Learned Helplessness, Depression and Control

Professor Ian Robertson
Course Outline

• Control: definitions and development
• **Learned helplessness, depression and control**
• Status Syndrome – socioeconomic factors in health
• Cognitive control – mechanisms of attentional control
• Neuroimaging and neuropsychological correlates of control
• Neurochemistry and neurophysiological covariates of control
• Molecular genetics and evolution of control
• The biology of social dominance
• Implicit processing in dominance hierarchies
• Lifespan development and control
• Control and personality
LEARNED HELPLESSNESS, DEPRESSION AND CONTROL
Clinical Diagnosis of Major Depressive Episode

- 5 or more of the following nearly every day for 2 weeks
  - Depressed mood
  - Diminished interest or pleasure in all or nearly all activities
  - Decreased appetite, weight loss in the absence of dieting, or weight gain
  - Insomnia or hypersomnia
  - Psychomotor agitation or retardation
  - Fatigue or loss of energy
  - Diminished concentration or indecisiveness
  - Thoughts of death, suicidal ideation (with or without a plan)
Scope of the Problem

- One of the most common health conditions in the world
  - 4.4% of the total overall disease burden (similar to heart disease and diarrheal illness)\(^2\)
- Prevalence in the United States is 5.4-8.9 percent.\(^4\)
- Lifetime incidence in the United States\(^1\)
  - 12% in men
  - 20% in women
The symptoms of depression

<table>
<thead>
<tr>
<th>Emotional Symptoms Include:</th>
<th>Physical Symptoms Include:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sadness</td>
<td>Vague aches and pains</td>
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<tr>
<td>Loss of interest or pleasure</td>
<td>Headache</td>
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<tr>
<td>Overwhelmed</td>
<td>Sleep disturbances</td>
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<tr>
<td>Anxiety</td>
<td>Fatigue</td>
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<tr>
<td>Diminished ability to think or concentrate, indecisiveness</td>
<td>Back pain</td>
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<tr>
<td>Excessive or inappropriate guilt</td>
<td>Significant change in appetite resulting in weight loss or gain</td>
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Serotonin$^{5HT}$ and Norepinephrine$^{NE}$ in the brain

Prefrontal Cortex

Raphe Nuclei (5-HT source)

Locus Ceruleus (NE Source)

Limbic System

Cooper JR, Bloom FE. *The Biochemical Basis of Neuropharmacology*. 1996.
There are at least two sides to the neurotransmitter story

Functional domains of Serotonin and Norepinephrine

- **Serotonin (5-HT)**
  - Sex
  - Appetite
  - Aggression

- **Norepinephrine (NE)**
  - Depressed Mood
  - Anxiety
  - Vague Aches and pain
  - Irritability
  - Thought process
  - Concentration
  - Interest
  - Motivation

- **Both serotonin and norepinephrine mediate a broad spectrum of depressive symptoms**

References:
... CHICKENS AND EGGS...
THE HPA AXIS

1. Hypothalamus
   - Releasing factor
   - Anterior pituitary
     - ACTH (through blood)
2. Adrenal cortex
   - Cortisol
PATHOPHYSIOLOGY OF STRESS

CORTISOL

ADRENALINE

INCREASED BLOOD SUGARS
STRESS EFFECTS ON THE BODY

STRESS HORMONE RELEASED – CORTISOL

INCREASED HR, BP, IMMUNE SYSTEM

MEMORY IS SHARPER

ABILITY TO DEAL WITH ACUTE STRESS
THE FEEDBACK SYSTEM OF STRESS

- Desensitisation of cortisol receptors leads to disturbances in noradrenaline and serotonin transmission.
- Hypersecretion of CRF.
- Impaired negative feedback by cortisol.
- Adrenal hypertrophy.
- Excess cortisol released.
- Circulation.
- Cortisol.
- Impaired negative feedback by cortisol.
Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene

Avshalom Caspi,¹,² Karen Sugden,¹ Terrie E. Moffitt,¹,²* Alan Taylor,¹ Ian W. Craig,¹ Honalee Harrington,² Joseph McClay,¹ Jonathan Mill,¹ Judy Martin,³ Antony Braithwaite,³ Richie Poulton³

