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Focus and Scope
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Acknowledgements

First and foremost, we would like to thank all those people that have dedicated countless hours and have made this publication a possibility. The editorial board and authors have meticulously reviewed and revised the following manuscripts in order to present the best possible product. We would also like to acknowledge the Psychology Department at the New School for Social Research for the continued support of this publication. There are several individuals in particular that have been integral in the continued success of the New School Psychology Bulletin that we would like to acknowledge: Dr. McWelling Todman and Robert Kostrzewa for securing funding, Dr. Howard Steele and the advisory board comprised of previous editors for their guidance, as well as Alyson Aladro for ensuring the Bulletin is as professional in appearance as other top-tier publications. Finally, we would like to acknowledge and thank our readers. It is your ongoing attention, support, and participation that guides the ongoing success of this publication.

Letter from the Editors

As the *New School Psychology Bulletin* (*NSPB*) moves forward with the second decade of publication, the core principles of the journal will continue to be the guiding force in the continued existence of this project. The present issue and the manuscripts contained herein are representative of the dedication on the part of the authors, the editorial board, the layout editor, and all other parties that have been involved with NSPB over the years. The ongoing success of NSPB is, without a doubt, the result of much painstaking effort that often goes unnoticed but should be recognized and applauded.

Our continued dedication to the publication of traditionally underrepresented work in the major journals truly makes NSPB unique in the position it occupies. As we move forward, the focus of the journal will continue to be on providing an outlet for quality graduate student work, as well as to serve as a teaching model for academic publishing. In so doing, it is our goal, and the definitive indicator of our success, that a new cohort of academic psychologists are able to positively contribute to the publication process in the future.

The publication of this issue marks the end of the present editorial team's tenure and an exciting transition. We are pleased to announce that three current editorial board members have been selected as our successors, with the hope that close collaboration between the present team and the new team will elevate NSPB to new heights. Throughout their tenure with NSPB they have demonstrated their dedication and commitment to the improvement of their peer's work, as well as to their own technical knowledge of academic publication. Alissa von Malachowski and Gabriella Santoro will be assuming responsibilities as Co-Editors and Clinton Merck will serve as Assistant Editor. In selecting our successors, we believe we have placed the future of NSPB in the capable care of these three ambitious and eager individuals.

The present issue is but a small sample of the diverse range of material submitted over the past few months. We find the broadness in scope an encouraging sign regarding the reach that NSPB has in the academic community. The reader will find articles on topics ranging from biological bases and modern treatment methods of PTSD to affective responses to music without explicit recognition.

As a parting thought, we would simply like to thank our predecessors for the opportunity to be involved with this publication. It has been a wonderful experience and will likely guide and inform us long in to our futures.

Travis Cyr, Ryan Tellalian & Nadia Nieves
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A Review of the Effects of Prolonged Exposure to Cortisol on the Regulation of the HPA Axis: Implications for the Development and Maintenance of Posttraumatic Stress Disorder

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The present review examines the extant literature in both human and animal experiments of the stress response. Specifically, this paper aims to demonstrate that the prolonged release, and subsequent higher basal levels of cortisol results in altered functioning of the regulatory systems that modulate the hypothalamic-pituitary-adrenal (HPA) axis. Furthermore, the intent is to show that these alterations in neural circuitry and neuroendocrines play a substantial role in the development and maintenance of posttraumatic stress disorder (PTSD). A review of the literature was conducted and summarized according to the three major regulatory systems that interact to facilitate the functioning of the HPA axis (i.e., hippocampus, amygdala, and prefrontal cortex). Finally, the author integrates the findings and provides a theoretical rationale for the development and maintenance of PTSD. Discussions of limitations and future directions are offered throughout.

Keywords: trauma, hormone exposure, stress, neurobiology, neuroplasticity, hypothalamic-pituitary-adrenal axis, translational research, glucocorticoid

Cortisol, also called hydrocortisone, is an endogenous steroid hormone classified as a glucocorticoid and is one of the endocrines produced by the adrenal glands (i.e., endocrine organs located superior to the kidneys). When mammals experience stress, the hypothalamic-pituitary-adrenal (HPA) axis mediates the release of cortisol (De Kloet & Rinne, 2007). The areas of the brain that comprise the HPA axis include the hypothalamus, pituitary gland, and projections to the adrenal cortex. Excitatory inputs converge on neurons located in the hypothalamic paraventricular nucleus, where corticotropin-releasing hormone (a peptide hormone often referred to as the stress hormone) is synthesized. Under stressful conditions the amygdala stimulates the paraventricular nucleus. This results in the release of corticotropin-releasing hormone into the portal circulation, which signals the anterior pituitary gland to release adrenocorticotropic hormone (ACTH, also known as corticotropin; Cullinan, Herman, Helmreich, & Watson, 1995; Franklin, Saab, & Mansuy, 2012). Release of ACTH then stimulates the adrenal cortex, which results in cortisol release into the bloodstream (Jovanovic et al., 2011).

Once released, cortisol, like other glucocorticoids, acts throughout the body and mediates changes in various processes such as inflammatory reactions, immune function, and metabolic regulation by interacting with mineralocorticoid and glucocorticoid receptors. While in the brain, glucocorticoids increase these stress responses and serve to contain or regulate the responses, thus facilitating recovery and behavioral adaptation (Franklin et al., 2012). Containment and regulation are important as a number of sites in the brain can be negatively affected by increased and prolonged exposure to cortisol (e.g., hippocampal dysfunction results in deficits in declarative memory function, reduced neural survival and plasticity, and promotion of inflammatory cascades; Bremner, 1999; Franklin et al., 2012). Although moderate levels of these hormones are responsible for mediation of normal cell functioning, glucocorticoids must be maintained at appropriate levels to preserve homeostatic function of systems (Franklin et al., 2012).

Through negative feedback inhibition, cortisol attenuates the stress response by acting on the hypothalamus, pituitary gland, hippocampus, and medial prefrontal cortex, which suppresses the HPA axis (DeBellis, 2010; van der Kolk, 1996). The negative feedback regulation of the HPA axis is facilitated by the rapid inhibition of corticotropin-releasing

hormone release, as well as a more prolonged down-regulation of corticotropin-releasing hormone and vasopressin expression in the neurons of the paraventricular nucleus (Di, Malcher-Lopes, Halmos, & Tasker, 2003; Franklin et al., 2012). By suppressing the HPA axis, basal cortisol levels are restored and the brain returns to homeostasis (DeBellis, 2010).

However, these reactions can be altered following intensely distressing, psychologically traumatic events. Under chronic, persistent stressors the effectiveness of the stress response is inhibited. Over sustained periods of stress, the negative feedback loop ultimately reduces resting glucocorticoid levels and their secretion in response to subsequent stressors (van der Kolk, 1996). These functional alterations have been observed in individuals diagnosed with posttraumatic stress disorder (PTSD) following a psychologically traumatic event (Southwick, Rasmusson, Barron, & Arnsten, 2005).

Posttraumatic Stress Disorder

PTSD is a mental health diagnosis that results from one or more traumatic events in a person's life whereby the person experiences intense fear, helplessness, and/or horror (American Psychiatric Association, 2013). The disorder is characterized by symptoms of re-experiencing the event (e.g., intrusive recollection of disturbing memories, distressing dreams or nightmares), behavioral avoidance and emotional numbing (e.g., avoidance of people, places, and events that serve as reminders of the event, loss or blunting of primary emotions), and hypervigilance (e.g., increased startle response, diminished concentration; APA, 2013; Roth & Fonagy, 2005). Epidemiological studies suggest that over half of all adults in the United States will experience traumatic stress at some point in their lives (Friedman, Resick, & Keane, 2007).

Although the behavioral manifestations and impact of psychological trauma are subject to individual differences, and the construct of PTSD is broad (particularly in light of recent updates to the criteria set forth in the DSM-5; Galatzer-Levy & Bryant, 2013), about one-third of the individuals who experience a trauma will go on to receive a PTSD diagnosis. Indeed, these individual differences may result from differential functioning of the HPA axis among those who are able to recover following a traumatic event (with little or no impairment from subjective psychological symptoms) compared to those who develop PTSD (Franklin et al., 2012; Southwick et al., 2005).

Although results from studies examining basal urinary cortisol levels among individuals with PTSD have been mixed though often finding lower baseline cortisol levels (Yehuda, Resnick, Schmeidler, Yang, & Pitman, 1998), studies examining cerebrospinal fluid (CSF) have shown that basal cortisol levels are higher among PTSD-diagnosed individuals than compared healthy controls (Baker et al., 1999; Baker et al., 2005). Furthermore, the CSF levels were shown to be higher compared to within-subject urinary levels. This suggests that urinary measurements may not reflect the actual level of cortisol within the central nervous system (Southwick et al., 2005). Moreover, studies that examined cortisol levels in individuals within the acute aftermath of trauma found that cortisol levels are increased and prolonged just after a traumatic event and in the face of acute stress (Bremner et al., 2003; Lemieux & Coe, 1995; Pitman & Orr, 1990). Thus, it has been suggested that immediate and prolonged exposure to cortisol results in HPA adaptation over time, which may in turn explain later reductions in cortisol levels and the resultant inefficiency in the normal HPA suppression and behavioral adaptation processes (Bremner, 2001; Handwerger, 2009).

Another reason for mixed findings may lie in the comorbidity between PTSD and depression (Morris, Compas, & Garber, 2012). Indeed, this has been suggested by studies that have found reduced activity of intracellular cortisol-deactivating enzymes 5alpha-reductase and 11beta-HSD2, resulting in high amounts of circulating cortisol (hypercortisolemia) among clinically depressed individuals in comparison to healthy controls (Romer et al., 2009). Similar findings have emerged in individuals with PTSD, demonstrating a three-fold less hepatic 5alpha-reductase presence and a deficiency of 11beta-HSD2 in the kidneys. These deficiencies slow the body's ability to clear cortisol, thus increasing the amount of bioavailable cortisol (Yehuda & Seckl, 2011). Ultimately, research in this area of PTSD is relatively young and, as a result, will likely be subject to debate until more methodologically sound studies are available.

At this time, there is sufficient evidence to suggest that cortisol levels are increased and prolonged in individuals who experience a traumatic event and that such exposure to higher levels of cortisol can result in changes to the neural circuits that mediate the fear and stress response, as well as emotional and memory processing. For the purposes of the present review, the focus will be on alterations in neurobiological functioning in the presence of PTSD. Specifically, the intent is to demonstrate that the prolonged release and subsequent higher basal levels of cortisol can result in altered functioning of the regulatory systems that modulate the HPA axis and play a substantial role in the development and maintenance of PTSD. To do so, brief reviews and integration of the literature will be provided for the three major regulatory systems that interact to facilitate functioning of the HPA axis: the hippocampus, the amygdala, and the prefrontal cortex. Furthermore, an overview of the impact of higher basal levels of, and prolonged exposure to, cortisol will be provided for each area.

Effects of Cortisol on the Hippocampus

The hippocampus is a brain region that is largely responsible for the formation, consolidation, and storage of memories—specifically declarative memory—and is indirectly implicated in emotion. Recent studies examining the effects of N-methyl-D-aspartate (NMDA) receptor subunit deletion (from the granule cells of the dentate gyrus) in genetically modified mice, suggest that the hippocampus may serve a functionally distinct role in anxiety. With NMDA receptor deletion in the ventral portion of the hippocampus, anxiety was reduced in addition to spatial memory impairment (Barkus et al., 2010). These dual roles make the hippocampus a prime structure in which to consider the implications of prolonged stress. Furthermore, the hippocampus has also been shown to negatively regulate the HPA axis by decreasing glucocorticoid secretion in rat and human brains following stimulation (Herman, Ostrander, Mueller, & Figueiredo, 2005). Following chronic stress conditions, glucocorticoid receptors in the hippocampus, which aid in dexamethasone-mediated negative feedback of the HPA, are down-regulated (Herman et al., 1989; Mizoguchi, Ishige, Aburada, & Tabira, 2003; Sapolsky, Krey, & McEwen, 1984). The down-regulation of glucocorticoid receptors disrupts the hippocampus' ability to provide inhibitory feedback under chronic stress conditions (Herman et al., 1989; Mizoguchi et al., 2003; Schloesser, Manji, & Martinowich, 2009).

Indeed, the hippocampus is one of the sites where a large number of Corticotropin-releasing hormone receptor 1 are found (Franklin et al., 2012). As a result, the structure is a primary target for glucocorticoids and exhibits a notable sensitivity to their binding, such that both marked neurochemical and electrophysiological changes are observed in their presence (Sapolsky, Uno, Rebert, & Finch, 1990). Furthermore, studies involving rodents have demonstrated that prolonged hippocampal exposure to cortisol can damage the structure and accelerate aging, create changes in electrical activity, and result in neuronal loss (Sapolsky et al., 1990). Sapolsky and colleagues (1990) sought to expand upon these findings by examining the impact of prolonged glucocorticoid exposure in four male vervet monkeys. The researchers surgically implanted pellets secreting either cortisol or cholesterol (control) into the hippocampus. After a year of exposure to these conditions, overall hippocampal size did not observably differ by group and the cholesterol group was essentially normal; however, in the cortisol group, damage was observed in the pyramidal neurons, which exhibited abnormal shrinkage or elongation of somas and abnormalities in dendritic stems and branches. The researchers concluded that the results were in agreement with literature suggesting that increased and prolonged exposure to cortisol accelerates hippocampal neuron loss. A more recent review by Sapolsky (2000) offers some insight as to why overall hippocampal size decreases were not observed, despite finding significant damage and neuron loss. Although overall hippocampal size did not differ, it is unclear to what extent this was examined in the study or whether examination was bilateral. Indeed, many studies that show decreased hippocampal size following stressors (usually traumatic events) do not demonstrate decreased size bilaterally (i.e., significantly smaller size in either right or left with a non-significant contralateral finding). Furthermore, Sapolsky et al. (1990) may not have found significant volume changes as a function of the magnitude of cortisol exposure (i.e., the amount

of cortisol may not have been sufficient to result in overall volume loss). Sapolsky's (2000) review found the common thread of trauma severity to be related to hippocampal volume loss. The more intense the trauma, the more likely we are to find a significant loss of volume in the hippocampus (Sapolsky, 2000). Perhaps the amount of cortisol exposure varies in a significant way as to result in differential hippocampal volume changes, or, more likely, it is the combined neurobiological and psychological effects following severe trauma that lead to these changes.

Such changes in the hippocampus, including volume loss and atrophy, create significant deficits in the hippocampus' ability to participate in the regulation of the HPA axis. Furthermore, the hippocampus' ability to regenerate these cells (neurogenesis) is impaired by exposure to stressors (Gould & Tanapat, 1999). Gould, McEwen, Tanapat, Galea, and Fuchs (1997) examined adult neurogenesis in the dentate gyrus of the hippocampus in tree shrews under stressful conditions. To examine whether granule cell production occurs under stress, the researchers injected bromodeoxyuridine (BrdU; a marker of DNA synthesis) into label cells in the dentate gyrus. Psychosocial stress was induced by removing an opaque partition that separated two adjacent cages, which resulted in competition and establishment of dominance between the shrews. The shrew that took the subordinate role was virtually immobile and showed characteristic signs of distress. After one hour, the partition was replaced and BrdU injections were given. Elevations in urinary cortisol levels were observed in the subordinate shrew compared to the dominant shrew and controls. Two hours after injection, the subordinate shrews were anesthetized, examined, and then compared to control shrews, which were not exposed to stress. The results indicated that a single exposure to psychosocial stress for one hour resulted in a significant decrease in BrdU-labeled cells in the dentate gyrus in comparison to controls and reflected a reliable change in the number of proliferating cells. Although these results may provide insight into the effects of stress on neurogenesis in the short-term, the long-term implications remain an empirical question. It would stand to reason that prolonged life stressors may exacerbate and perpetuate this effect, at least for some period of time; future studies are needed to examine both chronic stress conditions (e.g, trauma-exposure) and situational stress conditions (e.g., non-traumatic stress exposure) to explore differential effects of habituation.

In addition to changes in the structure and plasticity of cells in the hippocampus, inhibition of neurogenesis results in potentiated response from the HPA axis following stress. Schloesser et al. (2009) examined the impact of loss of neurogenesis in the hippocampus on the efficiency of the hippocampus to inhibit the HPA axis. Transgenic mice, genetically altered for complete suppression of the doublecortin-positive neuroblasts in the dentate gyrus, were used so that conditional suppression of adult neurogenesis was possible through viral kinase and promoter genes. Plasma concentrations of corticosterone (cortisol equivalent in mice) were measured in normal conditions (being in the home-cage environment) and under mild stress (being placed in a novel cage environment). A significant increase in corticosterone levels was observed in non-neurogenesis mice exposed to a mild stressor compared to non-neurogenesis mice that were not exposed to stress and controls. Their findings suggest that suppression of neurogenesis in the hippocampus results in an increased HPA axis response and that new neurons formed in the dentate gyrus are critical to the inhibitory regulation of the HPA axis by the hippocampus.

