Trauma and the HPA Axis

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"Trauma is personal. It does not disappear if it is not validated. When it is ignored or invalidated the silent screams continue internally heard only by the one held captive. When someone enters the pain and hears the screams healing can begin."

— Danielle Bernock

Trauma exposure often starts in childhood

Trauma exposure, particularly child maltreatment (e.g., neglect, emotional, physical and sexual abuse), has been established as one of the main determinants of emotional dysregulation and is also a known risk factor for psychiatric disorders, especially depression and PTSD (McLaughlin et al.,2012; McLaughlin et al., 2013).

Moreover, several prior studies have shown that trauma exposure is clearly associated with profound deficits in emotional regulation across the entire lifespan, including during preschool (Langevin, Hebert, Allard-Dansereau; Bernard-Bonnin, 2016), adolescence (Shields & Cicchetti, 1997; Vettese, Dyer, Li, & Wekerle, 2011) and even adulthood (Briere & Rickards, 2007; Thompson, Hannan, & Miron, 2014; Dunn et al., 2018).

Trauma occurs when we are faced with an experience that overwhelms our ability to process incoming information both at the time of that experience and in future situations (Barta, 2018).

Dr. Michael Barta suffered from trauma himself as a child which led him to addictions that ultimately landed him in jail and almost destroyed his life. In is book, *TINSA*, he wrote that trauma occurs when our natural defenses are unable to keep us safe from physical, emotional, or mental threats or harm (Barta, 2018).

Dr. Michael Barta's Adverse Childhood Experiences

Barta (2018) in his book, *TINSA,* defines ACEs a little differently as summarized below:

- Sexual assault or abuse
- Physical assault or abuse
- Psychological or emotional trauma
- Serious accidents, medical procedures, or illnesses
- Manmade or natural disasters
- Witnessing violence to include domestic abuse
- School violence to include bullying
- Traumatic grief or unwanted separation
- Terrorism or war
- Betrayal by others to include relational trauma

Trauma - Adverse Childhood Experiences

The ten reference categories experienced during childhood or adolescence are as below, with their prevalence in parentheses (Felitti and Anda, 2009):

Abuse

- Emotional – recurrent threats, humiliation (11%)
- Physical - beating, not spanking (28%)
- Contact sexual abuse (28% women, 16% men, 22% overall)

Household dysfunction

- Mother treated violently (13%)
- Household member was alcoholic or drug user (27%)
- Household member was imprisoned (6%)
- Household member was chronically depressed, suicidal, mentally ill, or in psychiatric hospital (17%)
- Not raised by both biological parents (23%)

Neglect

- Physical (10%)
- Emotional (15%)

Big T Trauma and Little t Trauma

- In my personal experience as a pediatric psychologist, far more of my patients have been subjected to "little t" traumas and I agree with Barta that these experiences have a tremendous impact on how children view themselves, their relationships, and their place in the world.
- Moreover, the long-term consequences of these traumas are tremendous and often lead to a total inability or impaired ability to access appropriate responses to threatening events and can lead to chronic hyperarousal, intense anxiety, panic, mood instability, poor emotional/behavioral regulation, feelings of powerlessness, helplessness, shame, and even immobility.
- Of all traumas, relational (or loss of connection) trauma is particularly devastating.
- The implications here are enormous. Specifically, in order to promote safe and healthy emotional regulation, we must be able to pinpoint where in the lifespan people hurt us physically, emotionally, mentally, or spiritually, whether intentionally or accidentally.
- If we can resolve our developmental wounds, we can move on and experience a more fulfilling life.

Big T Trauma and Little t Trauma

The experts in the field divide trauma into two categories:

Big T trauma: Traumas that are associated with horrific single events such as natural disasters, terrorism, and war.

Little t trauma: Trauma that are smaller in nature such as bullying, neglect, and betrayal.

ACE Scores and Outcomes

As Dr. Felitti in a 2009 lecture points out, studies reveal many shocking long-term horrible outcomes when we are exposed to ACEs and this raises exponentially according to how many of them we have been exposed to.

