The dynamic functional capacity theory: A neuropsychological model of intense emotions

Philip C. Klineburger and David W. Harrison
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Abstract: The music-evoked emotion literature implicates many brain regions involved in emotional processing but is currently lacking a model that specifically explains how they temporally and dynamically interact to produce intensely pleasurable emotions. A conceptual model, the dynamic functional capacity theory (DFCT), is proposed and provides a foundation for the further understanding of how brain regions interact to produce intensely pleasurable emotions. The DFCT claims that brain regions mediating emotion and arousal regulation have a limited functional capacity that can be exceeded by intense stimuli. The prefrontal cortex is hypothesized to abruptly deactivate when this happens, resulting in the inhibitory release of sensory cortices, the limbic system, the reward-circuit, and the brainstem reticular activating system, causing “unbridled” activation of these areas. This process is hypothesized to produce extremely intense emotions. This theory may provide—music-evoked emotion researchers and music therapy researchers—a theoretical foundation for continued research and complement current theories of emotion.

Subjects: Biopsychology; Clinical Neuropsychology; Evolutionary Psychology; Laterality (Left & Right Domains); Music Therapy; Psychological Science

Keywords: music; emotion; brain; capacity; prefrontal cortex; emotional regulation

1. Introduction

Music's ability to evoke intense emotions has received interest from researchers investigating the neural bases of emotions (Koelsch, Fritz, Cramon, Muller, & Friederici, 2006). A growing body of neuroscience literature consistently identifies brain structures involved in emotion and motivation

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that are modulated by listening to music (Ball et al., 2007; Blood & Zatorre, 2001; Koelsch et al., 2006), and whose activity correlates with varying levels of emotional intensity. For example, music consistently engages cortical, subcortical, brainstem arousal systems, paralimbic, and limbic structures such as the amygdala and hippocampus; the “reward-circuit” including the ventral tegmental area, nucleus accumbens, striatum, and orbitofrontal cortex; and the temporal, parietal, and prefrontal cortices (Peretz, 2010). The assumption that music-evoked emotions are a purely esthetic experience that is unsuitable for investigating the neural basis of emotions has been discredited as activation is consistently seen in regions of the brain associated with motivation and goal-oriented behavior. The data clearly demonstrate music’s utility for investigating the neural correlates of emotional processes (Peretz, 2010).

Reports of specific regions of cerebral activity are abundant in the music-evoked emotion literature. Recent progress in identifying their functional connections (Fritz, Koelsch, Grigutsch, & Sampler, 2007; Schmidt & Trainor, 2001) has contributed to the proposals of music processing neural networks. However, within this literature, support for the predominant theories of emotion such as the valence hypothesis (Tomarken, Davidson, & Henriques, 1990) and the right hemisphere model (Bowers, Bauer, & Heilman, 1993) is mixed, which highlights their limited applicability for understanding the full range of the powerful emotions that music is capable of provoking.

Despite renewed interest and research advances, these theories of emotion have received surprisingly less critical examination when considering the inextricable link between music and emotion, and in particular, the intense emotions that music evokes. In response to this, the dynamic functional capacity theory (DFCT) (Carmona, Holland, & Harrison, 2009; Mitchell & Harrison, 2010) hypothesizes that when the functional capacity of the prefrontal cortex (PFC) is exceeded, it functionally “shuts down” by deactivating and releasing inhibition over sensory, subcortical, and brainstem arousal systems. It is through this process that intensely pleasant and unpleasant emotions, behavioral disinhibition, and cognitive deficits may arise. The DFCT proposes that the PFC has a functional capacity that is limited by its physiological constraints and its functional neuroanatomy. Once this capacity is exceeded, the PFC is hypothesized to deactivate/disconnect from brain regions normally under its inhibition such as the sensory cortices, the limbic system involved in emotion and memory, the reticular activating system, and the “reward-circuit” involved in pleasure and reinforced behavior. The DFCT’s focus on the temporal dynamics of emotion has applications for better understanding how intense emotional states ranging from euphoria to rage develop, and to how the emotional and cerebral interactions that occur during music therapy play a role in its effectiveness. The DFCT’s hypothesis that the relationship between PFC activation and emotional intensity is not linear may help reconcile mixed findings in the music and emotion literature. Using the DFCT, researchers should investigate emotional intensity and PFC activation as a potential moderator of music therapy’s effectiveness. Researchers could also use the DFCT model to investigate how neurological insults can lower an individual’s functional capacity.

The DFCT builds on Luria’s Functional Cerebral Systems Model (1966) in that it incorporates contributions from all three of Luria’s functional units of the brain, but it specifically hypothesizes how temporal and dynamic cerebral interactions produce the intensely pleasant and unpleasant emotional states that characterize psychopathology, but also the wide array of emotional intensity that gives life meaning. Luria’s Functional Cerebral Systems Model describes three functional units of the brain including the brain stem reticular activating system, the posterior lobes of the brain, and the frontal lobes. The reticular activating system is responsible for regulating cortical tone or arousal, the posterior lobes obtain and store sensory information from the outside world, and the frontal lobes comprehend this information and are involved in decision-making as well as regulating emotions and behavior. It is through the interaction of these three functional units that consciousness, emotion, and behavior arises.

The DFCT has evolved from research on emotional and autonomic regulation, and individuals with diminished right frontal lobe capacity such as highly hostile individuals performing multiple cognitive tasks. Hostility has been previously associated with increased reactivity for auditory (Demaree &
Harrison, 1997), visual (Harrison & Gorelczenko, 1990; Herridge, Harrison, Mollet, & Shenol, 2003), and somatosensory modalities (Herridge, Harrison, & Demaree, 1997). Additionally, highly hostile individuals display an exaggerated acoustic startle response that fails to habituate, also indicating a diminished frontal lobe capacity to inhibit an acoustic brainstem reflex (Klineburger & Harrison, 2015) to predictable and repetitive stimuli. The DFCT has been mentioned briefly however, the neural mechanisms hypothesized to mediate PFC functional capacity have not been described in detail, and that is the primary purpose of this paper.

