



Basic Neuroscience of Complex Post-Traumatic Stress Disorder (CPTSD)



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Course Outline

- **Basic Understanding**
- **Regions of the Brain Implicated in C-PTSD**



Basic Understanding of CPTSD



What is Complex Post-Traumatic Stress Disorder (C-PTSD)?

Complex post-traumatic stress disorder (C-PTSD) is a psychological **response** to **prolonged** and **repeated interpersonal trauma**, in which escape is difficult or impossible. Cortman, C. & Walden, J. (2018); Herman, J.L. (1997)

C-PTSD is a more severe form of PTSD. The PTSD diagnosis was originally developed for adults who had suffered from a single-event trauma, such as rape, car accident, or war trauma. (National Child Traumatic Stress Network, 2013).

Repeated and prolonged trauma during childhood or adulthood presents a group of symptoms that differ from those described for PTSD. van der Kolk B (2005)

As a result, C-PTSD has been included as a diagnostic category in the International Classification of Diseases, 11th Edition (ICD-11).



Common C-PTSD Symptoms in Children and Adolescence

(Cook, A. et al. 2003)

- **Insecure attachment issues:** difficulty attuning to others' emotional states, problems with boundaries, lack of trust towards others and the world, social isolation, etc.
- **Biological issues:** sensory-motor developmental dysfunction, hypersensitivity to physical contact, excessive somatic symptoms, increased medical problems (e.g., asthma, skin problems, pelvic pains, autoimmune illnesses)
- **Emotional dysregulation:** difficulty describing feelings and other internal states, poor emotional regulation, and difficulties communicating needs, wants and wishes
- **Dissociation:** distinct alterations in states of consciousness, amnesia, depersonalisation, derealisation, two or more dissociative ego states with impaired memory integration



Common C-PTSD Symptoms in Children and Adolescence

(Cook, A. et al. 2003)

- **Behavioural control issues:** self-destructive behaviours, eating disorder, sleep problems, excessive compliance, oppositional behaviours, substance abuse, aggression against others, behavioural re-enactments of trauma
- **Cognition:** difficulty regulating attention; problems with a variety of executive functions, learning difficulty, difficulty planning and anticipating, problems in language development, difficulty processing new information; difficulty focusing and completing tasks; problems with orientation in time and space, acoustic and visual perceptual problems
- **Self-concept problems:** fragmented and disconnected autobiographical narrative, disturbed body image, low self-esteem, toxic shame, toxic guilt, and negative internal working models of self.



**COMPLEX
PTSD**

Common C-PTSD Symptoms in Adults

(ICD-1 1th Edition; Herman, J.L. (1997); Pelcovitz D., et al. (1997)

All diagnostic requirements for PTSD are met (re-experiencing of trauma, avoidance of thoughts and memories of trauma, and hyper-vigilance). In addition, Complex PTSD is characterised by severe and persistent:

- **problems with emotional regulation;** such as persistent **dysphoria** (a profound state of anxiety, agitation, depression, unease), chronic suicidal preoccupation, **self-injury**, explosive or extremely **inhibited anger**, and compulsive or extremely inhibited sexuality
- **problems with self-perception;** such as a sense of **helplessness**, toxic shame, guilt and self-blame (vicious inner critics), a sense of being defective and flawed, and a sense of being completely different from other human beings (may include a sense of specialness, utter aloneness, a belief that no other person can understand, or a feeling of nonhuman identity)
- **problems with consciousness:** such as **amnesia** or improved recall for traumatic events (hyper-amnesia), episodes of **dissociation, depersonalisation/derealisation**, and reliving experiences (either in the form of intrusive PTSD symptoms or in ruminative preoccupation)



Common C-PTSD Symptoms in Adults

(ICD-1 1th Edition; Herman, J.L. (1997); Pelcovitz D., et al. (1997))

- **problem with perception of the perpetrators;** such as a preoccupation with the relationship with a perpetrator (including a preoccupation with revenge), an unrealistic attribution of total power to a perpetrator, a sense of a special or supernatural relationship with a perpetrator, and acceptance of a perpetrator's belief system or rationalisations
- **problems with relations with others;** such as isolation and withdrawal, disruption in intimate relationships, a repeated search for a rescuer (may alternate with isolation and withdrawal), persistent distrust, and repeated failures of self-protection.
- **problems with systems of meaning;** such as a loss of sustaining faith in self (self-abandonment) and a sense of hopelessness and despair.