The results indicate that for every category of traumatic experience we have had as a child, we are dramatically more likely to be depressed as an adult.

If we have ACE scores of  we are:

- 260% more likely to have chronic obstructive pulmonary disease than someone with a score of 0
- 240% more likely to contract hepatitis, 460% more likely to experience depression
- 1,220% more likely to attempt suicide

If we have ACE scores of  we are:

- Five times more likely to become depressed as an adult and if we have had

If we have ACE scores of  , we are:

- 3,100 percent more likely to attempt suicide as an adult (Felitti et al., 2014; Felitti 2004; Felitti and Anda, 2009; Felitti et al., 1998).

Dr. Felitti offered the following graphs which nicely detail the dramatic impact that ACEs have on our society:

The chart below adapted by Dr. Rothschild nicely demonstrates the shifting in body sensations, physiological symptoms, and emotions as we move between autonomic states (Rothschild, 2017).

AUTONOMIC NERVOUS SYSTEM: PRECISION REGULATION
** WHAT TO LOOK FOR **

"Normal" Life

Threat to Life

		LETHARGIC Parasympathetic I (PNS I)	CALM Parasympathetic II (PNS II) *Ventral Vagus*	ACTIVE/ALERT Sympathetic I (SNS I)	FLIGHT/FIGHT Sympathetic II (SNS II)	HYPER FREEZE Sympathetic III (SNS III)	HYPO FREEZE Parasympathetic III (PNS III) *Dorsal Vagus* Collapse
PRIMARY STATE		**Apathy, Depression**	**Safe, Clear Thinking, Social Engagement**	**Alert, Ready to Act**	**React to Danger**	**Await Opportunity to Escape**	**Prepare for Death**
AROUSAL		**Too Low**	**Low**	**Moderate**	**High**	**Extreme Overload**	**Excessive Overwhelm Induces Hypoarousal**
MUSCLES		Slack	Relaxed/toned	Toned	Tense	Rigid (deer in the headlights)	Flaccid
RESPIRATION		Shallow	Easy, often into belly	Increasing rate	Fast, often in upper chest	Hyperventilation	Hypo-ventilation
HEART RATE		Slow	Resting	Quicker or more forceful	Quick and/or forceful	Tachycardia (very fast)	Bradycardia (*very* slow)
BLOOD PRESSURE		Likely low	Normal	On the rise	Elevated	Significantly high	Significantly low
PUPILS, EYES, EYE LIDS		Pupils smaller, lids may be heavy	Pupils smaller, eyes moist, eye lids relaxed	Pupils widening, eyes less moist, eye lids toned	Pupils very dilated, eyes dry, eye lids tensed/raised	Pupils very small or dilated, eyes very dry, lids very tense	Lids drooping, eyes closed or open and fixed
SKIN TONE		Variable	Rosy hue, despite skin color (blood flows to skin)	Less rosy hue, despite skin color (blood flows to skin)	Pale hue, despite skin color (blood flow to muscles)	May be pale and/or flushed	Noticeably pale
HUMIDITY	Skin	Dry	Dry	Increased sweat	Increased sweat, may be cold	Cold sweat	Cold sweat
	Mouth	Variable	Moist	Less moist	Dry	Dry	Dry
HANDS & FEET (TEMPERATURE)		May be warm or cool	Warm	Cool	Cold	Extremes of cold & hot	Cold
DIGESTION		Variable	Increase	Decrease	Stops	Evacuate bowel & bladder	Stopped
EMOTIONS (LIKELY)		Grief, sadness, shame, disgust	Calm, pleasure, love, sexual arousal, "good" grief	Anger, shame, disgust, anxiety, excitement, sexual climax	Rage, fear	Terror, may be dissociation	May be too dissociated to feel anything
CONTACT WITH SELF & OTHERS		Withdrawn	Probable	Possible	Limited	Not likely	Impossible
FRONTAL CORTEX		May or may not be accessible	Should be accessible	Should be accessible	May or may not be accessible	Likely inaccessible	Inaccessible
INTEGRATION		Not likely	Likely	Likely	Not likely	Impossible	Impossible
RECOMMENDED INTERVENTION		**Activate, Gently Increase Energy**	**Continue Therapy Direction**	**Continue Therapy Direction**	**Put on Brakes**	***Slam on Brakes***	***Medical Emergency CALL PARAMEDICS***

