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Review Paper

Neuroplasticity in Psychopathological Disorders: A Review

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ABSTRACT

Advancements in the field of neuroscience confirm that the brain possesses an ability to alter both its structure, as well as its functioning. While neuropsychological investigations have made salient the neurological underpinnings and consequences of psychological disorders, the specific role of this ability of the brain (termed as neuroplasticity) has received less attention. The present study reviews previous works on the connections between neuroplasticity and psychopathology, summarizing the neuroplastic changes that accompany (or antedate) anxiety disorders, mood disorders, schizophrenia, posttraumatic stress disorder, and eating disorders. Neurochemicals involved in these neuroplastic processes, as well as the potential implications of these processes on treatment of disorders, are also explored.

Keywords: *Neuroplasticity, mood disorders, anxiety disorders, eating disorders, PTSD, schizophrenia.*

Neuropsychology attempts to find connections between cognitions, behaviors, and the nervous system. An early awareness of the brain-behavior connection can be observed in ancient practices such as trephination, a technique used as far back as 10,000 BC (Rutkow, 2000). Trephination entailed the removal of a portion of the skull, often to relieve pressures built up in the brain. In certain North African tribes, trephination was used to rid patients of "evil spirits" (Rawlings & Rossitch Jr., 1994).

Santiago Ramón y Cajol is hailed as the father of modern neuroscience due to his discovery of the fundamental unit of the nervous system: the neuron (Costandi, 2016). Techniques of neuroscience have come a long way, and methods more sophisticated than the tissue staining that Cajol relied on have allowed scientists to examine neuronal functioning and neuronal connections in detail.

Neurological examination in the present age is not limited to postmortem examination of the structure of the brain and its constituents (Zillmer et al., 2007). A prominent electrophysiological procedure, electroencephalography (EEG), allows measurement of the electrical activity of neurons in the brain by capturing the activity using electrodes placed on the scalp, amplifying the signal, and demonstrating them through oscillators (Dadebayev et al., 2022). The resulting waveforms provide information about the brain activity at the time

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of measurement, but this information may lack specificity. Evoked potential (EP) follows a similar technique, but it is primarily used to measure responses to specific stimuli.

The structure of the brain can be studied using radiological techniques, with computed transaxial tomography (CT) being a popular choice (Diwakar & Kumar, 2018). CT entails generating photos based on a three-dimensional model of the brain obtained by combining information from X-ray images of two-dimensional sections. Magnetic resonance imaging (MRI), meanwhile, involves measurement of waves emitted by hydrogen atoms in the brain upon activation by a strong radio signal frequency in a magnetic field. The images so obtained have a higher resolution and more detail. However, this comes at the expense of the speed afforded by CT scans. Higaki et al. (2018) describe how artificial intelligence and deep machine learning are being used to increase the resolution of CT scans, and decrease the scanning time of MRIs.

Functional magnetic resonance imaging (fMRI) allows for study of brain activity. By measuring changes in blood oxygenation levels in different areas of the brain, neurophysiological processes can be traced. Positron emission tomography (PET) scans also permit for functional analyses, but the images obtained are low-fidelity, and radioactive atoms must be used to monitor metabolic activity in the brain. Zhu & Zhu (2019) note that, although MRI holds certain advantages over PET scans in neurological applications, an integration of MRI and PET techniques could provide even better results. Efforts are being made to increase quantitative accuracy of PET scans through implementation of machine learning algorithms and integration of data from different sources (Zaidi & El Naqa, 2021). Such PET/MRI scans can provide more accurate images, even with low radiation doses (Lee, 2021).

It must be noted that use of brain imaging techniques alone is insufficient for a comprehensive psychological diagnosis, unless the results are interpreted by neurologists in light of a patient's symptoms. The history of the patient and the specifics of the presenting complaints must be taken into consideration to provide better treatment (Parsons & Hamekke, 2014). Taking note of symptoms inconsistent with neurological disorders gains precedence in clinical settings, since an early diagnosis of conversion disorders (or other somatic symptom and related disorders) can prevent unnecessary expenditure on more comprehensive neurological assessments (Ali et al., 2015).

Neuropsychological deficits not only provide etiological bases for disorders, but may also occur as impairments after a disorder has precipitated. A study compared the episodic memory and executive functioning in those with anxiety disorders against the general population and found episodic memory to be negatively affected in those with anxiety disorders (Airaksinen et al., 2004). The test of episodic memory employed involved memorization and recall of 32 neutral words by the participants. Statistically significant deficits in recall as compared to controls were found in those with anxiety disorders- but those with generalized anxiety disorder and specific phobias did not follow this trend.

These findings are corroborated by recent research, which confirm the negative impact psychopathology can have on an individual's neurological capacity and also identify impaired neurological capacities as a maintaining factor in psychopathological disorders, such as through reward dysfunction (Oumeziane et al., 2019). Cognitive deficits associated with neurological dysfunction may also manifest in the form of maintaining factors, as observed by Ahern et al. (2019) in their article exploring the negative impact that deficits in

cold cognitions (those independent of emotion, like attention and memory) can have on hot cognitions (those informed by emotions, such as interpersonal problem-solving) in individuals suffering from depression.

Neuroplasticity

Brain damage can occur in a variety of manners. Ranging from tumors (uncontrolled growing masses of cells), to cerebral ischemias (disruption of the blood supply to the brain), to head injuries, and even bacterial brain infections, death of brain cells can occur for numerous reasons (Pinel, 2010).

To counteract these detriments, the nervous system possesses an innate ability to restructure and alter connections between neurons. Neuroplasticity can be defined as the "ability of the nervous system to respond to intrinsic or extrinsic stimuli by reorganizing its structure, function and connections" (Cramer et al., 2011, p. 1591). As indicated by the definition, modalities of neuroplasticity can vary. Neuroplasticity involves not only the formation of new neural connections (structural neuroplasticity), but also alterations in the function of pre-existing nerve cells and synapses (functional neuroplasticity).

The existence of neuroplasticity had already been speculated upon by Charles Darwin in the 1870s, and the term "neuronal plasticity" was even used by Cajal (Fuchs & Flügge, 2014). Decades later, William James (1918) implied that the brain has a capacity to alter its functions. The term "synaptic plasticity", however, was first described by Jerzy Konorski in 1948 (Bijoch et al., 2020).

