, thinking about why someone organized a piece of music in a certain way), leads to one aspect of musical tension (Koelsch, 2014). Another component of musical tension is the perceived stability of a musical piece.

*Stability of structure.* Another component of musical structure that impacts musical tension is the stability of musical structure. A stable beat produces relaxation, while straying from the stable beat produces musical tension. Examples of straying from a stable beat are accelerando (speeding up the beat), ritardando (slowing the beat down), syncopations (accenting an off-beat), and off-beat phrasing (not starting a musical phrase on a down beat). In tonal music, structure is also represented by a tonal centre (how one specific tone is used in relation to other tones, such as the key of a song). Straying away from the tonal centre creates tension; moving back to the tonal structure creates relaxation (Koelsch, 2014). An example of this is when composers use the 7th note in the scale as a leading tone to lead back to the root note (in the key of C, playing a B note that leads to a C note in a melody). The stability of musical structure is one component of musical tension.

*Predicting the probability of chords.* Another component of musical tension is the ability to predict the probability of the next chord or tone. In music, there is a probability of certain chords following other chords. For example, after a dominant chord (the 5th chord in a scale) the most likely chord is a tonic chord (the first chord in a scale). Thus, when a dominant chord is played (in the key of C, a G chord), the entropy of the probability distribution is low because one has a high prediction of what will come next, which will be a tonic chord (in the key of C, a C chord). When a tonic chord is
played, the uncertainty of the probability for the next chord increases to an “intermediate”
on the entropy of the probability distribution. For submediant chords (in the key of C, an Am chord), the entropy of the probability distribution increases to “high” because the
mind is not as sure what chord will come next. In other words, when the predictability of
the next chords decreases, it produces musical tension and anticipation, which produces
emotion (Koelsch, 2014). Figure 14 displays the degree of chords in the key of C.

Predicting the probability of tones. Chords are not the only component of
musical tension; progressing tones and harmonies also play a role. Again, the certainty,
or uncertainty, of tonal or harmonic predictions creates tension. When tones or
harmonies make it difficult to make musical predictions, it creates suspense on whether
the prediction will come true, and produces musical tension. When one’s prediction
comes true, it is perceived as rewarding. When the prediction is false, one will feel
unpleasant emotions. Even if one has heard a song that violates the prediction of musical structure, the listener still produces the music-evoked emotions, even if one knows the musical violation is going to happen.

*Skin conductance response.* A study measured the amplitude of skin conductance responses and self-report of felt tension when unexpected chords were played (chords that violate music theory). Skin conductance response measures the sweat glands of the skin because when the sympathetic nervous system is aroused, it actives the sweat glands. Thus, conductance of the skin can be used as an indicator of emotional responses (Martini & Bartholomew, 2011). In one study, results showed there is a direct correlation between the amplitude of skin conductance responses and felt tension (Koelsch, 2014). The amplitude of skin conductance response was large when the degree of unexpectedness was large. This unexpectedness stimulates changes in the superficial amygdala and the lateral orbitofrontal cortex. These brain changes correlate with rating of felt tension. Thus, the changes in the superficial amygdala and lateral orbitofrontal cortex correlate with feelings of unexpectedness (Koelsch, 2014).

*Musical intensions of composers.* Composers can intentionally include musical tension before a transition phase, so that when the composer resolves the tension, it leads to pleasantness. However, if the musical tension is not resolved, it is perceived as unpleasant. Therefore, a breach in expectancy will either produce resolution or unpleasantness. For example, when a composer includes a dominant seventh chord (in the key of C, a C chord with a B flat added to it), followed by a tonic chord (in the key of C, a C chord), the musical tension is resolved, and produces relaxation by resolving the anticipated chord. The same process is very similar for tonal (melody) structures. When
a musical piece uses the structure of build-up, musical breach, transitory phase, and resolution, the anticipation of resolution produces emotions, and then relaxation when the melody is resolved. Again, if the melody is not resolved, it produces unpleasant emotions (Koelsch, 2014). The components of musical tension were explained. Another component of music producing emotions is emotional contagion.

*Emotional Contagion.* An aspect of music-evoked emotions is emotional contagion. This is expressing emotion through music. Physiological changes occur while listening to “happy” music (in western music, a fast tempo and high pitch notes), such as, activating zygomatic muscles (the face muscles used to smile), increase skin conductance, and rate of breath. Sad music (minor songs, slow-paced, and lower pitched notes) leads to the activation of corrugator muscles (the muscle between the eyebrows that are used to scowl). Happy music can physiologically cause one to smile, while sad music can cause one to frown (Koelsch, 2014).

To conclude, some emotional responses to music are due to musical expectations and anticipation. These expectations come from musical patterns in which the listener may not be aware. The structure of music has the listener anticipating melodic resolutions. The anticipation, resolution, or unexpected musical breach produces emotion within the listener (Hallman et al., 2009). If the music is resolved, the listener feels pleasant feelings. If the expected and anticipated chord or tone meets the listener’s expectation, it is associated with reward. When the listener’s expectation or anticipation is not met, it is associated with unpleasantness (Koelsch, 2014). Next, the paper will focus on how music neurologically impacts the brain.
Neuroscience of Music