The Autonomic Nervous System Precision Regulation Chart is Available for purchase on Amazon for $8.99 (a very high recommend): Babette Rothschild (2017) https://www.amazon.com/Autonomic-Nervous-System-Table-Laminated/dp/039371280X/ref=sr_1_15?dchild=1&keywords=deb+dana&qid=1590326813&s=books&sr=1-15

Several of the following slides are adapted from Dr. Dawn-Elise Snipes excellent lecture on HPA-Axis Dysfunction in her review of the article cited below.

Click here to listen: https://www.youtube.com/watch?v=qLxgQdxedL4&ab_channel=DocSnipes

Based on

- **Post-traumatic stress disorder: the neurobiological impact of psychological trauma**

  Dialogues Clin Neurosci. 2011 Sep; 13(3): 263-278.

  https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3182008/

  This article lays out the many changes and/or conditions seen in the brain of people with PTSD.

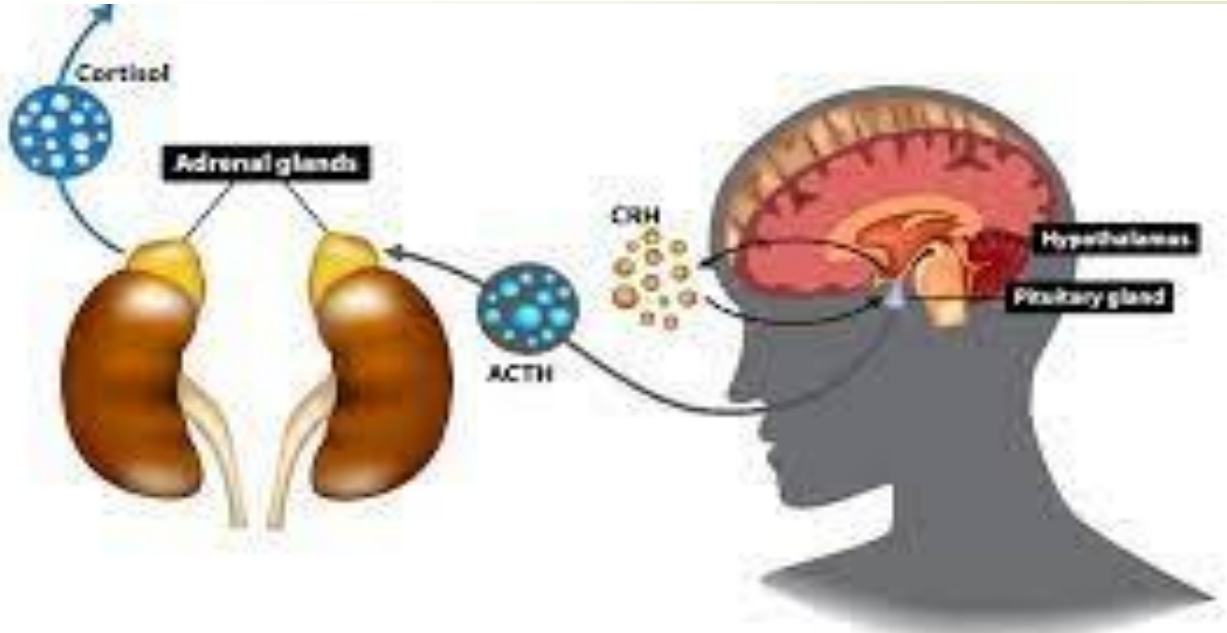
- As clinicians, awareness of these changes can help us educate patients about their symptoms and find ways of adapting to improve quality of life.

Exposure to excessive trauma impacts the brain – in particular, he HPA-Axis.