Initial evidence of neuroplasticity came later still, from Hubel and Wiesel's (1964) study on the effects of visual deprivation in kittens, which demonstrated the relationship between sensory experiences and brain development. Bliss and Lømo's (1973) work on long-term potentiality- a mechanism by which synapses can be strengthened for extended durations- confirmed the phenomenon of neuroplasticity. Through stimulation of hippocampal synapses, they were able to induce increases in the strength of these synapses.

Paul Broca's (1861) work on the identification of the specific role of the posterior, inferior region of the left frontal lobe in speech and the subsequent discovery of the relation between this region and aphasia (speech disorders caused by neuronal cell death in the left hemisphere; Kirshner, 2012) also added to the understanding of neuroplasticity. Aphasia recovery in adults illuminated the possibility of neurogenesis in adults- or indeed, the adaptation of other areas of the brain to compensate for the loss (Thompson, 2000).

Neuroplasticity and psychopathology

The recovery of function after brain damage has major implications for clinical psychological and psychiatric practice. Effective management of aphasia, for instance, pre-supposes an understanding of neuroplasticity (Crosson et al., 2019). The limits of plasticity can inform the limits of recovery, and treatment options rely on the reorganizing and restorative features of the brain. Physical exercise can promote neuroplastic processes which improve spatial learning and memory in those suffering from neurodegenerative disorders (Cassilhas et al., 2015). Additionally, McEwen (2002, as cited in Bingaman, 2013) explores the possibility that mindfulness meditation alters neural pathways in a manner that reduces activation of the amygdala, reducing anxiety in the process.

It is not solely the therapeutic potential of neuroplasticity that is pertinent to clinical settings, however. Pittenger (2022) expands upon three means by which disruption of neuroplastic processes influences psychopathology: (a) through cognitive deficits resulting from disrupted neuroplasticity; (b) enhancing pathogenic memories; (c) impairments in behavior modulation. Pittenger's study highlights the wide-ranging effects of such disruptions. In patients diagnosed with depression, for example, cognitive deficits (such as a lack of concentration) can emerge due to decreased hippocampal volumes resulting, in part, from a decreased capacity for neurogenesis (formation of new neurons). PTSD (posttraumatic stress disorder) patients, meanwhile, exhibit pathological memories which are rigidly held- a process partially mediated by altered neuroplastic processes in the amygdala.

Given the influence of neuroplasticity on the manifestation and treatment of psychopathological disorders, there is a need for further investigation of the role that neuroplasticity plays in such disorders, considering the avenues a clarification of this relationship between the two could open up for treatment opportunities. The current study aims to stimulate further research by reviewing previous works on neuroplasticity in prevalent disorders such as anxiety disorders, mood disorders, PTSD, and eating disorders. Works on schizophrenia are also reviewed to highlight neuroplastic changes in psychotic disorders.

Neuroplasticity in anxiety disorders.

The alterations in brain functions that come with psychological disorders- either as an antecedent or a cause- have been extensively studied. For instance, studies have been conducted to examine depression-related morphological changes in the brain (Frodl et al., 2008), to compare brain activation during Bipolar-I disorder and major depressive disorder (Cerullo et al., 2014), and to measure and map changes in the brain in schizophrenic patients (Andreason et al., 2011), amongst others. Disruption in the hypothalamus-pituitary-adrenal cortex (HPA) axis has been implicated in anxiety disorders (Faravelli et al., 2012; Kallen et al., 2007).

Patients with anxiety disorders are also found to have increased activation of the amygdala (Trimble, 1988). Further, excessive anxiety and chronic stress can decrease hippocampal volume and impair the functioning of the hippocampus and the prefrontal cortex (Mah et al., 2016).

Neuroplasticity-related disorders have been directly implicated in the onset of anxiety disorders in animal populations. For example, in a study conducted on rats, it was found that treatment of the gamma amino butyric acid-related dysfunctions in neuroplasticity by introducing BDNF reduced anxiety-like behavior in aged mice (Zhu et al., 2019).

Zheng et al. (2018) comment on the comorbidity of irritable bowel syndrome (IBS) and anxiety, implicating deficits in neuroplasticity in the development of IBS and anxiety. Their research posits that alterations in the HPA axis lead to increased levels of glucocorticoids and decreased levels of brain derived neurotropic factors (BDNFs), which in turn lead to decreased neuroplasticity. This deficit influences the autonomic nervous system to the detriment of the digestive system. IBS and anxiety, hence, co-occur in part due to alterations in neuroplasticity.

Adverse childhood experiences- known to play a causal role in anxiety and depression- may interfere with neuroplastic processes. Watt et al. (2019) employed measures of generalized anxiety disorder, depression, adverse childhood experiences, and a blood sample test to

study this relationship. Quantities of BDNF in the blood sample served as a measure of neuroplasticity. Subjects who had experienced four or more adverse childhood experiences were significantly more likely to have mental disorders. They also demonstrated lower levels of BDNF, indicating lesser neuroplasticity. The study notes, however, that these physiological deficits can be either a cause, a consequence, or both.

The efficacy of treatment of anxiety disorders comes, in part, from the neural changes that the treatments induce. Månsson, Salami, Frick, Carlbring, et al. (2016) illustrated the changes in grey matter volume of the amygdala that occur as a result of cognitive behavioral treatment of patients with social anxiety. Such individuals demonstrate an increased grey matter volume in the amygdala pre-treatment, which decreases over the course of therapy. Activation of the amygdala in response to self-referential criticism also decreases post-therapy. These changes imply that functional and structural neuroplasticity results from successful cognitive behavioral therapy in socially anxious populations. In a follow-up study, Månsson, Salami, Carlbring, et al. (2017) confirmed that decreases in the gray matter volume of the amygdala persisted one year after therapy; functional changes in the amygdala returned to pre-therapy levels. Owing to the contribution of neuroplasticity in improving symptoms of anxiety disorders, Chen et al. (2019) propose pharmacological interventions targeted at inducing neuroplastic changes.

Neuroplasticity in mood disorders.

Impaired neuroplasticity also plays a role in mood disorders, as indicated by reduced BDNF levels found in bipolar patients experiencing depressive and manic episodes (Kapczinski et al., 2008). BDNF levels return to normal levels once episodes end. Further, factors such as life stress and trauma that may play a causal role in mood disorders, also lead to reduced BDNF levels in bipolar patients.