Music is a treasured tool because it can evoke changes in emotion and stimulate the brain (Koelsch, 2014; Moffic, 2008). There is evidence that music can induce changes in subjective feelings, automatic stimulation, hormone arousal, emotional motor expression (smiling), and action movements (clapping, foot tapping, singing, dancing, and playing an instrument) (Koelsch, 2014). A meta-analysis conducted by Stefan Koelsch (2014) used functional neuroimaging studies, found parts of the brain critical in producing emotion are stimulated by music. These brain structures include the amygdala,
nucleus accumbens, hypothalamus, hippocampus, insula, cingulate cortex and orbitofrontal cortex (Koelsch, 2014). Moffic’s study concluded that biologically, while listening to instrumental music, neurons are fired in the brain structures involved with motivation, reward, and emotion. Reward, motivation and emotion are controlled in the limbic system (hippocampus, amygdala, hypothalamus, and thalamus) and cerebellum (Moffic, 2008). Shaui-Ting and his colleagues found through neuroimaging techniques, listening to music can stimulate the mesocorticolimic system (nucleus accumbens, ventral tegmental area, hippocampus, amygdala, orbitofrontal cortex, and ventromedial prefrontal cortex) (see figure 15) (Shuai-Ting et al., 2011). Feelings of unpleasantness were reported when the hippocampus, parahippocampal gyrus, amygdala, and temporal poles were activated (see figure 16) (Shuai-Ting et al., 2011). When words are added, music is also processed in the cerebral cortex (Moffic, 2008). This paper will explore in detail how the amygdala, hippocampus, and reward pathways are stimulated through music. It will look at how music can prompt neuroplasticity, neurogenesis, and how music impacts socialization.
The superficial amygdala plays the largest role in processing musical stimuli, the dopaminergic mesolimbic rewards pathway (containing the nucleus accumbens) is associated with musical pleasure, and the hippocampal formation is largely responsible for emotions connected to social attachments (Koelsch, 2014). “The power of music to change the neuronal activity within these brain structures has implications for the development of music-based therapies for the treatment of neurological and psychiatric disorders associated with dysfunction and morphological abnormalities in these structures” (Koelsch, 2014, para. 4). The amygdala is attached to other neural networks. It is in a central location to regulate emotional networks. This can be done by initiating, maintaining, or terminating emotions (Koelsch, 2014).

**Amygdala.**

*The superficial amygdala and communication.* The olfactory bulb (receives sensory input) sends information to the superficial amygdala, where the information is processed. The superficial amygdala plays the largest role in processing musical stimuli. The signals received from the olfactory bulb produce basic, universal emotions. The results from the meta-analysis showed the superficial amygdala is sensitive to stimuli with social significance (faces, music, sounds, voices), which can assist with communication. This conveys the superficial amygdala is perceptive to stimuli with basic socio-affective information (emotions that come from socializing) (Koelsch, 2014).

*The superficial amygdala and new situations.* The meta-analysis also showed the superficial amygdala and the nucleus accumbens, as well as the superficial amygdala and the mediodorsal thalamus, had stronger connections during joy-evoking music than during fear-evoking music. The study concluded the superficial amygdala, nucleus
accumbens, and the mediodorsal thalamus form a network. This network regulates someone’s natural inclination to approach new situations or to withdrawal from them when stimulated by socio-affective stimuli (Koelsch, 2014).

*Laterobasal amygdala on negative and positive rewards.* The laterobasal amygdala receives information from the auditory cortex and the auditory thalamus. It is the main amygdala input structure for auditory and sensory information. The laterobasal amygdala is involved with the evaluation and learning of negative and positive stimuli, as well as creating expectations of reinforcements that guides behavior. Studies showed that the laterobasal amygdala was activated during joyful music in some studies and activated for unpleasant or sad music in other studies. The differing results show the laterobasal amygdala was most likely coding the positive or negative reward value of musical stimuli (Koelsch, 2014). The amygdala is not the only brain structure stimulated by music; the hippocampus is also activated.

**Hippocampus.**

*The hippocampus and the HPA axis.* In studies where very familiar pieces were studied, hippocampus activation was associated with tenderness, peacefulness, joy, thrill, or sadness. Other studies showed that hippocampal activity were activated with joyful, unpleasant, and fearful emotions. Koelsch’s meta-analysis showed this evidence to be consistent with the hippocampus’s role in the regulation of the hypothalamus-pituitary-adrenal (HPA) axis, which controls the stress response. Connectivity between the hypothalamus and the hippocampus have been shown in neuroimaging studies to be activated by music-evoked joy. This supports the notion that the hippocampus is
involved with music-evoked positive emotions that have endocrine (hormonal) effects on reducing emotional stress, such as decreased cortisol levels (Koelsch, 2014).

**Damage to the hippocampus.** Structural damage to the hippocampus is believed to be due to stress-induced damage of hippocampal neurons and reduced neurogenesis in the dentate gyrus (located in the hippocampus) (Koelsch, 2014). For people who have PTSD, there is a relationship between decreased hippocampus volume and the ability to experience tender, positive emotions. This was shown by studies that examined the relationship between tender positive emotion (love, joy, compassion, and empathy) and neural relationships within the brain. The studies found there is a positive correlation between an individual’s tendency to experience positive emotion and hippocampal volume (Koelsch, 2014). If one’s hippocampal volume is low, one’s ability to experience positive emotions is low.