2. The dynamic functional capacity
The DFCT consists of several key tenets. The DFCT: (1) proposes that the functional neuroanatomy of the PFC makes it uniquely vulnerable to the exhaustion of finite resources and limits its physiological capacity for normal functioning that when exceeded results in impaired cognitive, behavioral, and emotional regulatory functions, (2) emphasizes the temporal dynamics of cerebral interactions as emotions intensify over time, provides a testable four-phase model of emotional activation characterized by distinct patterns of cerebral interactions, and proposes a nonlinear, “inverted-U”, relationship between emotional intensity and PFC activity, (3) identifies factors contributing to individual differences in capacity levels, and (4) proposes that the function of exceeding PFC capacity is adaptive.

2.1. PFC functional capacity is limited by its functional neuroanatomy and finite resources
The DFCT hypothesizes that the PFC’s functional neuroanatomy makes it extraordinarily powerful, yet particularly vulnerable to interference effects, cellular exhaustion due to its high metabolic demands, impairments of white matter, and presents evidence demonstrating its susceptibility to catecholamines and stress hormones. The anterior PFC’s larger size is attributable to the disproportionate development of connective fibers (white matter), rather than neuron cells (gray matter) (Fuster, 2004). The rostro-caudal model, proposed by Christoff and colleagues suggests that the rostro-caudal, or anterior to posterior, axis of the PFC supports a control hierarchy whereby posterior-to-anterior PFC mediates progressively abstract, higher order control (Christoff & Gabrielli, 2000). The development of this model has arisen from a working memory perspective, and from hierarchical models such as the relational complexity model (Christoff & Gabrielli, 2000), the cascade model (Koechlin & Summerfield, 2007), and the abstract representational hierarchy model (Miller & Cohen, 2001). Importantly, working memory was among the first cognitive abilities with a clearly demonstrated limited capacity.

From a working memory perspective, it is proposed that rostral and caudal PFC can be distinguished on the basis of processing domain-general, versus domain-specific representations (Courtney, 2004; Fuster, 2004), such that domain-specific regions in the posterior frontal lobes can be modulated by the domain-general rules anteriorly in the dorsolateral prefrontal cortex and PFC (Sakai & Passingham, 2001, 2006). Double dissociation between posterior and mid-dorsolateral PFC in monkeys (Petrides, 2006) supports the hypothesis that domain-generality versus domain-specificity distinguishes rostral from caudal PFC. In summary, evidence supporting these models suggests that the anterior PFC handles the most abstract, nebulous, and complex tasks, while the posterior PFC regions handle simpler, less complex tasks lower in the hierarchy.

Due to the multiple abstract, nebulous, and complex tasks carried out by anterior regions of the PFC such as the orbitofrontal cortex, dorsolateral prefrontal cortex, and the medial PFC, it is predicted that there would be increased opportunity for overlapping processes in these areas leading to dual-task interference and cellular exhaustion. This “crowding” of functional space in the PFC and its increased stress on these systems is one mechanism hypothesized by the DFCT to limit anterior PFC capacity. Cerebral systems share overlapping organization networks and when two cortically overlapping areas process different tasks, there is an increased demand on those networks leading to performance declines on one or both of the concurrent tasks. This is referred to as dual-task interference. Kinsbourne’s model of functional cerebral space (Kinsbourne, 1980) claims that facilitation or impairment of concurrent performance in multiple tasks depends on the degree of task relatedness.
and the degree to which the multiple neural networks involved in the tasks are “close” in physical space. If the tasks are dissimilar and involve common cerebral networks, then dual-task interference will occur and impair performance. Thus, the anterior regions of the PFC are predicted to be particularly vulnerable to dual-task interference. Moreover, increased resource demands of high task complexity and dual-task interference in the PFC are hypothesized to make it vulnerable to resource depletion and particularly sensitive to the neuroanatomical integrity of gray and white matter. However, if the tasks are highly related and the neural networks are in close proximity, performance will be expedited via sharing of networks, and this may contribute to the advanced cognitive functions of the anterior PFC. According to the DFCT, the PFC’s functional neuroanatomy facilitates “higher order” cognitive abilities while also limiting its functional capacity.

The shared space principle is applicable to the frontal-posterior associations. As described earlier, the frontal lobes mediate many abstract cognitive processes that compete and cooperate to utilize cerebral resources both cortically and subcortically to regulate behavior. The impact of cognitive tasks on cerebral activation and cardiovascular functioning in hostile populations supports this. Williamson and Harrison (2003) used concurrent fluency tasks sensitive to left and right frontal lobe activation (Foster & Harrison, 2004) to examine their influence on parasympathetic and sympathetic activation in high-hostile men. High-hostile men demonstrated increased systolic blood pressure in response to a design fluency task that challenged the capacity of the right frontal system (Foster & Harrison, 2004), whereas a verbal fluency task that challenged left frontal capacity (Benton & de Hamsher, 1976) resulted in decreased systolic pressure. Moreover, high-hostile men showed heightened perseverative errors in the design fluency tasks, a common clinical finding with frontal lobe deficits (Foster & Harrison, 2004). Those with limited frontal regulatory capacity facing dual-task challenges such as regulating anger or anxiety, while meeting additional cognitive challenges may fail to regulate their emotion, the cognitive task at hand, or fail at both as they compete for PFC resources and neural networks.