Neuroplasticity has been suggested to play a mediatory role between stress and depression (Liu et al., 2017). Neurological changes accompanying depression include reduced volumes of the hippocampus and the prefrontal cortex, and increased synaptogenesis in the amygdala. Serafini (2012), through a systematic literature review, explains how the relationship between depression and abnormal neuroplastic processes do not end there. Antidepressants reverse the neuroplastic processes that maintain depression by reversing reduced neurogenesis, for instance. Price and Duman (2020) present an integrative model of depression, drawing links between inflexible maladaptive cognitions and behaviors and abnormalities in neural circuits, emphasizing the role of deficient neuroplasticity in maintaining these issues.

Neurochemicals other than BDNF influence neuroplastic changes in mood disorders. Walther et al. (2019) reviewed literature on the antidepressant effect of testosterone to determine an underlying mechanism for this influence. They concluded that, although other theories explaining this mechanism may hold weight, an increase in neuroplasticity after testosterone administration contributes to this effect. Circadian disruption is known to be a salient feature in mood disorders. Valdés-Tovar et al.'s (2018) review focuses on the relation between melatonin- a neurotransmitter involved in regulation of circadian rhythms- and neuroplastic changes in the hippocampus which occur in depression. Decreased receptivity to glucocorticoids observed in depression also hinders neuroplastic processes (Steckler et al., 1999; Arango-Lievano et al., 2015).

In their review of past literature on neuroplasticity and how it relates to bipolar disorder, Gandhi et al. (2020) noted a scarcity of studies exploring this topic. Nonetheless, previous studies focused on the altered neural circuitry between the insula and the parietal lobe observed in patients of bipolar-I and bipolar-II. These alterations (and other such changes in the parietal lobe), contribute significantly to the cognitive, attentional, and memory-related complaints that bipolar patients present with. Enhancements in neuroplasticity induced through administration of lithium can counter these neurocognitive deficits due to the neurotrophic enhancements that it results in. The authors expand upon the highly beneficial effect that pharmacological intervention can have when paired with physical activity- which promotes neurogenesis in the hippocampus- and cognitive therapy. Other studies extend this specific influence of physical exercise to treatment of depression as well (Phillips, 2017).

Thus, abnormal neuralplasticity- particularly in the hippocampus, amygdala, and prefrontal cortex- plays a definite role in mood disorders (Hall et al., 2018); physical exercise and BDNF-enhancing medication can result in efficacious reversal of this effect.

Neuroplasticity in schizophrenia.

The biological basis of schizophrenia- in particular the contribution of genetic, neurochemical, and neuroanatomical (specifically the role of the dorsolateral prefrontal cortex) factors- has been extensively studied (Brisch et al., 2014; Gejman et al., 2010; Lewis, 2022; McCutcheon et al., 2020; Sullivan et al, 2019). Evidence for the contribution of neuroplasticity has also been presented.

Altered neuroplasticity in schizophrenia manifests as both- decreased neuroplasticity, and abnormally excessive neuroplasticity (Voss et al., 2019). McCullumsmith (2015) describes schizophrenia as a “disorder of neuroplasticity” (p. 313), drawing on past studies to substantiate this claim. One such study, a postmortem comparative analysis of brains of chronic schizophrenic patients conducted by Li et al. (2015), implicated increased associational activity in parts of the hippocampus in false memory formation in schizophrenic patients.

Neuroplasticity-based cognitive training methods have also proved efficacious in treating schizophrenic symptoms. Fisher et al. (2010) used targeted training tasks for improving auditory and visual functioning. Through rewarding performance on successive tasks (such as auditory stimulus distinction tasks) arranged in terms of their difficulty, the researchers were able to promote improved cognitive functioning and overall functioning in the subjects. The tasks used were aimed at increasing neuronal plasticity.

Wobrock et al. (2012) examined biological interventions for schizophrenia. A growth factor involved in brain development, erythropoetin, had a positive impact on the cognitions of patients with schizophrenia when administered on them. Repetitive transcranial magnetic stimulation- associated with enhanced long-term potentiation- also led to reduction of symptoms of schizophrenia. Finally, aerobic exercise improved the memory capacity of patients by increasing hippocampal volume.

Voss et al. (2019) shed light on the need for integrating neuroplasticity-enhancing substances with cognitive training aimed at improving neuroplasticity to effectively manage the disorder. The complex interplay of neurotransmitters that result in schizophrenia are thought to be best controlled by targeting multiple neurological circuits at once, and efforts directed towards improving neuroplasticity help achieve this goal. Convergent evidence that

cognitive training improves neuroplasticity in schizophrenia also comes from other studies; these also confirm that aerobic exercise can enhance this effect (Campos et al., 2017; Jahshan et al., 2017).

Neuroplasticity in PTSD.

A defining characteristic of posttraumatic stress disorder (PTSD) is maladaptive learning after experiencing an immensely stressful circumstance. Dealing with stressful situations leads to rapid physiological changes, and neuroplastic changes occur on multiple levels when stress is experienced (Deppermann et al., 2014). For one, the introduction of glucocorticoids through the HPA axis after experiencing such a stressor alters neuroplasticity- although glucocorticoid levels tend to be lower in individuals with PTSD than in the general population (Radley et al., 2011; Raglan et al., 2017). Symptoms of PTSD, including flashbacks, re-experiencing of the traumatic events, hyperarousal, and *peritraumatic amnesia* (failure to encode memory of events occurring before the trauma), are all accompanied by anatomical or morphological changes in the brain (Nash et al., 2014).

Exposure to extreme stress- like in PTSD- can decrease neuroplasticity of the brain. Seo et al. (2019) studied this effect in rats by administering inescapable shocks and subsequent behavioral trials to ensure depressive and anxious symptoms. Changes observed in the brain included impaired mitochondrial function and increased apoptosis (programmed cell death) in the hippocampus. Exercise could help mitigate PTSD symptoms and improve neuroplasticity, with an increase in BDNF resulting from physical activity being a potential cause for this change.

In another rat study (Liang et al., 2014), subjects were exposed to a predatory odor (a worn cat collar) with no opportunity to escape. Even seven days after exposure, the rats showed anxious behaviors. Neural circuits of the amygdala (assessed through functional magnetic resonance imaging) and surrounding connections were found to be altered as compared to controls, confirming an enhanced neuroplasticity in the rats.