**The hippocampus, sound stressors, and oxytocin.** The meta-analysis showed that chronic sound stressors from the environment can lead to functional and structural changes in the hippocampus and amygdala (Koelsch, 2014). Studies also show that the hippocampus contains oxytocin receptors that regulate oxytocin into the bloodstream by the pituitary gland (Koelsch, 2014). Oxytocin is known as the “bonding hormone” due to its ability to prompt feelings of intimacy and attachment between couples. It is released during and after childbirth. When oxytocin is released, it can increase feelings of trust and has the ability to reduce cortisol. A calmness one feels while listening to music can be due to oxytocin (MacGill, 2014). The secretion of oxytocin during music can also play a role in social attachments (Koelsch, 2014).
**The hippocampus and social attachments.** The hippocampus is involved with the development and maintenance of social attachments. Attachment-related feelings (feelings that come from being attached to someone, such as love), and the emotions that come from social attachments (happiness), have a positive chemical bond formation, which leaves one to conclude this occurs in the ventral striatum. The ventral striatum receives messages from the hippocampus. On the opposite side of the spectrum, loss of social attachments can lead to sadness. When emotions are produced from participating in music socially (listening with a group, playing music in a group, moving in unison with a group), the hippocampus plays a role in the production of those emotions (Koelsch, 2014). The hippocampus is not the only part of the brain stimulated by music. Reward pathways that include the nucleus accumbens, which are involved with the release of dopamine, are also activated by music.

**Dopamine.** Dopamine, a chemical that communicates between neurons, is released in the nucleus accumbens (Moffic, 2008; Shuai-Ting et al., 2011). The nucleus accumbens plays a significant role in the cognitive processing of motivation, pleasure, reward, reinforcement learning (conditioning), and addiction (Malenka, Nestler, & Hyman, 2009). It is involved with primary rewards (food, sex, drink) and secondary rewards (money, power). It plays a role in representing pleasant and unpleasant sensations, and initiates behavior that will lead to the reward (Koelsch, 2014). Figure 17 displays the dopamine rewards pathway.
A study conducted by Benovov, Dagher, Larcher, Salimpoor, and Zatorre specifically measured if dopamine is released while listening to music. Music is capable of producing intensely enjoyable emotional responses. Previous neuroimaging studies show that listening to pleasurable music stimulates emotional and reward circuits within the brain (the ventral striatum [bottom part of the striatum]). Pleasurable moments are difficult to quantify; however, physiological changes occur during extreme states of pleasure (Benovov, Dagher, Larcher, Salimpoor, & Zatorre, 2011).

Autonomic nervous system arousal (respiration rate, heart rate, electrodermal skin conductance, blood volume pulse amplitude, and peripheral temperature) was used to determine the precise time of peak emotional responses to music. This was coupled with positron emission tomography (PET) scans and fMRI to determine if dopamine is released during music, and if so, if it is due to the anticipation of the reward or the reward itself. Results showed that there is endogenous dopamine released during peak emotional arousal in the striatum while listening to music. Additionally, results indicated that the caudate nucleus released dopamine during the anticipation of music and the nucleus

![Dopamine rewards pathway](http://www.michelleinc.com/blog_tools_tips/youre_wired_to_take_chances)
accumbens released it during the emotional peak of music (Benovov et al., 2011). Music also stimulates reward pathways, which also release dopamine.

**Reward pathways.** The nucleus accumbens is apart of the mesolimbic dopamine system, a reward pathway. The mesolimbic dopamine system is the most important reward pathway in the brain. It controls one’s response to rewards, and thus, determines motivation and incentive drives. Simply, when the mesolimbic dopamine reward pathway system is activated, one will repeat the behavior the individual did to get the reward (Icahn School of Medicine at Mount Sinai, n.d.).

**Reward pathway in music.** The nucleus accumbens, caudate nucleus, ventromedial orbitofrontal cortex, pre-genual cingulate cortex, amygdala, anterior insula, and the mediodorsal thalamus are activated in response to music-evoked emotions. The pattern of these seven brain structures being activated, are the exact same pattern of activation in response to food, sex, and money, which are primary and secondary rewards. These brain structures serve as an evolutionary reward network that ensures the survival of the species (Koelsch, 2014). Thus, music-evoked emotions are correlated with primary and secondary rewards. The process of the reward pathway starts with the ventral tegmental area (VTA) (Icahn School of Medicine at Mount Sinai, n.d.).

**Process of the reward pathway.** The VTA contains dopamine, and informs the human if a stimulus is rewarding or not. The VTA sends dopamine to the ventral striatum, which controls the effects of the reward. The amygdala establishes the association between the environmental cues and the reward or aversive event. The hippocampus stores the learned event. The hypothalamus controls any movement necessary due to the reward or aversive event (Icahn School of Medicine at Mount Sinai,
n.d.). Next, the relationship between music, neuroplasticity, and neurogenesis will be explained.

**Music, neuroplasticity, and neurogenesis.** Brain-imaging techniques show music activates networks in the brain. Through biomedical research, it has been demonstrated that “music is a highly structured auditory language involving complex perception, cognition, and motor control in the brain, and thus it can effectively be used to retrain and reeducate the injured brain” (McIntosh & Thaut, 2010, p. 2). Helping the injured brain involves activating the networks that process music, because these networks also process other functions (McIntosh & Thaut, 2010). Secondly, music learning changes the brain (McIntosh & Thaut, 2010).

The areas of the brain used in music are the same areas active when processing language, auditory perception, attention, memory, executive control and motor control (McIntosh & Thaut, 2010). Music can be used to not only activate these areas, but it can also be used to develop “patterns of interaction” between them (McIntosh & Thaut, 2010, p. 3). For example, Broca’s areas of the brain (located in the left hemisphere in the prefrontal cortex) is used to scrutinize the structure of a sentence and is the same area of the brain used to process the structure of a musical composition, such as a wrong note. This area of the brain also processes order of bodily movement and is also used for tracking musical rhythms. Additionally, the Broca’s area is activated when putting thoughts into words (McIntosh & Thaut, 2010). “Scientists speculate, therefore, that Broca’s area supports the appropriate timing, sequencing, and knowledge of rules that are common and essential to music, speech, and movement (McIntosh & Thaut, 2010, p. 4).
Findings show that learning music changes the brain by having the auditory and motor areas communicate more effectively, resulting in these areas physically growing larger. Studies showed after a beginner piano player had a few weeks of training, the area of her brain that controls hand movement became more interconnected and larger in size. “It quickly became clear that music can drive plasticity in the human brain, shaping it through training and learning” (McIntosh & Thaut, 2010, p. 4). Studies that show music can prompt neuroplasticity and neurogenesis can be used to help heal the brain.