Science, 2003
Learned Helplessness

• First psychological model of depression
Contingency

- controllability of reinforcers
- a strong contingency between the response and a reinforcer means the response controls the reinforcer
- most of the research has focused on control over aversive stimulation
- contemporary research originated with studies by Seligman and colleagues
- they investigated the effects of uncontrollable shock on subsequent escape-avoidance learning in dogs
- the major finding was that exposure to uncontrollable shock disrupted subsequent learning
- this phenomenon called the learned-helplessness effect
The learned-helplessness effect

Experiment by Seligman and Maier (1967)
- demonstrated the basic LH effect
- 3 groups of dogs

Phase 1:

Group 1: Escape
- restrained and given unsignaled shock to hindfeet
- could terminate the shock by pressing either of 2 panels on either side of snout

Group 2: Yoked
- placed in same restraint and given same # and pattern of shocks
- could not terminate the shocks by pressing the panels; they were shocked whenever Escape animals were shocked

Group 3: Control just put in restraint
The learned-helplessness effect

Phase 2:
- all dogs were treated alike
- put in a 2-compartment shuttlebox and taught a normal escape/avoidance reaction
- dogs could avoid shock by responding during a 10-s warning L or escape shock once it came on by jumping to other side of compartment
- if subject did not respond in 60 s, the shock was terminated

Thus, the experiment tested whether prior inescapable shock affected escape/avoidance learning
The learned-helplessness effect

Results:

The Escape group learned as easily as the Control group.

The Yoked group showed an impairment.

This deficit in learning is the learned-helplessness effect.
The learned-helplessness effect

- the yoked group received the same number of shocks as the escape group, so the failure to learn is not simply due to having received shock in phase 1

- rather, the failure to learn was due to the inability to control shock in phase 1

- no matter which response they performed, their behavior was unrelated to shock offset in phase 1

- according to Seligman and Maier, the lack of control in phase 1 led to the development of the general expectation that behavior is irrelevant to the shock offset

- this expectation of lack of control transferred to the new situation in phase 2, causing retardation of learning
Learned Helplessness
Consequences

• Decreased effort and persistence
• Reduced learning
  – Belief that outcomes do not depend on behavior
  – Reduced learning of future behavior–outcome
    contingencies
  – Behavior–outcome important for self-efficacy

* Negative affect – depression, stress response.
Human Learned Helplessness

• Diener & Dweck (1978)
  • Problem solving – make guesses and determine hypothesis
  • Given authentic (can use feedback to determine hypothesis) vs. random feedback (cannot determine hypothesis)
  • New task – people given authentic feedback tried harder and did much better
Learned helplessness in humans: Critique and reformulation.

- Abramson, Lyn Y.; Seligman, Martin E.; Teasdale, John D. Journal of Abnormal Psychology. Vol 871978, 49–74

- *Two problems with learned helplessness:*

  (a) It does not distinguish between cases in which outcomes are uncontrollable for all people and cases in which they are uncontrollable only for some people (universal vs personal helplessness), and

  (b) it does not explain when helplessness is general and when specific, or when chronic and when acute
Learned helplessness in humans: Critique and reformulation.

• A reformulation based on a revision of attribution theory is proposed

• According to the reformulation, once people perceive noncontingency, they attribute their helplessness to a cause.

• This cause can be
  – stable or unstable,
  – global or specific,
  – internal or external.

The attribution chosen influences whether expectation of future helplessness will be chronic or acute, broad or narrow, and whether helplessness will lower self-esteem or not.
Explanations of the LH effect

The learned-helplessness hypothesis

- based on the conclusion that animals can perceive the contingency between their behavior and the reinforcer
- so, the original theory emphasized the lack of control over outcomes
- according to this position, when the outcomes are independent of the subject’s behavior, the subject develops a state of learned helplessness which is manifest in 2 ways:
  - there is a motivations loss indicated by a decline in performance and heightened level of passivity
  - the subject has a generalized expectation that reinforcers will continue to be independent of its behavior
  - this persistent belief is the cause of the future learning deficit
The LH hypothesis has been challenged by studies showing that it is not the lack of control that leads to the LH outcome, but rather the inability to predict the reinforcer

- receiving predictable, inescapable shock is less damaging than receiving unsignaled shock
  - if inescapable shock is signaled, then see less learning deficit
  - if you present a cue that tells the animal the shock is coming, then see less learning deficit
  - animal still can’t escape the shock (i.e., still uncontrollable), but they know its coming

- presentation of stimuli following offset of inescapable eliminates the LH deficit
  - this was demonstrated in an experiment by Jackson & Minor, (1988)
Jackson & Minor (1988)  
4 groups of rats  

**Phase 1:**  

**Escape Group:**  
- received unsignaled shock that rats could terminate by turning a small wheel  

There were 2 Yoked groups  

**Feedback Group:**  
- house-light was turned off for a few seconds when shock ended  

**No-Feedback Group:**  
- no stimulus was given when shock was turned off  

**No-Shock Control Group:**  

**Phase 2:**  
- all rats trained in a shuttlebox where they could run to other side to turn off shock
Results:

Escape and No shock groups performed better than the Yoked Group – this is the typical LH effect.