The HPA- Axis is major component of the homeostatic response is the hypothalamic-pituitary-adrenal (HPA) axis, an intricate, yet robust, neuroendocrine mechanism that mediates the effects of stressors by regulating numerous physiological processes, such as metabolism, immune responses, and the autonomic nervous system

What is the HPA Axis

- Hypothalamic-Pituitary-Adrenal Axis AKA the Threat Response System
  - Controls reactions to stress and regulates many body processes, including digestion, the immune system, mood and emotions, sexuality, and energy storage and expenditure
  - The ultimate result of the HPA axis activation is to increase levels of cortisol in the blood during times of stress.
  - Cortisol's main role is in releasing glucose into the bloodstream in order to facilitate the "flight or fight" response. It also suppresses and modulates the immune system, digestive system and reproductive system.



HPA-Axis Dysfunction can result in

Hypercortisolism

- The body reduces its HPA axis activation when it appears that further fight/flight may not be beneficial. (Hypocortisolism)
- Hypocortisolism seen in **stress-related disorders such as CFS, burnout and PTSD** is actually a protective mechanism designed to conserve energy during threats that are beyond the organism's ability to cope.
- Dysfunctional HPA axis activation will result in
  - Abnormal immune system activation
  - Increased inflammation and allergic reactions
  - IBS symptoms such as constipation and diarrhea,
  - Reduced tolerance to physical and mental stresses (including pain)
  - Altered levels of sex hormones

Fatigue

- Fatigue is actually an emotion generated in the brain, which prevents damage to the body when the brain perceives that further exertion could be harmful.
  - Fatigue in sports is largely independent of the state of the muscles themselves and is more related to:
    - Physical factors
      - Core temperature
      - Glycogen levels
      - Oxygen levels in the brain
      - Thirst
      - Sleep deprivation
      - Levels of muscle soreness/fatigue

HPA-Axis Dysfunction and Fatigue

Fatigue

- Fatigue cont…
  - Psychological factors reducing fatigue
    - Emotional state
    - Knowledge of the endpoint
    - Other competitors/motivation
    - Visual feedback
- Fatigue is one sign that the body is getting ready to downregulate the HPA-Axis
- In counseling practice, how can we reduce fatigue and help clients restore HPA-Axis functioning?

HPA-Axis Dysfunction and Fatigue – cont.

HPA-Axis Dysfunction and Cortisol

Cortisol is a hormone that is mainly released at times of stress. Cortisol has many important functions in the body. Having the right cortisol balance is essential for human health and you can have problems if you produce too much or too little cortisol.

What is cortisol?

Cortisol is a steroid hormone that is produced by the adrenal glands, which sit on top of each kidney. When released into the bloodstream, cortisol can act on many different parts of the body and can help:

- the body respond to stress or danger
- increase the body's metabolism of glucose
- control blood pressure
- reduce inflammation

Cortisol is also needed for the fight or flight response, which is a healthy, natural response to perceived threats. The amount of cortisol produced is highly regulated by your body to ensure the balance is correct.

Low Cortisol and PTSD

Low Cortisol and PTSD

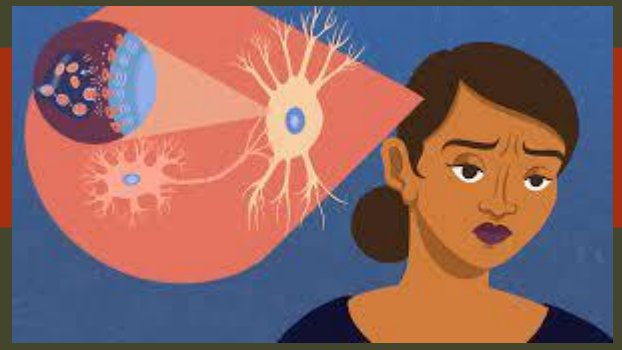
- Low cortisol has been found to relate to more severe PTSD hyperarousal symptoms.
  - Sensitized negative feedback loop in veterans diagnosed with PTSD by means of a greater gluticorticoid responsiveness. (0-100)
    - Generally low cortisol, but when a threat is perceived there is an exaggerated stress response. (Flat or furious)
  - Evidence points toward a role of trauma experience in sensitizing HPA axis regulation, independent of PTSD development.
    - Those with prior trauma may be more at risk of PTSD from later traumas (Area for prevention)