The Brain Regions Implicated in C-PTSD



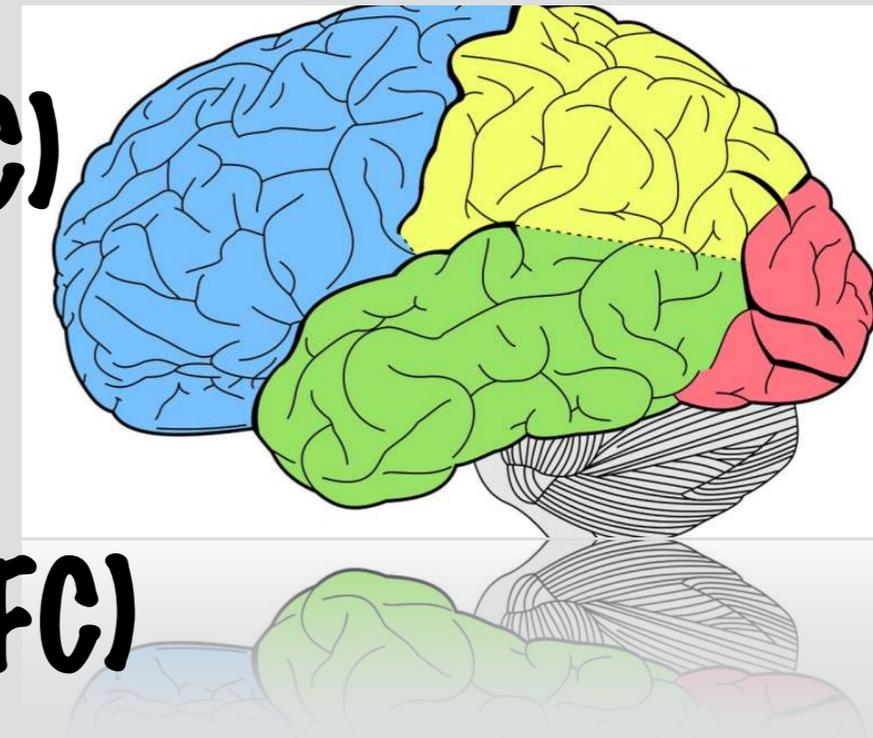
Brain Regions Implicated in C-PTSD

(Woodward, S.H., et al. 2006; McGovern R.A. & Sheith, S.A., 2017; Hamner, M.B., et al., 1999)

Using functional brain scans (fMRIs, DTI, and SPECT), a C-PTSD brain shows distinct abnormalities in a variety of brain regions.

These regions are:

- Anterior Cingulate Cortex (ACC)
- Amygdala
- Medial-Prefrontal Cortex (mPFC)
- Hippocampus
- HPA axis



Anterior Cingulate Cortex (ACC)