Given that prolonged and increased levels of cortisol are observed in the aftermath of psychologically traumatic events, and that the bioavailability of cortisol appears to be maintained over a longer term among those with PTSD, it is not difficult to begin to understand the vicious cycle that takes hold. The increased and chronic exposure to cortisol results in impairment of the hippocampus, which results in poorer anxiety coping and reduced inhibitory modulation of the HPA axis; this, in turn, leads to increased HPA activity under stress while leaving the individual with continuously increased anxiety response, as well as memory deficits resulting from hippocampal volume reduction (Bremner, 1999). Furthermore, the extinction of conditioned fear responses—the reduction and ultimate extinction of a conditioned fear response following repeated presentations of conditioned stimuli without the unconditioned, originally feared, stimuli—is contingent on the ability to efficiently form new

associative relationships. Fear responses to stimuli present during a traumatic event are often generalized to similar, innocuous stimuli. For example, the sound of gun fire is generalized to cars backfiring or a loud sound from a heavy falling object, thus resulting in an exaggerated startle or cover response. To this end, the hippocampus has been shown to be involved primarily in the acquisition of the fear response to stimuli (conditioned response) and may mediate the awareness of the conditioned stimulus to unconditioned stimulus relationship (Knight, Smith, Cheng, Stein, & Helmstetter, 2004). However, it is the amygdala that serves to aid in the processing of emotional content (Herbert et al., 2009), changes in environmental relationships among stimuli (Knight et al., 2004), and the principle site of convergence between the conditioned stimulus and unconditioned stimulus (Hashikawa et al., 2013; Knight et al., 2004). Not surprisingly, amygdalar functioning is also altered by increased exposure to cortisol (Ardayfio & Kim, 2006).

Effects of Cortisol on the Amygdala

Kluver and Bucy's (1939) lesion studies first implicated the amygdala as the structure mediating the emotional significance of stimuli. Specifically, the amygdala is primarily responsible for the automatic, non-conscious processing of threatening stimuli. The amygdala receives signals from sympathetically activated regions (e.g., brainstem, thalamus, and sensory) responsible for orienting attention toward a threat, which facilitates an individual's ability to target, identify, and assign emotional information to a stimuli in the interest of self-preservation. Thus, under stressful conditions, the amygdala aids in the ability to quickly discriminate between threatening and neutral stimuli, encode the emotionally salient information via projections to the hippocampus, and help prepare the body for action (Kensinger & Corkin, 2004).

As previously discussed, one of the amygdala's duties is to promote the stress response following stimulation from the locus coeruleus. However, with 13 distinct subnuclei, the amygdala is implicated in a number of emotional processing and stress modulating processes (Ressler, 2010). Among these is the process of fear extinction. Fear extinction appears to be dependent on intercalated amygdala cells within the central nuclei and, when disrupted, impairs extinction (National Institute of Health [NIH], 2010; Ressler, 2010). The central nuclei of the amygdala is responsible for the regulation of cortisol release via projections to the paraventricular nucleus of the hypothalamus (activating the HPA axis), as well as modulation of behavioral responses to fearful stimuli. Lesions in the central nuclei have been shown to result in elimination of conditioned fear responses in rodents (Ressler, 2010), as well as reduced ACTH and glucocorticoid release following stress (Herman et al., 2005).

Knight et al. (2004) utilized functional magnetic resonance imaging (fMRI) to examine brain activity during the acquisition and extinction of fear conditioning. During acquisition, human subjects were either exposed to a light paired with an electrical shock or a light without an electrical shock. During extinction, half of the subjects in the active group continued to receive the pairing while the remainder, as well as the control subjects, received the light without the shock. Results showed increased hippocampal activity during acquisition, whereas increased amygdalar activity was observed when experimental conditions changed, specifically in the absence of the shock following acquisition.

In addition to the amygdala's role in acquisition under uncertain stressful conditions or conditions that run counter to the learned expectation, the amygdala acts to moderate the consolidation of emotionally charged information (for a review see Paré, 2003). Buchanan and Lovallo (2001) examined the effects of cortisol versus placebo on human memory performance. Their findings demonstrated that exposure to cortisol during acquisition and encoding resulted in better long-term recall performance for emotionally charged stimuli compared to neutral stimuli. Therefore, under stressful conditions, memories are more efficiently consolidated when the stimuli are perceived as threatening or fear-congruent.

Another neuroimaging study conducted by van Stegeren et al. (2007) found that individuals with higher levels of cortisol showed significantly greater amygdalar activation when viewing emotional content compared to individuals with lower levels of cortisol. However, cortisol did not exert this effect in isolation. An interaction between the basal level of cortisol and levels of arousal-induced noradrenergic

activation in the basolateral complex of the amygdala appeared to highlight a synergistic relationship that may result in enhanced consolidation of emotional memories. This interactive relationship between increased cortisol and norepinephrine has been found in other studies, as well, and has been shown to result in an exaggerated amygdalar response equivalent to that found in individuals with PTSD (Gueze et al., 2012; Kukolja et al., 2008).

Kukolja and colleagues (2008) examined the role of the glucocorticoid-noradrenergic interaction in amygdalar activation using pharmacological interventions to artificially increase the levels of cortisol and norepinephrine. Sixty-two healthy human participants were assigned to one of four conditions: (a) placebo, (b) a selective norepinephrine reuptake inhibitor, (c) hydrocortisone, or (d) a combination of both drugs. Subjects were then placed in an fMRI scanner where amygdalar activation was measured during the presentation of emotional faces. Results indicated that a negative response bias was created under the combination condition, such that amygdalar activity was decreased in response to positive emotional content and increased in response to negative emotional content. This finding may provide insight into the restricted range of emotions (particularly positive) exhibited by individuals with PTSD.

These studies show that the amygdala is both responsible for the release of cortisol through its role in the HPA axis and, in the interaction with norepinephrine, affected by increased levels of cortisol. Studies examining the amygdala as a target for glucocorticoids have shown that the central and medial nuclei express both glucocorticoid and mineralocorticoid receptors, which make it reactive to basal levels of cortisol in addition to stress-related release (Herman, Ostrander, Mueller, & Figueiredo, 2005). Akana, Chu, Soriano, and Dallman (2001) examined the site-specific and state-dependent effects of glucocorticoids on the amygdala and the regulation of ACTH, insulin, and fat depots in rats. The adrenal glands were surgically removed from the rats and corticosterone was administered in low doses by the researchers. Half of the rats were kept in room-temperature environments, while the other half were subjected to cold (5° C) over five days before being restrained (as the stressor). Results indicated that implants of glucocorticoids in the central nucleus do not modulate the acute stress response; rather, they seem to be implicated in the alteration of neural output from the central nucleus to preganglionic sympathetic neurons while under chronic stress. Thus, in individuals with higher basal cortisol levels, the amygdala will likely continue to respond in the usual feed-forward fashion to potentiate the HPA axis while exerting, to some extent, an influence over the sympathetic branch of the autonomic nervous system. Though not explicitly stated in the literature reviewed, increased influence of the amygdala on autonomic function, as a result of exaggerated activity, may explain some of the pervasive hyper-arousal symptoms experienced by individuals with PTSD.

Effects of Cortisol on the Prefrontal Cortex

In addition to exaggerated amygdalar response, individuals with PTSD have been shown to exhibit inefficient functioning of the prefrontal cortex (Gueze et al., 2012). Indeed, a number of studies show support for prefrontal cortex influence on amygdalar function. For example, the ventro-medial prefrontal cortex is thought to play a role in the inhibition of the amygdala and may facilitate fear extinction (Delgado, Nearing, LeDoux, & Phelps, 2008).

The prefrontal cortex plays a role in higher order functions such as cognition, affect regulation, and social reasoning. Moreover, regions of the prefrontal cortex (particularly the anterior cingulate, medial, and ventromedial regions) have also been implicated in the stress response (Herman et al., 2005). Like the hippocampus, the prefrontal cortex plays a role in the inhibition of the HPA axis and takes on the added duty of inhibiting the amygdala. Furthermore, just as in both the hippocampus and the amygdala, the prefrontal cortex is home to a high density of glucocorticoid receptors, primarily in the medial region (Sanchez, Young, Plotsky, & Insel, 2000). Because of the role of the medial prefrontal cortex in higher order functions and its diverse ascending and descending projections, it is thought that this region is crucial to the overall function and regulation of the HPA axis (Kern et al., 2008). In rodent models, lesions to this area result in significant increases in adrenocorticotropic hormone and cortisol release under stress. It has been suggested that the ventral region of the medial

prefrontal cortex may exert an excitatory influence on the HPA axis, perhaps allowing the prefrontal cortex to play a larger role in the maintenance of HPA axis equilibrium.

Kern et al. (2008) sought to examine distinct patterns of prefrontal cortex involvement in neuroendocrine stress control. They hypothesized that both positive and negative associations between stress-induced glucose metabolic rate and saliva cortisol concentrations would be present depending on specific locations within the prefrontal cortex (positive associations in lateral regions, and negative associations in medial dorsal regions). The researchers assigned 14 human subjects to either a stress or control condition. Subjects attended three sessions and baseline positron emission tomography (PET) scans were established approximately 14 days after the first session (session one involved only informed consent and study procedure explanation) and another scan exactly one week later. Subjects in the stress condition were confronted with an established, structured psychosocial stress situation (a modified version of the Trier Social Stress Test; Kirschbaum, Pirke, & Hellhammer, 1993). The control condition followed a structured situation that was determined to be similar to the stress condition, without the stress component. Results implicated Brodmann areas 9 and 10 in the medial prefrontal cortex as part of the regulatory circuitry that modulates responses to stressful stimuli. Lateral regions were found to be associated with increased cortisol levels and appear to lend support to the relationship between this region and subjective discomfort experienced during stressful social interactions and the resultant withdrawal behavior. The authors conclude that their data lend support for Brodmann area 10 in voluntary regulation of negative emotion, particularly during socially threatening situations. Another finding with regard to Brodmann area 9 is its implication in voluntary down regulation of negative affect states, which is inactivated during perceived negative emotions. The authors conclude that Brodmann area 9 may function as an integration circuit, allowing the regulation of affective states while coordinating active coping behaviors under stress. Despite potential limitations due to small sample size, these results provide promising evidence that the prefrontal cortex provides substantial regulation of both neural circuits that modulate the HPA axis and the regulation of subjective affective content while under stress. However, as previously mentioned, the prefrontal cortex has a large number of glucocorticoid receptors. Just as other areas are vulnerable to alteration as a result of increased basal cortisol levels, detrimental effects on the functional efficiency of the prefrontal cortex have been observed.

Carrion, Weems, Richert, Hoffman, and Reiss (2010) examined the impact of increased cortisol on prefrontal cortex volume among adolescent humans with PTSD. Using magnetic resonance imaging (MRI) and salivary cortisol measurements, the authors found that higher levels of cortisol were related to significantly decreased prefrontal cortex tissue volume (particularly in the ventral and inferior regions) among subjects with PTSD symptoms. Although these findings are restricted to adolescents, the changes in structural volume found in this study are consistent with the effects of prolonged exposure to cortisol observed the amygdala and hippocampus. Thus, it may be prudent to consider, however cautiously, these findings in the context of impairments observed in adult PTSD. If these changes are taking place more generally, reduced volume in prefrontal cortical tissue may help to explain the overall restriction of positive emotions, social withdrawal and avoidance, irritability, and concentration difficulties experienced by individuals with PTSD. Furthermore, inefficiencies resulting from these changes in the prefrontal cortex may also explain the resistance of traumatic memories to the extinction process.

Livneh and Paz (2012) sought to examine the neural basis for the resistance to extinction of aversive memories. As with individuals suffering from PTSD, impaired fear extinction results in a maladaptive and persistent anxiety response despite the absence of actual threat. Whereas the amygdala has been shown to enhance the processing of emotional memories, the dorsal anterior cingulate cortex is thought to interact with the amygdala (via direct connections) and aid in the regulation of expressing learned fear responses. Furthermore, in terms of conditioned learning, the dorsal anterior cingulate cortex has been shown to play an important role in the processing of uncertainty (Huettel, Stowe, Gordon, Warner, & Platt, 2006; Krain, Wilson, Arbuckle,

Castellanous, & Milham, 2006) and functions differentially in the face of continuous and partial reinforcement (Dunsmoor, Bandettini, & Knight, 2007; Hartley, Fischl, & Phelps, 2011; Milad et al., 2007). Livneh and Paz (2012) hypothesized that the amygdala and this region of the prefrontal cortex would function differentially under different reinforcement schedules (continuous and partial). To test their theory, Livneh and Paz put two monkeys through a tone-odor conditioning task. A partial reinforcement schedule was used on randomly intermingled days with a continuous reinforcement schedule. Memory expression was measured by volume of breath intake (measured by a pressure-sensitive mask attached to the nose) that followed the tone prior to odor release, while activity in brain centers was measured by MRI-based electrodes. Results suggest that under continuous reinforcement schedules, activity in the amygdala precedes behavioral responses, whereas prefrontal cortex activity precedes behavioral responses under partial reinforcement. Furthermore, the persistence of the behavioral response, and ultimately the resistance to extinction, was observed in the prefrontal-mediated partial reinforcement condition but not the amygdalar-mediated continuous reinforcement condition. The authors conclude that this finding suggests that the tone-odor associations were acquired differentially, depending on reinforcement schedule. Thus, while the amygdala appears to function most efficiently when rapid, simple sensory associations must be made, more complex forms of conditional learning, like the probabilistic learning in the partial reinforcement trials, appear to recruit the dorsal anterior cingulate cortex, which results in resistance to extinction. Damage to the prefrontal cortex impairs these processes, thus it stands to reason that reduction in cortical volume in areas of the prefrontal cortex may lead to impaired, or at least inefficient, complex conditional learning.

Summary and Conclusion

The aim of the present review was to demonstrate that the prolonged release, and subsequent higher basal levels of cortisol, results in altered functioning of the regulatory systems that modulate the HPA axis. Furthermore, the aim was to show, to the extent possible, that these alterations in neural circuitry and neuroendocrines play a substantial role in the development and maintenance of PTSD. A review of the literature was conducted and summarized according to the three major regulatory systems that interact to facilitate functioning of the HPA axis: Hippocampus, amygdala, and prefrontal cortex.

Ultimately, while research regarding normal stress reactions has a long history of publication, much less has been done in respect to PTSD. Where possible, relevant research specific to PTSD was reviewed and research-informed speculation offered in its absence. Furthermore, where studies in humans are available they are often reliant on relatively young technologies (e.g., functional MRI), which reduce the confidence with which researchers can make definitive causal statements. Although support may be lent from animal studies, methodological differences between animal and human studies, as well as the usual translational problems, exist. However, despite these limitations, some consistent findings have been demonstrated across studies that may begin to paint a coherent picture of the interactions between the many processes involved in the stress response.

Overall, research has demonstrated that basal levels of cortisol may be increased in individuals who experience PTSD. Although some of the earlier literature appears to be mixed, some finding lower urinary levels of cortisol while others find higher levels, more recent studies utilizing more reliable measures of bioavailability (e.g., CSF) seem to be tending toward a model of higher basal cortisol levels following trauma. In the context of studies finding reduced efficiency in the metabolism of cortisol following traumatic stressors, it may help explain why studies find lower levels of cortisol in urinary samples, as it is not being processed and flushed by the kidneys due to enzymatic deficiencies.

This increase in the bioavailability of cortisol, which has broad reaching effects on the body and is free to cross the blood-brain barrier, may continuously bind to HPA regulatory structures and create a cycle of inefficient stress responses. Whereas normal function of the stress response utilizes cortisol to aid in the containment of stress responses, when cortisol is not maintained at appropriate levels, detrimental effects are observed in regulatory structures. Furthermore, prolonged exposure to high levels of cortisol has been

shown to result in significant alterations in the cells that comprise the structures. For example, chronic cortisol exposure was shown to significantly reduce hippocampal volume as a result of cellular atrophy, which ultimately disrupted neurogenesis. As a result of this disruption, the inhibitory regulation of the HPA axis by the hippocampus is prevented, which leads to an increased HPA response. Similarly, the central and medial nuclei of the amygdala express both glucocorticioid and mineralocorticoid receptors, which make it reactive to basal levels of cortisol in addition to stress-related release. For this reason, the amygdala will likely continue to promote and potentiate the HPA axis while also exerting influence over the sympathetic branch of the autonomic nervous system. Like the hippocampus, the prefrontal cortex exerts an inhibitory effect on the HPA axis, as well as on the amygdala, creating a down regulating effect. Thus, lesions to the prefrontal cortex not only reduce inhibitory effects on the HPA, but also free the amygdala to promote effects, thus increasing sympathetic activation. In all three cases, the ultimate response is some form of general failure to inhibit the HPA axis, which may ultimately contribute to the amount and circulation of cortisol that remains bioavailable.