Even in human subjects, changes in neuroplasticity are observed post-trauma. Changes occur in the limbic brain circuits- in particular, the hippocampus and the amygdala- which play an integral role in the stress response. Interestingly, refugees from war-torn regions who rated worse on mental health measures than others showed increased levels of BDNF and nerve growth factor in one study (Arnetz et al., 2020). This led the authors to conclude that dysfunctional neuroplasticity occurs in the limbic system, indicating salience of dysfunctional memories of traumatizing events. Abnormal limbic development in adolescents experiencing PTSD has also been hypothesized to separate them from resilient adolescents and controls (Cisler & Herringa, 2021).

Cognitive behavioral therapy is efficacious in mitigating PTSD symptoms. Part of this effect may come from neurological changes it is capable of inducing. Instead of undoing the neural connections associated with pathological memories, however, this effect may be because of formation of stronger inhibitory connections that diminish the maladaptive circuits (Quirk & Mueller, 2008).

Thus, neurological changes in PTSD include development of maladaptive neural pathways that result in symptoms such as pathological pathways; a heightened serum BDNF level mediates this effect. Similar to schizophrenia, cognitive therapy and physical activity can

help revert neuroplastic processes to resemble those of healthy people- the exact mechanism of this change, however, differs (Quirk & Mueller, 2008; Fisher et al., 2010).

Neuroplasticity in eating disorders.

The reward systems of the brain are known to be impaired in individuals with eating disorders, and compulsive eating has been likened to substance addiction (Avena & Bocarsly, 2012; Davis & Claridge, 1998). Since addiction is accompanied by neuroplasticity in the mesocorticolimbic system (involved in regulating dopaminergic activity), the association between eating disorders and addiction has led researchers to believe that similar neuroplastic changes occur in eating disorder patients (Natenshon, 2016). Decreases in basal metabolic rates in the prefrontal cortex are also observed in patients with binge eating disorders and bulimia nervosa (Jáuregui-Lobera & Martínez-Quiñones, 2020).

Neuroplastic changes and deficits are also found in patients with anorexia nervosa. Malnutrition and weight loss associated with the condition can impair neuroplasticity and neurogenesis, with a low BDNF level and brain atrophy being direct results of chronic stress and malnutrition (Keeler et al., 2021). This can, in turn, lead to cognitive difficulties mirroring those seen in patients with depression- a condition that anorexia nervosa is frequently comorbid with. Nunn et al. (2012) hypothesize that dysregulation of norepinephrine observed in patients with anorexia nervosa results in altered activity of the insula, which may explain the negative body-image held by patients with the disorder.

By acknowledging the contribution of neuroplasticity to eating disorders, treatment plans can be developed that seek to reinforce neural pathways that alter self-image for the better. Neurofeedback training (providing feedback to a subject about their neural waves to improve some aspect of functioning) can prove a carrier for this change, considering its utility in ameliorating cognitive distortions (Natenshon, 2016; Val-Laillet et al., 2015). Additionally, Skokou (2022) suggests that the neuroplastic effects of drugs such as lamotrigine (an anticonvulsant) and vortioxetine (an antidepressant) can be exploited to aid the treatment of eating disorder.

Table 1: Summary table- Role of Neuroplasticity in Psychological Disorders

Disorder type	**Areas affected**	**Neurochemicals involved**	**Pertinent neuroplastic changes**	**Treatment implications**
Anxiety disorders	Altered volume of hippocampus (Mah et al. (2016)	Lowered BDNF (Watt et al., 2019)	Increased activation of amygdala (Trimble, 1988)	Targeted pharmacological interventions (Chen et al., 2019)
	HPA-axis dysregulation (Faravelli et al., 2012; Kallen et al., 2007)	Glucocorticoids (Zheng et al., 2018)		Neuroplastic change accompanying cognitive therapy (Månsson, Salami, Frick, Carlbring, et al., 2016; Månsson, Salami, Carlbring, et al., 2017)
		GABA (Zhu et al., 2019)		

Mood disorders	Altered volume of amygdala (Hall et al., 2018)	Lowered BDNF (Kapczinski et al., 2014)	Neurocircuitry involved in inflexible cognitions (Price & Duman, 2020)	Role of antidepressants and mood stabilizers in enhancing neuroplasticity (Searfini, 2012; Gandhi et al., 2020)
	Decreased volume of prefrontal cortex (Liu et al., 2017)	Glucocorticoids (Steckler et al., 1999; Arango-Lievano et al., 2015)		Utility of physical exercise (Gandhi et al., 2020; Philips, 2017)
	Decreased volume of the hippocampus (Liu et al., 2017	Melatonin (Valdés-Tovar et al., 2018)		
		Testosterone (Walther et al., 2019)		
Disorder type	**Areas affected**	**Neurochemicals involved**	**Pertinent neuroplastic changes**	**Treatment implications**
Schizophrenia	Dorsolateral prefrontal cortex (Sullivan et al., 2019)	Dopamine (Brisch et al., 2014; McCutcheon et al., 2020)	False memory formation (Liu et al., 2015)	Utility of physical exercise (Wobrock et al., 2012)
	Hippocampus (Li et al., 2015)	Glutamate (McCutcheon et al., 2020)		Neuroplastic changes accompanying cognitive training (Fisher et al., 2009; Campos et al., 2017; Jahshan et al., 2017; Voss et al., 2019)
				Targeted pharmacological interventions (Guercio et al., 2019; Voss et al., 2019)
PTSD	Limbic system (Cisler & Herrings, 2021)	Increased BDNF and nerve growth factor (Nah et al., 2014; Arnetz et al., 2020)	Formation of pathological memories (Arnetz et al., 2020; Nash et al., 2014)	Suppression of pathological memories through introduction of neuroplastic changes (Quirk & Mueller, 2008)

	HPA-axis dysregulation (Radley et al., 2011)	Glucocorticoids (Radley et al., 2011; Raglan et al., 2017)	Abnormal learning (Nash et al., 2014)	Utility of physical exercise (Seo et al., 2019)
	Decreased hippocampal volume (Seo et al., 2014)			Neuroplastic changes accompanying cognitive behavioral therapy(Quirk & Mueller, 2008)
Disorder type	**Areas affected**	**Neurochemicals involved**	**Pertinent neuroplastic changes**	**Treatment implications**
Eating disorders	Mesocorticolimbic system (Natenshon, 2016)	Dopamine (Avena & Bocarsly, 2012)	Impaired neuroplasticity due to malnutrition (Keeler et al., 2021)	Efficacy of neurofeedback training (Nathenshon, 2016; Val-Laillet et al., 2015)
	Prefrontal cortex (Jáuregui-Lobera & Martínez-Quiñones, 2020)	Lowered BDNF (Keeler et al., 2021)	Altered insular activity and neurological bases of a negative body-image (Nunn et al., 2012)	Targeted pharmacological interventions (Skokou, 2022)
		Norepinephrine (Nunn et al., 2012)		

CONCLUSION

The present study reviews existing literature on neuroplasticity and how it pertains to specific psychological disorders. The findings are summarized in Table 1. The review provides, for future researchers, a means of assessing gaps in research which warrant further investigation. Future studies can extend the principles of neuroplasticity to inform formulation and evaluation of targeted interventions. Further, since the scope of the review was limited to five categories of disorders, efforts to generate or consolidate information on a wider range of disorders could be made.