Music and Social Attachments

The attachment-related emotions evoked through music are related to the social functions of music, such as the creation, maintenance, and strengthening of social attachments. The history of music, and the current social contexts of music, involves one actively participating in the musical activity: for example, going to a concert and clapping, dancing, tapping one’s foot, playing an instrument, or singing in a group. These activities involve social functions, such as communication, collaboration, and social cohesion, which serve to ensure the continuation of the human species. When people are involved with music in a group (playing, dancing, tapping, listening), they share a goal, are focusing on the same entity, have a shared intention, and have mutual emotionality. Simply listening to music as a group prompts social engagement (Koelsch, 2014).

Studies show when people move together to a beat, it increases trust and cooperation in both adults and children. Moving in unison increases group identity. A person participating in cooperative behavior is pleasurable, and activates the nucleus
accumbens. Group cooperation increases trust and one’s likelihood of future cooperation (Koelsch, 2014).

To summarize the main parts of the paper up to now, the presence of stress (cortisol) causes the dendrites in the hippocampus and prefrontal cortex to retract. This reduces the number of synapses and weakens the neuropathways that involve the hippocampus and prefrontal cortex. The prefrontal cortex controls complex functions, such as emotional regulation, planning for the future, decision-making, integrating information, and reflecting. The hippocampus is involved with learning and memory. If these two structures are not working effectively, it can decrease communication, causing higher functioning to be weakened.

On the other hand, cortisol strengthens communication within the amygdala, making it respond to stimuli faster than it did before. Since cortisol reduces the functioning of the hippocampus and prefrontal cortex, the hippocampus and prefrontal cortex have a difficult time determining if the stimulus is dangerous or not. In order to survive, the amygdala presumes the stimulus is dangerous, activating the HPA axis. This produces even more cortisol, and the cycle continues. Conditioning strengthens the fear response and produces symptoms of PTSD. However, there is hope the cycle can stop because the brain is able to form new pathways (synapses) and produce new neurons. If stress subsides, the synapses can return to their original state.

At the most basic level, music is a pressure wave. The ear turns the pressure wave into a compression wave, where it can be processed in the brain. Music stimulates the superficial amygdala (processes stimuli that have social significance [faces, sounds, voices]); the laterobasal amygdala (evaluates stimuli and creates expectations for
behavior reinforcement); the hippocampus (associated with joyful music, decreased cortisol levels when joyful music was played, and plays a role in social attachments); and the reward pathway, which releases dopamine. Music can play a role in stopping the cortisol-stress cycle because brain-imaging techniques show that music can trigger neuroplasticity and neurogenesis. The next part of the paper will explore how this information can be utilized in social work practice.

**Social Work Practice Implications**

The implications are directed towards all social workers, even ones who do not have a musical background, or who know how to play an instrument. Music helps adults regulate and experience emotions and mood (Koelsch, 2014) and can be used to neurologically help clients function. Music can be used in social contexts, which can address symptoms of PTSD. Implications for neurological impacts of music will be explored first.

**Neurological Implications**

**Provoking moods.** Since feelings, hormones, emotional motor expression (smiling), and action movements (clapping, foot tapping, dancing, etc.) can be induced through music (Koelsch, 2014), they can be regulated by music. Therefore, music can be used to deliberately provoke a certain mood, or try to stimulate certain hormones. For those with PTSD, this could prove to be a vital tool for self-regulation.

**Identifying emotions.** The superficial amygdala processes musical stimuli, produces universal emotions, and is sensitive to stimuli with social significance (Koelsch, 2014). Therefore, music can be used to help prompt a basic emotion between people, and/or be used as a tool to help communicate basic emotions. This could be applied to a
group setting, or between the therapist and client, when trying to address symptoms of PTSD that interfere with socialization or/and emotional self-disclosure. For example, a therapist could model emotional self-disclosure by playing an instrumental song he/she perceives to be sad. If the therapist does not play an instrument, he or she can play the song through a media device. The therapist can name the emotion he/she is experiencing, sadness. This same exercise can be done with an instrumental song the therapist perceives as happy. The client can then take the role of sharing a song perceived to be a basic emotion (a song evoking feelings of sadness, happiness, fear, etc). Naming an emotion can prompt self-disclosure or be a good exercise in a group setting.

**Reducing social avoidance.** Furthermore, the superficial amygdala, nucleus accumbens, and the mediodorsal thalamus form a network that has strong connections when joyful music is played. This network is used to regulate one’s natural inclination to approach new situations or to withdraw from them when in a social situation (Koelsch, 2014). Perhaps joyful music can be played to strengthen this network, and be used to help aid the person who withdraws from social stimuli to calm him/her in these situations. Therefore, social avoidance could be reduced through joyful music.