With regard to the effects of these alterations on the development and maintenance of PTSD, each structure also appears to have a corresponding expression that is observable in PTSD. Though not always explicitly connected in the literature, fairly clear relationships appear to exist. As shown above, impairment of the hippocampal functioning results in poorer anxiety coping, leaving the individual with continuously increased anxiety response as well as memory deficits. Meanwhile, the amygdalar response bias toward negatively-charged emotional content following increased cortisol exposure map on to PTSD symptoms related to restricted range of emotions, irritability, and dysphoria. Furthermore, increased influence of the amygdala on autonomic function, as a result of exaggerated activity related to cortisol exposure, may also be related to hyperarousal symptoms experienced by individuals with PTSD. The amygdala is also related to the extinction of aversive reactions to traumatic and stressful memories. When this process is impaired there is disruption in the ability to extinguish conditional fear learning, which may relate to the frequency of recurrent, unwanted memories of traumatic events, as well as nightmares and other re-experiencing symptoms in PTSD. The prefrontal cortex has been shown to work closely with the amygdala and provide higher order functions that influence emotional content and mediate the appraisal of complex conditional learning paradigms. Together with the amygdala, reduced volume in prefrontal cortical tissue may aid in the overall restriction of positive emotions, social withdrawal and avoidance, irritability, and concentration difficulties experienced in PTSD, as well as difficulty extinguishing more complex, conditioned fear and anxiety following trauma.

One of the issues experienced during the present literature review was the need to interpret some results cautiously due to methodological flaws or limitations, or small sample sizes, which lack sufficient statistical power to establish reliable relationships between variables. Aside from quality issues and generalizability limitations, although a body of literature does exist, there is a surprisingly limited amount of translational research between psychology and neuroscience. Although the two fields have begun to cross-collaborate over the last decade, a great deal more work needs to be done with regard to examining the behavioral implications (like those expressed in PTSD) in the context of these complex neural systems. More collaborative research will not only advance the science and bridge the gaps between basic science and clinical practice, but also begin to provide reliable models of psychological reactions to specific alterations in neural systems.

While this may not happen in the immediate future, the clinical implications of such research might include more effective medications and better informed psychological interventions. Moreover, understanding the neural reactions to stressful events, both in the immediate aftermath and the long-term, would provide invaluable information regarding the development of preventative interventions that may allow us to minimize or eliminate the chances of developing a psychological disorder. In particular, given the findings reviewed herein, it is clear that an increased bioavailability of cortisol has a number of short and long-term deleterious

effects on the overall function of the stress response. Indeed, there is sufficient evidence to conclude that such prolonged exposure to cortisol serves to promote the development of PTSD. However, much more work is needed, particularly in human samples, and future research must consider not only cortisol, but the mechanisms involved in its regulation (e.g., enzymatic degradation to reduce the unchecked bioavailability of the hormone). Additionally, due to evidence that individuals with PTSD exhibit deficiencies that slow the body's ability to breakdown and pass cortisol, it is clear that examining urinary hormone levels will not yield reliable data. For this reason, researchers in this area will need to expand their training or collaborate with qualified individuals who can perform CSF sample extractions in order to glean more reliable comparisons. Such data may help to inform short-term interventions to improve prognosis. For example, if it is found that enzymatic deficiencies play a role in the chronicity of PTSD, perhaps pharmacological interventions (to improve cortisol degradation) may be developed to either reduce the chance of developing PTSD in the aftermath of trauma, or at least curb the impact prolonged cortisol exposure would have on the brain, thus making psychological interventions more effective.

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Virtual Reality Exposure Therapy for Military Veterans with Posttraumatic Stress Disorder: A Systematic Review

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Military personnel in a war situation appear to be especially susceptible to posttraumatic stress disorder (PTSD), given that a high number of recently deployed service members are diagnosed with the disorder. Exposure therapy is frequently used in the treatment of this population. To our knowledge, however, there are no detailed systematic reviews of a relatively new exposure therapy, virtual reality exposure (VRE), in the treatment of combat veterans with PTSD. Therefore, the present work provides a comprehensive, 15-year overview (1999 to 2013) of research on the therapeutic effectiveness of VRE in treating PTSD in military combat veterans. A considerable body of empirical evidence suggests that VRE can decrease PTSD symptoms within the veteran population and follow-up data do show promise for maintaining positive treatment outcomes. Further research is necessary to determine the long-term effects of this treatment.

Keywords: posttraumatic stress disorder, military veterans, virtual reality exposure therapy, exposure therapy

Posttraumatic stress disorder (PTSD) is a psychological disorder that develops during a time of increased stress following the experience of a traumatic event, in which one's life is at risk or threatened, or the person witnesses another person experience a traumatic event, involving death, injury, or a threat to that person's welfare (DSM-V; American Psychiatric Association, 2013). The prevalence rate for PTSD is about 3.5% of the United States' population, although military personnel in a war situation appear to face a higher risk level for developing PTSD (DSM-V; American Psychiatric Association, 2013; Gates et al., 2012). The actual prevalence rates for PTSD among United States (U.S.) military personnel are difficult to estimate, mainly because of secondary gain, in which veterans might benefit from the diagnosis (see Gates et al., 2012). However, epidemiological research conducted with former military personnel suggests that there is a lifetime PTSD prevalence of 31% for men and 26.9% for women in Vietnam veterans, a 10.1% prevalence in all Gulf war veterans, and a 13.8% prevalence in Afghanistan and Iraq war veterans (Department of Veterans Affairs, 2013).

This higher prevalence of PTSD for veterans likely stems from the fact that military personnel have an increased chance of experiencing a wide array of traumas ranging from rape, capture, torture, physical assault, physical injury, and witnessing a fellow soldier's death (Briere & Scott, 2006). Multiple deployments, which became increasingly common in the Afghanistan and Iraq conflicts, increase the risk for developing mental health problems, and military personnel who have deployed multiple times are more likely to show an increase in mental health issues, as are those who stayed longer in theater (Mental Health Advisory Team V, 2008). Thus, it is imperative to find effective methods of treating war veterans.

Treatments for PTSD have evolved over the years to include behavior therapy, biofeedback, eye movement desensitization reprocessing (EMDR), stress inoculation therapy (SIT), cognitive therapy, contemporary cognitive behavior therapy (CBT), and exposure therapies (e.g., systematic desensitization, flooding; Haugen, Evces, & Weiss, 2012; Wiederhold

& Wiederhold, 2005).

Exposure therapy shows some of the strongest empirical support for successfully treating PTSD (Ready, Pollack, Rothbaum, & Alarcon, 2006; Rothbaum, Hodges, Ready, Grapp, & Alarcon, 2001), and it is therefore currently considered the treatment of choice (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010). This therapy involves the repetition of the traumatic memory using imaginal or real exposure. The goal of exposure treatment is to decrease anxiety through identifying, discussing, and emotionally processing the traumatic event(s). Evoking the same emotions and feelings that occurred in the original traumatic event is key to exposure treatment's success (Bush, 2008). Emotional processing requires the activation of the fear structure during the treatment. Once the fear structure becomes activated, to decrease anxiety, the client's emotional engagement is targeted for change (Reger & Gahm, 2008). A hierarchy of fears is established, and feared stimuli are addressed beginning with the least aversive. Repeatedly approaching a feared stimulus during exposure allows the fear structure to be activated and then paired with safe outcomes (Foa & Kozak 1986). When the least aversive fear has been sufficiently reduced, the next fear is approached, and so on (Wiederhold & Wiederhold, 2005). Gradually, this leads to adequate processing of the traumatic event and extinction of fear (Foa & Kozak, 1986).

Three types of exposure therapies have traditionally been recognized as being effective treatments of PTSD: a) systematic desensitization, which involves training the client in relaxation and then introducing a series of increasingly anxiety-provoking situations to which the client practices relaxation until the most feared situation no longer causes significant anxiety (McGlynn, Mealiea, & Landau, 1981); b) imaginal exposure, where the individual repeatedly imagines his/her traumatic event with the goal of the client habituating to chronic anxiety (Foa & Chambliss, 1978; Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002); and c) in vivo exposure, which requires that the individual is placed in an environment to confront the traumatic or feared situation (Wiederhold & Wiederhold, 2005).

Although exposure therapy has been shown to be effective for treating combat-related PTSD (Ready et al., 2006; Rothbaum et al., 2001), there may be several limitations to this approach. Because one of the diagnostic criteria for PTSD is persistent avoidance of stimuli associated with the trauma (DSM-V; American Psychiatric Association, 2013), it is not surprising that many patients refuse to participate in exposure therapy. Others have difficulty approaching or staying with their traumatic memories at a level that adequately facilitates the emotional engagement necessary for habituation to occur (Rothbaum et al., 1999). It may be possible, however, to address these issues with recent technology that has allowed expansion of the in vivo experience through the use of virtual reality exposure (VRE).

Virtual Reality Exposure Therapy

Through the fairly recent expansion of an in vivo experience into VRE, the client can be immersed into the event that triggered his or her PTSD (e.g., combat) via a multisensory computer simulation without relying on self-generated memories. In fact, virtual reality exposure attempts to offer both in vivo exposure and imaginal exposure techniques (Wiederhold & Wiederhold, 2005). Through this method, the limitations of the patient's imagination and memory are overcome (Riva & Vincelli, 2001), avoidance is reduced, and the likelihood of emotional engagement is increased. The present work critically examines recent literature on this promising type of therapy and highlights the evidence supporting the use of VRE for PTSD in veteran populations.

VRE has advantages over other exposure therapies and other treatment modalities for veterans with PTSD. Virtual reality occurs in real time and offers a sense of presence while encouraging the veteran to return to those prior memories and thoughts in a safe environment. Paradoxically, virtual reality is not reality; nothing can truly harm the individual in this treatment. Successful uses for virtual reality have included the treatment of eating disorders (Wiederhold & Wiederhold, 2005), sexual disorders (Riva et al., 2004), phobias (Hodges et al., 1999; Rothbaum et al., 1995), and during neuropsychological evaluations and medical procedures (Wiederhold & Wiederhold, 2005). In relation to PTSD treatment, researchers have noted that if other exposure treatments such as prolonged exposure are not effective,

virtual reality may be the most effective exposure therapy in assisting individuals with decreasing PTSD symptoms (Reger & Gahm, 2008).

Virtual reality uses computer-based technology in a three-dimensional world, whereby the individual navigates a computer simulation of the triggering traumatic environment in real time (Wiederhold & Wiederhold, 2005). During VRE sessions, the client wears a head-mounted display (HMD), which includes headphones, a display screen for each eye, and a head-tracking device (Rothbaum et al., 2001; Rothbaum, Ruef, Litz, Han, & Hodges, 2003; Wiederhold & Wiederhold, 2005). In some virtual environments the veteran has her or his own chamber with a computer and other measurement devices, which keeps attention focused on the simulation. Researchers contend that head-mounted displays are immersive; they allow the client to explore and experiment with cognitions and emotions while perhaps decreasing a sense of threat (Riva, Bacchetta, Cesa, Conti, & Molinari, 2001; Rizzo, Schultheis, Kerns, & Mateer, 2004).

Virtual reality exposure treatment simultaneously stimulates the senses—the visual, auditory, and olfactory systems—which immerse the veteran in virtual environments during exposure sessions (Cukor, Spitalnick, Difede, Rizzo, & Rothbaum, 2009). Visual stimuli include night vision images, soldiers, civilians, buildings, and vehicles. Specific sounds include weapon fire, explosions, mortar fire, helicopter and vehicle noises, wind blowing, human voices, and radio communications. Olfactory stimuli can be released through a scent palette contained in the chamber; an example of sensory stimuli would be a burning smell. Other stimuli provided can be tactile and kinesthetic; an example would be feeling vibrations on the floor or on the seat, such as in a helicopter VRE session (Gerardi, Rothbaum, Ressler, Heekin, & Rizzo, 2008).

Sensory exposure can be tailored to each veteran's needs. For instance, Virtual Vietnam and Virtual Iraq are simulation environments developed specifically to match what these veterans encountered sensory-wise while deployed. Within the Virtual Vietnam environment there are two subtypes of environments, the Huey Helicopter and an open field (Hodges et al., 1999). The Virtual Iraq environment, again, has two subtype environments, the Middle Eastern City and desert road (Rizzo et al., 2009).

The Present Work: Review of Studies Using VRE with Combat Veterans

The purpose of the present research project is to provide a comprehensive, systematic review of the scientific literature regarding virtual reality exposure's therapeutic success of treating PTSD in military combat veterans and to promote awareness about virtual reality exposure as a viable, contemporary treatment for military personnel with PTSD. As VRE is a fairly new treatment that has not been widely used or recognized, the aim of this work is to provide an organized compilation and analysis of the extant literature on VRE that may serve as a valuable tool for determining its viability as a treatment option.

Although the literature features valuable meta-analyses by other pioneers in the field (see Parsons & Rizzo, 2008; Powers & Emmelkamp, 2008), they have focused on VRE therapy for treating anxiety and specific phobias. Other systematic reviews on virtual reality exposure have also provided excellent narratives on the effectiveness of VRE in treating anxiety disorders (see Meyerbroker & Emmelkamp, 2010) and clinical treatments in general (see Riva, 2005). In addition, Paul, Hassija, and Clapp (2012) provided a review of promising technological advances in treating posttraumatic stress disorder, including VRE. However, their review was brief and included other groups such as the survivors of the World Trade Center attacks. The present work aims to contribute to the body of knowledge by reporting on recent research on VRE therapy directed at improving the lives of combat-exposed veterans with posttraumatic stress disorder.

Method

We conducted a systematic review of the scientific literature from 1999 to 2013, as the first virtual reality exposure session to treat a Vietnam veteran took place in 1999 (Rothbaum et al., 1999). Databases used were PsychINFO, PubMed (Medline), Published International Literature on Traumatic Stress (PILOTS), and the Staff College Automated Military Periodicals Index (SCAMPI), allowing for the careful

consideration of peer-reviewed journal articles and books that detailed studies of VRE therapy in military veterans. Selected key-words for searches were as follows: Posttraumatic Stress Disorder, military veterans, virtual reality exposure, VRE, combat experience, and military psychology. Military veterans, for the purpose of this systematic review, included any military personnel who were diagnosed with combat-related PTSD. This included retired military personnel and active duty personnel. To be included, the article had to address each factor; that is, PTSD, military veterans, and VRE. The resources that the search yielded were carefully examined by reading the abstracts to determine whether or not they met the inclusion criteria. Below we describe the empirical research studies that meet the aforementioned criteria.

Results

VRE Treatment of U.S. Veterans of the Vietnam War

Rothbaum et al. (1999) conducted the first study of virtual reality exposure treatment effectiveness with a veteran. This was a case study of a 50-year-old male Vietnam veteran diagnosed with PTSD and major depressive disorder. The veteran completed the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), Combat Exposure Scale (CES; Keane et al., 1989), Beck Depression Index (BDI; Beck, Epstein, Brown, & Steer, 1988), and Impact of Events Scale (IES; Horowitz, Wilner, & Alvarez, 1979) prior to and after treatment. During VRE treatment, the veteran was immersed in the two environments of Virtual Vietnam—the jungle and Huey helicopter environments. Subjective Units of Distress (SUDS) ratings were gathered every 5 minutes during treatment. Treatment took place over seven weeks, with 14 sessions lasting 90 minutes each. Results suggested that the veteran had benefited from the treatment as evidenced by his decrease in overall symptom scores related to PTSD (34% decrease on clinician-rated scores and 45% decrease on self-rated scores), intrusion and avoidance symptoms, and depression (Rothbaum et al., 1999). Although this veteran still met the criteria for depression, his overall symptoms had improved from posttreatment to the six-month follow-up (Rothbaum et al., 1999). This study provided evidence that virtual reality exposure can effectively decrease PTSD symptoms and even comorbid diagnoses.

In another study, Rothbaum et al. (2001) explored the effectiveness of the virtual reality exposure therapy in the treatment of nine male veterans from the Vietnam War. Assessments were conducted at pretreatment, posttreatment, and three- and six-month follow-ups. Treatment consisted of ten 90-minute sessions conducted two times a week for an average of five to seven weeks. The approach required a discussion of the traumatic event in the present tense and also prompted the veteran to keep his eyes open during this discussion and during the VR simulation so the therapist could attempt to match the virtual environment to what the veteran was describing (e.g., helicopters). The therapist assessed SUDS ratings every 5 minutes during exposure sessions.

Of the nine veterans in the study, eight veterans at the six-month follow-up showed reductions in PTSD symptoms as evidenced by CAPS, which showed a range of 15-67% decrease in overall PTSD symptomatology. IES total scores also showed decreases in intrusion and avoidance symptoms from baseline to the three- month follow-up, but there was an increase in symptoms at the six-month follow-up. BDI results also showed a decrease following treatment, and again, an increase which tapered off at the six-month follow-up. Taken together, Rothbaum et al.'s series of studies conducted in 1999 and 2001 provide evidence of the effectiveness of VRE; however, the results from such a small number of participants may not be generalizable. In addition, Rothbaum et al.'s (2001) study was an open clinical trial whereby both researchers and participants were aware of the treatment. Thus, expectancy effects and demand characteristics may have affected the results.