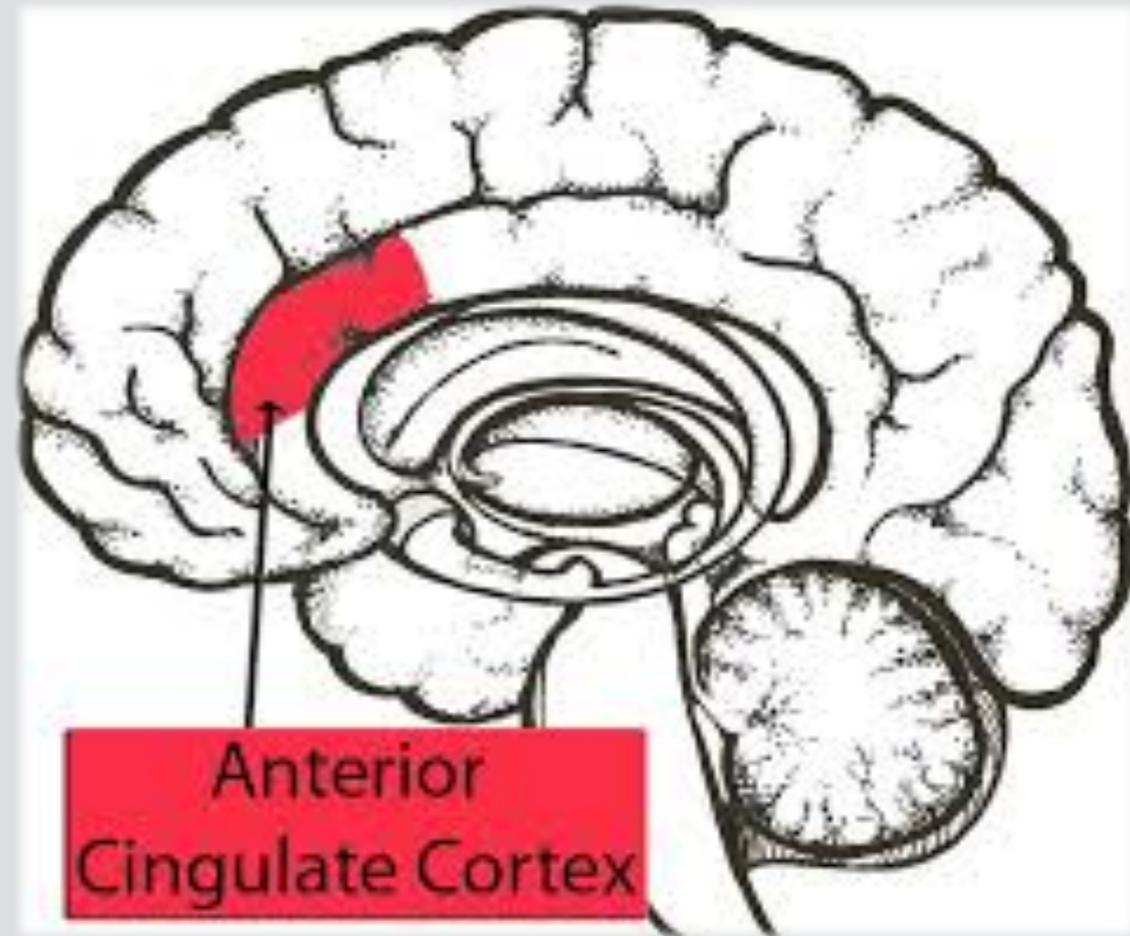
In C-PTSD, the ACC serves as a **gear-shifter** that gets stuck in exaggerated emotional and behavioural responses to conditioned fear (hyper-arousal).

(Hamner, M.B., et al., 1999; Young D.A. et al. 2018)

ACC malfunction has also been linked to **obsessive threat sensitivity** and **compulsive threat response** found in both OCD and PTSD

(McGovern RA and Sheith SA, 2017; Hamner, M.B., et al., 1999)

Complex trauma shrinks the ACC volume, affecting its functions; such as self-sensing, cognitive flexibility, fear and intrusive thoughts regulation. Woodward, S.H., et al. (2006)