**Reducing the startle response.** The laterobasal amygdala processes positive and negative stimuli, guides processes for reinforcing behavior, and codes the positive or negative reward value of musical stimuli (Koelsch, 2014). A symptom of PTSD is having difficulty with processing stimuli and being conditioned to presume danger. Thus, music could potentially play a role in balancing the processing of stimuli, so the amygdala resumes to the state it was before the traumatic event conditioned it. This could reduce the amygdala’s startle response, which will decrease cortisol production.
Studies will need to be done to determine if positive stimuli (a calming, happy, or pleasant song picked by the client) being played while processing negative stimuli (the events of the trauma), can help balance the startle response long-term. Another study would need to be done to explore if exposure to a positive stimulus (pleasant song) during times of PTSD symptoms reduces the severity of the symptoms.

**Reducing cortisol production.** The hippocampus is stimulated by music and the activation of the hippocampus is associated with producing peaceful, joyful, unpleasant, or fearul emotions (Koelsch, 2014). The connections between the hippocampus and the hypothalamus in the hypothalamus-pituitary-adrenal (HPA) axis have been proven through neurological studies to be stimulated by music-evoked joy (Koelsch, 2014). There is evidence that the activation of the hippocampus, which stimulates the HPA axis through positive emotions that come from music, can have hormonal effects that reduce cortisol levels (Koelsch, 2014). The HPA axis and the production of cortisol play a significant role in PTSD (Ehlert et al., 2001). Since music is associated with reducing cortisol levels through the HPA axis, music can be purposefully used to help reduce the stress response in PTSD. This can be done during a therapy session, if a client becomes too triggered and needs assistance stabilizing. It can also be used as a tool for the client to reduce cortisol levels while he or she is out of the office. It could be included in crisis planning to help the client self-regulate.

**Neurogenesis in the hippocampus.** Studies show people who have PTSD have a decreased hippocampus volume and have a reduction in experiencing positive emotions (Koelsch, 2014). Neurogenesis and neuroplasticity have been shown to be stimulated by listening to music or by playing music (McIntosh & Thaut, 2010). Therefore, music can
be used to stimulate the hippocampus, potentially prompt neurogenesis, and produce a larger hippocampus. The change in the hippocampus could increase positive emotions, regulate negative emotions, or help balance the HPA axis so that it does not produce cortisol. Studies will need to be completed to determine if music can help during exposure-based treatments.

**Neuroplasticity.** Additionally, strengthening “patterns of interaction” (McIntosh & Thaut, 2010) between the areas that are stimulated by music, and increasing the size of these brain structures, can produce a variety of beneficial outcomes. Music used to promote neurogenesis and neuroplasticity could help people with PTSD heal faster and/or more easily. Activities include learning an instrument, drumming, or producing any musical sound. Encouraging music lessons (from a music teacher, YouTube, videos, or books) can be helpful. For people who may be unable to learn an instrument, there are musical applications that can be downloaded on tablets, smartphones, or computers that will allow people to make music by touching a button.

**Reducing chronic sound-stressors.** Studies showed that chronic sound stressors from the environment can lead to functional and structural changes in the hippocampus and amygdala (Koelsch, 2014). An example of chronic sound stressors is someone who is exposed to loud, unpleasant sounds on a regular basis (soldiers, yelling in domestic abuse, etc.). Being able to reduce the impact of the sounds (using earplugs) could potentially reduce the negative effect it has on the amygdala and hippocampus. It will need to be examined if wearing earplugs or headphones that play pleasant music reduces arousal in situations or locations that trigger symptoms of PTSD.
Stimulate memories. Since the hippocampus plays a role in memory, and the re-processing of memories are used to heal PTSD (for example, in EMDR), then music could be used to potentially stimulate memories. For example, if a client is unable to make progress in therapy and does not remember a traumatic memory; and therefore cannot reprocess it and heal, music could be used to try to jog the client’s memory. This can be done by asking the client the type of music he/she mostly listened to during the time the traumatic memory occurred. Then, the therapist could play songs that were popular for that genre during that time, which could trigger a memory. Before this activity is done, the client should be taught grounding techniques (so the client does not become consumed by the flashback). The client should also be given time to process the experience before the session is over. Lastly, the therapist should know if the client is able to self-sooth outside of the session, and consider this before participating in this exercise.

Easing the process of exposure therapy. The hippocampus plays a role in learning. Symptoms of PTSD are partially caused by a type of learning called classical conditioning. The client conditioned himself or herself to believe or act a certain way for their survival, for example, the veteran who hits the floor when there is a loud sound. Music should be explored to see if it can quicken or ease the process of exposure therapy or relearning. It should also be explored if long-term exposure of the hippocampus responding positively to an external stimuli (music) can retrain the brain to normal hippocampal functioning.

Dopamine. The mesolimbic dopamine system is activated by music, which releases dopamine (Koelsch, 2014; Moffic, 2008). The release of dopamine plays a role
in reward pathways. Music can scientifically make people feel good (the release of dopamine). This solidifies that music should be utilized as a coping skill or in crisis prevention plans.

**Musical socialization and attachment-related emotions.** Participating in music as a group prompts communication, collaboration, sharing a goal, and social cohesion (Koelsch, 2014). The relationship between hippocampal activity and attachment-related emotions due to musical socialization can be used for people who have PTSD to address the negative changes in thoughts and moods (isolation, feelings of detachment, and being unable to experience positive emotions) (APA, 2013; Koelsch, 2014). Music can be used to trigger social functioning, which fulfills a basic human need, one that people with PTSD often feel like they are missing.

