Post Traumatic Stress Disorder

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Overview Of PTSD

I. Epidemiology and Risk Factors

II. Conceptualization of PTSD as a psycho-neurobiological disorder

III. Brief overview of DSM-V criteria for PTSD

IV. Discuss evidenced-based treatments for PTSD
Epidemiology of PTSD
National Comorbidity Study

- 7.8% of adults in the U.S. (lifetime)
- Type of trauma most often the basis for PTSD:
  - rape in women (46% risk)
  - combat in men (39% risk)
- 1/3 of cases have duration of many years
- 88% of cases have psychiatric comorbidity

Likelihood of getting PTSD after Experiencing a Trauma

It depends on the event and the person

Men experience more traumatic events

Women are more likely to develop PTSD

After a traumatic event, who gets PTSD?

- 20% of women
- 8% of men get PTSD

Kessler et al., 1995
Combat-Related PTSD: Epidemiology

Lifetime Prevalence:

- 30% in Vietnam veterans
- 5-10% of Gulf War I deployed veterans
- 10-20% in Operation Enduring Freedom and Operation Iraqi Freedom


Likelihood of PTSD....

Rape
- Men 65%
- Women 45%

Combat
- Men almost 40%

Physical Abuse
- Almost 50% of women
- 20%+ men
Risk for PTSD: After the Trauma

Degree of Social Support

Degree of Life Stress
What puts you at risk for PTSD?

Being female

Being poor

Less education

Bad childhood

Previous psychological problems
What puts you at risk for PTSD?

- Severity of trauma (ie, threat, duration, injury, loss)
- Prior traumatization
- Ethnicity
- Prior mood and/or anxiety disorders
- Family history of mood or anxiety disorders
What puts you at risk for PTSD?

*Strength or severity of the stressor

Characteristics of the trauma:

- Greater perceived life threat
- Feeling helpless
- Unpredictable, uncontrollable
Neurobiological Correlates of PTSD
Hypothalamus → CRH → Anterior Pituitary → ACTH → Adrenal Cortex → CORT

Corticotropin Releasing Hormone (CRH)

Anterior Pituitary

Adrenocorticotropic Hormone (ACTH)

Adrenal Cortex

HPA Axis

Negative Feedback
Cortisol in PTSD

Persistently low, with spikes during times of stress

A relatively small stressor to most people will trigger a biochemical cascade in someone with PTSD, manifesting as general hyper-reactivity and avoidant numbing, respectively.

No other emotional condition, including depression, panic attacks, or anxiety disorders will produce this profile.
Neuropeptide Y
The NPY system in stress, anxiety and depression.

Increased emotionality is seen upon inactivation of NPY transmission, while the opposite is found when NPY signaling is made overactive.

The most extensive evidence available for amygdala and hippocampus, some evidence for regions within the septum, and locus coeruleus.

Antistress actions of NPY are mimicked by Y1-receptor agonists. Blockade of Y2 receptors produces anti-stress effects.
NPY vs CRF

<table>
<thead>
<tr>
<th>NPY</th>
<th>CRF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety ↓</td>
<td>Anxiety ↑</td>
</tr>
<tr>
<td>Reward pathway</td>
<td>Stress response</td>
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BNST (Bed nucleus of the stria terminalis) acts as a scale to create a balance of CRF and NPY
Pathways

Wakefulness

Reward

Orexin (hypothalamus)

Nucleus accumbens

Ventral tegmental area (dopamine)

Raphe nuclei (serotonin)

Locus coeruleus (norepinephrine)
Norepinephrine
GABA neurotransmitter system

**Actions at GABAA Receptors**
Glutamate Receptors
Ghrelin qualifies as an orexigenic hormone.

It is produced by X/A-cells of oxyntic glands, abundantly present in the mucosal layer of the fundus region of the stomach.

Ghrelin is produced in small quantities in other parts of the digestive tract. It is also produced in the pancreas, in ghrelin neurons in the hypothalamus, in glomeruli of the kidney and in syncytiotrophoblast cells of placenta.
which ghrelin affects the NPY/AgRP neurons in the arcuate nucleus: the one produced by the stomach or by ghrelin-containing neurons in the hypothalamus?