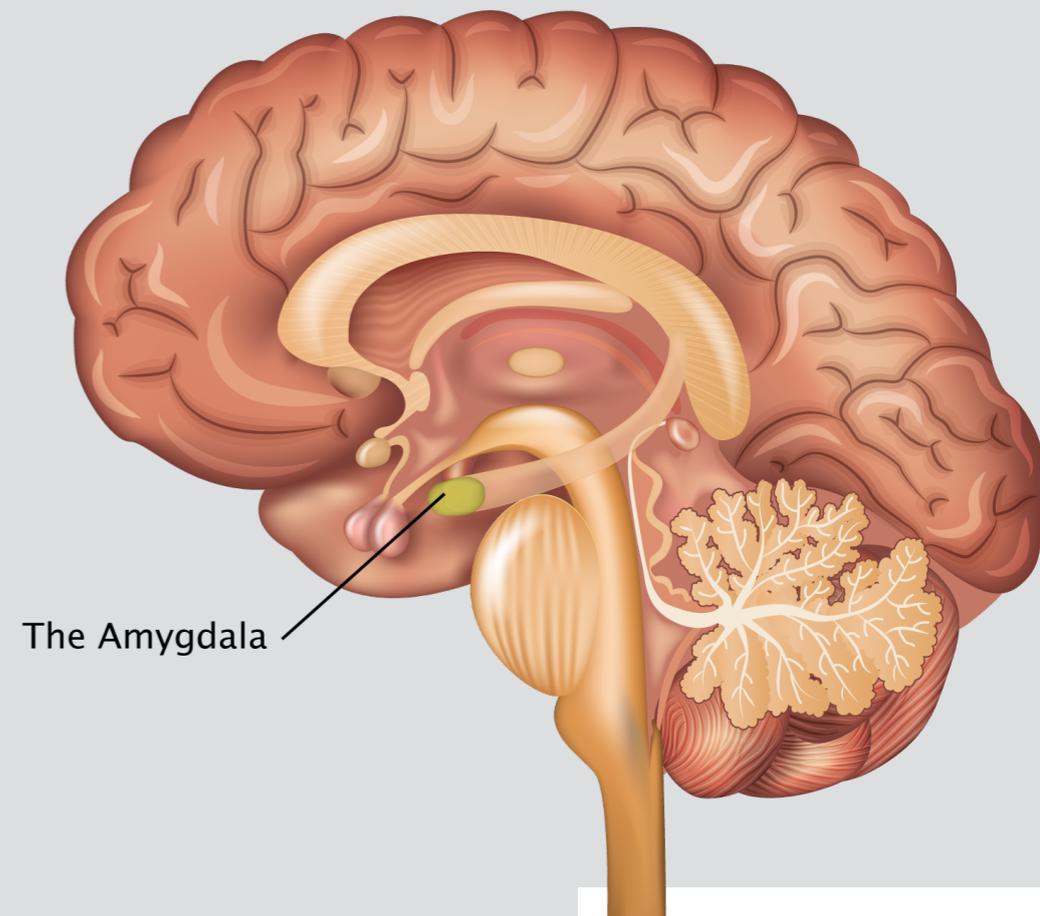
Amygdala

The amygdala is the fear and anxiety centre in the brain (chief security officer) involved in the activation of the fight/flight response. Simon, D. et al, 2014; Thorsen et al, 2018.

The amygdala plays a primary role in the acquisition and expression of conditioned fear and in the magnification of emotional memory. Koenigs, M., & Grafman, J. (2009)

Hyper-activation of the amygdala centre has been observed in complex trauma. Koenigs, M., & Grafman, J. (2009)

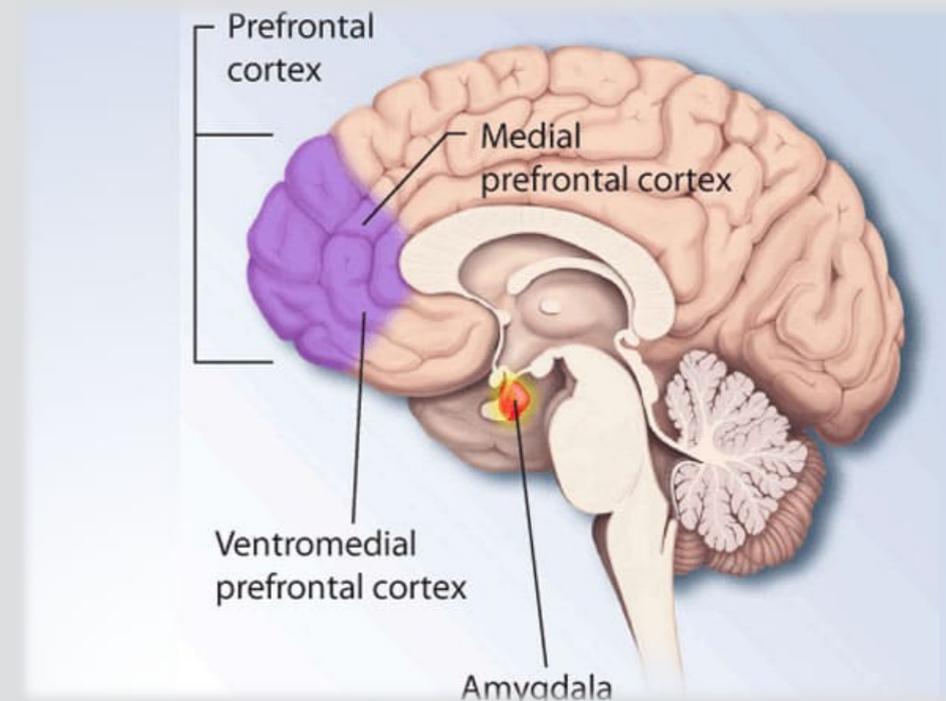
Individuals with borderline personality disorder and early abuse (CPTSD) have been found to have smaller amygdala volume. Vermetten, E. et al. (2011)