Rothbaum et al. (2003) continued their examination of the effectiveness of VRE in another case study of a 52-year-old male Vietnam veteran who exhibited PTSD, depression, and other anxiety symptoms. Data collected on the veteran included detailed accounts of five traumatic events he encountered during the Vietnam War. The veteran participated in twice-weekly sessions of VRE using a Huey helicopter environment or a Vietnam jungle environment. The study's goal was to assess anxiety symptoms

by measuring physiological responses during virtual reality exposure therapy. Heart rate and skin conductance ratings were measured every minute for 25 minutes followed by a posttreatment measurement. Physiological assessments showed that this veteran's overall heart rate was high when he was asked to recall and discuss his traumatic memories; however, as the treatment progressed, his heart rate decreased when recalling and discussing the same memories.

Interestingly, the client's physiological reactivity and self-reported distress were not always correlated (Rothbaum et al., 2003). Nonetheless, his SUDS ratings decreased as well. The participant's BDI scores indicated a marked posttreatment decrease in depression compared to pretreatment. CAPS scores decreased significantly from 40 (pretreatment) to 30 (posttreatment) at the six-month follow-up. However, there was an increase in symptoms at the three-month follow-up with a CAPS score of 49 from the baseline of 40. The researchers hypothesized that this increase could be due to a lack of coping skills and adjustment prior to terminating therapy and treatment. More research is needed to determine causes and possible explanations for such increases in PTSD symptoms after posttreatment.

In another study, Ready and colleagues (2006) examined 14 male Vietnam veterans who participated in two 90-minute therapy sessions per week in an outpatient setting. Veterans received eight to 20 total sessions of virtual reality exposure. The first session was an orientation session, with veterans experiencing a neutral virtual reality environment session (i.e., mock trial). Session two focused on the traumatic memory and the virtual reality treatment (Virtual Vietnam), and became progressively intense as treatment continued. Maintaining a discussion with the veteran about the traumatic memory was important in order to create a setting for habituation in combination with the virtual environment component. As previously mentioned, a key objective in this treatment is to not overwhelm the veteran, so SUDS ratings were obtained every 5 minutes to determine the level of anxiety. If the ratings were too high, a neutral stimulus was presented in the virtual module in order to lower anxiety.

For these veterans, the treatment ended when he was able to successfully discuss the traumatic memory in detail without obtaining a high level of negative emotion (i.e., fear) or anxiety, again measured by the SUDS ratings (Ready et al., 2006). The study concluded with a posttreatment assessment at the three- and six-month follow-up. The researchers found lowered posttreatment symptom scores compared to pretreatment scores, showing support for the effectiveness of virtual reality exposure treatment. Posttreatment scores lowered from the three-month follow-up to the six-month follow-up, showing evidence that after the treatment had been completed, the veterans learned the proper coping skills to successfully manage posttraumatic stress symptoms. The researchers also found reductions in specific criterion variables in avoidance and arousal symptoms of PTSD from the CAPS. Further, veterans' self-reports of intrusion symptoms from the IES and depression level, measured by the BDI were significantly lower at posttreatment. This study demonstrated significant overall reductions in PTSD symptoms and depressive symptoms as a result of using virtual reality exposure treatment, but it should be noted that limitations in this study include a small sample size and lack of a control group.

During a more recent study with 11 male Vietnam veterans, Ready, Gerardi, Backscheider, Mascaro, and Rothbaum (2010) investigated whether virtual reality exposure therapy (n = 6) or present-centered therapy (PCT; n = 5) would have more impact in decreasing PTSD symptoms. While virtual reality exposure therapy focuses on a corrective experience of the "there and then" by including the details of the trauma, processing the trauma, and incorporating reprocessed information, present-centered therapy emphasizes the "here and now" by maintaining a focus away from the traumatic stimuli through the use of psychoeducation and problem-solving techniques (Ready et al., 2010).

Virtual reality exposure therapy was completed following the guidelines with SUDS ratings assessed every five minutes and all veterans recounted their traumatic memories during treatment. Both treatment groups completed ten 90 minute sessions. A clinician blind to the treatment group conducted the CAPS and BDI for all veterans at pretreatment, posttreatment, and at the six-month follow-up. There were no statistically significant differences between CAPS scores pre and posttreatment or between BDI

scores pretreatment and posttreatment. The virtual reality exposure group showed CAPS improvement from pretreatment to posttreatment, and from pretreatment to the six-month follow-up. The present-centered therapy group also showed improvement from pretreatment to posttreatment, and from pretreatment to the six-month follow-up. Upon further analyses, after combining both treatment groups, there was significant improvement in CAPS scores from pretreatment to posttreatment and pretreatment to the 6-month follow-up (Ready et al., 2010). Thus, VRE was shown to be effective, but in their study, it was not more effective than PCT at reducing PTSD symptoms. Ready et al. (2010) acknowledged their low sample size and the issues with generalizing from such. The authors noted they encountered difficulty in recruiting Vietnam veterans, as these veterans show significant treatment resistance, especially with technology. Nonetheless, this study is the most current known to have success in using VRE therapy with Vietnam veterans, thus demonstrating promise; further research should endeavor to compare VRE treatment with more established treatments to determine its effectiveness.

VRE Treatment of U.S. Veterans of the Iraq and Afghanistan War

Besides the veterans of the Vietnam War, VRE has also been used in the treatment of the veterans of the Iraq and Afghanistan wars. Wood et al. (2007) examined a 32-year-old Iraq male veteran with 12 years of current active duty service who had been diagnosed with PTSD. To assess his symptoms, The Posttraumatic Stress Checklist-Military (PCL-M), Beck Anxiety Inventory (BAI) and CES were used, along with psychophysiological measures of skin conductance, respiration rate, and heart rate. All assessments were acquired pretreatment, midway through the treatment at the five session mark, and posttreatment. Also, SUDS ratings were assessed every 5 minutes during exposure. The veteran participated in ten, 90 minute virtual reality graded exposure therapy (VRGET) sessions, one time a week. During the first two sessions, the individual became acclimated to the equipment and process of the therapy, and learned about meditation training techniques. In these initial sessions, the veteran also described, in detail, his traumatic event account and physiological measurements were obtained. During the subsequent sessions, each therapy session was divided into four, 20 minute increments (a review of the previous session, meditation training, virtual reality graded exposure therapy, followed by a debriefing and discussion of current symptoms and issues).

The client's scores on the BAI indicated that his anxiety remained consistently "moderate" throughout the sessions. On the PCL-M the veteran met a formal PTSD diagnosis on both the pre- and mid-treatment assessments; however, the posttreatment score was significantly lower and the veteran no longer met the criteria for PTSD. With respect to physiological measurements, skin conductance ratings decreased following treatment and the participants reported no arousal symptoms. Lastly, the veteran's heart rate was lowest at posttreatment following the recovery phase of treatment, meaning he was able to also decrease his arousal and allow for habituation to occur. Whereas this veteran exhibited desired improvement, it is important to note that outcome of a case study cannot necessarily be generalized to all veterans.

Similar to the studies mentioned above, Gerardi et al. (2008) reported successful VRE treatment of a 29 year-old male Iraq veteran with over 10 years of military experience who was diagnosed with PTSD. The veteran spent 4 weeks, with one session a week lasting 90 minutes, in virtual reality exposure therapy with an average of 50 minutes a session in the virtual environment. SUDS ratings were collected every 5 minutes during the treatment. The veteran's posttreatment PTSD Symptom Scale Self-Report (PSS-SR) score reflected a decrease from pretreatment. Similarly, depressive symptoms decreased as evidenced by BDI scores. However, although posttreatment scores on CAPS exhibited a 56% decrease in PTSD symptoms, he still met an official diagnosis of PTSD according to the CAPS. Nonetheless, the majority of these results, along with the veteran's self-reported data, showed an increase in quality of life, including less intense PTSD symptoms following the VRE treatment. This case study offers additional support for the need of nomothetic research into VRE effectiveness.

Along the same lines, Reger and Gahm (2008) conducted a case study with a 30-year-old man with

active duty Army status. The veteran client had completed nine years of military service with one prior deployment to Iraq. He had experienced several traumatic events while deployed (e.g., witnessing a friend's death and enemy death) and was diagnosed with PTSD. This Army veteran completed six 90-minute sessions of VRE for a total of four weeks. Treatment also consisted of psychoeducation, relaxation training, and in vivo exposure. Following the treatment, the veteran's posttreatment score on the PCL-M was 29. The veteran self-reported a higher quality of life including fewer PTSD symptoms and an increase in overall functioning. At the seven week follow-up, the veteran reported improved functioning compared to the level of functioning he reported prior to the treatment. When researchers asked his opinion on VRE, he stated "I don't think I would be where I am today without it," (p. 944). His family also noted his improvement. We should add, however, that there were no objective scores provided at this point, so these anecdotal accounts, although encouraging, must be interpreted with caution.

More recently, Rizzo, Difede, and colleagues (2009) conducted a study of VRE effectiveness with Iraq and Afghanistan veterans which included female (n = 1) and male (n = 19) participants. All veterans had met a formal diagnosis for PTSD and had not benefited from other treatment modalities. Virtual reality sessions occurred twice a week for 90-120 minutes on average over a five-week time period. Self-report measures on the PCL-M and BAI were collected at pretreatment, posttreatment, and at a three month follow-up. The PCL-M results showed 16 participants no longer met an official PTSD diagnosis and 17 of the 20 participants reported decreases of at least 50% in PTSD symptoms following treatment. The BAI showed significant decreases in anxiety levels following treatment, with a 33% decrease in anxiety symptoms (Rizzo, Difede, et al., 2009). These results are again encouraging, as 80% of participants no longer met a formal PTSD diagnosis. The evidence from this study further points toward virtual reality exposure's effectiveness in decreasing PTSD symptoms with small samples of participants.

Miyahira, Folen, Hoffman, Garcia-Palacois, and Schaper (2010) presented the case of a male Iraq veteran diagnosed with chronic PTSD. This veteran had two prior deployments and a total of six years of military service. He completed six sessions of virtual reality exposure therapy, in combination with cognitive behavioral therapy sessions, for a total of 10 treatment sessions. Clinical assessments utilized were the CAPS, BDI, Quality of Life Inventory (QOLI), and the Posttraumatic Stress Diagnostic Scale, which evaluates PTSD symptoms. Assessments were completed pretreatment, posttreatment, and at the three-month follow-up. All assessments showed a decrease in PTSD symptoms, avoidance symptoms, and depression symptoms from pretreatment to posttreatment and three-month follow up. One unfortunate exception to this pattern of success was that scores on the QOLI, a self-report of quality of life, did not improve. One possible explanation for a low quality of life score, according to the researchers, may be the veteran's impending re-deployment to Iraq and report of having a newborn child from whom he would be separated when deployed.

Among research on virtual reality exposure therapy to treat military veterans with PTSD, we could locate only one study which described and discussed the treatment of a female veteran with combat-related PTSD. Wood et al. (2009) presented the case study of a 26-year-old female Seabee (a Naval Construction Battalion solider) who completed three deployments in Iraq with six total years of active duty service. This client was diagnosed with PTSD and a mild traumatic brain injury. Her prior mental health treatment consisted of the antidepressant paroxetine, which did not decrease her symptoms. During the treatment with VRE, the client was administered the PCL-M, the BAI, and the CAPS at pretreatment, posttreatment, and three-month follow up. Additional assessments included psychophysiological measures of skin conductance and peripheral finger temperature ratings (Wood et al., 2009).

The female veteran completed 20 virtual reality exposure sessions over 20 weeks and each session lasted 90 minutes. According to her BAI scores, there was a significant decrease in anxiety from pretreatment (28) to posttreatment (3), with a three-month follow-up score of 6 (indicating minimal anxiety). On the PCL-M this veteran scored 65 at pretreatment, 27 at posttreatment, and 24 at the three-month follow-up. Finally, the CAPS also showed a downward

trend of decreasing PTSD symptoms (83 at pretreatment; 11 at posttreatment; and 12 at three month follow-up). Of further note, skin conductance ratings lowered throughout treatment. Skin temperature did increase throughout treatment, which was expected, but showed no evidence of increase at the three-month follow-up (Wood et al., 2009). According to Wood et al. (2009), this female veteran's results showed improved PTSD scores compared with those of male veterans with PTSD. The client also reported improvements in her quality of life and more control over her symptoms following treatment (Wood et al., 2009). The results of this study showing the effectiveness of VRE in treating combat-related PTSD is consistent with the results of other VRE research using the case study methodology or research using small samples. Limitations include a lack of directly comparing gender effects, and the inclusion of a single female, as her results might not generalize to all females diagnosed with this disorder.

Reger and colleagues (2011) recently examined VRE therapy's effectiveness in treating active duty Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF) veterans. The sample included 24 veterans with at least one combat deployment, 75% of whom had a PTSD diagnosis and 25% of whom were diagnosed with anxiety disorder, not otherwise specified (NOS). Thirteen participants had not responded to other treatments. These veterans had experienced significant traumas such as improvised explosive device attacks involving body recovery or death, killing, or witnessing a fatal suicide attack.

Veterans received VRE therapy following the standard protocol (cf. Foa, Hembree, & Rothbaum, 2007) with the exception of session two. Here, Reger et al. (2011) introduced VR using an initially pleasant virtual environment. Following this, they introduced the Virtual Iraq environment. Researchers assessed symptoms using the PCL-M at pretreatment, throughout treatment, and posttreatment. Results showed a statistically significant drop in participants PCL-M scores from pretreatment (M = 60.92, SD = 11.03) to posttreatment (M = 47.08, SD = 12.70). Participants who averaged seven sessions of VRE reported PTSD symptom reductions with statistical and clinical significance.

Moreover, in this study, prior treatment did not have a significant effect on PCL-M scores (Reger et al., 2011). At pretreatment, PCL-M scores were significantly higher among participants with PTSD than those with anxiety disorder NOS. However, at posttreatment, differences in PCL-M scores were no longer significant between the groups (Reger et al., 2011). Twenty of the participants met the criteria for PTSD diagnosis at pretreatment, but at posttreatment, nine participants no longer met the criteria for PTSD and 15 participants had improved at least 11 points on the PCL-M.

Limitations of Reger et al.'s (2011) study include the lack of a comparison group, and that the outcome measures did not include clinician administered measures or blind assessments. Nonetheless, they took a rare nomothetic approach to the study of VRE effectiveness, and their results do underscore the promise of VRE in treating PTSD in veterans. One important feature of their study was the timing of the treatment. Often, veterans do not receive treatment for PTSD until decades after their trauma. Here, the average time from trauma until treatment was 2.33 years. Further research should account for time lags from trauma until VRET to assess its effect on outcomes.

Also fairly recently, Mclay et al. (2011) conducted a randomized, controlled trial to compare virtual reality graded exposure therapy (VR-GET) to treatment-as-usual (e.g., group therapy) for active duty service members with PTSD. Their subjects were 20 veterans of Iraq or Afghanistan campaigns who had a minimum CAPS score of 40. Ten participants were randomly assigned to the VR-GET group and ten were assigned to the treatment-as-usual (TAU) group. VR-GET combines graded VRE with physiological monitoring and skills training. The skills training component helps the patient become aware of cognitive reactivity levels and autonomic arousal cues and then employs techniques to facilitate a sense of control in high anxiety situations. This may allow clients to confront their trauma in a more present way. The VR-GET group participated in 1–2 sessions per week for ten weeks. The first two sessions were devoted to completing an intake interview, obtaining a complete trauma history including the participants' symptoms, disclosure of their most traumatic event, psychoeducation with respect to meditation skills and attention control skills, and practicing these skills while

recalling their most significant trauma. In the following sessions, participants began the Virtual Iraq or Afghanistan while a therapist controlled the intensity and provided a graded exposure experience. SUDS scores and physiological monitoring guided the therapist in selecting the appropriate intensity. The TAU group could use prolonged exposure, cognitive processing therapy, EMDR, pharmacotherapy, group therapy, or other common treatment for PTSD.

Results showed that 70% of the VR-GET group had at least a 30% CAPS score improvement. The TAU group did not demonstrate the same improvement (Mclay et al., 2011). These results offer more data supporting VR-GET effectiveness for combat-related PTSD in veteran populations. Study limitations included small sample size and variability in the control group.

McClay and colleagues (2011) conducted another study of VRET effectiveness. In an open-label, single group study, 20 active duty soldiers who served in Iraq and Afghanistan participated in VRET as part of a treatment development project. All participants had a diagnosis of combat related chronic PTSD. VRET was provided using the Virtual Iraq environment. Sessions took place twice per week for 90 to 120 minutes. VRE sessions were 45 minutes long and the remainder of the session consisted of processing the exposure. The number of sessions ranged from 10-15. The PCL-M, Patient Health Questionnaire-9 (PHQ-9), and BAI were administered two weeks before treatment, one week after completing treatment, and at three months posttreatment.