HPA-Axis Dysfunction and Endocrine Factors

Endocrine Factors

- Core endocrine features of PTSD include abnormal regulation of Cortisol and thyroid hormones
- Hypocortisolism in PTSD occurs due to increased negative feedback sensitivity of the HPA axis.
- Studies suggest that low Cortisol levels at the time of exposure to psychological trauma may predict the development of PTSD. (Prior trauma exposure may predispose to PTSD)
- Glucocorticoids interfere with the retrieval of traumatic memories, an effect that may
  - Independently prevent or reduce symptoms of PTSD
  - Or contribute to difficulty treating PTSD

HPA-Axis Dysfunction and Neurochemical Factors



Neurochemical Factors

- Core neurochemical features of PTSD include abnormal regulation of catecholamine, serotonin, amino acid, peptide, and opioid neurotransmitters, each of which is found in brain circuits that regulate/integrate stress and fear responses.
- the catecholamine family of neurotransmitters, including dopamine (DA) and norepinephrine (NE), derive from the amino acid tyrosine
- When a stressor is perceived the HPA Axis releases CRH which interacts with NE to increase fear conditioning and encoding of emotional memories, enhance arousal and vigilance, and integrate endocrine and autonomic responses to stress.
- there is an abundance of evidence that NF, accounts for certain classic aspects of PTSD symptomatology, including hyperarousal, heightened startle, and increased encoding of fear memories

HPA-Axis Dysfunction and Neurochemical Factors

Neurochemical Factors

- Serotonin (5HT)
  - Poor serotonin transmission in PTSD may cause impulsivity, hostility, aggression, depression, and suicidally
  - Serotonin binding to 5HT1A receptors do not differ between patients with PTSD and controls.

Note that there are many different serotonin receptors so it’s complicated

Serotonin Receptors (Soap Box)

5-HT1A
- Addiction
- Aggression
- Anxiety
- Appetite
- Blood Pressure
- Heart Rate
- Impulsivity
- Memory
- Mood
- Respiration
- Sexual Behavior
- Sleep
- Sociability

5-HT1B
- Addiction
- Aggression
- Anxiety
- Learning/Memory
- Mood

5-HT1D
- Anxiety

5-HT2A
- Addiction
- Anxiety
- Appetite
- Cognition
- Imagination
- Learning
- Memory
- Mood
- Perception
- Sexual Behavior
- Sleep

5-HT2B
- Anxiety
- Appetite
- GI Motility
- Sleep

5-HT2C
- Addiction
- Anxiety
- Appetite
- Mood
- Sexual Behavior
- Sleep

5-HT3
- Addiction
- Anxiety
- GI Motility
- Learning
- Memory
- Nausea

5-HT4
- Anxiety
- Appetite
- Learning
- Memory
- Mood

5-HT5A
- Sleep

5-HT6
- Anxiety
- Cognition
- Learning
- Memory
- Mood

5-HT7
- Anxiety
- Autoreceptor
- Memory
- Mood
- Respiration
- Sleep

https://en.wikipedia.org/wiki/5-HT_receptor

HPA-Dysfunction and GABA

- Gamma-aminobutyric acid (GABA) is an amino acid that functions as the primary inhibitory neurotransmitter for the central nervous system (CNS). It functions to reduce neuronal excitability by inhibiting nerve transmission. GABAergic neurons are located when the hippocampus, thalamus, basal ganglia, hypothalamus, and brainstem. The balance between inhibitory neuronal transmission via GABA and excitatory neuronal transmission via glutamate is essential for proper cell membrane stability and neurologic function.