**Problems:**

- very little ghrelin is transported across the blood-brain barrier in the direction of blood-to-brain: how does it reach its receptor?
- vagotony prevents ghrelin-mediated appetite

©the blood-brain barrier
Ghrelin

stress-related increases in circulating ghrelin, a peptide hormone, are necessary and sufficient for stress-associated vulnerability to exacerbated fear learning and these actions of ghrelin occur in the amygdala.
Functional Neuroanatomy of Traumatic Stress

- **Amygdala**: Extinction to fear through amygdala inhibition
- **Hippocampus**: Long-term storage of traumatic memories
- **Pituitary**: Output to cardiovascular system
- **Hypothalamus**: CRF, ACTH
- **Cerebral Cortex**: Glutamate
- **Parietal Cortex**: Stress, Attention and vigilance - fear behavior, Dose response effect on metabolism
- **Adrenal**: Cortisol
- **Locus Coeruleus**: NE

Other areas mentioned:
- **Prefrontal Cortex**
- **Orbitofrontal Cortex**
- **Cortisol**
- **Cerebral Cortex**
- **NE**
Functional Neuroanatomy of PTSD

- HIPPOCAMPUS
- MEDIAL PFC & Ant Cingulate
- AMYGDALA
Fear Circuitry Brain Structures

- **Amygdala**
  - Threat detection and fear conditioning
    - Exaggerated activation in response to trauma-related memories
    - Exaggerated activation for non trauma-related stimuli
    - Activation positively related to PTSD symptom severity

- **Medial Prefrontal Cortex**
  - Extinction (learn stimuli no longer aversive)
    - Anterior Cingulate Cortex (rACC): Diminished activation in PTSD

- **Hippocampus**
  - Memory encoding (e.g., context during fear conditioning)
    - Diminished activation in PTSD and lower hippocampal volumes
The dimensions interact with one another and combine in different ways to result in PTSD.

**Biological Dimension**
- Sensitized autonomic system
- HPA axis dysfunction
- Hippocampus atrophy
- Sensitized neural circuit

**Sociocultural Dimension**
- Low socioeconomic status
- Gender differences
- Immigration/refugee status

**Psychological Dimension**
- Pre-existing anxiety or depression
- Cognitive skill level
- Meaningfulness of trauma

**Social Dimension**
- History of childhood neglect or abuse
- Lack of social support
- Social isolation
“Just when I thought I knew what I was doing it all changed again…”

Not so much, really.
Main Changes in DSM-V for PTSD

PTSD moved from the anxiety disorders to a new class, “trauma and stressor-related disorders”

Definition of “trauma” slightly changed
- No longer need “fear, helplessness, or horror” (A2)
- Types of trauma (A1) somewhat narrowed (no longer can include unexpected death of family/close friend due to natural causes)
Main Changes in DSM-V for PTSD

- The 3 clusters of DSM-IV are now 4 clusters:
  - Intrusions
  - Avoidance
  - Negative alterations in cognitions and mood
  - Alterations in arousal and reactivity

- New subtype: with dissociative symptoms
Screening Questions for PTSD

“What’s the worst thing that ever happened to you?”
“How did you react when it happened?”
“Do memories of ______ still bother you? Did you get over it?”
“Do you avoid situations that might remind you of _____? Have your relationships suffered because of _____?”
“Have you become more nervous since ____? Is it hard for you to relax because of _____?”
PC-PTSD Screening

Brief, 4 item Screen for Primary Care
Does not ask patient the traumatic event
Asks Y/N symptoms in the past month
  Nightmares, Intrusive thoughts, On guard or easily startled, Feeling detached
Cut off score of 3 recommended

- Sensitivity
  - Women: .70, Men: .94

- Specificity
  - Women: .84, Men: .92

©Prins, et al. (2003). The primary care PTSD screen (PC-PTSD)
Longitudinal Course of PTSD Symptoms

Treatments for PTSD
Why PTSD Victims Might Be Resistant to Getting Help

Sometimes hard because people expect to be able to handle a traumatic event even on their own

People may blame themselves

Traumatic experience might be too painful to discuss

Some people avoid the event all together

PTSD can make some people feel isolated making it hard for them to get help

People don’t always make the connection between the traumatic event and the symptoms; anxiety, anger, and possible physical symptoms

People often have more than one anxiety disorder or may suffer from depression or substance abuse
Psychiatric Comorbidities

88% of men and 79% of women with PTSD meet criteria for another psychiatric disorder.