**Group cohesion.** A group of people moving in unison increases trust, cooperation, and group identity. It also is perceived to be pleasurable and activates the nucleus accumbens. Cooperation within a group increases trust and future cooperation (Koelsch, 2014). For people with PTSD, this is important because their trust has been broken numerous times. Therefore, a facilitator could start off a group with a musical exercise that involves people clapping a beat, swaying, or any other movement that could increase group cohesiveness, which can prompt trust and cooperation (Koelsch, 2014). This last section explored how the neurological results of music could help heal PTSD. The next section will focus on social and therapeutic implications for social work.

**Social and Therapeutic Implications**

**Music to build rapport.** As mentioned earlier, some forms of traditional therapy treatments for PTSD, such as EMDR, CBT, and CPT, may seem invasive for someone
who has PTSD. Putting thoughts and emotions into words may be too difficult for someone who has severe PTSD. Music is another way to express himself/herself without the pressure of using words. Thus, using music to build rapport is a less intrusive technique than talking. An example of this is having the client and therapist share their favorite song. A conversation can start about the song, for example, why he/she likes the song. Or, maybe the therapist and client have similar tastes in music, and can discuss other artists or share other songs in a similar genre.

Another example of using music to build rapport is asking the client to play a song (can be played from a media device or with an instrument) that resembles what he/she is currently feeling. The songs can be used as a tool to talk about feelings or thoughts. For example, maybe there are lyrics in the song that speaks to the client. The client can share the lyrics and the therapist can probe why the lyrics speak to the client. Maybe there are tonal components of a song that mirrors his/her mood. For example, songs in a minor scale may prompt depressive mood states, while a song with a strong bass beat may promote energetic, elated mood states. Therefore, using music to allow clients a different avenue to express themselves may give them the freedom and ease to feel comfortable with the therapist, and help progress the process of discussing current emotions. This could eventually lead to the therapist being able to assess client moods or risk (suicidal ideation), based on the conversation on the song the client played. Music cannot only be used to build rapport, it can also be used as a tool for grounding in therapy.

**Music used for grounding.** Clients with PTSD can feel like they are re-experiencing the traumatic event, which can also include having them feel like they are
experiencing it at the present time. Volkman (1993), as cited by Carr et al., states that music can help the client remain grounded. Music can be used as a tool to help them by having them actively focus on music. It can help distinguish between the traumatic memories and present moment (Carr et al., 2011).

**Addressing symptoms of PTSD.**

**Avoidance.** One symptom of PTSD is avoidance, such as avoiding places, people, environments, or any other stimuli that reminds them of the trauma. Using drugs or alcohol is considered avoidance behavior. Avoiding emotions, or numbing, is also avoidance. Music group therapy is in a social setting that addresses avoidance symptoms. Through musical improvisation, group members have to be aware of others around them and how they can contribute to the music. Being able to express feelings through music may address the barrier of emotional detachment and limited affect (Carr et al., 2011).

**Hyperarousal.** Hyperarousal is another symptom of PTSD. This includes poor concentration, an exaggerated startle response, aggression, irritability, sleeplessness, and hypervigilance. An example of an exaggerated startle response from conditioning is when a veteran is walking down the street, a lawn mower backfires, and he/she instinctively hits the ground. Another example is if someone who has an abusive past, gets tapped on the shoulder, and he/she automatically elbows the person behind them. Exaggerated startle responses and hypervigilance can be addressed by encouraging the client to tolerate silence and loud sounds (Carr et al., 2011). Allowing the client to be in a safe and predictable environment, the loud sounds could potentially desensitize the client. Also, making music forces the client to focus on the sounds he/she makes and the
sounds others are making. The intentional focus it requires to complete this task addresses the concentration deficit (a symptom of hyperarousal) in PTSD (Carr et al., 2011).

**Arousal Regulation.** Arousal regulation can be addressed by fostering relaxation through music. Orth found that most clients chose music that are predictable, slow paced, has an even rhythm, and uses standard instruments (Orth, 2005). Therefore, the therapist and client can make a list of songs that the client finds soothing. It is suggested that clients choose songs that include a predictable, even, and slow-paced rhythm that also uses standard instruments because music provokes emotions subconsciously (Koelsch, 2014). The components of music Orth suggests are consistent with the components of music that produce pleasant emotions (Koelsch, 2014; Orth, 2005).

Another therapeutic implication is using drumming for people who have PTSD.

**Drumming.** The emotions associated with PTSD, anger and irritability, can be expressed in a safe way through musical instruments (Carr et al., 2011). A study conducted by Bensimon and colleagues with soldiers who are diagnosed with PTSD found that drumming was the most effective way to express anger (Bensimon, Amir, & Wolf, 2008). The participants partook in circle group drumming activities. Some of the exercises included: having two or more participants drumming to the same beat; having one member play a rhythmic pattern, with the group responding back with the rhythmic pattern; having one person play a rhythm, and the person next to him or her immediately playing it thereafter, and continuing around the circle. The rhythms were a combination of basic and complex patterns.
The results showed that the drum circle prompted openness, togetherness, sharing, closeness, connectedness, and intimacy. Drumming can trigger traumatic memories, but did so in a safe environment. Drumming loudly (forte-fortissimo fff) gave the participants a sense of relief, satisfaction, and empowerment. The participants described it as “drumming out the rage” (Bensimon, Amir, & Wolf, 2008). They also may have regained a sense of control and confidence by mastering basic and complex rhythms in circle group drumming (Bensimon et al., 2008). A safe and predictable structure for expression and experimentation can promote feelings of safety (Bensimon et al., 2008 & Orth, Verburgt, Nieuwenhijzen, & Wijzenbeck, 2006). Therefore, this study could be mimicked and agencies could conduct a similar drum-circle group. Also, producing music can prompt neurogenesis (motor and auditory parts of the brain get larger) and neuroplasticity (the pathways in the brain get stronger) (Koelsch, 2014). Not only can drumming be used to help heal PTSD, but utilizing music to share one’s story through narrative therapy could benefit the client.