Neurochemical Factors

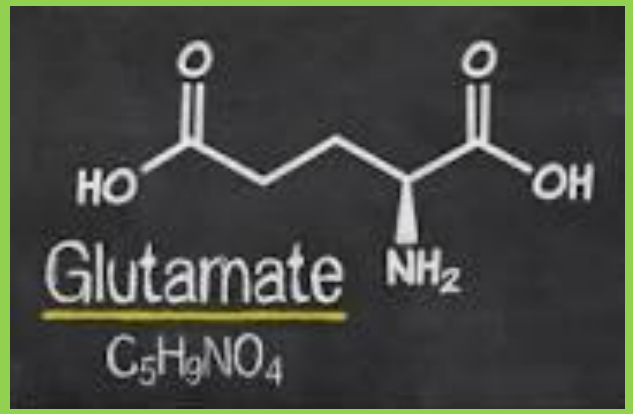
- GABA has profound anxiolytic effects in part by inhibiting the CRH/NE circuits
  - Patients with PTSD exhibit **decreased peripheral benzodiazepine binding sites.**
- May indicate the usefulness of emotion regulation and distress tolerance skills due to potential emotional dysregulation
  - We need to reduce excitotoxicity in order to reduce distress, improve stress tolerance and enable the acquisition of new skills

HPA-Axis Dysfunction and Glutamate

Glutamate is an important neurotransmitter present in over 90% of all brain synapses and is a naturally occurring molecule that nerve cells use to send signals to other cells in the central nervous system. Glutamate plays an essential role in normal brain functioning and its levels must be tightly regulated. Abnormalities in glutamate function can disrupt nerve health and communication, and in extreme cases may lead to nerve cell death.
Glutamate

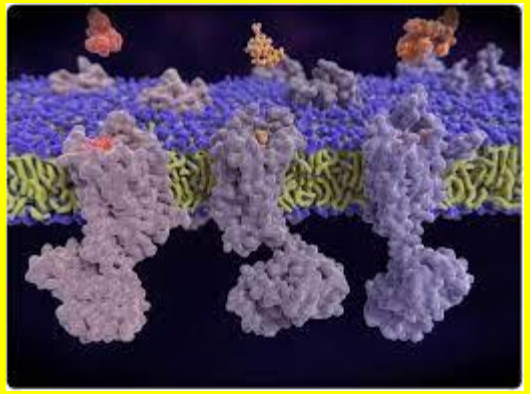
Neurochemical Factors

- NMDA (Glutamate) Receptors
  - The NMDA receptor system has been implicated in synaptic plasticity, as well as **learning and memory**
  - Glutamate binds to NMDA receptors. High levels of glutamate are secreted during high levels of stress
  - Ketamine blocks NMDA receptors
  - Overexposure of neurons to glutamate is known to be excitotoxic, and may contribute to the loss of neurons in the hippocampus of patients with PTSD
  - **Elevated glucocorticoids increase the sensitivity of NMDA receptors, rendering the brain more vulnerable to excitoxic insults at times of stress.**
- Points to ponder
  - It may take clients with PTSD more time to master new skills
  - If the brain becomes excitotoxic during stress, inhibiting learning and memory, then exposure therapies may also be dangerous.



HPA-Dysfunction and Endogenous Opioids

The endogenous opioid system is comprised of a wide array of receptors and ligands that are present throughout the central and peripheral nervous system, the gastrointestinal tract, and the immune system. This explains the multitude of physiological functions it is responsible for including analgesia, mood regulation, and modulation of the stress response. It also plays a pivotal role in modulating the brain's reward center with behavioral and social implications on mood disorders and addiction. Exogenous opioid therapy hijacks the endogenous system



Neurochemical Factors

- Endogenous opioids act upon the same CNS receptors activated by exogenous opioids such as morphine or heroin.
- Opioids (depressants) exert inhibitory influences on the HPA axis.
- Alterations in endogenous opioids may be involved in certain PTSD symptoms such as numbing, stress-induced analgesia, and dissociation.
- Naltrexone, appears to be effective in treating symptoms of dissociation and flashbacks in traumatized persons.
- Highlights the risk for opiate abuse of persons with PTSD
- How can we assist with physical and emotional distress tolerance