Despite the evidence supporting Kinsbourne’s notion of dual-task interference (see Meyer & Kieras, 1997; Pashler, 1994, 1999; Telford, 1931; Welford, 1952), the neurophysiological basis of dual-task interference is not fully understood (Herath, Klingberg, Young, Amunts, & Roland, 2001). One hypothesis addressing the neurophysiological basis of dual-task interference is the cortical field hypothesis (Roland & Zilles, 1998) which conceptually follows from Kinsbourne’s model of “shared functional cerebral space”, but provides a finer level of analysis and is supported by electrophysiological data. The cortical field hypothesis states that large fields in the upper layers of the cerebral cortex (layers I–III) depolarize in response to stimuli and preceding motor activity by making use of the elaborate horizontal connectivity in layer I–III of the cerebral cortex. The cerebral cortex is the most recently evolved structure of the brain and is composed of six layers of neurons that receive and project to various regions of the brain. These large depolarization fields have been observed in the auditory, somatosensory, olfactory, parietal, and prefrontal cortices (de Curtis & Takashima, 1999; Horikawa, Nasu, & Taniguchi, 1998; Kleinfeld & Delaney, 1996; Salzberg, 1973; Sawaguchi, 1994). Studies using voltage-sensitive dyes exhibit fields of massive depolarization in the upper layers of cortex that are much larger than the size of hypercolumns, which are still regarded as the functional and computational units of the cerebral cortex (Orbach, Cohen, & Grinvald, 1985). This widespread horizontal depolarization of cortical fields is believed to subserve the high computational elements of the cerebral cortex by creating dynamic depolarization fields for all its functional contributions (Roland, 2002). A negative consequence of this is the increased possibility for interference in the cooperative computations of layers I–III for two simultaneously performed tasks requiring overlapping fields that cannot be performed simultaneously.

Several dual-task neuroimaging experiments have found prefrontal and parietal activation specifically related to the performance of a dual task (Corbetta, 1998; D’Esposito et al., 1995; Dove, Pollmann, Schubert, Wiggins, & Yves von Cramon, 2000; Koechlin, Basso, Pietrini, Panzer, & Grafman, 1999), while others have failed to show additional cortical activity associated with dual tasks (Adcock, Constable, Gore, & Goldman-Rakic, 2000; Bunge, Klingberg, Jacobsen, & Gabrieli, 2000;
Goldberg et al., 1998; Klingberg, 1998; Passingham & Weinberger, 1996). Most recently, Watanabe and Funahashi (2014) provided direct neurophysiological evidence for dual-task interference in the lateral prefrontal cortex, an area known for its role in working memory. Using single-electrode recording of neurons in the lateral prefrontal cortex, they found that the performance of monkeys exhibited dual-task interference, and prefrontal neuron activities showed a decreased ability to represent task-relevant information to a degree proportional to the increased demand of the concurrent counterpart task. Most relevant to the DFCT and emotional regulation, using functional magnetic resonance imaging (fMRI), Herath et al. (2001) showed that the performance of dual-reaction time tasks activated cortical regions in excess of those activated by the performance of the component single tasks, and dual-task interference was specifically associated and correlated with increased activity in a cortical field of the right inferior frontal gyrus. This area has been previously implicated in emotion regulation and hostility, which lends to the notion that areas of the brain that regulate emotion may face the burden of dual-task interference.

One primary assumption of the DFCT is that PFC functions (e.g. working memory, executive functions, behavioral inhibition, emotional regulation, etc.) rely on finite energy resources that supply neurons and the glial cells that support them. These resources include oxygen and glucose delivering blood, neurotransmitter production, and axonal transport. Systematic differences in blood flow in various areas of the brain with a maximum over the frontal lobes (20–30% above average) and a minimum over the occipital lobes (Ingvar & Lassen, 1977) support the PFC’s larger energy requirements. The idea that cognitive functions rely on finite resources is not a new one; Baumeister’s Strength Model of Self-Control (Baumeister, Heatherton, & Tice, 1994) posits that self-control depends on limited energy resources (Baumeister et al., 1994) and is vulnerable to deterioration over time from repeated exertions, resembling a muscle that fatigues. Baumeister and colleagues (1994) provide evidence that decreased glucose levels resulting from self-regulation mediates self-regulation failure. Self-regulation draws on a common domain-general resource (Wagner & Heatherton, 2010), implicating the anterior PFC and its domain-general processing according to the rostro-caudal organization of the PFC. Self-regulation involves a balance between subcortical brain regions representing the emotional and reward value of a stimulus, and the prefrontal regions associated with self-control (Wagner & Heatherton, 2010) via inhibitory control.

If functional capacity relies on finite resources, it would be predicted that those with high capacity may require less resources to operate efficiently, an idea that is supported by the neural efficiency model (Haier et al., 1988). Using positron emission tomography (PET), Haier and colleagues (1988) found negative correlations between Raven’s advanced progressive matrices scores and absolute regional metabolic rates in different brain areas indicating that the brains of more intelligent individuals consumed less energy. Intelligence was related to how efficiently, rather than to how strongly, the brain worked. Haier et al. (1988) suggested that this capacity could derive from the disuse of task-irrelevant brain areas and more focused use of task-relevant areas. Consistent with the DFCT, lower resource consumption associated with high neural efficiency and the disuse of task-irrelevant brain areas would decrease the probability of dual-task interference.