Results show that of the 42 patients who entered treatment, 20 completed treatment and posttreatment measures (Mclay et al., 2011). Of those, 15 (75%) no longer met diagnostic criteria for PTSD and demonstrated an improvement of at least 50% on PCL-M scores (Mclay et al., 2011). At the three month assessment, 13 participants (76%) continued to show at least a 50% improvement from baseline on PCL-M scores. A significant difference was reported between pretreatment scores and posttreatment scores on the PCL-M, as well as between pretreatment and 3-month follow up scores. Scores between posttreatment and three month follow-up were not statistically significant. Results of PHQ-9 scores indicated a significant difference between pretreatment and posttreatment and between pretreatment and three month follow-up, indicating a reduction in depression was maintained. Moreover, BAI mean scores dropped significantly from 19.9 (moderate anxiety) pretreatment to 14.7 (mild anxiety) posttreatment. This study offers additional evidence of VRET effectiveness for active duty PTSD sufferers, as well as for treatment resistant PTSD patients. However, the lack of a control group and a non-randomized design may limit the internal validity of this study.

VRE Treatment of Non-U.S. Veterans

VRE treatment studies have not been limited to veterans of the U.S. military. The conflicts in Iraq and Afghanistan have forced new populations to deal with the issue of PTSD in veterans.

Tworus, Szymanska, and Ilnicki (2010) noted that Poland has not been involved with military conflict since the time PTSD was officially recognized by the medical literature. Thus, when Polish soldiers became involved in Iraq and then Afghanistan, cases of PTSD presented a problem for Polish military psychiatrists and psychologists who had no experience with treating the disorder. The clinicians turned to the United States for assistance with treatment alternatives and in so doing, collaborated to implement VRE in the treatment of Polish veterans diagnosed with PTSD.

Tworus and colleagues (2010) presented the case of a 30-year-old soldier from the Polish military who experienced three traumatic incidents over the short period of about one month. The soldier was diagnosed with severe PTSD and hospitalized for approximately four months. He received group and individual psychotherapy, as well as pharmacotherapy. The soldier was stabilized and released from the hospital. Three months later the patient was re-hospitalized due to PTSD symptoms and an intense fear of weapons that rendered him unfit for military service. The veteran was evaluated with the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), Mississippi scale, Watson's PTSD Interview, CES, Stress Events Questionnaire, and the Psychopathology Checklist-Military: Stress Event Impact Questionnaire. The soldier received VRE therapy. The first three sessions focused on concentration training to allow the soldier to manage severe intrusive thoughts associated with

PTSD. The soldier then completed 22 VRE sessions scheduled twice per week for 30–45 minutes. Autonomic arousal was measured while he experienced the virtual Iraq environment. Notably, during VR sessions the veteran was able to experience the environment and then extrapolate the scene in his mind to include images that were not displayed. Though initially distressing, eventually the soldier had no difficulty being in the virtual Iraq environment. At this point, the soldier began three sessions of in vivo exposure by participating in an actual shooting exercise. Initially the exposure caused intense anxiety that included fear, trembling, and muscle pain. At the conclusion of the third session the soldier no longer experienced these symptoms.

The soldier was able to return to duty without symptoms of PTSD. At three month follow up, the soldier remained symptom free. To verify that the soldier was not covering up symptoms or engaging in the emotional numbing common to PTSD sufferers, the soldier was asked to complete an additional VR session. Autonomic arousal was monitored and confirmed that the soldier maintained improvement (Tworus et al., 2010).

Although case studies do not necessarily allow for generalization, this case shows that VRE therapy can be effective for treating PTSD in veterans who have not responded to psychotherapy and pharmacotherapy. It also offers evidence that VRE therapy can be effective for treating PTSD in veteran populations cross-culturally. That being said, there are limitations in this case report. For example, the authors did not provide the pretreatment and posttreatment assessment test results. Rather, the authors simply indicated that the patient showed improvement. Even more important to note, the patient was coerced into VRE therapy. That is, he was told that his participation was a condition for release from the hospital (Tworus et al., 2010). Still, this case is an example of a positive outcome, and this case study adds to the mounting evidence of success of VRE therapy for the treatment of PTSD using primarily case studies (e.g., Miyahira et al., 2010), or small sample sizes (e.g., Mclay, 2011).

Gamito et al. (2009) investigated Portuguese veterans with PTSD in a controlled virtual reality exposure study. Five Portuguese veterans received virtual reality exposure therapy and completed either traditional psychotherapy or were in a waiting list group. From pretreatment (before session one) to midtreatment (after session five), the VRE group showed a statistical reduction in obsession-compulsion scores on the Symptoms Check List-Revised (SCL-90R). In addition, even though not statistically significant, avoidance, intrusion, and hyperarousal symptoms showed descriptive reductions (Gamito et al., 2009). Ostensibly, statistical significance may not have been reached in this study due to the low sample size. In addition, the researchers reported that these assessments were conducted during treatment, and not thereafter. Regardless, since symptoms were reduced in clients, this treatment should be further tested cross-culturally.

Gamito et al. (2010) completed another study to investigate whether VRE therapy or imaginal exposure were more effective. Portuguese veterans with PTSD were assigned to receive VRE ($n = 4$), imaginal exposure ($n = 2$), or a control group ($n = 3$), consisting of a waitlist. All participants were provided with 12 sessions of treatment with the exception of the waiting list group, who did not receive any treatment. Measurements used to assess symptoms were the Impact of Events Scale (IES-R), SCL-90-R, and the BDI, which were conducted at pretreatment and posttreatment. Following the 12 sessions of treatment, the virtual reality exposure group showed an 8% decrease in overall PTSD symptoms found by the CAPS, while the imaginal exposure group showed a 1% decrease, followed with the waiting list group with a 6% decrease in PTSD symptoms (Gamito et al., 2010). While these results did not meet statistical significance, they followed the predicted pattern, and the lack of significance may be due to the low sample size. There were, however, some statistical differences between the groups. According to scores on the SCL-90-R, a significant decrease was found along the depression dimension for the virtual reality exposure therapy group from pretreatment to posttreatment, while there was an increase in depression with the imaginal exposure group. The BDI was assessed with the virtual reality exposure therapy group only and resulting in a 40% statistically significant decrease from pretreatment to posttreatment in depression symptoms. In addition, veterans who

experienced VRE reported less anxiety and depression than the veterans in the other groups (Gamito et al., 2010).

Taken together, Gamito and colleagues' (2009; 2010) findings suggest cross-cultural evidence of the promise for VRE in treating PTSD. Future studies should test VRE with non-U.S. participants. Future studies should also utilize larger number of participants to increase the likelihood that typical outcomes are representative of the target population.

Discussion

The goal of this study was to critically examine the literature related to the use of virtual reality exposure therapy in the treatment of PTSD in veterans. We found 13 pertinent empirical studies, all of which used the case study methodology or utilized small sample sizes, but provided evidence of the success of VRE treatment. The main purpose of VRE is to decrease PTSD symptoms, thus augmenting quality of life. All studies documented decreases in PTSD symptom clusters (avoidance, hyperarousal, and intrusion), and in almost all studies the veterans reported increases in quality of life. In addition, evidence indicated that VRE treatment may work better than other treatment models (e.g., imaginal exposure therapy). Thus, VRE therapy appears to hold promise in treating military veterans with combat-related PTSD.

Advantages of Virtual Reality Exposure

Based on the evidence presented in the studies above, as underscored by many experts in the area, virtual reality exposure therapy has many strengths and poses numerous advantages over other treatment modalities for PTSD.

One of the biggest advantages of VRE is that the veteran is able to control the level of stimulation with regard to the amount of anxiety and the virtual environment. For instance, if the session becomes too overwhelming, the therapist can discuss this event with the veteran and the veteran may slow the pace during virtual reality or change the presented VR stimuli. Patient-directed VR navigation instills a feeling of control, which allows PTSD patients to develop a new sense of accomplishment (Bush, 2008). This is critical in PTSD recovery, as sufferers often feel like they are not in control of their own actions, thoughts, or feelings.

An additional advantage of VRE is allowing full engagement. Unlike imaginal exposure, whereby the individual imagines the traumatic event, virtual reality exposure allows the individual to remain immersed and engaged during the entire session through discussions of the event and the virtual environment (Ready et al., 2006; Rothbaum et al., 2001). With the triggering environment created by technology, the client can focus mental resources on anxiety and coping, allowing for more emotional engagement compared to imaginal exposure (Riva, 2009).

Another benefit of VRE in treating combat-related PTSD, for both therapist and veteran, is easier access to the triggering environment because it is conducted in an office setting and not in the field (Cukor et al., 2009). In addition to "case of access," there is little risk of physical harm during virtual reality exposure sessions compared to other exposure treatments and it is therefore safer and more private, making it easier to bring the traumatic memory to practice (Riva, 2009; Rothbaum et al., 1995). Taking a client into combat or onto a battlefield would be necessary to elicit emotional processing during in vivo exposure. With VRE, however, the triggering situation is realistically simulated in a comfortable setting (Rothbaum, Rizzo, & Difede, 2010), and therapists can repeat exposure experiences with ease compared to other exposure treatments, e.g., in vivo (Rothbaum et al., 2010).

The use of technology in VRE is yet another advantage to this treatment approach. Technology in work or at play has become second nature to the younger generation, and with younger military personnel returning from combat, learning how to navigate a new piece of technology should not be difficult (Rizzo, Newman, et al., 2009). Virtual Iraq appears to be an attractive treatment that may allow more Iraq and Afghanistan veterans the ability to utilize technology and treatment to decrease their symptoms (Reger, Gahm, Rizzo, Swanson, & Duma, 2009; Rizzo, Newman, et al., 2009). Further, stigma reduction occurs as less one-on-one time is spent with the psychologist and more time is devoted to the use of a computer. The veteran may initially feel safer in the virtual environment than in the traditional therapeutic context (e.g., individual talk-therapy), given that he

or she does not have to engage in constant person-to-person contact.

Indeed, one of the difficulties therapists encounter in the treatment of PTSD is the avoidance and escape behavior clients exhibit (Bush, 2008; Cukor et al, 2009). These clients often consider travel time and distance to the treatment center as time consuming and may use this as an excuse to avoid treatment. However, VRE therapy helps to eliminate the use of this excuse and the avoidance behavior. Some virtual environments are mobile, and depending on the circumstances, the therapist can meet the veteran in a more comfortable setting (Bush, 2008; Cukor et al., 2009). For veterans who live far from treatment centers or have other psychiatric symptoms, such as social phobias, they can complete treatment in the comfort of their home.

Disadvantages of Virtual Reality Exposure

Virtual reality exposure (VRE) therapy is not without its disadvantages. Avoidance and escape tendencies may interfere with the initial use of the virtual reality system. That is, veterans may use their discomfort with technology as an escape. This may be particularly true for older veterans who are not comfortable with the use of technology (Ready et al., 2010). As mentioned above, avoidance and escape are prevalent in those suffering from PTSD (Bush, 2008).

Along these lines, another limitation of VRE might be that the simulation can serve to perpetuate avoidance of human contact that is already present in PTSD symptomatology. In other words, more time spent in the virtual environment is less time spent directly engaged with the therapist. Because the aim is to decrease PTSD symptomatology, therapists may want to decrease virtual environment exposure and increase the actual time spent in human-to-human interaction with the veterans as their PTSD symptoms improve.

Moreover, whereas the use of technology may be a draw, particularly to younger veterans, Riva (2005) emphasized the lack of complete personalization of the virtual environments. As underscored by Hodges et al. (1999), not every memory or event can be perfectly created in the virtual environment. Each individual in treatment will have experienced different trauma in combat. For example, a virtual explosion may not replicate the precise memory for a veteran who suffered a trauma in this manner.

Also, with respect to the technology employed in VRE therapy, as mentioned earlier, cost is a major barrier to implementing its widespread use. Virtual reality exposure's expenditure requires money and time in order to begin initial treatment, both for the therapist and the veteran. A virtual reality system ranges in price from $5,000 to $200,000, with some estimates for an entire system reaching as high as $1 million (Riva, 2005; Wiederhold & Wiederhold, 2005). Further related to funding issues is the lack of trained professionals certified to conduct this type of therapy. This treatment tends to be unfamiliar, which can cause anxiety in clinicians and therapists (Bush, 2008). In addition, it is costly to train clinicians and technical support individuals to create, maintain, and operate virtual environments (Riva, 2005; Rothbaum et al., 2010). Thus, there appears to be large hurdles in terms of providing this treatment for the people who need it.

Among the drawbacks of VRE, one must consider the minor side effects that can occur from use of the virtual environment and device—motion sickness, cybersickness (motion sickness caused by a virtual environment), dizziness, and nausea have been reported in clients (Riva & Vincelli, 2001). While these symptoms are minor compared to the presenting symptoms of PTSD, proper care should be taken to prevent any side effects which could deter the veteran from further progress and even cause him or her to terminate therapy.

Future Directions

Whereas evidence suggests there are many benefits to using VRE to help combat-exposed veterans conquer PTSD symptoms, as this review has found, and as Ready et al. (2006) discussed, the need exists for future research with respect to its effectiveness and limitations.

There has been little research on VRE effectiveness conducted with larger sample sizes. Most of the studies discussed herein included either a single case or a very few participants (e.g., case studies by Reger & Gahm, 2008; Wood et al., 2009). Although case studies and low sample-size research provide valuable sources of hypotheses, virtual reality exposure is

a non-traditional and fairly new treatment for reducing PTSD symptoms, and further studies would benefit from larger participant sample sizes and further replication studies within the military population, as the diagnoses of combat-related PTSD continue to increase (Rothbaum et al., 2003).

In addition, most research involving military veterans treated for combat-related PTSD has been conducted with Vietnam veterans (Creamer & Forbes, 2004). Future research should examine other veteran populations beyond the Vietnam War and include recent veterans such as the Gulf War, and more recently Iraq and Afghanistan, to allow for generalizability across the veteran population. Moreover, additional research needs to be conducted with veterans in other countries to test the cross-cultural effectiveness of the VRE therapy. With respect to study design, creating treatment plans using randomized controlled clinical trials might provide stronger empirical evidence about how this therapy fairs against the other types of treatments.

Another limitation to the generalizability of studies of VRE treatment for PTSD is the limited number of female participants and the overall limited data on female veterans with PTSD (Creamer & Forbes, 2004). According to the Department of Veterans Affairs, as of September 2009, there were approximately 23 million United States veterans, and 1.8 million are female. That is, roughly 8% of the veteran population is comprised of women (Department of Veterans Affairs, 2010). In addition, female military veterans are twice as likely as men to develop PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Not emphasizing the treatment needs of female veterans is a grave oversight on researchers' and therapists' part (Breslau, 2002), and it is clear that future research should focus on this vulnerable population so that gender differences in the experience and treatment of PTSD and the effectiveness of VRE therapy in the treatment of female veterans can be determined.

Moreover, the current published research includes follow-up information on the veterans at three- and six-month posttreatment. Future studies should examine longer effects of this treatment by following these veterans perhaps at nine- or twelve-month posttreatment.

Summary: The Promise of VRE Treatment

Based on the evidence presented in this work, we contend that virtual reality exposure therapy is a promising technological treatment for use with military veterans, and we concur with other researchers who have suggested that this treatment become a standardized treatment for veterans with PTSD (Cukor et al., 2009). In addition, in the wake of evidence of its effectiveness, further research is needed to discover methods of creating a virtual reality module at a less expensive price; this would have obvious benefits to the therapist, the organizations purchasing the device, and insurance companies.

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Predicting Food Cravings: A Piece of Cake or a Hard Nut To Crack?

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The present study aimed to extend previous research investigating the relationships between predictors of food cravings in order to produce some preliminary findings in relation to the variable of sweet food cravings and its relationship to body image avoidance. The sample consisted of 139 university students (36 men and 103 women) at least 18 years old. Hierarchical multiple regression analysis was performed on body image avoidance, trait anxiety and BMI to examine predictors of food cravings. Both body image avoidance and gender separately predicted cravings for sweet foods, with women experiencing more sweet food cravings than men. However, no significant relationship was found between body image avoidance and general food cravings. BMI was not related to trait anxiety. Further implications of the results and directions for future research are discussed.

Keywords: food cravings, body image, body avoidance, anxiety, university, sweets

Food cravings are extremely common phenomena experienced by both men and women. In a study of undergraduate students in the USA, 94% of women and 75% of men reported experiencing food cravings (Zellner, Garriga-Trillo, Rohm, Centeno, & Parker, 1999). Food cravings are consistent across the lifespan (Rodin, Mancuso, Granger, & Nelbach, 1991), with chocolate being the most craved food, regardless of the country under investigation (Gendall, Sullivan, Joyce, Fear, & Bulik, 1997). Gender differences have been noted, with men generally craving carbohydrates (Lafay et al., 2001) and women preferring sweet foods (Wansink, Cheney, & Chan, 2003).

Food cravings are defined as an intense desire to consume a particular food, or type of food, that is difficult to resist (White, Whisenhunt, Williams, Greenway, & Netemeyer, 2002). Consumption of the craved food usually follows a food craving (Hill & Heaton-Brown, 1994; Martin, O'Neil, Tollefson, Greenway, & White, 2008; White et al., 2002). This consumption is considered to be the explicit, measurable aspect of a food craving (White et al., 2002) and links excessive food craving to over-eating and other disordered eating behavior. However, it is important to note that there may not always be a direct relationship between cravings and consumed food.