REFERENCES

Ahern, E., Bockting, C. L. H., & Semkovska, M. (2019). A Hot-Cold Cognitive Model of Depression: Integrating the Neuropsychological Approach Into the Cognitive Theory Framework. *Clinical Psychology in Europe*, *1*(3). https://doi.org/10.32872/cpe.v1i3.34396

Airaksinen, E., Larsson, M., & Forsell, Y. (2005). Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. *Journal of Psychiatric Research*, *39*(2), 207–214. https://doi.org/10.1016/j.jpsychires.2004.06.001

Ait Oumeziane, B., Jones, O., & Foti, D. (2019). Neural Sensitivity to Social and Monetary Reward in Depression: Clarifying General and Domain-Specific Deficits. *Frontiers in Behavioral Neuroscience*, *13*. https://doi.org/10.3389/fnbeh.2019.00199

Ali, S., Jabeen, S., Pate, R. J., Shahid, M., Chinala, S., Nathani, M., & Shah, R. (2015). Conversion Disorder- Mind versus Body: A Review. *Innovations in Clinical Neuroscience*, *12*(5–6), 27–33. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4479361/

Andreasen, N. C., Nopoulos, P., Magnotta, V., Pierson, R., Ziebell, S., & Ho, B. C. (2011). Progressive Brain Change in Schizophrenia: A Prospective Longitudinal Study of First-Episode Schizophrenia. *Biological Psychiatry*, *70*(7), 672–679. https://doi.org/10.1016/j.biopsych.2011.05.017

Arango-Lievano, M., Lambert, W. M., Bath, K. G., Garabedian, M. J., Chao, M. V., & Jeanneteau, F. (2015). Neurotrophic-priming of glucocorticoid receptor signaling is essential for neuronal plasticity to stress and antidepressant treatment. *Proceedings of the National Academy of Sciences*, *112*(51), 15737–15742. https://doi.org/10.1073/pnas.1509045112

Arnetz, B. B., Sudan, S., Arnetz, J. E., Yamin, J. B., Lumley, M. A., Beck, J. S., . . . Jamil, H. (2020). Dysfunctional neuroplasticity in newly arrived Middle Eastern refugees in the U.S.: Association with environmental exposures and mental health symptoms. *PLOS ONE*, *15*(3). https://doi.org/10.1371/journal.pone.0230030

Avena, N. M., & Bocarsly, M. E. (2012). Dysregulation of brain reward systems in eating disorders: Neurochemical information from animal models of binge eating, bulimia nervosa, and anorexia nervosa. *Neuropharmacology*, *63*(1), 87–96. https://doi.org/10.1016/j.neuropharm.2011.11.010

Bijoch, L., Borczyk, M., & Czajkowski, R. (2020). Bases of Jerzy Konorski's theory of synaptic plasticity. *European Journal of Neuroscience*, *51*(9), 1857–1866. https://doi.org/10.1111/ejn.14532

Bingaman, K. A. (2013). The Promise of Neuroplasticity for Pastoral Care and Counseling. *Pastoral Psychology*, *62*(5), 549–560. https://doi.org/10.1007/s11089-013-0513-0

Bliss, T. V., & Lomo, T. (1973). Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path. *The Journal of Physiology*, *232*(2), 331–356. https://doi.org/10.1113/jphysiol.1973.sp010273

Brisch, R., Saniotis, A., Wolf, R., Bielau, H., Bernstein, H.-G., Steiner, J., . . . Gos, T. (2014). The Role of Dopamine in Schizophrenia from a Neurobiological and Evolutionary Perspective: Old Fashioned, but Still in Vogue. *Front Psychiatry*, *5*(47). https://doi.org/10.3389/fpsyt.2014.00047

Campos, C., Rocha, N. B., Lattari, E., Nardi, A. E., & Machado, S. (2017). Exercise Induced Neuroplasticity to Enhance Therapeutic Outcomes of Cognitive Remediation in Schizophrenia: Analyzing the Role of Brai nderived Neurotrophic Factor. *CNS & Neurological Disorders - Drug Targets*, *16*(6), 638–651. https://doi.org/10.2174/1871527316661612231429 18

Cassilhas, R. C., Tufik, S., & de Mello, M. T. (2015). Physical exercise, neuroplasticity, spatial learning and memory. *Cellular and Molecular Life Sciences*, *73*(5), 975–983. https://doi.org/10.1007/s00018-015-2102-0

Cerullo, M. A., Eliassen, J. C., Smith, C. T., Fleck, D. E., Nelson, E. B., Strawn, J. R., . . . Strakowski, S. M. (2014). Bipolar I disorder and major depressive disorder show similar brain activation during depression. *Bipolar Disorders*, *16*(7), 703–712. https://doi.org/10.1111/bdi.12225

Chen, M., Pritchard, C., Fortune, D., Kodi, P., & Grados, M. (2019). Hydrogen sulfide: a target to modulate oxidative stress and neuroplasticity for the treatment of pathological anxiety. *Expert Review of Neurotherapeutics*, *20*(1), 109–121. https://doi.org/10.1080/14737175.2019.1668270

Cisler, J. M., & Herringa, R. J. (2021). Posttraumatic Stress Disorder and the Developing Adolescent Brain. *Biological Psychiatry*, *89*(2), 144–151. https://doi.org/10.1016/j.biopsych.2020.06.001

Costandi, M. (2016). *Neuroplasticity*. Cambridge, MA: The MIT Press.