2.2. The temporal dynamic cerebral interactions of exceeding functional capacity: A four-phase process

The process of exceeding the functional capacity of the PFC is hypothesized to occur in four distinct and testable phases (see Figure 1). During the first phase, subcortical and sensory cortices activate in response to a stimulus. In the second phase, fronto-parietal networks activate as the PFC integrates, comprehends, and selectively attends to relevant incoming sensory information. At this point, sensory regions, brainstem arousal systems, and PFC activity are somewhat in balance as the emotional intensity is mild or moderate. The PFC is able to regulate the emotion and arousal impacts of the stimulus. As the emotional intensity, stress, or cognitive demand increases in the second phase, global increases in brain activation consume large amounts of energy resources and the neuroanatomical integrity of the white matter tracts connecting these brain regions is put to the test. PFC capacity is exceeded in the third phase when frontal system resources are exhausted and/
or when there is insufficient neuroanatomical integrity (e.g. decreased white matter). This phase of the model is characterized by abrupt PFC deactivation. This causes a release of inhibition over sensory regions of the brain, brainstem nuclei, the reticular activating system, and results in intense emotional arousal. In the fourth phase, neural resources (e.g. neurotransmitters, glucose, oxygen, etc.) are replenished, or when the stimulus is no longer present, the PFC functioning returns to baseline levels.

Each of these phases is testable. In phase one, increased temporal, parietal, and occipital lobe activation would be evidenced by increased beta power (EEG), increased BOLD (fMRI), while frontal systems would exhibit relatively stable levels of activity. In the second stage, increases in the frontal, temporal, and posterior lobes would show increased beta power or BOLD on fMRI. Increased activation of fronto-parietal networks in the second phase specifically includes the posterior parietal lobe and regions of the PFC such as the dorsolateral prefrontal cortex, medial prefrontal cortex, and orbitofrontal cortex. Increased BOLD in regions of the brainstem containing the reticular activating system would also be predicted at this time reflecting increased arousal. This second phase is characterized by widespread activation of neural networks and as the stimulus or emotion intensifies, these global increases consume energy resources and will approach the third phase. In the third phase, the PFC is hypothesized to abruptly deactivate as its capacity is exceeded and this would be evidenced by increased delta EEG or decreased BOLD signal in the PFC. Concurrently, sensory cortices that are now uninhibited by the frontal lobes would show increases in beta EEG or BOLD. Brainstem arousal systems would show a similar increase in BOLD as well as limbic structures such as the amygdala and hippocampus. The reward circuit including the ventral tegmental area and nucleus accumbens are also predicted to show increased BOLD in the third phase. The fourth phase is predicted to show decreased activation in regions implicated in the third phase and a return to baseline activity. Each phase is predicated on varying levels of emotional intensity. Due to the continuous nature of self-reported varying levels of emotional intensity, researchers can implement methods that track participants’ emotional intensity levels, such as through a rating dial.

2.3. Factors contributing to individual differences in functional capacity level
The PFC’s regulatory capacity relies on neuroanatomical integrity such that traumatic brain injuries (TBIs), cerebrovascular accidents, hypoxic events, etc., are hypothesized to diminish capacity by impairing white matter, metabolic mechanisms, and causing cell death. Also, the rigid and sharp structures in the frontal and temporal bones of the skull make the frontal and temporal lobes particularly susceptible to damage from TBIs. The orbitofrontal cortex responsible for emotional regulation (Eslinger, Grattan, & Geder, 1996), and the white matter tracts connecting the frontal lobes with subcortical structures (Bigler, 2004; Wilde et al., 2006) are frequently damaged, which commonly leads to executive function deficits and emotional dysregulation reflecting diminished functional capacity. Patients with primary generalized anxiety disorder have a comparatively less myelinated uncinate fasciculus (Phan et al., 2009), which connects the PFC, amygdalae, and temporal lobes. Patients with panic disorder show lesser activation in the orbitofrontal cortex in response to anxiety-inducing stimuli (Kent et al., 2005), and abnormalities in the orbitofrontal cortex in the right
hemisphere in particular are shared by several different anxiety disorders (Rauch, Savage, Alpert, Fischman, & Jenike, 1997). This is consistent with the notion that keeping anxiety in check relies at least in part on effective down-regulation, or suppression, of the amygdala by the orbitofrontal cortex (Milad et al., 2007).

Extreme stress in young animals is associated with reduced basal activity in the medial PFC in adulthood (Stevenson, Marsden, & Mason, 2008). Effective stress regulation relies on intact executive functions (EF) and the neuroanatomic substrates that subserve and overlap with those known to control EF. For example, individual differences in response inhibition during a Stroop interference task are associated with greater systolic and diastolic blood pressure reactivity (Waldstein & Katzel, 2005), indicating reduced autonomic regulation. Executive function deficits have been implicated in anxiety disorders, depression, and bipolar disorder (Gruber, Rathgeber, Bräunig, & Gauggel, 2007). Dolan et al. demonstrated that neuropsychological symptoms of depression, including cognitive deficits, are associated with profound hypometabolism in the medial PFC. Likewise, individuals with bipolar and unipolar depression exhibit decreased cerebral blood flow and glucose metabolism rates in the PFC (Drevets et al., 1997). Thus, deficits in neuroanatomical integrity, such as reduced myelination and hypometabolism, are hypothesized to limit PFC capacity by restricting resources and decreasing the efficiency of communication between the PFC and other brain regions. Thus, an individual’s threshold, or capacity level, can diminish when resources (e.g. glucose) are scarce—for example, in hypoglycemia—or sleep exhaustion.

The PFC can functionally operate while working within, but not beyond, its capacity; an individual’s functional capacity can be conceptualized as having a “threshold” that can be exceeded in an “either–or” fashion, similar to the “all or nothing” of a neuron’s action potential. As a muscle will fail under a load it cannot support, so too can other organs with physiological limits such as the PFC. Just as individuals of varying strength can lift varying amounts of weight, individuals of varying PFC functional capacity can regulate differing levels of emotion and stress. Thus, an individual’s capacity level reflects a relatively stable trait that can however fluctuate under circumstances. Those with high functional capacity are predicted to be able to endure high levels of stress, while maintaining cognitive ability and emotional regulation. Like bodybuilders lifting heavy weights, they may seek out and thrive in contexts of high demand and they may possess a wide range in which they can functionally operate because they have a high “ceiling” or “threshold” of capacity. Those with low functional capacity may be able to operate under circumstances of low stress and emotion but are unable to operate under mild or high levels of stress and emotional provocation. Compared to strong bodybuilders, low capacity individuals are like novice bodybuilders, tiring after fewer repetitions (or less weight).