A recent surge of media attention on the addition of sugar, to everyday foods; the popularization of books such as *Sweet Poison* (Gillespie, 2008), may have contributed to increased consumer interest in sugar consumption and the physical side effects. However, there appears to be contradictory findings in the literature investigating food cravings of sweet, sugary foods. Research has indicated that sweet foods are craved more frequently than other types of food (Davis et al., 2011; Hill & Heaton-Brown, 1994; Martin et al., 2008; Rodin et al., 1991); however there are discrepancies such that Benton (2010) recently found that sugar was not addictive, nor did it play a role in eating disorders.

Body Mass Index (BMI)

The internationally accepted, standardized classifications of overweight and obese are based on the BMI, which allows a comparable analysis of prevalence rates worldwide (World Health Organization, 2012) and is a weight-to-height ratio, calculated by dividing one's weight in kilograms by the square of one's height in meters. The consensus is that the normal range is between 18.5 and 24.9 kg/m^2 (for both sexes and for all ages over 18 years). Obesity is defined as a BMI above $30kg/m^2$, while overweight is defined as a BMI above $25kg/m^2$. Morbid obesity is classified as a BMI above $40kg/m^2$.

Several studies have found that food cravers have higher BMI's than non-cravers (Gendall, Sullivan et al., 1997; White et al., 2002). Others have found that as BMI increases so too does body image avoidance and dissatisfaction (Algars et al., 2009; Barreto, Ferrandez, & Guihard-Costa, 2009; Lu & Hou, 2009; McCabe & Ricciardelli, 2004; Watkins, Christie, & Chally, 2008). Furthermore, BMI is one of the strongest predictors of body image avoidance, with overweight and obese groups reporting more body image avoidance than normal weight groups (Barreto et al., 2011; Lavender & Anderson, 2010; Slevec & Tiggemann, 2011; Watkins et al., 2008; Yates, Edman, & Aruguete, 2004). Finally, BMI is a significant predictor of anxiety in both men and women (Barry, Pietrzak, & Petry, 2008), and has been found to be positively correlated with trait anxiety in a female sample (Hillman, Dorn, & Huang, 2010).

Body Image Avoidance and Food Cravings

Body image avoidance can be defined as the dissatisfaction with one's physical appearance. It can lead to unhealthful responses including poor eating behaviors, a decrease in levels of physical activity, substance abuse and isolating social interactions (Australian Medical Association [AMA], 2009), and is extremely prevalent worldwide (Cheung et al., 2011; Forbes et al., 2012). There appears to be a lack of published research linking food cravings with body image avoidance, but a strong link has been established between body image avoidance and disordered eating styles (Michou & Costarelli, 2011; Stice & Bearman, 2001; Watkins, et al., 2008). Moreover, body image avoidance has been found to strongly predict disordered eating behaviors such as Binge Eating Disorder, in both men and women (Cheung et al., 2011, McFarland & Petrie, 2012; Urbszat, Herman, & Polivy, 2002; Thompson, Coovert, & Stormer, 1999; Tylka, 2001).

Anxiety and Food Cravings

In addition to these studies examining the relationship between BMI and body image avoidance, further investigations have concentrated on the impact of anxiety on food cravings. Individuals who have frequent food cravings are suggested to have higher levels of anxiety than those who have infrequent food cravings (Hill et al., 1991; Moreno-Dominguez et al., 2012; White & Grilo, 2005), and those individuals with high anxiety are at increased risk of developing an eating disorder than those with low anxiety (Scherr, Ferraro, & Weatherly, 2010). However, research is inconsistent. Another study has found no differences in anxiety between cravers and non-cravers (Gendall et al., 1997).

The differences in results may be because anxiety is considered a multifaceted construct, consisting of various dimensions including state and trait anxiety (Endler & Kocovski, 2001). State anxiety describes the experience of unpleasant feelings when confronted with specific situations and when the object or situation that is perceived as threatening goes away, the person no longer experiences anxiety (Endler & Kocovski, 2001). Trait anxiety can be defined as the anxiety an individual experiences on a day to day basis. (Spielberger, Gorsuch, & Lushene, 1983) and has a positive relationship with disordered eating attitudes (Michou & Costarelli, 2011) and BMI (Hillman, Dorn, & Huang, 2010). Recent research has investigated the ability of trait, rather than state anxiety, to predict food intake but not cravings (Schneider, Appelhans, Whited, Oleski, & Pagoto, 2010). The present study aimed to investigate the nature of trait anxiety only, in order to determine if it had an impact on food cravings and potential eating psychopathology, rather than a single state situation.

Hypotheses

Positive relationships were expected between BMI and food cravings, body image avoidance, and trait anxiety. In addition, it was hypothesized that gender and body image avoidance would account for more variance in sweet food cravings than trait anxiety.

Method

Participants

Fifteen cases were removed due to missing data. Upon inspection of the descriptive analysis, it was revealed that there were only 10 underweight participants, all of which were female. The final sample therefore, consisted of 139 university students (n_{male} = 36, n_{female} = 103), all over 18 years. In terms of BMI

Table 1
Participant Demographics (N = 139)

Characteristic	%(n)
Gender	
M	25.9 (36)
F	74.1 (103)
Age	
18–25	38.8 (54)
26–35	28.1 (39)
35 and over	33.1 (46)
Education	
High school	42.4 (50)
Vocational/TAFE	24.5 (34)
Bachelors degree	25.2 (35)
Masters degree	7.1 (11)
Relationship status	
Partnered	28.1 (39)
Not partnered	71.9 (100)
Body Mass Index (M = 25.7, SD = 5.80)	
Healthy	58.3 (81)
Overweight	24.5 (34)
Obese	17.3 (24)

status, 58.3% fell into the healthy weight category, 24.4% fell into the overweight category and 17.3% fell into the obese weight category. See Table 1 for further details.

Procedure

Ethical approval was obtained from Bond University Research Ethics Committee, Bond University, Queensland, Australia. After responding to a call to participate, students were forwarded an email containing an electronic link to a web-based survey. Participation was voluntary and informed consent was obtained prior to survey completion. Inclusion criteria specified that participants needed to be a currently enrolled student between 18–60 years old.

Measures

Participants completed demographic information, which included their self-reported height and weight. The primary researcher then calculated the BMI to classify participants into categories based on their weight and height (WHO, 2012). A BMI below 18.5 is classified as underweight, BMI from 18.5 to 24.99 is considered within the normal range, a BMI from 25 to 29.99 is classified as overweight range and a BMI over 30 is considered obese. Within the obese category, Class I obesity is defined as a body mass index ≥ 30 but < 35. Class II obesity is defined as a BMI ≥ 35 but < 40. Class III obesity is defined as a BMI ≥ 40 (WHO, 2012).

The Body Image Avoidance Questionnaire (BIAQ; Rosen, Srebnik, Saltzberg, & Wendt, 1991) was used to measure the frequency of avoidance of situations that might cause concern about one's physical appearance (Rosen et al., 1991). Participants were to indicate on a 6-point Likert scale (*0 = never, 1 = rarely, 2 = sometimes, 3 = often, 4 = usually, 5 = always*), how often they engaged in "certain behaviors at the present time". The BAIQ has been found to contain satisfactory psychometric properties, with an internal consistency coefficient of .89 and a test-retest correlation of .87 (Rosen et al., 1991).

The 40-item self-report State and Trait Anxiety Inventory Form Y (STAI; Spielberger et al., 1983) was used to measure state and trait anxiety. Participants were to respond to statements indicating their current (State) feelings using a four point Likert scale, (*1 = not at all, 2 = somewhat, 3 = moderately so, and 4 = very much so*), and how they "generally feel" (Trait), on a four point Likert scale, (*1 = almost never, 2 = sometimes, 3 = often, and 4 = almost always*). Quek et al., (2004), found internal consistency for the state and trait subscale and also the overall anxiety score (Cronbach's alpha = .87, .83 and .86 respectively). Vitasari et al., (2011), also found excellent internal consistency for both subscales and the total anxiety score (Cronbach's alpha of .80, .78 and .85 respectively).

Participants were to rate the frequency of individual food cravings over the past month on a 5-point Likert scale ranging from 1 ("*never*") to 5 ("*almost every day*"). The 37-item self-report Food Craving Inventory (FCI; White et al., 2002) was used to measure four subscales: high fats (8 items; e.g. fried chicken, sausage), carbohydrates/starches (8 items;

Table 2
Summary of Intercorrelations, Means and Standard Deviations for Age, Gender, Marital Status, Education Level, BMI, BIAQ, State and Trait Anxiety, the Sweets subscale of the FCI and the total score of the FCI. (N = 139)

Variable	1	2	3	4	5	6	7	8	9	10	M	SD
1. Age	--	.01	.61**	.24**	.29**	.03	-.13	-.06	-.13	-.12	--	--
2. Gender		--	-.01	.01	-.19*	.38**	.25**	.25**	.20*	.11	--	--
3. Martial status			--	-.12	-.39**	-.16	-.00	-.03	.20*	.16	--	--
4. Education level				--	.16	.19*	.02	.03	.03	-.11	--	--
5. BMI					--	.21*	-.04	.02	-.04	-.03	25.76	5.80
6. BIAQ						--	.27**	.33**	.23**	.11	28.24	11.99
7. State anxiety							--	.81**	.16	.18*	38.81	11.39
8. Trait anxiety								--	.18*	.13	40.77	11.17
9. FCI–Sweets									--	.81**	17.06	5.78
10. FCI–Total										--	55.38	15.02

Note: * $p < .05$, ** $p < .01$.

e.g. baked potato, pasta), sweets (e.g. 8 items; chocolate, ice cream) and fast food fats (4 items; e.g. pizza, hamburger). To limit errors due to cultural differences, the authors replaced two of the food items with the Australian equivalent of the American food. Cinnamon rolls were replaced with finger buns and candy was replaced with lollies. Possible scores range from 0 to 140, with higher scores indicating increased food cravings. The FCI has been found to be both reliable and valid when used in a variety of populations including a community sample of individuals of varying weight (White et al., 2002) and an obese sample with binge eating disorder (White & Grilo, 2005).

Results

The data was analysed using the software Statistical Package for Social Studies (SPSS version 19.0). All statistical analyses were considered significant at alpha level $p = .05$. Prior to analysis, all variables were examined for data entry errors, missing data, linearity, normality, and the presence of univariate and multivariate outliers. In 15 cases, complete missing data existed, therefore it was decided to delete all 15 cases. Partial data was missing in three cases, so were they removed. Preliminary analysis was undertaken using Pearson product-moment correlation to investigate whether the demographic variables (age, education level, marital status and gender) covaried with the predictor variables (food cravings) or the criterion variable (body mass index, body image avoidance or anxiety). Further analysis was then undertaken to investigate the relationship between food cravings and several associated criterion variables using Pearson product-moment correlation. Reporting of correlations was pursuant to the strength coefficient categories of small = .10–.29, medium = .30–.49 and large = .50–1.0 reported by Tabchnick and Fidell (2007). In order to determine whether food cravings could be predicted by gender, BMI, body image avoidance and trait anxiety, a hierarchical multiple regression analysis was performed.

As demonstrated in Table 2, a number of significant relationships were found relating to gender. A small significant negative relationship was found between gender and BMI ($r = -.19$, $p = .027$), indicating that men had higher BMI's than women. A significant

Table 3
Means and Standard Deviations of Variables

	Trait Anxiety		BIAQ		Sweets–FCI		Total–FCI	
	M	SD	M	SD	M	SD	M	SD
Gender								
M	36.05	7.80	20.67	8.90	15.17	5.21	52.50	15.94
F	42.42	11.72	30.88	11.83	17.73	5.84	56.39	14.63
Age								
18–25	40.74	10.38	28.61	12.85	18.37	5.94	59.61	13.77
26–35	41.67	11.33	25.69	10.23	16.49	5.24	52.51	14.57
36 and over	40.04	12.09	29.95	12.20	16.02	5.84	52.85	15.94
Marital status								
Partnered	41.79	12.16	30.33	14.14	15.77	5.67	51.95	16.13
Not partnered	40.37	10.80	27.42	11.00	17.57	5.77	56.72	14.43
BMI								
Healthy	41.05	11.30	26.62	10.71	17.32	17.32	5.63	13.67
Overweight	38.79	10.77	28.26	13.64	16.56	16.56	5.97	16.52
Obese	42.62	11.33	33.67	12.49	16.92	16.92	6.19	17.45

medium positive relationship was found between gender and body image avoidance ($r = .38$, $p < .001$), such that women had higher levels of body image avoidance than men. Gender was also significantly positively correlated with both state ($r = .26$, $p = .002$) and trait anxiety ($r = .25$, $p = .003$), indicating that women had higher levels of anxiety than men. Analysis between gender and total score of the FCI revealed no relationship. However, a significant negative relationship was found between gender and the high-fats subscale ($r = -.19$, $p = .029$), such that men had more cravings for high-fat foods than women. Analysis revealed that gender and the subscale for sweets were significantly positively correlated ($r = .20$, $p = .022$), as was the relationship between gender and carbohydrates ($r = .17$, $p = .047$); women craved more of these types of food than did men.

Body Mass Index and Food Cravings. The relationship between BMI and food cravings was investigated using Pearson product-moment correlation. As seen in Table 2, support was not found for the hypothesis that participants with a higher BMI would have more food cravings than those with a lower BMI. As seen in Table 3, healthy range BMI participants admitted more food cravings than both the overweight and obese groups.

Body Mass Index and Body Image Avoidance. To investigate the hypothesis that the obese and overweight group would have more body image avoidance than the normal group, a one-way between-groups ANOVA was performed. Participants were divided into three groups (healthy, overweight and obese) based on the criteria recommended by the World Health Organization. A statistically significant difference was found for BIAQ scores between the three BMI groups: $F(2, 136) = 3.31$, $p = .04$. The effect size was $\eta^2 = .05$. As can be seen in Table 3, Post-hoc comparisons using the Tukey HSD test indicated that the mean scores for Group 1 ($M = 26.62$, $SD = 10.71$, $p < .05$) were significantly different from Group 3 ($M = 33.67$, $SD = 12.49$, $p < .05$). Group 2 ($M = 28.26$, $SD = 13.64$) did not differ significantly from either Group 1 or 3. As shown in Table 3, support was partially found for the hypothesis, as the obese group, but not the overweight group, had significantly more body image avoidance than their normal weight counterparts.

Body Mass Index and Trait Anxiety. Pearson

Table 4
Hierarchical Multiple Regression Analysis with Sweet Food Cravings predicted from Age, Marital Status, Education Level, Gender, Body Mass Index, Body Image Avoidance Questionnaire and Trait Anxiety (N = 139)

Predictor	ΔR^2	β	*B*	SE *B*	95% CI for *B*
Step 1	.04				
Constant			14.58	2.32	[9.98, 19.17]
Age of participant		-.03	-.15	.56	[1.26, .05]
Highest level of education		.06	.32	.50	[-.68, 1.31]
Marital status		.18	.60	.35	[-.08, 1.29]
Step 2	.04				
Constant			12.66	2.42	[7.86, 17.45]
Gender		.20	2.59	1.09	[4.3, 4.74]
Step 3	.01				
Constant			10.61	3.26	[4.16, 17.05]
BMI		.09	.66	.70	[-.72, 2.04]
Step 4	.03				
Constant			9.26	3.27	[2.80, 15.72]
BIAQ		.21	.10	.05	[.01, .19]
Step 5	.01				
Constant			7.43	3.61	[.28, 14.58]
Trait anxiety		.10	.05	.05	[-.04, .14]

Note: CI = confidence intervals

product-moment correlation indicated there was no relationship between BMI and trait anxiety. Furthermore, as shown in Table 3, overweight participants did not have significantly more trait anxiety than their normal weight counterparts.

Body Image Avoidance and Food Cravings. As demonstrated in Table 2, and contrary to the expected outcome, a non-significant relationship was found, indicating that body image avoidance was not related to general food cravings.

A hierarchical regression analysis was undertaken for the criterion variable of the FCI sweets subscale. A preliminary analysis revealed that age, marital status and education level co-varied with other variables within the study, they were entered at Step 1. Gender also co-varied with other variables within the study, however, as it was also a predictor variable, it was entered at Step 2. Based on the existing food craving literature, BMI was entered at Step 3, body image avoidance was entered at Step 4 and trait anxiety was entered at Step 5.

Table 4 details the results of the hierarchical regression analysis for sweet food cravings. After Step 5, when all variables were entered into the regression equation, a significant amount of variance in sweet food cravings was accounted for (R = .13, adjusted R = .082; $F(7, 131) = 2.77$, p = .01. The R value of 12.9 for the model with all four variables indicates that almost 13% of the variance in sweet food cravings was predicted by gender, BMI, body image avoidance and anxiety.

On Step 1 of the hierarchical multiple regression analysis, the demographic variables of age, marital status and education level, accounted for a non-significant amount of 4.1% of the variance in sweet food cravings, $R^2_{change} = .041$, $F_{change}(3, 135) = 1.94$, p = .126. Therefore, at their point of entry, the demographic variables were not significant predictors of

sweet food cravings.