Yoked/feedback group learned as well as the Escape and No shock groups.
The Jackson & Minor (1988) experiment demonstrated that receiving a feedback stimulus following shock offset eliminated the typical learning deficit.

So, the learning deficit is not due to simple lack of control, as suggested by the LH Hypothesis, but rather, it is due to a lack of predictability.

But this takes us later to the question of safety signals.
Safety Signals– *Learned Safety*

- Instinctive and learned fear are essential for survival
- pathological forms of learned fear cause anxiety disorders, posttraumatic stress disorders, and can lead to depression
- Do effective inhibitory constraints exist that prevent the inappropriate expression of learned fear?
- Conditioning
Classical Conditioning

UCS – meat powder – UCR salivation
Neutral stimulus bell no effect
Pair CS and UCS several times
CS bell
CR salivation
Conditioned Inhibition 1

- a learning paradigm whereby a neutral CS develops the ability to inhibit responses to learned predictors of aversive or rewarding stimuli (Pavlov, 1927; Rescorla, 1969)
- Fear conditioning – a positive correlation (pairing) of a previously neutral CS and an aversive US.
- During conditioned inhibition a CS that is negatively correlated (explicitly unpaired) with an aversive US becomes a positive signal (predictor) for safety and reduces the expression of conditioned fear.
Conditioned Inhibition 2

• Since the animal associates the target signal with protection from an impending aversive event, conditioned inhibition may form of learned safety

• a process by which the animal learns to take advantage of sources of safety and security in the environment

• The term “safety signal” generally refers to a stimulus that is inversely or negatively correlated to an aversive event
Learned Safety – can it help prevent depression?

• Can learned safety, as a predictor of a break from continuously imminent, stress-producing danger, have antidepressant effects?

• Could learned safety be one of the mechanisms by which CONTROL has its beneficial effects?

• See Jackson and Minor (1988) study above: predictability may afford signals of safety
Learned Safety – *Pollack et al Neuron 2008*

- **Safety conditioning**: delivery of four shock US is followed by the presentation of four tone CS.
- **Fear conditioning**: the number of CS and US presentations is matched to the safety conditioning paradigm (four paired CS–US).
- Training over 3 days, one session per day.
- A memory recall test, consisting of a single CS presentation, is carried out 24 hr after the last training day.
Learned safety in the brain

Conditioned safety reduces anxious behaviour – ‘freezing’
• CS in safety group reduces ‘anxiety’
• CS in fear conditioning group increases ‘anxiety’
• The signal is specific – another tone does not reduce anxiety.
Conditioned safety reduces anxious behaviour – ‘freezing’

- Safety group explore more in innately threatening ‘open’ situations.
- Fear group explore less in these
- Vice versa for innately ‘secure’, ‘closed’ situations
- Think of possible comparisons with agoraphobia
Conditioned safety has ‘antidepressant’ effects

• A Mice show less ‘despair’ (stopping swimming in forced swim test) in presence of conditioned safety signals

• B Size of the effect similar to effect of antidepressant

• C 4 weeks of stress (uncontrollable mild chronic stress) INCREASED learned safety response

• D Sugar preference measure of ‘anhedonia’ (key feature of depression): this normalised in chronically stressed mice in presence of safety signals
Learned Safety Promotes the Survival of Newborn Cells in the Dentate Gyrus of the Hippocampus
Learned Safety Leads to Increased Expression of BDNF in the Dentate Gyrus of the Hippocampus
Diener et al. (2009) Psychological Medicine

Normally enhanced frontal PINV during unexpected change from an escape paradigm to uncontrollability.

Also during unpredictable response outcome contingencies in general.

Mild electric shock to finger – could avoid by button press.

Unexpectedly, schedule changes so that response has no effect then changes back.
Caveats

• Over-control
  – Eg anorexia nervosa

• Overestimation of control (Illusion of Control)
  – Illusory control – depressed people more realistic, eg in gambling tasks.