Medial-Prefrontal Cortex (mPFC)

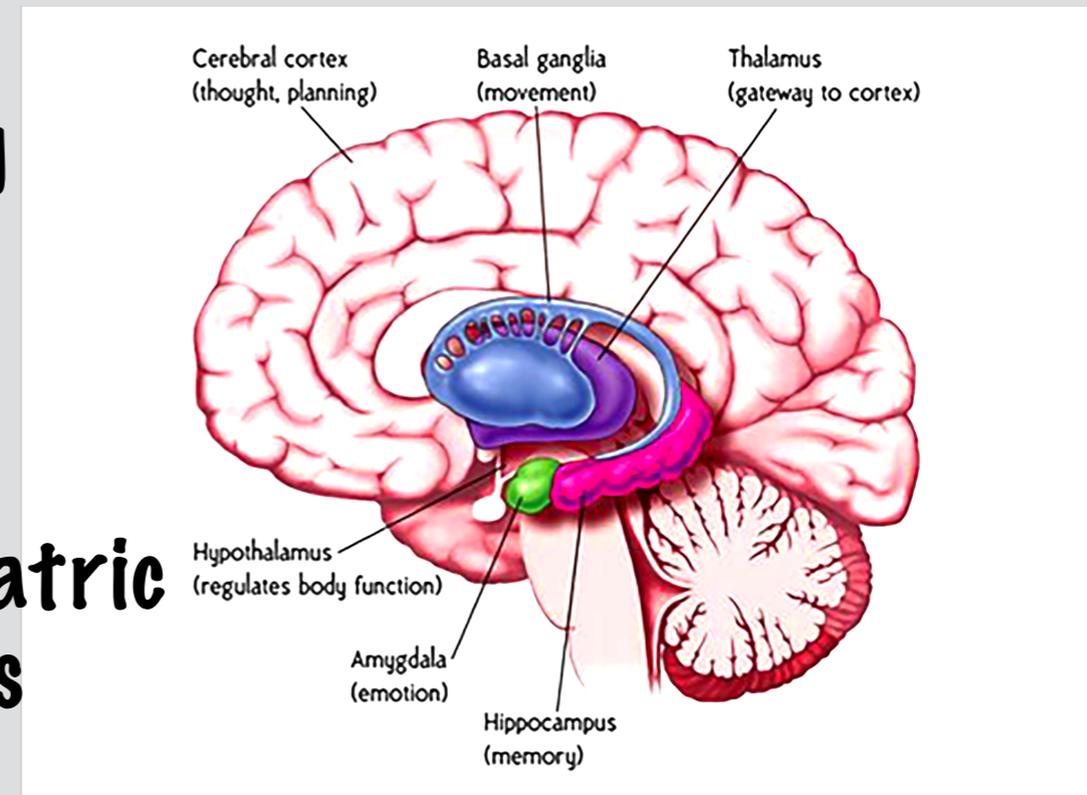
Koenigs, M. & Grafman, J. (2009)

- mPFC is involved in the **extinction** of conditioned fear and in the appropriate regulation of **negative emotion**
- The mPFC serves as a **breaking system** for the amygdala. A defect in this inhibitory capacity has been linked to exaggerated PTSD symptoms
- Functional imaging studies of complex trauma show **hypo-activity** in the mPFC.
- Individuals with PTSD exhibit **decreased volumes** of the pre-frontal cortex. Rauch, S.L. et al. (2003)