Music as narrative therapy. Narrative therapy is based on the premise that people are shaped by their story (events in their life), and problems can be solved by exploring the effects, meaning, and context in which the story was formed. It allows the client to “rewrite” their future to solve their problem (Morgan, 2000). Orth used a gradual approach for music therapy techniques which includes composition, recording relaxing music, musical improvisation, and song writing “to formulate a narrative of traumatic and autobiographical experiences” (Orth et al., 2006). Writing music to share and express a client’s story could be very empowering and therapeutic for the client. For someone who has PTSD, composing and/or writing lyrics about his/her trauma experience can give
them a voice and allow them to express themselves in ways they have not before. Giving
the client power and control over the trauma, allowing the client to decide what note or
what word they will use to express an aspect of their pain, can be very healing. The
process of writing or composing a song can help the client see their experience in a
different light. To summarize, these implications addressed the neurological impact
music has on the brain, and how that could potentially assist with treating PTSD. It also
explored the social aspect of how music can be used to treat symptoms of PTSD.

Conclusion

In conclusion, music can be used in an addition to standard treatment to address
symptoms for PTSD. For example, it can be used as a way to build rapport and allow the
client to express himself or herself nonverbally, since some clients with PTSD have a
difficult time talking about the trauma. Music can be used as a way to prompt memories
(because music stimulates the hippocampus) when a client is unable to reprocess a
memory due to not remembering it. It can also be used as a tool to ground the client,
because music can prompt feelings of calmness. Music gives the client something to
focus on in the here-and-now, so that he/she can remember that he/she are in a safe place.
Using music as a way to talk about and name emotions can be used as tool for self-
disclosure. Music in a group setting can reduce isolation and feelings of detachment, and
increase pleasurable emotions. Drumming can be used to express rage and anger about
the trauma. While done in a drumming circle, it can produce relief, satisfaction, and
empowerment. Emotions are produced by music (through musical structure and/or the
release of dopamine) and can be used intentionally to help regulate the client by planning
with the client to have a list of songs that increases his or her mood. This can be used as
a positive distraction technique during a crisis. Neurologically, music can be used to stimulate the parts of the brain that are negatively impacted by trauma (hippocampus, amygdala, nucleus accumbens), to try to prompt neurogenesis and neuroplasticity so that the brain is healed to healthy functioning. This would include balancing the HPA axis response, increasing the effectiveness of the hippocampus, and reducing the unnecessary stimulation of the amygdala.
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### Appendix A
Summary of Brain Structures and Their Significance

<table>
<thead>
<tr>
<th>Brain Part</th>
<th>Location</th>
<th>Function</th>
<th>Relationship to Trauma</th>
<th>Relationship to Music</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrum</td>
<td>Consists of:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Frontal lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Parietal lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Occipital lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Temporal lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Left Hemisphere</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Right Hemisphere</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal Lobe</td>
<td></td>
<td>Helps control judgment, behavior, attention, creative thought, problem solving, intellect, abstract thinking, muscle movements, physical reactions, smell and personality.</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Controls: Attention, integrating information, planning, reflecting on past behaviors and considering consequences before making a decision.</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