At Step 2, after controlling for the demographic variables, gender accounted for an additional 3.9% of the variance in sweet food cravings, R^2 = .039, F = (1, 134) = 5.64, p = .019. At the point of entry into the model, consistent with hypothesis, gender was a significant predictor of sweet food cravings.

At Step 3, after controlling for the demographic variables and gender, BMI accounted for a non-significant .6% of the variation in sweet food cravings, R = .006, F = (1, 133) = .884, p = .349. At this point, BMI was not a significant predictor of sweet food cravings.

At Step 4, after controlling for the demographic variables, gender and BMI, participants BIAQ scores accounted for a significant additional 3.3% of the variance in sweet food cravings, R = .033, F = (1, 132) = 5.01, p = .027, at its point of entry into the equation, consistent with the hypothesis, participants' body image avoidance (as measured by the BIAQ) was a significant predictor of sweet food cravings.

At Step 5, after controlling for the demographic variables, gender, BMI and BIAQ, participants trait anxiety scores accounted for a non-significant .9% of the variance in sweet food cravings, R = .009, F(1, 131) = 1.38, p = .242. Therefore at its point of entry, trait anxiety was not a significant predictor of sweet food cravings.

With all four predictors entered into the equation at Step 5, gender and body image avoidance (as measured by the BIAQ), were the only predictors to significantly predicted sweet food cravings. With regards to unique variance, gender contributed 3.8% unique variance to food cravings, whilst body image avoidance contributed 3.3% unique variance to food cravings.

Gender and Sweet Food Cravings. Gender accounted for more variance in sweet food cravings than did trait anxiety. In order to ascertain which gender had more sweet food craving, an independent sample t-test was conducted. Analysis revealed a statistically significant difference in scores for men (M = 15.17, SD = 5.21) and women (M = 17.73, SD 5.84); t(137) = -2.33, p = .022, two-tailed). The magnitude of the difference in the means (mean difference = -2.56, 95% CI: -4.74 to -0.38) was small to medium (η^2 = .04). Inspection of means in Table 3, show that women had significantly more sweet food cravings than men.

Body Image Avoidance and Sweet Food Cravings. Body image avoidance significantly accounted for more variance in sweet food cravings than trait anxiety. This indicates that higher levels of body image avoidance significantly predicted increased cravings for sweet foods. As shown in Table 2, those with higher body image avoidance scores had significantly more sweet food cravings than those with lower body image avoidance scores.

Discussion

The present study sought to investigate whether gender, BMI, body image avoidance and trait anxiety were useful in predicting sweet food cravings. BMI was not found to be related to either food cravings or trait anxiety and this was unexpected. A considerable amount of past research has shown that there was a relationship between BMI and food cravings (Gendall & Sullivan, 1997; White et al., 2002). However, the samples used were often clinically diagnosed participants, for example obese patients with binge eating disorder (White & Grilo, 2005). It is possible that the experience of food craving is different for those with a clinical diagnosis of an eating disorder than those without. Perhaps food cravings experienced by the university sample in the present study, were more related to other factors, for example stress, rather than BMI. Future research could investigate food cravings in both a clinical and non-clinical sample, to examine differences in the food craving experience across culture and diagnosis.

It was an aim of this study to expand on findings of previous research by Zhao et al., (2009) who found that BMI predicted anxiety, but did not use a valid and reliable measure of anxiety. Surprisingly, in this study it was found that BMI was not related to trait anxiety, although previous research has found support for this hypothesis (Hillman et al., 2010; Schneider, et al., 2010). Substantial research has also shown a robust relationship between BMI and general anxiety only (Barry, Pietrzak, & Petry, 2008; Hillman et al., 2010). It is possible that the lack of significant results in the present research is a result of the change of investigation from general anxiety to the specific component of trait anxiety. Future research would

benefit from including both state and trait anxiety.

The hypothesis that BMI would be related to body image avoidance was partially supported, as the obese group, but not the overweight group, had significantly more body image avoidance than the normal weight group. However, there was no significant difference between the overweight group and the healthy weight group in terms of their body image avoidance. These results are not consistent with previous research that has found both obese and overweight groups report more body image avoidance than their normal weight counterparts (Barreto et al., 2011; Lavender & Anderson, 2010; Slevec & Tiggemann, 2011; Watkins et al., 2008; Yates et al., 2004). These differing results could be due to the lack of understanding of what constitutes a healthy BMI or the colloquial use of the term 'overweight' which may include obese individuals. Many people incorrectly believe a BMI over 25 is still within the healthy range, when in fact this reflects the overweight or obese range (Basterra-Gortari, Bes-Rastrollo, Forga, Martinez, & Martinez-Gonzalez, 2007). While the current study did not ask participants to calculate their own BMI, future studies could measure participants' understanding of BMI against their own self-reported status to control for this issue. The healthy weight range participants did not differ from the overweight participants in terms of body image avoidance, perhaps due to the participant's individual differences that were not measured, for example, disordered eating or the presence of an eating disorder was not evaluated. In the present research, several variables known to relate to food cravings were measured in an attempt to predict food cravings, however, it was not possible to measure all individual differences. Future research could use a more clinical measure of eating disorders to control for this issue.

An aim of this study was to provide preliminary results relating to body image avoidance and food cravings, as research in this area appears to be limited. Based on the previously established relationship between body image avoidance and disordered eating styles (see Michou & Costarelli, 2011; Stice & Bearman, 2001; Watkins, et al., 2008) and the similarities between the disordered eating literature and that of the food cravings literature, it was hypothesized that body image avoidance would be related to food cravings. Surprisingly, there was no significant relationship between body image avoidance and general food cravings. However there was a predictive relationship as body image avoidance was predictive of sweet food cravings. Perhaps the reason is the lack of men in the present study. Males typically experience less general cravings than women (Rozin, Levine & Stoess, 1991; Weingarten & Elston, 1991), and when they do crave food, it is more for carbohydrates (Lafay et al., 2001). It is possible that due to the small number of male participants in the present study, the amount of general food cravings was not large enough to produce a significant effect.

The current study found that body image avoidance was predictive of sweet food cravings. This finding supports previous disordered eating research where it was found to be predictive of body image avoidance in both men and women (Cheung et al., 2011; McFarland & Petrie, 2012; Thompson, Coover,t & Stormer, 1999; Tylka, 2001; Urbszat, et al., 2002). This study highlighted that gender was also predictive of sweet food cravings, with women having significantly more sweet food cravings than men. Since food cravings are usually followed by consumption of the craved food (Hill & Heaton-Brown, 1994; Martin et al, 2008; White et al., 2002); it would be assumed, then, that women also have increased rates of being overweight. However, at a worldwide level, reports suggest men are more often overweight than women (Algars et al., 2009; Cheung et al., 2011; Neighbors & Sobal, 2007; Yates, Edman, & Aruguete, 2004). Future studies should investigate other potential factors e.g., portion size and frequency of eating as reasons for this discrepancy.

Limitations of the current study include the retrospective nature of the questionnaires. Participants were asked to retrospectively evaluate their food cravings over the past 30 days, how often they engaged in body image avoidance behaviors, and how they generally felt with regard to their anxiety. It is possible that participants' responses were influenced by memory distortions, which have been shown to occur when recalling past events (Schacter, 1999). The present study examined only normal weight, overweight and obese participants. A sample more representative of the general population, including underweight and morbidly obese, would ensure results are

generalizable to a wider population. Additionally, this study did not have an even distribution of women to men in order to examine true gender effects and differences; therefore, future research would benefit from an equal ratio of men to women. Finally, including the demographic questions prior to the other measures may have inadvertently introduced a stereotype threat (a risk of confirming, as self-characteristic, a negative stereotype about one's group, either based on BMI or food cravings; Steele & Aronson, 1995).

Conclusion

This study contributes to the research on sweet and general food cravings, and their relationship to other related variables, despite its limitations. This study revealed participants' body image avoidance was a significant predictor of sweet food cravings; those with higher body image avoidance scores had more sweet food cravings than those with lower body image scores. In addition, women had significantly more sweet food cravings than men. However, while higher BMI ratings were not related to increased food cravings, those in the obese group had significantly more body image avoidance than the normal weight participants.

Treatment programs for addressing the obesity epidemic have included combined dietary and physical activity approaches (Fujioka, 2002; McGuire, Wing, Klem, Lang, & Hill, 1999), but those with higher BMI scores may benefit from the inclusion of specific skill training relating to body avoidance, specifically with regard to the role of sweet food cravings and consumption. The results of this study suggest that it is possible to identify predictive variables of sweet food cravings. As worldwide consumption of sugar and sweet foods increases (Davis et al., 2011; Gillespie, 2008), and as the obesity epidemic worsens (Withrow & Alter, 2010), innovative and effective treatment approaches to the whole issue are critical.

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Affective Responses to Music Without Recognition: Beyond the Cognitivist Hypothesis

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A recent topic of concern for those interested in the science of music is whether affective responses to music are the result of *recognition* or actual *affective experience*. Cognitivist researchers have found that individuals *recognize* rather than *feel* an affective response when listening to music, while emotivist proponents posit that people have an intrinsic affective experience to music. While it has been promoted that biological methods must be used in order to answer this recognition-experience problem cited above, the current authors employed a more traditional technique (i.e., paper and pencil self-report surveys). Data from the present study show that participants reported statistically similar levels of five different categories of affect, regardless of whether they recognized the intended emotion of the musical clips. Results suggest that the induction of affect while listening to music is not reliant upon recognition, and are supportive of the emotivist position regarding musical emotions. These results may have implications regarding the ultimate origins of musicality in humans.

Keywords: affective response, musical stimuli, emotions, mood, cognitivist, emotivist

Psychological research on musical phenomena has long been plagued by both theoretical and methodological criticisms. A major theoretical criticism of the field of music-based affect research has been that of the cognitivist position: That people merely *recognize* the intended affective response of a piece of music rather than *feel* any affective response when listening to music (Konečni, 2008; Manuel, in press; Payne, 1980; Pratt, 1952). This stands in contrast to the emotivist position that people actually experience affective responses to music analogous to other real-world affective responses (Davies, 1994; Robinson, 1994). Although some research has provided empirical support for the notion that listening to certain types of music can elicit affective change in individuals (Hill & Palmer, 2010; Scherer & Zentner, 2001), there continues to be a need for empirical support within this specific field of study. Further, most studies supporting the claims that music elicits affective responses fail methodologically to account for the recognition problem by not empirically asking participants to report the intended affect, or intended emotion of the musical clip.

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The Recognition Problem

That listening to music elicits affective responses in individuals has been well-documented over the past two decades (Blood, Zatorre, & Bermudez, 1999; Hill & Palmer, 2010; Krumhansl, 1997; Trainor & Schmidt, 2003). Despite this substantial research, debate continues over the processes by which humans have affective responses as a result of listening to music (Peretz, 2001). Some studies (Hill & Palmer, 2010) showing changes in affective states after listening to music have not sufficiently accounted for the possibility that people merely recognize the affect or emotion intended by the composer in a piece of music, as opposed to feeling the emotion elicited by the music. The current trend in psychological research suggests that highly technical approaches, such as physiological measurements, may be the most promising avenues by which researchers can solve the recognition-experience problem (Chamberlain

& Broderickk, 2007; Kassam & Mendes, 2013). However, before proceeding to such approaches, the current authors believe a traditional method can provide some insight, as well. Therefore, the present study used a traditional self-report survey as the experimental methodology.

Methodological Constraints in Music Research

Advances in methodology and technological materials also tend to advance the systematic study of psychological processes (e.g., psychology of emotions and facial expressions; Ekman & Friesen, 1976). However, contrary to the methodological advances in other areas of research on emotion, mood, and affect, the psychological study of music's effects on these constructs have used highly variable sets of stimuli, potentially leading to a slow start in methodological consistency. For example, Krumhansl (1997) used orchestral excerpts to represent the emotions of sadness and happiness. Krumhansl then tested participant reactions to those excerpts by measuring heart rate, skin conductance, blood pressure and skin temperature. Etzel, Johnsen, Dickerson, Tranel, and Adolphs (2006) used similar physiological measurements (e.g. heart rate, diastolic blood pressure), but they used musical excerpts from various films to elicit those respective emotions instead of the orchestral excerpts used by Krumhansl. These physiological indicators of emotion and music represent only one example of the lack of methodological consistency in the study of musical emotions. However, Vieillard et al. (2008) have recently made a significant move toward the advancement of methodological consistency in research on musical emotions, by creating an archive of emotionally categorized music.

The current study presented participants with many musical clips each lasting very short durations (e.g., 15 seconds) during the experimental procedure. It is less likely that participants will feel any complex emotional reaction to music during a short duration of presentation. Emotions are "a complex set of interrelated sub-events concerned with a specific object" (Russell & Barrett, 1999, p. 806), suggesting a time component longer than that required for an affective response. Affect is considered a less complex, but always-occurring feeling (Ekkekakis, 2012), suggesting that it may be easier to measure in short durations of time.

It is, therefore, more appropriate to consider the reactions reported by participants in the present study to be affective responses. Thus, the cognitivist and emotivist positions are translated here into affective responses instead. The cognitivist hypothesis argues that individuals merely recognize the intended affect of music rather than experience any affective response to the music (Payne, 1980; Pratt, 1952). This hypothesis would predict that there should be a significant difference between the affective responses of those who correctly and incorrectly recognize the intended emotion of the music; correct and incorrect recognition being determined by the congruency of a participant's responses with the intended emotion of a piece of music as dictated by the composer. Conversely, if there is no relationship between the participant's affective response to music and that individual's cognitive recognition of the music's intended emotion, one would expect no significant differences in affective responses between those who correctly and incorrectly recognized the intended emotion of the musical clips. The latter hypothesis is consistent with predictions of the emotivist position (Davies, 1994; Robinson, 1994).

Hypothesis

The present study was designed such that the two plausible statistical outcomes would lend support in two different directions. One outcome would lend support for the cognitivist position (a main effect for recognition), while the other outcome would fail to lend support for the cognitivist position (no main effect for recognition) and thereby indirectly lend support for the competing emotivist position. Although a null finding would seem to indicate indirect support for the emotivist position, it is well-established that using null findings as evidence is problematic. Therefore, a main effect for recognition, and support for the cognitivist position, would be the strongest possible empirical outcome from the current study.

Method

Participants

Participants in this study were 205 undergraduate psychology students from a regional university in the southern U.S. One hundred forty-two of the

participants were female, 61 of the participants were male, and two participants did not indicate their gender (age: M = 20.95, SD = 2.23). One hundred seventeen participants listed themselves as Caucasian, 71 as African American, seven as Asian-American, three as Hispanic, and four as Other. Three of the participants did not indicate their ethnicity. Students in various university psychology courses were offered extra credit by their professors for participating in the study. Those who did not participate were given alternative options for extra credit.

Materials

Musical stimuli. Musical clips developed by Vieillard et al. (2008) were used to attempt mood induction in the participants. The original Vieillard et al. (2008) archive[1] consists of 56 musical excerpts. The 56 excerpts are divided into four groups (i.e., happy, sad, fearful, and peaceful), with each group containing 14 musical excerpts. The "happy" excerpts were composed in a major key signature with an average tempo of 137 Metronome Marking (MM). The "sad" excerpts were composed in a minor key signature at a slow tempo (M_{MM} = 46) . The "fearful" excerpts were composed with minor chords on the third and sixth degrees, leading to the use of many out-of-key notes and making the piece sound "odd" and "eerie." The "peaceful" excerpts were composed in the major key signature with an intermediate tempo (M_{MM} = 74) in addition to the compositions including arpeggio accompaniment (Vieillard et al., 2008). These excerpts were constructed similar to the trends of most Western music (Cooke, 1959). Henceforth, the various clips and their associated experimental conditions will be referred to as happy, sad, fearful, and peaceful music, respectively.

Audience response system. An audience response system (TurningPoint, 2008) was used to collect data for this project. The response system requires the experimenter to create interactive slides through the TurningPoint toolbar in Microsoft PowerPoint. When interactive slides are created, each participant is given a response card resembling a small remote control device. When a slide is shown on a projection screen students may indicate how they feel by pressing a number, 1 through 5, on their response device. Each number corresponds to a specific feeling depending upon the question number (e.g., Right now I feel: 1–*Quite dejected*, to 5–*Quite cheerful*).

Feeling and Mood questionnaire. The participants' feelings and moods were assessed via the Semantic Differential Feeling and Mood Scale (SDFMS; Lorr & Wunderlich, 1988). The SDFMS is a scale consisting of 35 items which are divided into five subcategories of seven questions each: Elation (A), Relaxed (B), Unsure (C), Fatigue (D), and Grouchiness (E). The paper/pencil forms require participants to place a check in the box describing how they feel at a given moment (e.g., *quite elated*, *slightly elated*). For example, a single question assessing elation (SDFMS-A) states: "Right now I feel: (1) [*Quite Dejected*], (2) [*Slightly Dejected*], (3) [*Neutral*], (4) [*Slightly Cheerful*], or (5) [*Quite Cheerful*]. The SDFMS is not designed to assess emotions, but instead moods and feelings, the latter being a core component of affect. The SDFMS has an internal consistency of .74 (Lorr & Wunderlich, 1988). It most accurately assesses affective responses to the presentation of stimuli, and has been used and described as such (Ho & MacDorman, 2010; Wasylkiw, Fabrigar, Rainboth, Reid, & Steen, 2010).