Cramer, S. C., Sur, M., Dobkin, B. H., O'Brien, C., Sanger, T. D., Trojanowski, J. Q., . . . Vinogradov, S. (2011). Harnessing neuroplasticity for clinical applications. *Brain*, *134*(6), 1591–1609. https://doi.org/10.1093/brain/awr039

Crosson, B., Rodriguez, A. D., Copland, D., Fridriksson, J., Krishnamurthy, L. C., Meinzer, M., . . . Leff, A. P. (2019). Neuroplasticity and aphasia treatments: new approaches for an old problem. *Journal of Neurology, Neurosurgery & Psychiatry*, *90*(10), 1147–1155. https://doi.org/10.1136/jnnp-2018-319649

Dadebayev, D., Goh, W. W., & Tan, E. X. (2022). EEG-based emotion recognition: Review of commercial EEG devices and machine learning techniques. *Journal of King Saud University - Computer and Information Sciences*, *34*(7), 4385–4401. https://doi.org/10.1016/j.jksuci.2021.03.009

Demarin, V., Morović, S., & Béné, R. (2014). Neuroplasticity. *Periodicum Biologorium*, *116*(2), 209–211. Retrieved from https://hrcak.srce.hr/126369

Deppermann, S., Storchak, H., Fallgatter, A., & Ehlis, A. C. (2014). Stress-induced neuroplasticity: (Mal)adaptation to adverse life events in patients with PTSD – A critical overview. *Neuroscience*, *283*, 166–177. https://doi.org/10.1016/j.neuroscience.2014.08.037

Diwakar, M., & Kumar, M. (2018). A review on CT image noise and its denoising. *Biomedical Signal Processing and Control*, *42*, 73–88. https://doi.org/10.1016/j.bspc.2018.01.010

Drigas, A. S., Karyotaki, M., & Skianis, C. (2018). An Integrated Approach to Neuro-development, Neuroplasticity and Cognitive Improvement. *International Journal of Recent Contributions from Engineering, Science &Amp; IT (iJES)*, *6*(3), 4. https://doi.org/10.3991/ijes.v6i3.9034

Faravelli, C., Lo Sauro, C., Lelli, L., Pietrini, F., Lazzeretti, L., Godini, L., . . . Ricca, V. (2012). The Role of Life Events and HPA Axis in Anxiety Disorders: A Review. *Current Pharmaceutical Design*, *18*(35), 5663–5674. https://doi.org/10.2174/138161212803530907

Fisher, M., Holland, C., Subramaniam, K., & Vinogradov, S. (2010). Neuroplasticity-Based Cognitive Training in Schizophrenia: An Interim Report on the Effects 6 Months Later. *Schizophrenia Bulletin*, *36*(4), 869–879. https://doi.org/10.1093/schbul/sbn170

Fuchs, E., & Flügge, G. (2014). Adult neuroplasticity: More than 40 years of research. *Neural Plasticity*. https://doi.org/10.1155/2014/541870

Gandhi, A. B., Kaleem, I., Alexander, J., Hisbulla, M., Kannichamy, V., Antony, I., . . . Khan, S. (2020). Neuroplasticity Improves Bipolar Disorder: A Review. *Cureus*, *12*(10). https://doi.org/10.7759/cureus.11241

Gejman, P. V., Sanders, A. R., & Duan, J. (2010). The Role of Genetics in the Etiology of Schizophrenia. *Psychiatric Clinics of North America*, *33*(1), 36–66. https://doi.org/10.1016/j.psc.2009.12.003

H. Natenshon, A. (2016). Clinical Application of Neuroplastic Brain Research in Eating Disorder Treatment. *The Open Access Journal of Science and Technology*, *4*. https://doi.org/10.11131/2016/101219

Hall, F. S., Li, A., & Li, B. (2018). Neural Plasticity in Mood Disorders. *Neural Plasticity*, *2018*. https://doi.org/10.1155/2018/3745251

Higaki, T., Nakamura, Y., Tatsugami, F., Nakaura, T., & Awai, K. (2018). Improvement of image quality at CT and MRI using deep learning. *Japanese Journal of Radiology*, *37*(1), 73–80. https://doi.org/10.1007/s11604-018-0796-2

Hubel, D. H., & Wiesel, T. N. (1964). Effects of monocular deprivation in kittens. *Naunyn-Schmiedebergs Archiv Für Experimentelle Pathologie Und Pharmakologie*, *248*, 492–497. https://doi.org/10.1007/BF00348878

Jahshan, C., Rassovsky, Y., & Green, M. F. (2017). Enhancing Neuroplasticity to Augment Cognitive Remediation in Schizophrenia. *Frontiers in Psychiatry*, *8*. https://doi.org/10.3389/fpsyt.2017.00191

James, W. (1918). *The Principles of Psychology* (Vol. 1).

Jáuregui-Lobera, I., & Martínez-Quiñones, J. V. (2018). Neuromodulation in eating disorders and obesity: a promising way of treatment? *Neuropsychiatric Disease and Treatment*, *14*, 2817–2835. https://doi.org/10.2147/ndt.s180231

Kallen, V., Tulen, J., Utens, E., Treffers, P., De Jong, F., & Ferdinand, R. (2008). Associations between HPA axis functioning and level of anxiety in children and adolescents with an anxiety disorder. *Depression and Anxiety*, *25*(2), 131–141. https://doi.org/10.1002/da.20287

Kapczinski, F., Frey, B. N., Kauer-Sant'Anna, M., & Grassi-Oliveira, R. (2008). Brain-derived neurotrophic factor and neuroplasticity in bipolar disorder. *Expert Review of Neurotherapeutics*, *8*(7), 1101–1113. https://doi.org/10.1586/14737175.8.7.1101

Keefe, R. S. E. (1995). The contribution of neuropsychology to psychiatry. *American Journal of Psychiatry*, *152*(1), 6–15. https://doi.org/10.1176/ajp.152.1.6

Keeler, J. L., Treasure, J., Juruena, M. F., Kan, C., & Himmerich, H. (2021). Ketamine as a Treatment for Anorexia Nervosa: A Narrative Review. *Nutrients*, *13*(11), 4158. https://doi.org/10.3390/nu13114158

Kirshner, H. S. (2012). Aphasia. In V. S. Ramachandran (Ed.), *Encyclopedia of Human Behavior (Second Edition)* (pp. 177–186). Academic Press. https://doi.org/10.1016/B978-0-12-375000-6.00029-X

