Encyclopedia of Trauma: An Interdisciplinary Guide

Brain and Trauma

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Psychological trauma can have lasting effects on the brain. These effects underlie symptoms of posttraumatic stress disorder (PTSD), as well as depression, substance abuse, dissociative disorders, and borderline personality disorder (BPD). Brain areas affected by trauma that also mediate the stress response include the amygdala, hippocampus, and medial prefrontal cortex (including the anterior cingulate).

Trauma spectrum psychiatric disorders represent the behavioral manifestation of stress-induced changes in brain structure and function. Stress results in acute and chronic changes in neurochemical systems and specific brain regions, which result in long-term changes in brain “circuits” involved in the stress response.

Hormones (including norepinephrine and cortisol) play a critical role in the stress response. However, chronic stress can lead to dysfunction of these systems. Norepinephrine (adrenaline) is released in both the brain and the body, and has several functions that are critical for survival. Norepinephrine sharpens the senses, focuses attention, raises the level of fear, quickens the heart rate and blood pressure, and in general prepares us for the worst. The norepinephrine system is like a fire alarm that alerts all areas of the brain simultaneously. This system sacrifices the ability to convey specific information to specific parts of the brain in order to obtain more speed. Norepinephrine focuses the senses by activating the neurons that collect information, preparing the body for fight or flight. At the same time, it stimulates the heart to beat more rapidly and blood pressure to increase, causing a rapid transfer of oxygen and nutrients needed for survival to all the cells of the body. Chronic stress in animals leads to increased levels of norepinephrine.

PTSD is associated with long-term dysregulation of the noradrenergic system. Psychophysiology studies have demonstrated an increase in sympathetic nervous system responses (e.g., heart rate, blood pressure, and galvanic skin response) to traumatic reminders. Other studies showed increased nor-epinephrine in plasma and urine at baseline and in response to traumatic reminders. Administration of the alpha2 antagonist yohimbine, which causes increased release of norepinephrine in the brain, resulted in an increase in PTSD-specific symptomatology, as well as
greater release of norepinephrine metabolites in plasma, in PTSD patients. Alterations in brain responses to stimulation of the norepinephrine system with yohimbine were also found in PTSD patients as measured with positron emission tomography (PET).

Figure 1 Impact of Trauma on the Brain. Trauma has lasting effects on the brain, including norepinephrine and cortisol systems and brain areas including the hippocampus, amygdala, and medial prefrontal cortex (mPFC).

The cortisol system also plays an important role in the stress response. Cortisol redistributes energy to enhance survival, suppressing functions not needed for immediate survival, such as reproduction, the body's immune response, digestion, and the feeling of pain, and shunting energy to the brain and muscles. Although useful in the short run, this may occur at the expense of long-term viability of the body. With chronic cortisol elevation, there is increased risk of gastric ulcers, thinning of the bones, cognitive dysfunction, and other problems.

The corticotropin-releasing factor (CRF) is released in the brain during stress, and causes release of adrenocorticotropic-releasing hormone (ACTH) from the pituitary, which in turn stimulates release of cortisol from the adrenal gland. Stress can result in long-term increases in CRF-potentiated release of cortisol with subsequent stressors. PTSD has been associated with long-term dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. Baseline levels of urinary cortisol were either decreased or unchanged in chronic PTSD, but decreased levels were found in 24-hour samples of plasma cortisol levels, and stressors or traumatic reminders are associated with
potentiated release of cortisol. PTSD was associated with a super-suppression of the cortisol response to lower doses of the synthetic form of cortisol, dexamethasone (0.5 mg), a finding that is the opposite of patients with major depression who are nonsuppressors with the standard 1 mg DST test. PTSD patients also had elevated levels of CRF in the cerebrospinal fluid.

Trauma is also associated with changes in brain areas involved in memory, including the hippocampus, amygdala, and medial prefrontal cortex. The hippocampus, a brain area involved in verbal declarative memory, is very sensitive to the effects of stress. Stress in animals was associated with damage to neurons in the CA3 region of the hippocampus (which may be mediated by hypercortisolemia, decreased brain-derived neurotrophic factor, and/or elevated glutamate levels) and inhibition of nerve growth.

The amygdala plays a critical role in the acquisition of fear responses. The medial prefrontal cortex modulates emotional responsiveness through inhibition of amygdala function. The medial prefrontal cortex is also involved in the extinction of conditioned fear responses through inhibition of amygdala function. Animal studies also show that early stress is associated with a decrease in branching of neurons in the medial prefrontal cortex.

Studies have shown changes in hippocampal volume in patients with PTSD as measured by magnetic resonance imaging (MRI).

Other studies in PTSD have found smaller hippocampal volume and/or reductions in N-acetylaspartate (NAA), a marker of neuronal integrity. PTSD patients also have deficits in verbal declarative memory, which is mediated at least in part by the hippocampus. Patients with other trauma-related disorders, including dissociative identity disorder, bipolar disorder, and depression related to early abuse, showed smaller volume of the hippocampus. Other studies found a failure of hippocampal activation measured with functional brain imaging during the performance of memory tasks in PTSD.

Smaller hippocampal volume was reversed with treatment by using paroxetine or phenytoin. It has been hypothesized that stress-induced hippocampal dysfunction may mediate many of the symptoms of PTSD that are related to memory dysregulation,
including both explicit memory deficits as well as fragmentation of memory in abuse survivors. It is unclear at the current time whether these changes are specific to PTSD, whether certain common environmental events (e.g., stress) in different disorders lead to similar brain changes, or whether common genetic traits lead to similar outcomes.

Animal studies also show that early stress is associated with a decrease in branching of neurons in the medial prefrontal cortex. Patients with PTSD had smaller anterior cingulate volume (part of the medial prefrontal cortex) based on MRI measurements in PTSD. Some studies have found reduced anterior cingulate NAA in the prefrontal cortex. Other findings in PTSD include a decrease in gray matter density and smaller volume of the corpus callosum in neglected children and adults with PTSD.

Imaging studies of brain function in PTSD are consistent with dysfunction of the medial prefrontal cortex, amygdala, and hippocampus. Exposure to traumatic reminders in the form of traumatic slides and/or sounds or traumatic scripts was associated with an increase in PTSD symptoms, decreased blood flow and/or failure of activation in the medial prefrontal cortex/anterior cingulate, including Brodmann's area 25, or the subcallosal gyrus, areas 32 and 24, as measured with PET, single-photon emission spectroscopy (SPECT), or functional magnetic resonance imaging (fMRI). Other findings in studies of traumatic reminder exposure include decreased function in hippocampus, thalamus, visual association cortex, parietal cortex, and inferior frontal gyrus, and increased function in amygdala, posterior cingulate, and parahippocampal gyrus. Other studies found decreased medial prefrontal function with recall of traumatic words or during performance of the emotional Stroop task (naming the color of traumatic words), and increased amygdala function with exposure to fearful faces or with classical fear conditioning. Performance of working memory tasks was associated with decreased inferior frontal and parietal function studies in patients with bipolar disorder and a history of early abuse have similarly shown decreased medial prefrontal function.

Imaging studies have found decreased binding of benzodiazepine receptors in the frontal cortex in PTSD, a reduction in anterior cingulate opiate receptor binding, and an increase in hippocampal beta2 nicotinic acetylcholine receptor binding in PTSD.

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