Hippocampus

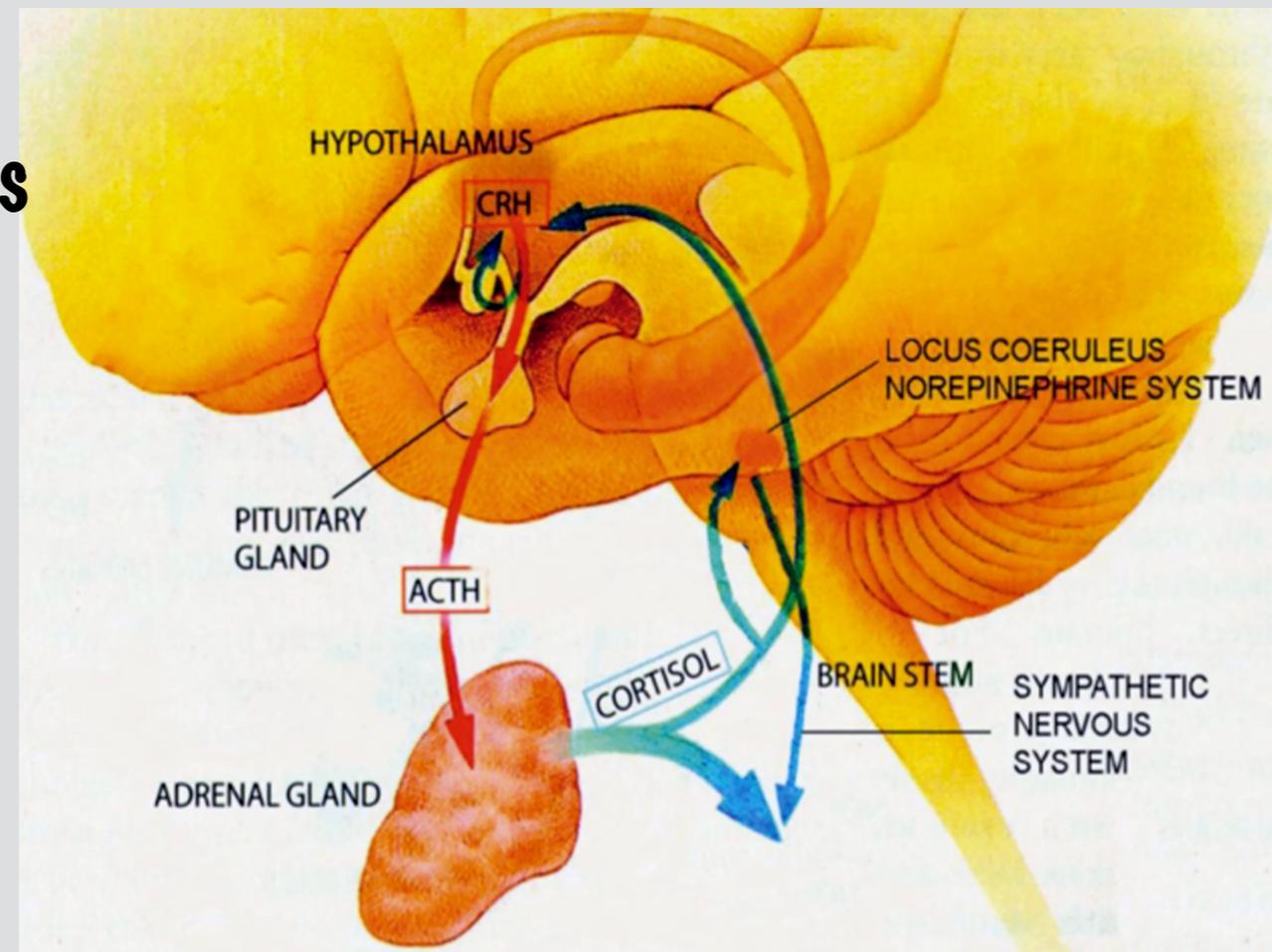
- Hippocampus plays a critical role in memory, learning, and stress regulation. Brunson, K.L. et al 2003
- The anterior hippocampus (aHPC) has a central role in encoding short-term memory, anxiety-related behaviour, stress response, emotional memory and unconditioned fear. Abdallah, C. G. Et al. 2017
- The posterior hippocampus (pHPC) has a central role in encoding long-term memory, spacial memory, autobiographical memory (implicit), and contextual fear conditioning. Abdallah, C. G. Et al. 2017
- PFC - aHPC dysconnectivity has been observed in individuals with PTSD, leading to hyperarousal or numbing symptoms. Abdallah, C. G. Et al. 2017
- Smaller hippocampal volume has been reported in several stress-related psychiatric disorders, including post-traumatic stress disorder (PTSD). Vermetten, E. Et al. (2011)