Krystal, J. H., Tolin, D. F., Sanacora, G., Castner, S. A., Williams, G. V., Aikins, D. E., . . . D'Souza, D. C. (2009). Neuroplasticity as a target for the pharmacotherapy of anxiety disorders, mood disorders, and schizophrenia. *Drug Discovery Today*, *14*(13–14), 690–697. https://doi.org/10.1016/j.drudis.2009.05.002

Lee, J. S. (2021). A Review of Deep-Learning-Based Approaches for Attenuation Correction in Positron Emission Tomography. *IEEE Transactions on Radiation and Plasma Medical Sciences*, *5*(2), 160–184. https://doi.org/10.1109/trpms.2020.3009269

Lewis, D. A. (2009). Neuroplasticity of excitatory and inhibitory cortical circuits in schizophrenia. *Dialogues in Clinical Neuroscience*, *11*(3), 269–280. https://doi.org/10.31887/dcns.2009.11.3/dalewis

Li, W., Ghose, S., Gleason, K., Begovic, A., Perez, J., Bartko, J., . . . Tamminga, C. A. (2015). Synaptic Proteins in the Hippocampus Indicative of Increased Neuronal Activity in CA3 in Schizophrenia. *American Journal of Psychiatry*, *172*(4), 373–382. https://doi.org/10.1176/appi.ajp.2014.14010123

Liang, Z., King, J., & Zhang, N. (2014). Neuroplasticity to a single-episode traumatic stress revealed by resting-state fMRI in awake rats. *NeuroImage*, *103*, 485–491. https://doi.org/10.1016/j.neuroimage.2014.08.050

Lillard, A. S., & Erisir, A. (2011). Old dogs learning new tricks: Neuroplasticity beyond the juvenile period. *Developmental Review*, *31*(4), 207–239. https://doi.org/10.1016/j.dr.2011.07.008

Liu, Y. Z., Wang, Y. X., & Jiang, C. L. (2017). Inflammation: The Common Pathway of Stress-Related Diseases. *Frontiers in Human Neuroscience*, *11*. https://doi.org/10.3389/fnhum.2017.00316

Mah, L., Szabuniewicz, C., & Fiocco, A. J. (2016). Can anxiety damage the brain? *Current Opinion in Psychiatry*, *29*(1), 56–63. https://doi.org/10.1097/YCO.0000000000000223

Makinson, R. A., & Young, J. S. (2012). Cognitive Behavioral Therapy and the Treatment of Posttraumatic Stress Disorder: Where Counseling and Neuroscience Meet. *Journal of Counseling &Amp; Development*, *90*(2), 131–140. https://doi.org/10.1111/j.1556-6676.2012.00017.x

Månsson, K. N., Salami, A., Carlbring, P., Boraxbekk, C. J., Andersson, G., & Furmark, T. (2017). Structural but not functional neuroplasticity one year after effective cognitive behaviour therapy for social anxiety disorder. *Behavioural Brain Research*, *318*, 45–51. https://doi.org/10.1016/j.bbr.2016.11.018

Månsson, K. N. T., Salami, A., Frick, A., Carlbring, P., Andersson, G., Furmark, T., & Boraxbekk, C. J. (2016). Neuroplasticity in response to cognitive behavior therapy for social anxiety disorder. *Translational Psychiatry*, *6*(2), e727–e727. https://doi.org/10.1038/tp.2015.218

McCullumsmith, R. E. (2015). Evidence for Schizophrenia as a Disorder of Neuroplasticity. *American Journal of Psychiatry*, *172*(4), 312–313. https://doi.org/10.1176/appi.ajp.2015.15010069

McCutcheon, R. A., Krystal, J. H., & Howes, O. D. (2020). Dopamine an glutamate in schizophrenia: biology, symptoms and treatment. *World Psychiatry*, *19*(1), 15–33. https://doi.org/10.1002/wps.20693

McEwen, B. S., & Lasley, E. N. (2002). *The end of stress as we know it.* Washington, D. C.: Joseph Henry Press.

Nash, M., Galatzer-Levy, I., Krystal, J. H., Duman, R., & Neumeister, A. (2014). Neurocircuitry and neuroplasticity in PTSD. In M. J. Friedman, T. M. Keane, & P. A. Resick (Eds.), *Handbook of PTSD: Science and practice* (2nd ed., pp. 251–274). The Guilford Press.

Nicoll, R. A. (2017). A Brief History of Long-Term Potentiation. *Neuron*, *93*(2), 281–290. https://doi.org/10.1016/j.neuron.2016.12.015

Nunn, K., Frampton, I., & Lask, B. (2012). Anorexia nervosa – A noradrenergic dysregulation hypothesis. *Medical Hypotheses*, *78*(5), 580–584. https://doi.org/10.1016/j.mehy.2012.01.033

Olsen, C. M. (2011). Natural rewards, neuroplasticity, and non-drug addictions. *Neuropharmacology*, *61*(7), 1109–1122. https://doi.org/10.1016/j.neuropharm.2011.03.010

Parsons, M. W., & Hammeke, T. A. (Eds.). (2014). *Clinical Neuropsychology: A Pocket Handbook for Assessment, Third Edition* (3rd ed.). American Psychological Association.

Phillips, C. (2017). Physical Activity Modulates Common Neuroplasticity Substrates in Major Depressive and Bipolar Disorder. *Neural Plasticity*. https://doi.org/10.1155/2017/7014146

Pinel, J. P. J. (2010). *Biopsychology* (8th ed.). Boston, MA: Pearson.