• Decreased attentional capacity
• Decreased cortical thickness
<table>
<thead>
<tr>
<th>Brain Region</th>
<th>Function Descriptions</th>
<th>Neurological Impact of PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most frontal part of the frontal lobe</td>
<td>emotional regulation, following social norms, personality, plan for the future.</td>
<td>• Architectural changes due to chronic stress</td>
</tr>
<tr>
<td>Parietal Lobe</td>
<td>• Helps control movement • Senses touch, pressure, and pain • Controls language • Processes the senses.</td>
<td></td>
</tr>
<tr>
<td>Temporal Lobe</td>
<td>• Interprets sounds and language. • Controls memory functions.</td>
<td></td>
</tr>
<tr>
<td>Occipital Lobe</td>
<td>• Helps interpret vision input (color, word recognition).</td>
<td></td>
</tr>
<tr>
<td>Cerebellum</td>
<td>• Controls essential body functions (posture, balance, and coordination).</td>
<td>Contributes to: • Reward • Motivation • Emotion</td>
</tr>
<tr>
<td>Brain Stem</td>
<td>Consists of the midbrain, pons, and medulla. • “Reptilian brain.” • Controls physiological responses (heartbeat, breathing, blood pressure).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Contains the amygdala,</td>
<td>• Composed of glands that help regulate</td>
</tr>
</tbody>
</table>
| Limbic System | hippocampus, hypothalamus, and thalamus. | hormones and emotions.  
• In charge of complex behaviors, such as reproducing, fighting, or fleeing.  
• Used to assign truth and meaning to experience  
• Critical in producing emotion | Sensitization | Controls:  
• Reward  
• Motivation  
• Emotion |
|-----------------|---------------------------------|-----------------|-----------------|-----------------|
| Hippocampus     | Learning and storing memories  
• Emotion and reward are linked to the hippocampus.  
• Critical in producing emotion | People with PTSD tend to have a smaller hippocampal volume  
• Contextual fear conditioning  
• Hypervigilance, exaggerated startle response, increased behavioral activation, paranoia, generalization of traumatic event  
• Declarative memory deficiencies  
• Dissociation  
• Stores the learned event | Associated with unpleasant feelings when activated by music  
|                      |                                |                  |                                |                                |
| Amygdala          | Helps the body to react to emotions such as fear, aggression, and anger.  
• Associated with how fears are processed | Reduced ability to regulate the fear response  
• Unable to determine if a stimuli is safe or not  
• People with PTSD are | See “laterobasal amygdala” and “superficial amygdala.”  
• Associated with unpleasant feelings when activated by music |
| Laterobasal Amygdala | • Critical in producing emotion  
• Apart of the reward network. | associated to have a smaller Amygdala  
• Establishes the association between the environmental cues and the reward or aversive event | • Involved with primary and secondary rewards |
|----------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Superficial          | • The main amygdala input structure for auditory and sensory information.  
• Evaluation and learning of negative and positive stimuli,  
• Creates expectations of reinforcements which guide behavior.  
• Codes the positive or negative reward value of musical stimuli | • Largest role in processing musical stimuli.  
• Perceptive to stimuli with basic socio-affective information.  
• The SA and the MT had stronger connections during joy-evoking music than during fear-evoking music.  
• The SA, MT, and NA form a network that regulates someone’s natural inclination to |
<table>
<thead>
<tr>
<th>Amygdala (SA)</th>
<th></th>
<th>Used to control mood, hunger, temperature, thirst, and the pituitary gland.</th>
<th>Controls mood and pituitary gland. Expected chords or tones stimulate the SA.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothalamus</td>
<td></td>
<td>Apart of the HPA-axis Critical in producing emotion.</td>
<td>The HPA axis produces cortisol Controls any movement necessary due to the reward or aversive event</td>
</tr>
<tr>
<td>Thalamus</td>
<td></td>
<td>Monitors the sensations the body is experiencing.</td>
<td></td>
</tr>
<tr>
<td>Mediodorsal Thalamus (MT)</td>
<td></td>
<td>Apart of the reward network.</td>
<td>Activated by music-evoked emotions. The SA and the MT had stronger connections during joy-evoking music than during fear-evoking music. The SA, MT, and NA form a network that regulates someone’s natural inclination to approach new situations or to withdrawal from</td>
</tr>
<tr>
<td></td>
<td>Critical in producing emotion</td>
<td>Apart of the reward network</td>
<td>Released dopamine during the emotional peak of music</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------------------</td>
<td>-----------------------------</td>
<td>------------------------------------------------------</td>
</tr>
<tr>
<td>Nucleus Accumbens (NA)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopaminergic Mesolimbic</td>
<td>Controls one’s response to rewards</td>
<td>Determines motivation and incentive drives.</td>
<td>Evolutionary benefit</td>
</tr>
<tr>
<td>Rewards Pathway</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesocorticolimbic System</td>
<td>Contains the nucleus accumbens, ventral tegmental area, hippocampus, amygdala, orbitofrontal cortex, and centromedial prefrontal cortex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Region</td>
<td>Function Description</td>
<td>Example</td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>--------------------------------</td>
<td></td>
</tr>
<tr>
<td>Ventral Tegmental Area</td>
<td>• Contains dopamine&lt;br&gt;• Informs if a stimulus is rewarding or not.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudate Nucleus</td>
<td>• Apart of the reward network.</td>
<td>• Activated by music-evoked emotions.</td>
<td></td>
</tr>
<tr>
<td>Evolutionary Reward Network</td>
<td>Contains: The nucleus accumbens, caudate nucleus, ventromedial orbitofrontal cortex, pre-genual cingulate cortex, amygdala, anterior insula, and mediodorsal thalamus.</td>
<td>• Involved with primary and secondary rewards&lt;br&gt;• Releases dopamine during the anticipation of music</td>
<td></td>
</tr>
<tr>
<td>Ventral Striatum</td>
<td>The bottom portion of the striatum.</td>
<td>Previous neuroimaging studies show that listening to pleasurable music stimulates emotional and reward circuits within the brain (the ventral striatum).</td>
<td></td>
</tr>
</tbody>
</table>
Dentate Gyrus

Stem cells are able to reproduce here.

Structural damage to the hippocampus is believed to be due to stress-induced damage of hippocampal neurons and reduced neurogenesis in the dentate gyrus

Broca’s Area

Left hemisphere of the prefrontal cortex.

- Scrutinizes the structure of a sentence.
- Processes the order of bodily movement.
- Puts thoughts into words.

- Used to process the structure of music.
- Tracks musical rhythms.
- Supports the appropriate timing, sequencing, and knowledge of rules that are common and essential to music, speech, and movement

Note. The table summarizes brain structure information from the paper. A “-“ means that information was not included in the paper. Brain structures that are not included in the table are summarized below. The insula, cingulate cortex, and lateral orbitofrontal cortex are critical in producing emotion (Koelsch, 2014). The orbitofrontal cortex is stimulated when unexpected chords or tones are played (Koelsch, 2014). The auditory system processes sensory dissonance and consonance and regulates changes in the laterobasal amygdala (Koelsch, 2014). The Auditory thalamus and auditory cortex (part of the auditory system) sends information to the laterobasal amygdala (Koelsch, 2014). The auditory cortex regulates changes in the laterobasal amygdala (Koelsch, 2014). The parahippocampus gyrus and the temporal poles are associated with unpleasant feelings when activated by music (Shuai-Ting et al., 2011). When words are added to music, music is processed in the cerebral cortex (Moffic, 2008). The olfactory bulb processes odors and other sensory input; sends signals to the superficial amygdala; and produces basic, universal emotions (Koelsch, 2014).