Hypothalamic-Pituitary-Adrenal (HPA) Axis

Jonathan E. S. & Charles B. N. (2011); de Quervain, D.J. 2008; Resnick, H.S. et al. 1995

- The hypothalamic-pituitary-adrenal axis is the body's major response system for stress, producing cortisol and adrenaline.
- The hippocampus and prefrontal cortex (PFC) inhibit the HPA axis activities.
- The amygdala and brain stem neurones stimulate the HPA axis
- Low cortisol levels at the time of exposure to trauma may predict the development of PTSD
- Sustained cortisol exposure degenerates the hippocampal neurones, interferes with the retrieval of traumatic memories, and impairs neurogenesis
- Complex trauma can trigger thyroid abnormalities by breaking down the hypothalamic-pituitary-thyroid (HPT) axis



Neurobiological Regions Affected in Complex Post-Traumatic Stress Disorder

| Feature | Change | Effect |
|-------------------------------------|--|--|
| Hippocampus | Reduced volume and activity | Alters stress responses and extinction |
| Amygdala | Increased activity | Promotes hypervigilance and impairs discrimination of threat |
| Cortex | Reduced prefrontal volume | Dysregulates executive functions |
| | Reduced anterior cingulate volume | Impairs the extinction of fear responses |
| | Decreased medial prefrontal activation | Poor executive functions, impairs the extinction of fear responses |
| Hypothalamic-pituitary-adrenal axis | Hypocortisolism | Disinhibits CRH/NE and upregulates response to stress |
| | | Drives abnormal stress encoding and fear processing |
| | Sustained, increased level of CRH | Blunts ACTH response to CRH stimulation |
| | | Promotes hippocampal atrophy |
| Hypothalamic-pituitary-thyroid axis | Abnormal T3: T4 ratio | Increases subjective anxiety |

Jonathan E. S. & Charles B. N. (2011)

Neurochemicals Affected in Complex Post-Traumatic Stress Disorder

| Feature | Change | Effect |
|----------------|--|---|
| Catecholamines | Increased dopamine levels | Interferes with fear conditioning by mesolimbic system |
| | Increased norepinephrine levels/activity | Increases arousal, startle response, encoding of fear memories |
| | | Increases pulse, blood pressure, and response to memories |
| Serotonin | Decreased concentrations of 5 HT in: <ul style="list-style-type: none"> • Dorsal raphé • Median raphé • Dorsal/median raphé | Disturbs dynamic between amygdala and hippocampus |
| | | Compromises anxiolytic effects (body's natural anxiety relief effort) |
| | | Increases vigilance, startle, impulsivity, and memory intrusions |
| Amino acids | Decreased GABA activity | Compromises anxiolytic effects (body's natural anxiety relief effort) |
| | Increased glutamate | Fosters derealisation and dissociation |
| peptides | Decreased plasma NPY concentrations | Leaves CRH/NE unopposed and upregulates response to stress |
| | Increased CSF β -endorphin levels (endogenous opioid) | Fosters numbing, stress-induced analgesia, and dissociation |

Q&A Session

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enquiries@mindbodybreakthrough.net**

“There are no constraints on the human mind, no walls around the human spirit, no barriers to our progress except those we ourselves erect.”

—Ronald Reagan, 40th U.S President