2.4. Adaptive functions of limited PFC capacity
The DFCT hypothesizes that PFC deactivation during extreme stress or emotion can be adaptive, and there is strong evidence that the PFC’s ability to “turn off” is a result of being highly sensitive to its neurochemical environment (Arnsten, 2009). Further, all biological systems require rest and replenishment after times of stress (Caccioppo & Bernston, 2007). The DFCT proposes that exceeding capacity serves an adaptive, self-protective function that may be unique to the PFC due to the extensive interconnectivity in layers I–III of the cerebral cortex. It is hypothesized that the PFC attempts to prevent excitotoxicity from highly stressful stimuli by deactivating (decreased neural firing), or by disconnecting itself from overwhelming sensory input through recurrent inhibitory systems which would result in a temporary, refractory state of rest. Excitotoxicity of neurons occurs in the presence of excessive excitatory neurotransmitters such as glutamate that cause dangerously high rates of action potentials which exceed the physiological capability of neurons with respect to duration (chronic stressors) or intensity (acute stressor). Increased extracellular glutamate concentrations result from neuronal depolarization, depletions of glucose and oxygen in hypoglycemia and hypoxia, respectively, TBI (Cheng & Mattson, 1991; Siesjo & Wieloch, 1985), or defects in the glutamate reuptake systems (Volterra et al., 1992) that remove excess glutamate from synapses. Arnsten (1998) has proposed a theory of “rapid neuroplasticity” in which the PFC rapidly deactivates or disconnects
from other brain regions through a mechanism inherent to the PFC that weakens PFC network connections in order to prevent overexcitability through a negative feedback mechanism in PFC microcircuits. Other factors such as low glucose levels and severe hypoglycemia can cause neuronal death and cognitive impairment due to excitotoxicity and DNA damage (Suh et al., 2003). Additionally, oligodendrocytes in the forebrain and PFC that support communication with the rest of the brain are highly vulnerable to excitotoxicity (Mcdonald, Althomsons, Hyrc, Choi, & Goldberg, 1998). Thus, the DFCT proposes that low blood glucose and other energy resources may be one mechanism that can diminish PFC capacity.

The “all or nothing” refractory rest period hypothesized by the DFCT that occurs once capacity is exceeded may allow for a faster and more efficient return to optimal levels of cognitive, emotional, and behavioral regulation by the PFC while promoting cellular repair. This “rest period” also reflects typical neuronal functioning in which a refractory (hyperpolarization) period occurs immediately after a neuron fires. This refractory period prevents a neuron from firing continuously (Nieuwenhuys, 2007) which can damage a neuron. It has recently been shown that cortical neurons spontaneously, and in response to strong activation, fluctuate between “up” and “down” states (Branchereau, Van Bockstaele, Chan, & Pickel, 1996; Lewis & O’Donnell, 2000). While in a “down” state, the polarity of neuronal membrane is lowered, or hyperpolarized, making it less responsive to incoming signals and less likely to fire. While in an “up” state, the neuronal membrane is slightly depolarized, making it more easily excitable and likely to fire. These “up” and “down” states have been reported in regions of the cortex (Steriade, Nunez, & Amzica, 1993), as well as the dorsal and ventral striatum (O’Donnel & Grace, 1995). These areas are involved with the salience of reward and goal-directed movement, and are highly interconnected with the PFC. Becoming hyperpolarized and shifting to a “down” state may be one mechanism through which PFC neurons attempt to prevent excitotoxicity. Through local inhibitory interneurons that provide lateral inhibition, neurons in layers I–III can cooperatively inhibit themselves, possibly orchestrating a localized refractory period by shifting to a “down” state in a manner consistent with the behavior of cortical fields.

Exceeding capacity may also allow for more creative responses to environmental challenges (e.g. Foster et al., 2011), and could allow for uninhibited, novel intake and synthesis of sensory information that would normally be selectively inhibited by frontal lobe attentional systems. Foster et al. (2011) found that depressed individuals who are shown to have PFC neuropathology exhibited more spreading activation and creativity in a verbal-based lab task. The link between creativity and PFC deactivation has also been demonstrated by Limb and Braun (2008). In this experiment, professional jazz pianists either freely improvised to an auditory accompaniment of a prerecorded jazz quartet or reproduced memorized jazz sequences. Improvisation, in comparison to the production of over-learned material was characterized by widespread activation in sensorimotor and language areas, the dorsolateral prefrontal cortex, focal activation of the medial prefrontal cortex, but deactivation in the lateral orbital prefrontal cortex. The authors argued that this “loosening” of PFC networks via PFC deactivation may mediate creative responses, and Shimamura (2008) similarly argues that shutting off one’s PFC may enhance creativity. The dorsolateral prefrontal cortex receives, comprehends, and utilizes multimodal sensory projections from the association cortex of the posterior parietal lobe in order to develop and initiate informed cognitive and behavioral responses. Thus, exceeding PFC capacity may allow for the uninhibited intake and synthesis of this sensory information, alter sensory perception, or recruit adjacent overlapping areas that are not typically involved in these processes. It is hypothesized that this might help create a novel and creative response which could be adaptive when the reliance and exhaustion of previously utilized PFC regions fail to circumvent challenges.