Pittenger, C. (2013). Disorders of memory and plasticity in psychiatric disease. *Dialogues in Clinical Neuroscience*, *15*(4), 455–463. https://doi.org/10.31887/dcns.2013.15.4/cpittenger

Price, R. B., & Duman, R. (2019). Neuroplasticity in cognitive and psychological mechanisms of depression: an integrative model. *Molecular Psychiatry*, *25*(3), 530–543. https://doi.org/10.1038/s41380-019-0615-x

Quirk, G. J., & Mueller, D. (2007). Neural Mechanisms of Extinction Learning and Retrieval. *Neuropsychopharmacology*, *33*(1), 56–72. https://doi.org/10.1038/sj.npp.1301555

Radley, J. J., Kabbaj, M., Jacobson, L., Heydendael, W., Yehuda, R., & Herman, J. P. (2011). Stress risk factors and stress-related pathology: Neuroplasticity, epigenetics and endophenotypes. *Stress*, *14*(5), 481–497. https://doi.org/10.3109/10253890.2011.604751

Raglan, G. B., Schmidt, L. A., & Schulkin, J. (2017). The role of glucocorticoids and corticotropin-releasing hormone regulation on anxiety symptoms and response to treatment. *Endocrine Connections*, *6*(2), R1–R7. https://doi.org/10.1530/ec-16-0100

Rawlings, C. E., & Rossitch, E. (1994). The history of trephination in Africa with a discussion of its current status and continuing practice. *Surgical Neurology*, *41*(6), 507–513. https://doi.org/10.1016/0090-3019(94)90018-3

Rocha, M. V., Nery, F., Galvão-de- Almeida, A., de Castro Quarantini, L., & Miranda-Scippa, Â. (2017). Neuroplasticity in Bipolar Disorder: Insights from Neuroimaging. In T. Heinbockel (Ed.), *Synaptic Plasticity* (pp. 193–209). IntechOpen. https://doi.org/10.5772/67288

Rutkow, I. M. (2000). Trephination. *Arch Surg*, *135*(9). https://doi.org/10.1001/archsurg.135.9.1119

Seo, J. H., Park, H. S., Park, S. S., Kim, C. J., Kim, D. H., & Kim, T. W. (2019). Physical exercise ameliorates psychiatric disorders and cognitive dysfunctions by hippocampal mitochondrial function and neuroplasticity in post-traumatic stress disorder. *Experimental Neurology*, *322*, 113043. https://doi.org/10.1016/j.expneurol.2019.113043

Serafini, G. (2012). Neuroplasticity and major depression, the role of modern antidepressant drugs. *World J Psychiatry*, *2*(3), 49–57. https://doi.org/10.5498/wjp.v2.i3.49

Shaffer, J. (2016). Neuroplasticity and Clinical Practice: Building Brain Power for Health. *Frontiers in Psychology*, *7*. https://doi.org/10.3389/fpsyg.2016.01118

Skokou, M. (2022). Comment on Keeler et al. Ketamine as a Treatment for Anorexia Nervosa: A Narrative Review. Nutrients 2021, 13, 4158. *Nutrients*, *14*(10), 2118. https://doi.org/10.3390/nu14102118

Steckler, T., Holsboer, F., & Reul, J. M. (1999). Glucocorticoids and depression. *Bailliere's Best Practice & Research. Clinical Endocrinology & Metabolism*, *13*(4), 597–614. https://doi.org/10.1053/beem.1999.0046

Sullivan, C. R., Koene, R. H., Hasselfeld, K., O'Donovan, S. M., Ramsey, A., & McCullumsmith, R. E. (2018). Neuron-specific deficits of bioenergetic processes in the dorsolateral prefrontal cortex in schizophrenia. *Molecular Psychiatry*, *24*(9), 1319–1328. https://doi.org/10.1038/s41380-018-0035-3

Thompson, C. K. (2000). Neuroplasticity. *Journal of Communication Disorders*, *33*(4), 357–366. https://doi.org/10.1016/s0021-9924(00)00031-9

Trimble, M. R. (1988). The neurology of anxiety. *Postgraduate Medical Journal*, *64*(2), 22–26. Retrieved from https://europepmc.org/article/med/3064063

Valdés-Tovar, M., Estrada-Reyes, R., Solís-Chagoyán, H., Argueta, J., Dorantes-Barrón, A. M., Quero-Chávez, D., . . . Benítez-King, G. (2018). Circadian modulation of neuroplasticity by melatonin: a target in the treatment of depression. *British Journal of Pharmacology*, *175*(16), 3200–3208. https://doi.org/10.1111/bph.14197

Val-Laillet, D., Aarts, E., Weber, B., Ferrari, M., Quaresima, V., Stoeckel, L., . . . Stice, E. (2015). Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. *NeuroImage: Clinical*, *8*, 1–31. https://doi.org/10.1016/j.nicl.2015.03.016

Voss, P., Thomas, M. E., Guercio, G. D., & de Villers-Sidani, E. (2019). Dysregulation of auditory neuroplasticity in schizophrenia. *Schizophrenia Research*, *207*, 3–11. https://doi.org/10.1016/j.schres.2018.04.025

Watt, T., Ceballos, N., Kim, S., Pan, X., & Sharma, S. (2019). The Unique Nature of Depression and Anxiety among College Students with Adverse Childhood Experiences. *Journal of Child &Amp; Adolescent Trauma*, *13*(2), 163–172. https://doi.org/10.1007/s40653-019-00270-4

Wobrock, T., Hasan, A., & Falkai, P. (2012). Innovative Treatment Approaches in Schizophrenia Enhancing Neuroplasticity: Aerobic Exercise, Erythropoetin and Repetitive Transcranial Magnetic Stimulation. *Current Pharmaceutical Biotechnology*, *13*(8), 1595–1605. https://doi.org/10.2174/138920112800784763

Zaidi, H., & El Naqa, I. (2021). Quantitative Molecular Positron Emission Tomography Imaging Using Advanced Deep Learning Techniques. *Annual Review of Biomedical Engineering*, *23*(1), 249–276. https://doi.org/10.1146/annurev-bioeng-082420-020343

Zheng, Z., Zeng, Y., Yang, W., & Wu, J. (2014). Irritable bowel syndrome may be induced by decreased neuroplasticity. *Neuroendocrinology Letters*, *35*(8), 655–665. Retrieved from https://www.nel.edu/userfiles/articlesnew/NEL350814R02.pdf

Zhu, G., Sun, X., Yang, Y., Du, Y., Lin, Y., Xiang, J., & Zhou, N. (2019). Reduction of BDNF results in GABAergic neuroplasticity dysfunction and contributes to late-life anxiety disorder. *Behavioral Neuroscience*, *133*(2), 212–224. https://doi.org/10.1037/bne0000301

Zhu, Y., & Zhu, X. (2019). MRI-Driven PET Image Optimization for Neurological Applications. *Frontiers in Neuroscience*, *13*. https://doi.org/10.3389/fnins.2019.00782

Zillmer, E. A., Spiers, M. V., & Culbertson, W. C. (2007). *Principles of Neuropsychology* (2nd ed.). Belmont, CA: Wadsworth Publishing.

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