Men: alcohol abuse/dependence; MDD; conduct disorders; drug abuse/dependence.

Women: MDD; simple phobias; social phobias; and alcohol abuse/dependence.

U.S. Department of Veteran Affairs, National Center for PTSD
Trauma Affects Personality

Difficulty trusting

Persistent sense of shame

Unstable relationships

Borderline Personality Disorder

Prefrontal cortex damage:

- impulsivity, poor planning and judgment
During a Traumatic Event

Norepinephrine- Mobilizing fear, the flight response, sympathetic activation, consolidating memory

Too much = hypervigilence, autonomic arousal, flashbacks, and intrusive memories

Serotonin- self- defense, rage and attenuation of fear

Too little = aggression, violence, impulsivity, depression, anxiety
Treatment

Individual Therapy

Group Support (especially for Chronic PTSD)

Medication
Psychological Treatments for Chronic PTSD

Psychotherapy
- Exposure therapy
- Cognitive processing therapy
- Anxiety management

Additional treatments
- Eye Movement Desensitization and Reprocessing (EMDR)
- Hypnotherapy
- Psychodynamic therapy
- Expressive therapies
Traumatic Disorders

Treatments for PTSD

- Antidepressants
Medications

SSRIs – Sertraline (Zoloft), Paroxetine (Paxil), Escitalorpram (Lexapro), Fluvoxamine (Luvox), Fluxetine (Prozac)

Affects the concentration and activity of the neurotransmitter serotonin

May reduce depression, intrusive and avoidant symptoms, anger, explosive outbursts, hyper arousal symptoms, and numbing

FDA approved for the treatment of Anxiety Disorders including PTSD
Functional Neuroanatomy of PTSD

MEDIAL PFC & Ant Cingulate

HIPPOCAMPUS

AMYGDALA
BDNF

Brain-derived neurotrophic factor (BDNF) is a 25-kDa CNS protein implicated in neuronal cell growth and differentiation.
5-HT and BDNF-regulated intracellular signal transduction pathways
Adrenergic-Inhibiting Agents: 
*Alpha1-Adrenergic Blockers*

Prazosin* 7 to 15 mg qhs

Alpha1- post-synaptic adrenoceptor receptor antagonist

Alpha1 receptors widely distributed in the brain, including the amygdala and hippocampus

Alpha1 receptors modulate sleep and startle responses

Double-blind RCT in 40 veterans, 13.3 +/- 3 mg 1
- Robust improvement in sleep quality and distressing dreams
- Medium to large effect size in each PTSD Sx cluster
- Adverse reactions include: syncope, dizziness, drowsiness, decreased energy, headache

*Not FDA approved for the treatment of PTSD

Treatment for Children

FDA approved Prozac for depression in children

FDA approved Zoloft for OCD in children

Cognitive-Behavioral therapy- exposure, anxiety management, Cognitive restructuring

Play Therapy
Conclusions

Many of our patients are suffering from unrecognized trauma

They most likely will not tell us unless we ask the right questions, at the right time, in the right way

If they don’t have the words to tell us, we have to help them find the words

When they are ready to tell us their stories, we have to be willing to hear them
Trauma Affects Language

Alexithymia: Inability to verbally describe emotions

The “I was so upset I couldn’t think straight” phenomenon, magnified.
Prevalence of Trauma and Probability of PTSD


![Prevalence of Trauma](chart1.png)

![Probability of PTSD](chart2.png)
Exposure Therapy- Education about common reactions to trauma, breathing retraining, and repeated exposure to the past trauma in graduated doses. The goal is for the traumatic event to be remembered without anxiety or panic resulting.

Cognitive Therapy- Separating the intrusive thoughts from the associated anxiety that they produce.

Stress inoculation training- variant of exposure training teaches client to relax. Helps the client relax when thinking about traumatic event exposure by providing client a script.