Arnsten (1998) proposes that in responses to danger, the PFC can be rapidly taken “offline” to switch control of behavior to more primitive brain regions such as the amygdala and basal ganglia that mediate instinctive reactions. High levels of catecholamine released during stress exposure drives the production of cAMP, which disconnects PFC networks while strengthening connections between the amygdala, striatum, and related structures (Arnsten, 1998). It is possible that a
consequence of this increased arousal level could be increased metabolism of stored fats into glucose to be released into the bloodstream and ultimately replenish depleted resources in the brain. It may also allow older, more primitive brain regions associated with survival that are normally under inhibitory regulation to take control (Arnsten, Paspalas, Gamo, Yang, & Wang, 2010). In this way, PFC deactivation might promote self-protective reflexive behaviors that would normally be under inhibitory control.

3. Neuropsychology of music-evoked emotions

There is ample evidence that music activates limbic and subcortical structures that are intimately involved with emotional processes (Peretz, 2010). For example, several experiments have found that the amygdala activates in response to both pleasant and unpleasant music (Ball et al., 2007; Eldar, Ganor, Admon, Bleich, & Handler, 2007; Koelsch et al., 2006). This finding challenges previous views implicating the amygdala as a structure that only responds to threat cues. Blood & Zatorre, (2001) found increased regional cerebral blood flow (rCBF) in the amygdala, anterior hippocampal formation, ventral striatum, midbrain, anterior insula, anterior cingulate cortex, and the orbitofrontal cortex during emotional responses to participants’ self-selected music that elicited intense emotional arousal. Participants in this experiment reported feeling “chills” that accompanied intensely pleasurable emotional arousal characterized by sensations such as “goose bumps”, or “tingles up and down the spine.” Reports of music-evoked “chills” have been compared to feelings of elation and euphoria with a strong visceral component. Ball and colleagues (2007) used original piano pieces as pleasant stimuli and dissonant versions of the same musical piece as unpleasant stimuli, and found blood oxygen-level dependent (BOLD) increases in the basolateral amygdala in response to both versions. Replicating and extending these findings, Fritz et al. (2007) found decreased BOLD in the central aspect of the amygdala with increasing emotional intensity, and increased BOLD in the superior aspect of the amygdala with increasing emotional intensity. Furthermore, they found a functional connection between the central aspect of the amygdala and temporal pole, whereas the superior aspect of the amygdala was functionally connected with the ventral striatum (part of reward circuit) and the orbitofrontal cortex.

Changes in the anterior hippocampal formation in response to music have also been reported (Blood & Zatorre, 2001; Eldar et al., 2007; Koelsch, 2010). The hippocampus is anatomically and functionally connected with the amygdala, is involved in memory formation, and has dense reciprocal connections with structures that regulate behaviors essential for survival and autonomic regulation. Efferent projections from the hippocampus reach the nucleus accumbens for reward salience, other limbic, paralimbic, and non-limbic structures, as well as parts of striatum involved in motor learning (Nieuwenhuys, 2007). These findings place the hippocampus in a pivotal position for the processing of music-evoked emotions. For example, Blood and Zatorre (2001) found that increased chill intensity, or emotional intensity, with consonant music correlated with decreased amygdala and anterior hippocampus rCBF, whereas dissonant music increased rCBF in the hippocampus, parahippocampal gyrus, and temporal pole. Several experiments suggest that the mid-portion of the parahippocampal gyrus processes acoustic roughness, which is perhaps relevant for the decoding of affective content of vocal signals, supporting Sperber and Hirschfeld’s (2004) view that we process music as a super expressive voice.

It is clear then that music’s cultural persistence is due in part to its pleasurable and reinforcing actions on the “reward-circuit” of the brain, a collection of interconnected structures that influence and engage motor movements directed towards obtaining rewards. Activity in the nucleus accumbens correlates with motivation- and reward-related experiences of pleasure (Berridge, 2003), and is active in humans during sexual activity, intake of drugs, eating chocolate, and drinking water when dehydrated (Berridge, 2003; Nicola, 2007), and it is also active while listening to music. The nucleus accumbens, ventral striatum (which contains the nucleus accumbens), amygdala, hippocampus, midline thalamus, and the orbitofrontal cortex are interconnected structures of the reward circuit. The ventral striatum is strongly innervated by dopaminergic fibers from the ventral tegmental area, also called the mesolimbic dopamine system for its dopaminergic connections from midbrain and limbic structures.
Importantly, nucleus accumbens activity correlates with motivation- and reward-related experiences of pleasure (Berridge, 2003) and is considered a “limbic motor interface” (Nieuwenhuys, 2007) as it influences movement towards rewarding targets. Interestingly, the pleasure from listening to music activates the nucleus accumbens in a similar manner as primary reinforcers (Blood & Zatorre, 2001). Using PET, Brown, Martinez, and Parsons (2004) reported ventral striatum activation during listening to unfamiliar pleasant music and others have shown ventral tegmental area activation as well (Koelsch et al., 2006). Investigating differences in pleasantness as a result of the predictability of music, Menon and Levitin (2005) found ventral striatum activation was connected with ventral tegmental area and hypothalamus activation. Koelsch (2010) argued that the hemodynamic changes observed in the ventral striatum in that study (Koelsch et al., 2006) reflected dopaminergic activity. Indeed, the ventral tegmental area and substantia niagra dopaminergically innervate the nucleus accumbens of the reward circuit. Dopamine, as discussed earlier, is also critically involved in PFC functioning. Using PET, Salimpoor et al. (2008) found dopamine binding in the nucleus accumbens during intense music-evoked pleasure, whereas Goldstein (1980) administered a dopamine antagonist (naloxone) that reduced the pleasure ratings of subjects listening to music that normally provided pleasurable emotions. These experiments provide evidence that music can engage the very core of evolutionary adaptive neuroaffective mechanisms with their ability to evoke intense emotions. These experiments also implicate catecholamines (e.g. dopamine) in the experiencing of music-evoked emotions, suggesting that dopaminergic involvement via the mesolimbic system during intensely pleasant emotions can exceed PFC capacity, lead to PFC deactivation, and result in intensely pleasurable feelings.