HPA-Axis Dysfunction Changes in Brain Structure – Hippocampus

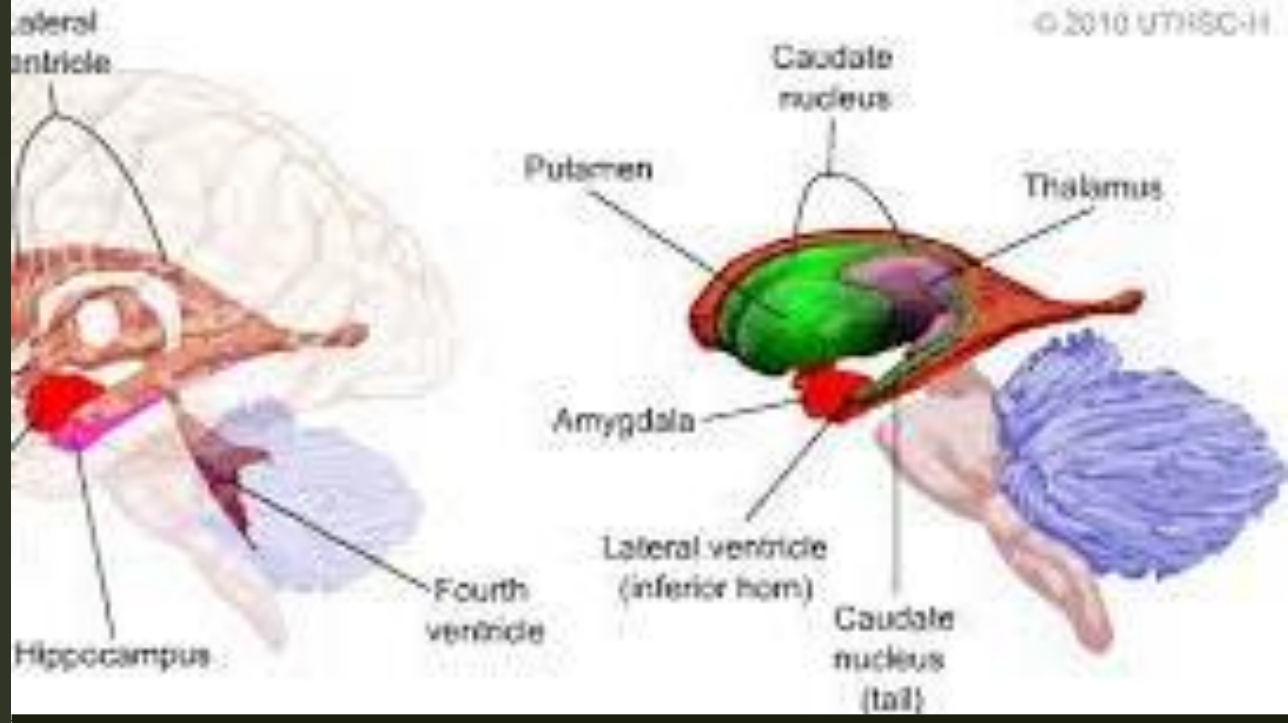
The hippocampus (via Latin from Greek ἱππόκαμπος, 'seahorse') is a major component of the brain of humans and other vertebrates. Humans and other mammals have two hippocampi, one in each side of the brain. The hippocampus is part of the limbic system, and plays important roles in the consolidation of information from short-term memory to long-term memory, and in spatial memory that enables navigation. The hippocampus is located in the allocortex, with neural projections into the neocortex in humans, as well as primates. The hippocampus, as the medial pallium, is a structure found in all vertebrates. In humans, it contains two main interlocking parts: the hippocampus proper (also called *Ammon's horn*), and the dentate gyrus.