Procedure

All data collection took place in the afternoon in order to control for unintended fatigue effects not induced by the musical clips themselves[2]. The experiment was conducted on groups of students clustered in the different classes the researchers recruited from. Each of the four classes was randomly assigned to listen to only one of the four different types of music (i.e., happy, sad, peaceful, or scary).

Prior to each class, the musical clips and TurningPoint slides were loaded onto the classroom's computer. As students arrived for class, they were notified of the possibility of participating in the study.

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[1]Musical clips are available at the website of the University of Montreal's International Laboratory for Brain Music and Sound Research (BRAMS): www.brams.umontreal.ca/plab/publications/article/96#downloads

[2]Although time of day was monitored for data collection in an effort to keep data collection times consistent, no data were recorded for the day of the week. Thus, we cannot rule out possible fatigue effects due to day of the week.

Interested students were provided with informed consent forms. Any questions about the experimental procedure were answered by the researchers prior to beginning the study.

After signing the informed consent documents, participants were given an audience response device. The researcher then briefly explained the methodology of the experiment and proceeded to show participants two sample slides in order to familiarize each student with the audience response system.

The researcher then began to play the designated type of music for a period of five minutes over each classroom's audio system, with the volume set to be identical for each of the four classes. The music consisted of fourteen musical clips designed with the same intended emotion in mind, and each having a period of only approximately 10–11 seconds. The participants heard each 150 second clip twice, without interruption.

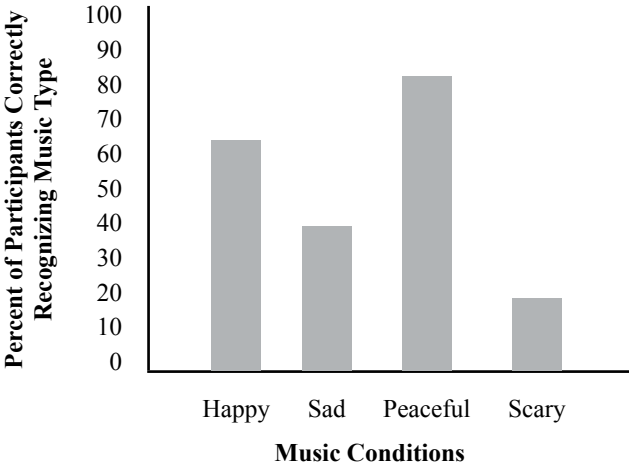
Subsequently, the experimenter stopped the music and posted one last slide asking the participants to guess the intended emotion of the music that had been played throughout the experiment. Participants were debriefed by telling them that we were measuring affective, or emotional, reactions to music.

Results

Prior to any of the primary analyses, the data were examined to check the accuracy of recognizing an intended emotion of the musical excerpts. Overall accuracy was 48%; however, the accuracy changed depending on the specific musical condition (Figure 1). Based on correct or incorrect recognition, each participant and their SDFMS responses were assigned to the "correct" or "incorrect" recognition conditions for further analysis.

To test whether a cognitive component (recognition) is needed for affective responses in music listening, a series of five separate 4 (music type) x 2 (recognition; correct/incorrect) analyses of variance were conducted, one for each of the five subcategories of the SDFMS. All post-hoc comparisons were performed using a Bonferroni adjustment on SPSS statistical software. A priori power analyses using MorePower 6.0.1 (Campbell & Thompson, 2012) indicated that a sample size of 56 would be needed in order to find a statistically significant interaction between music type and recognition[3]. The analyses presented below were performed on data from 205 participants, a number well beyond the prescribed sample size.

Figure 1. Musical emotion recognition performance for each of the experimental conditions, and the average recognition collapsed across experimental conditions.



SDFMS-A: Elation

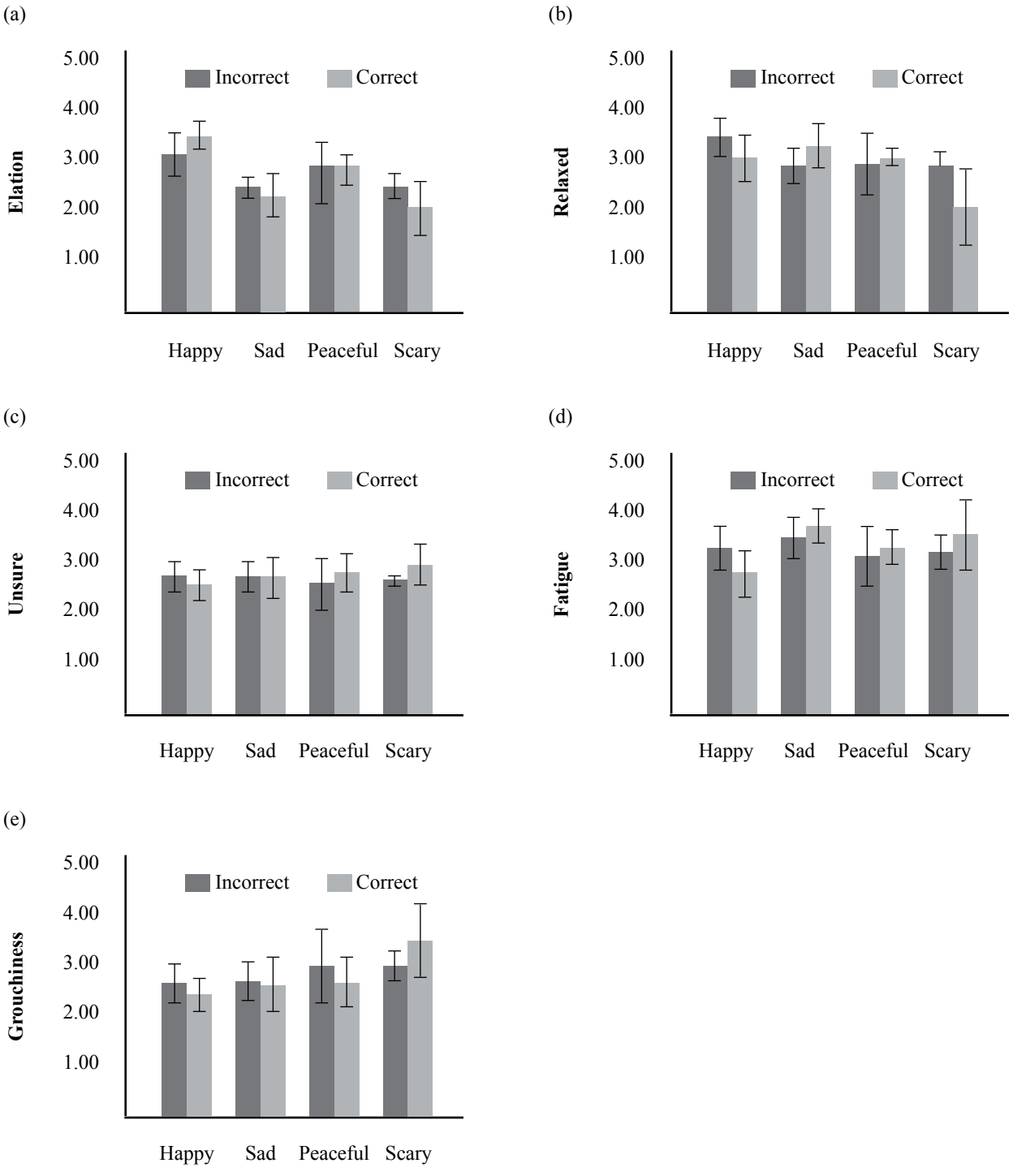
Results showed a statistically significant main effect for music type, $F(3, 197) = 8.13$, $p < .001$, partial $\eta^2 = .11$. Multiple comparisons showed that happy music ($M = 3.62$, $SD = 0.75$) elicited stronger feelings of elation than sad ($M = 3.03$, $SD = 0.69$) and scary music ($M = 2.94$, $SD = 0.69$) but did not elicit stronger feelings of elation than peaceful music ($M = 3.26$, $SD = 0.75$). There was not a significant main effect for recognition, $F(1, 197) = 0.34$, $p = .559$, partial $\eta^2 < .01$, or a significant interaction between recognition and music type, $F(3, 197) = 1.18$, $p = .318$, partial $\eta^2 = .02$ (Figure 2a).

SDFMS-B: Relaxed

For the dependent variable of relaxation, there was a statistically significant main effect for music type, $F(3, 197) = 4.12$, $p = .007$, partial $\eta^2 = .06$,

[3]Parameters entered for MorePower 6.0.1 power analysis: ANOVA was selected, 2x2 was selected for the IM design factors, 2x2 was selected as the IM effect of interest, alpha level of .05, power of .80, and a medium eta-squared effect size of .13 were entered. Solve for sample size was also selected.

Figure 2. The relationship between various affective response types for those who correctly and incorrectly guessed the intended mood of the musical clips. Graphs are organized by SDFMS subcategory: (a) elation, (b) relaxed, (c) unsure, (d) fatigue, and (e) grouchiness.



Note: Error bars are 95% confidence intervals.

although post-hoc comparisons showed that none of the music types were significantly different from one another when compared one-on-one. Again, there was not a significant main effect for recognition, $F(1, 197) = 1.18$, $p = .280$, partial $\eta^2 = .01$. There also was not a statistically significant interaction between recognition and music type, $F(3, 197) = 2.60$, $p = .053$, partial $\eta^2 = .04$ (Figure 2b).

SDFMS-C: Unsure

For Unsure, there was not a statistically significant main effect for music type, $F(3, 197) = 0.94$, $p = .422$, partial $\eta^2 = .01$. Post-hoc comparisons showed no significant differences between the four music types. There also was not a significant main effect for recognition, $F(1, 197) = 1.89$, $p = .171$, partial $\eta^2 = .01$ or a significant recognition by music type interaction, $F(3, 197) = 0.87$, $p = .458$, partial $\eta^2 = .01$ (Figure 2c).

SDFMS-D: Fatigue

Fatigue showed a statistically significant main effect for music type, $F(3, 197) = 3.07$, $p = .029$, partial $\eta^2 = .05$. Further examination of this effect showed that sad music ($M = 3.57$, $SD = 0.81$) elicited significantly higher rates of fatigue in participants than happy music ($M = 3.01$, $SD = 0.98$). There were no other significant differences. Neither the main effect for recognition, $F(1, 197) = 0.40$, $p = .529$, partial $\eta^2 < .01$, nor the interaction between music type and recognition, $F(3, 197) = 1.27$, $p = .283$, partial $\eta^{22} = .01$, were statistically significant (Figure 2d).

SDFMS-E: Grouchy

The dependent variable grouchiness showed a statistically significant main effect for music type, $F(3, 197) = 3.82$, $p = .011$, partial $\eta^2 = .06$. Post-hoc comparisons showed that the scary music ($M = 2.93$, $SD = 0.73$) elicited significantly higher levels of grouchiness in participants than both happy music ($M = 2.48$, $SD = 0.66$) and peaceful music ($M = 2.60$, $SD = 0.59$). There were no other significant differences. Consistent with the other dependent variables, there was neither a statistically significant main effect for recognition, $F(1, 197) = 0.22$, $p = .637$, partial $\eta^2 < .01$, nor a statistically significant interaction, $F(3, 197) = 1.28$, $p = .281$, partial $\eta^2 = .02$ (Figure 2e).

Discussion

The cognitivist hypothesis suggests that individuals, when listening to emotion-latent music, merely recognize the intended mood of music, thereby enabling them to internalize an emotional response to the music. However, results of this study suggest that individuals tend to have similar self-reported moods regardless of whether they correctly recognize the intended mood of the musical clip. Further, there were no significant interactions between music type and recognition for any of the various dependent variables (i.e., SDFMS subcategories), suggesting that the recognition explanation is not moderated by music type (Figure 2).

Post-hoc analyses showed the intuitive result that "happy" music makes participants feel higher levels of elation than "sad" and "scary" music. Other intuitive results were that "sad" music made participants feel higher levels of fatigue than "happy" music, and that grouchiness was significantly higher after participants listened to "sad" music compared to both "happy" and peaceful music. These intuitive findings help validate the utility of the Vieillard et al. (2008) musical clips for further use in music research.

Some of the findings were, however, unintuitive. Specifically, "peaceful" music did not induce higher levels of relaxation than the "scary" music, which seems a highly plausible outcome. Also, the unsure subscale did not reveal any differences between categories of musical clips. Collectively, these tertiary findings show some possible limitations to the Vieillard et al. (2008) musical clips; either the dependent measure is not sensitive enough to detect changes in participants after listening to the clips, or some of the musical clips are more effective than others at eliciting specific affective responses.

These results could also be due to the methodology used in this specific study. Participants were presented with music in large classrooms and tested as groups. This may create distracting effects that could have created the null findings for recognition. Although class meeting times were similar, there may have been individual differences in classes which could have led to artificially high or low affective responses in certain musical emotion conditions.

Another possible limitation of the study concerns the mismatch between the intended emotions of the Vieillard et al. (2008) musical clips and the subscales of the SDFMS. Although feeling and affect are important aspects of the more complex construct of emotion, and there is some overlap between the clips and the SDFMS (e.g., happy musical clips leads to higher scores on elation subscale), a more direct match may lend itself to more sensitive measurement. This increase in methodological sensitivity may lead to more fruitful results, and such improvements should be pursued with future research.

While cognitive-based hypotheses certainly have their places in experimental psychology, the process of eliciting affective reactions in people via music may not rely on inordinate amounts of cognitive processing. On the contrary, the processing of music-based affective responses may be quite automatic, as defined by Hasher and Zacks (1979), in the sense that it does not require the cognitive processing needed to accurately recognize an intended emotion of a musical excerpt.

Various researchers have suggested that common emotional and affective responses in humans and animals serve an adaptive function (Cosmides & Tooby, 2000; Darwin, 1871/1997); however, less arguments have been made for the adaptiveness of emotional and affective reactions to music. Since there is a concern over the similarities, between common emotions and those experienced when listening to music, this may be one reason why research on music-based emotions has been slow to start (Lundqvist, Carlsson, Hilmersson, & Juslin, 2009). The data presented in the current paper suggest that affective responses to music are relatively automatic in the sense that they do not require effortful cognitive processes; although reaction time was not measured, automaticity could be deduced from the observation that affective response was the same for those who correctly and incorrectly guessed the intended emotion of the musical clips. This apparent automaticity may lend credence to the notion that musical behavior (playing, perceiving, and emotionally experiencing) is an adaptation rather than, as Pinker (1997) suggests, an exaptation.

Future research should examine the nature of this automaticity found in the current study from different theoretical perspectives. Also, research is needed in order to understand the process by which participants in the current study experienced the emotions, leading to affective responses on self-report measures. Various researchers have suggested that emotional contagion—recognizing the emotion of a piece of music and then internalizing it, leading to actual experienced emotion—may explain emotional and affective responses to music (Dibben, 2004; Lundqvist et al., 2009; Scherer & Zentner, 2001). However, the current data suggest that conscious awareness of the correct emotion of a piece of music is not necessary in order to have affective experiences as a result of the music.

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NSPB: How To Get Involved

CALL FOR PEER REVIEWERS

The NSPB is currently seeking members for the 2014–2015 Editorial Board.

The NSPB aims to foster the scientist-practitioner model within a university psychology department and highlight the diverse empirical research that is being conducted by both experimental and clinical students, faculty and alumni. Being a peer reviewer is an excellent opportunity to obtain valuable editing and reviewing skills, and it provides experience in the art of scientific writing. The main duties include reviewing submitted manuscripts for content, style, and length, and returning to the Editors-in-Chief in a timely fashion.

Why should you become a peer reviewer?

- Enhance your Curriculum Vitae
- Become familiar with the publication process
- Important for academic positions, internships, and career opportunities
- Practice and enhance knowledge of APA style

Applicants should:

- Have knowledge of APA style
- Have strong critical reading skills
- Show an understanding of scientific writing
- Be willing to learn the publication process and pracatice use of APA style

What we'll need from you:

- Your CV
- Statement of interest
- Writing sample in APA style

For more information:
nspb.net/peer-review

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The NSPB is currently seeking both theoretical and empirical manuscript submissions from psychology graduate students for the Fall 2014 NSPB issue.

The articles may cover:

- Experiments in all brances of psychology
- Theoretical articles
- Clinical case studies
- Literature reviews
- Commentary on the field

Why should you publish?

- Enhance your Curriculum Vitae
- Recognition and communication
- Research or theory dissemination
- Professional accomplishment
- Personal satisfaction
- Important for academic positions, internships, externships, and career opportunities

What is needed?

- An original theoretical or empirical contribution to the literature
- Scientific writing skills
- Critique of ideas (i.e., from a conference)
- Knowledge of and strict adherence to APA style, including tone and neutrality
- Time and effort to conduct research and compile the manuscript
- A willingness to revise and resubmit your manuscript as many times as it takes
- The experience the NSPB provides using a training model and an experiental learning process for reviewers and authors