4. Application of the DFCT to intense music-evoked emotions
Music has the ability to evoke intense emotions by challenging the PFC’s limited capacity for emotional regulation. Music has the ability to activate the reticular activating system via the auditory cranial nerve (8th cranial nerve) as it enters the brainstem and projects to regions of the PFC including the orbitofrontal cortex and ventromedial prefrontal cortex, two regions implicated in emotion and arousal. Magoun (1952) first demonstrated dramatic shifts in arousal as a result of reticular activating system stimulation when he produced wakefulness from sleep in cats by electrically stimulating ascending somatic and auditory paths of the reticular activating system. Music’s potential to affect arousal at the level of the brainstem and reticular activating system may be supported by finding from Blood and Zatorre (2001); rCBF in the midbrain and right orbitofrontal cortex correlated with intensity of “chills” ratings and may reflect orbitofrontal cortex and ventromedial prefrontal cortex recruitment as a result of increasing emotional intensity. Increased reticular activating system and orbitofrontal cortex activity in this scenario likely reflects the second phase of the DFCT, whereas the PFC is able to regulate the resultant emotion. The DFCT predicts a global cerebral activation including the co-activation of posterior sensory and frontal systems in the second phase. A left cortical network including left primary auditory area, posterior temporal, and inferior parietal cortex was found in response to pleasant music (Flores-Gutiérrez et al., 2007) consistent with this notion.

During this time, global energy expenditure and the crowding of shared tasks in the anterior PFC may create dual-task interference, increased metabolic demands and resource exhaustion leading to the third phase of the DFCT where capacity is exceeded, and the PFC deactivates and disconnects from brainstem arousal systems and posterior sensory regions. Indeed, rCBF decreases from baseline in the ventromedial prefrontal cortex were correlated with increasing “chills” intensity (Blood & Zatorre, 2001) possibly reflecting exceeded ventromedial prefrontal cortex functional capacity for emotional regulation. In this case, the “chills” reported in Blood and Zatorre (2001) may in part be a result of the PFC’s inhibitory release of the somatosensory cortex of the parietal lobes, contributing to the profound visceral experience described as feeling “chills”. Furthermore, lesions to the right somatosensory cortex disproportionately impaired emotional experiences to music but left autonomic responses relatively unaffected (Johnsen, Tranel, Lutgendorf, & Adolphs, 2009), supporting the role of posterior sensory systems in emotional experience. Furthermore, based on participants’ self-report data, Blood and Zatorre (2001) suggested that pleasantness and emotional intensity must reach a certain level before “chills” can be experienced, indicating that a “threshold” must be
exceeded to experience intense emotions. The DFCT may contribute a perspective on brain “activation” versus “deactivation” as it relates to emotional intensity; PFC activation may indicate mild emotional intensity, but PFC deactivation is hypothesized to indicate emotional intensification.

Midbrain and ventral tegmental area activation (Blood & Zatorre, 2001) through its dopaminergic projections to the frontal lobes (Peretz, 2010) could disconnect PFC networks while strengthening the amygdala and related structures as the release of dopamine drives cAMP production which disconnects PFC networks while strengthening the amygdala and related structures (Arnsten, 1998). In this way, pleasurable music may effectively exceed orbitofrontal cortex and ventromedial prefrontal cortex capacity, causing abrupt PFC deactivation or disconnection in the third phase. In support of this, during music listening, Koelsch et al. (2006) found a functional connection between the orbitofrontal cortex, superior aspect of the amygdala, and ventral striatum. Koelsch et al. (2006) found that increased BOLD in the superior aspect of the amygdala signals correlated with increasing emotional intensity. This functional network involved in pleasurable music is consistent with the DFCT model; strong “bottom-up” activation is believed to challenge the PFC’s emotional regulatory ability. PFC hypometabolism or activation may reflect diminished PFC capacity, or the underutilization of PFC resources to inhibit strong impulses from the reward circuit may allow it to gain control of behavior and emotion.

Reviewing the neurobiology of music and emotion, Peretz (2010) asks whether subcortical involvement responds to top-down influences from the cortex and whether the subcortical relay can modulate the cortical processing of music. It does appear that the orbitofrontal cortex, lateral orbitofrontal cortex, ventromedial prefrontal cortex, striatum, ventral tegmental area, nucleus accumbens, hippocampus, and amygdala are functionally connected in the case of music and emotion, but the direction of influence is more difficult to determine. It is most likely bidirectional and depends on PFC capacity. In response to Peretz’s (2010) question, the DFCT proposes that the emotional intensity and pleasure of music is potentiated through limbic and subcortical structures’ dopaminergic influence on the PFC that can deactivate it, allowing subcortical structures associated with emotion and reward to act uninhibited.

5. Clinical applications, conclusions, and future directions

The DFCT proposes several neural mechanisms hypothesized to mediate intra-cerebral dynamics of intense affective responses to music that may contribute to the music-evoked emotion research, but may also have broader implications for the understanding of the emotional extremes that characterize neurological disorders and psychopathology. The DFCT may also contribute to theoretically driven, rather than data-driven, methodological improvements in music therapy research for the treatment of the affective, behavioral, and cognitive sequelae of stroke, TBI, and dementia.