Changes? In Brain Structure

- A hallmark feature of PTSD is reduced hippocampal volume.
  - The hippocampus is implicated in the control of stress responses, memory, and contextual aspects of fear conditioning.
  - Prolonged exposure to stress and high levels of glucocorticoids (cortisol) damages the hippocampus
  - Hippocampal volume reduction in PTSD may reflect the accumulated toxic effects of repeated exposure to increased glucocorticoid levels (Flat or Furious)
  - Decreased hippocampal volumes might be a pre-existing vulnerability factor for developing PTSD.

HPA-Axis Dysfunction Changes in Brain Structure – Amygdala

The amygdala, region of the brain primarily associated with emotional processes. The name *amygdala* is derived from the Greek word *amygdale*, meaning "almond," owing to the structure's almondlike shape. The amygdala is located in the medial temporal lobe, just anterior to (in front of) the hippocampus. Similar to the hippocampus, the amygdala is a paired structure, with one located in each hemisphere of the brain. The amygdala is part of the limbic system, a neural network that mediates many aspects of emotion and memory. Although historically the amygdala was considered to be involved primarily in fear and other emotions related to aversive (unpleasant) stimuli, it is now known to be involved in positive emotions elicited by appetitive (rewarding) stimuli.



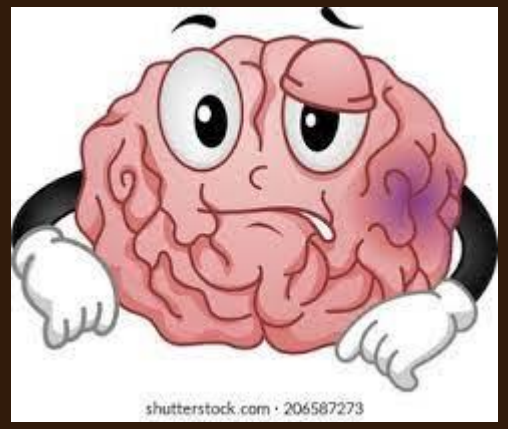
- The amygdala is a limbic structure involved in emotional processing and is critical for the acquisition of fear responses.
  - Functional imaging studies have revealed hyper-responsiveness in PTSD during the presentation of stressful scripts, cues, and/or trauma reminders.
  - PTSD patients further show increased amygdala responses to **general emotional stimuli that are not trauma-associated**, such as emotional faces.
- So clients with PTSD may be more emotionally responsive across the board (dysregulation)

HPA-Dysfunction impact healthy development of neurobiological systems

Changes? In Brain Structure

- Early adverse experience, including prenatal stress and stress throughout childhood, has profound and long-lasting effects on the development of neurobiological systems, thereby "programming" subsequent stress reactivity and vulnerability to develop PTSD.
  - Adult women with childhood trauma histories exhibit sensitization of both neuroendocrine, and autonomic stress responses

HPA-Axis Dysregulation -- In summary



- A variety of changes take place in the brains and nervous systems of persons with PTSD
- Pre-existing issues causing hypocortisolism (the brain has already down regulated) increases the likelihood of the development of PTSD
- This points to the importance of prevention and early intervention of adverse childhood experiences
- People with hypocorticolism may or may not have PTSD
- Hypocorticolism sets the stage for the Flat and the Furious → toxic levels of glutamate upon exposure to stressors → reduction of hippocampal volume persistent negative brain changes

- People with PTSD are more reactive to emotional stimuli, even stimuli unrelated to trauma
- Hypocorticolism results when the brain perceives that continued effort is futile.
  - Feelings of "fatigue" set in (akin to reduced stress tolerance)
  - Reducing fatigue can be accomplished, in part, with psychological factors including
    - Motivation/Knowledge of "competitors"
    - Feedback (frequent successes)

HPA-Axis Dysregulation -- In summary

Summary

- 46% of people in the US are exposed to adverse childhood experiences. (Early Intervention)
- Instruction in skills to handle emotional dysregulation
  - Mindfulness
  - Vulnerability prevention and awareness
  - Emotion Regulation
  - Distress Tolerance
  - Problem Solving
- Of those exposed to trauma, education about and normalization of heightened emotional reactivity and susceptibility to PTSD in the future may be helpful