Stress Biology: Basic Overview

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Introduction

- Important issue in basic and clinical neuroscience research
- Fundamental to survival
- Strongly related to several brain disorders
- Crucial to provide more meaningful advances in the stress field
    - Translational approach, integrating basic knowledge and clinical practice.
- Describe the history of stress research and stress basic concepts, explore the complex Neuroanatomy, and time domains of the stress response.
- Raises clinical implications from these concepts and discusses future research

What is Stress?

- Internal state can be caused by physical demands on body or by environmental and social situations that can be potentially harmful
- Physical, environmental, and social cause of stress states= stressors
- Hallmark of stress response is the activation of the ANS and HPA axis
    - Excessive adrenocortical and autonomic function is deleterious
- Brain- key organ of the stress response because it determine what is stressful and controsses responses to potentially stressful experiences

History of Stress Research

- Canon coined the term homeostasis
  - His study of different responses to different stimuli which reacts differently in face of a stressful situation
- Many contemporary culture regard illness as the product of its environment
- Stress was coined by Hans Selye (1907) and invoked the adrenocortical system as the crucial responder to stress
- Any novelty or perturbation of the system was associated with an elevation of adrenocortical activity
- Another pathway: the sympathetic nervous system

Stress Responses

- Behavioral Responses
- Emotional Responses
- Cognitive Responses
- Biochemical Responses
- Physiological Responses
- Physical Responses

Development of Resilience

- Stress can cause a temporary activation of circuits– when the stressor is removed
- Circuit is unprovoked, no systems are produced
- Certain types of stress (childhood) can sensitive brain circuits
- Higher rates of depression
- Brain can also avoid producing symptoms
- Short-term stress can develop resilience to stress, not as many symptoms

Vulnerability of Genes

- Long genotype of SERT is more resilient
  - Less amygdala reactivity to fearful faces
  - Less likelihood for depression
  - Higher likelihood for tolerating SSRIs
- Short genotype of SERT is more likely to develop an affective disorder
  - More cognitive symptoms
  - Less responsive to tolerance to SSRI treatment
- Dopamine System
- Functional Polymorphism

Physiologic Responses

- Stressor disrupts an organism’s equilibrium
  - Bring about an adaptive response
  - Restoring homeostasis
- Immune Changes to Stress
  - Psychoneuroimmunology
  - Biological Connections
  - Clinical Implications
  - Cytokine Influences
  - CNS and Immune System

Neurotransmitter Response

- Activate noradrenergic system s in the brain
- Activate serotonergic system
- Increases dopaminergic neurotransmission
- Amino Acid and Peptidergic Neurotransmitters

Endocrine Response to Stress

- Secretion of CRH from the Hypothalamus
  - Anterior pituitary to trigger release of ACTH
- ACTH acts as the adrenal cortex to stimulate the synthesis
- Glucocorticoids
  - Promotes energy utilization
  - Increases cardiovascular activity
  - Inhibits functions like growth, reproduction, and immunity
- Major depression has been recognized as associated with overactivity of the HPA axis– with elevated cerebrospinal CRH levels

Stress Response

SHORT-TERM:

- Increased heart rate
- Increased blood pressure
- Dilation of bronchioles
- Changes in blood flow pattern
- Converts glycogen to glucose
- Increased metabolic rate

LONG-TERM:

- Increased blood volume and blood pressure
- Retention of sodium and water
- Converts to glucose
- Increased blood sugar
- Suppression of ecosystem

Acute Stress and Immunity

- Acute stressors produce changes in the immune system due to immunoregulatory cells
  - Elicit decreases in cellular immune responses and increases in markers of inflammation
  - Among depressed patients– acute stress leads to increases of inflammatory cytokine activity
  - Also at a greater risk for IL-6 and other proinflammatory markers because of acute sleep loss as a reaction to stress

Chronic Stress and Immunity

- Associated with reliable decreases of cellular and innate immunity
  - Increases in proinflammatory cytokine activity
- Both depressed and stressed people show declines of cellular response to shingles and impairments in responses to vaccines
- Immune activation leads to changes of peripheral physiology and behaviors that are similar to a stress response
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Summative Video