Despite music’s obvious recreational value, music therapy research has hinted at promising clinical applications. In stroke patients, music therapy has been successfully used for the rehabilitation of fine and gross motor control when subjects play simple melodies on a keyboard (Altenmüller, Marco-Pallares, Munte, & Schneider, 2009). Listening to self-selected music after stroke appears to improve recovery in the domains of verbal memory and focused attention, and can lead to less depressed mood (Särkämö et al., 2008). Listening to positive music can decrease visual neglect (Soto et al., 2009). In individuals with Parkinson’s disease, isometric musical stimuli can regulate gait and arm control, presumably as a result of music-evoked arousal, priming of motor systems via auditory stimulation, and entrainment of motor systems to the beat of the music (Hoemberg, 2005). Despite these promising clinical applications of music therapy, the neural mechanisms and effect of emotion involved in these changes are not fully understood, and experiments with rigorous methodology such as randomized controlled trials are lacking (Koelsch, 2010).

Similarly, the potentially promising use of music therapy for dementia is hindered by methodological and theoretical shortcomings. Koger, Chapin, and Brotons (1999) showed highly significant effects of music/music therapy with dementia yet noted that analyses of moderating variables were unable to determine the variability between studies using various forms of music therapy. The DFCT
hypothesizes that emotional intensity may be an important moderator to consider in future music therapy research as varying levels of emotional intensity engage cortical and subcortical interactions and may have an impact on learning. A meta-analysis of music therapy with dementia using randomized controlled trials also showed improvements in behavioral, cognitive, social, and emotional functioning but noted that the methodological quality and reporting of studies was too poor to make any firm conclusions about music therapy’s true value (Vink, Bruinsma, & Scholten, 2003). A subsequent meta-analysis yielded similar results showing mostly favorable outcomes for music therapy in reducing depression, anxiety, agitated behaviors, and quality of life, but also highlighted methodological weaknesses (Moreno & Bidelman, 2014). The variability in music therapy implementation (e.g. passive music listening versus playing instruments) is surely one contributing factor to this ambiguity, but theoretical contributions from the DFCT may help improve their methodology and highlight the important role of emotional intensity. Kumar et al. (1999) used music therapy with dementia and measured blood samples before and after four weeks of 30- to 40-min sessions of music therapy five times per week. They found significant increases in norepinephrine, epinephrine after four weeks, but these levels returned to pre-therapy levels at a six-week followup. The previously discussed implication for catecholamines (epinephrine and norepinephrine) in music-evoked emotions may indicate emotional intensity as a possible mediating effect of emotion in music therapy. Specifically, the DFCT may suggest that sufficient levels of emotional arousal during music listening may contribute to the effectiveness of music therapy and as such should be measured and taken into consideration in future music therapy experiments.

Neurological music therapy has established evidence-based therapeutic techniques to re-train and re-educate brain-behavior functions in neurological disorders and injuries, particularly in the area of motor recovery, and is supported by neuroimaging studies indicating a clearly defined network of parietal, cerebellar, and frontal areas involved in processing rhythmity (Schlaug, Marchina, & Norton, 2009; Thaut, Peterson, & McIntosh, 2005). A preliminary neurologic music therapy study showed promising results in improving executive functions, emotional adjustment, decreased depression, and anxiety following TBI (Hegde, 2014). Hegde (2014) has called for theoretical development and additional research to better understand how the emotional component of music plays a role in cognitive rehabilitation for individuals with TBI. Melodic intonation therapy is the use of singing to improve speech in severely non-fluent aphasics. Melodic intonation therapy has shown functional gains in propositional speech that generalized to unpracticed words and phrases, and was supported by neuroimaging indicating melodic intonation therapy’s unique engagement of the right hemisphere through singing and tapping with the left hand to prime the sensorimotor and premotor cortices for articulation that accounted for its effect over non-intoned speech therapy (Wan, Rüber, Hohmann, & Schlaug, 2010). Merrett, Peretz, and Wilson (2014) review the neurobiological, cognitive, and emotional mechanisms in melodic intonation therapy and argued that a better understanding of the interaction between the roles of the cognitive, behavioral, and emotional components of melodic intonation therapy may allow for the design of protocols that maximize the effectiveness of singing therapy for aphasia. Baird and Samson (2014) found that listening to music after a severe TBI facilitated the recall of autobiographical memories. Training programs aimed at improving auditory-cognitive abilities have received mixed success, but recent studies suggest that music training provides robust, longlasting benefits to auditory functions. Additionally, consistent nucleus accumbens activation by pleasant music (Blood & Zatorre, 2001) may indicate a use for music in pain management; the nucleus accumbens is rich in opiate receptors and its efferent projections are largely opioid (Gardner & Vorel, 1998). Thus, music may be a unique way to approach pain management.

To fully examine intense music-evoked emotions, the use of self-selected music that is personally meaningful, emotionally powerful, and capable of exceeding PFC capacity, rather than standardized music samples, is recommended. In order to test the four-phase model of the DFCT, within subject comparisons across time, and correlations of cerebral responses across the entire music listening experience up to and beyond subjective reports of intense emotional experience are required. To test the temporal assumptions of the DFCT, high temporal resolution techniques, such as EEG, will be useful as abrupt PFC deactivation is proposed to mediate intense emotional responses. To test the
cerebral dynamics assumption of the DFCT, neuroimaging techniques with high spatial resolution, such as fMRI, are also recommended, and the use of diffusion tensor imaging would help identify cerebral interactions throughout the four phases of the DFCT. To test the assumption of resource depletion inherent in exceeding capacity, techniques that measure glucose metabolism, blood flow, and oxygen absorption, such as PET and fMRI could be used. A combination of EEG and fMRI techniques, using within subject comparisons, and the use of self-selected music may be a practical start to testing the assumptions of the DFCT.

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