PAIN PERCEPTION: THE MIND BODY CONNECTION

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disclosures

I have no conflicts of interest
objectives

- Understand pain as perception.
- Highlight the anatomic connections between the sensory and affective components of pain.
Pain is a sensation caused by injury or harm

- Differences to same therapy
- Failure of interventions
- Poor association between disability + impairment

**the biological model**

*does NOT explain:*
the psychological model

Limitations:
- Does not account for inherited and cognitive factors in the learning process
- Suggests that pain in absence of pathology indicates psychological issue
the gate theory

Adapted from Wall & Melzack (1965) (Beynon, 2013)
bio-behavioral model

- An interplay of pre-disposition + biology + psychology
- Integrates appraisal + behaviors + new social roles
- Explains the inter-individual variability of pain expression.
- It is suffering > pain that brings patients to the doctor.

Adapted from Loeser's model of pain
“Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”

IASP 1975
“It is essential to remember that stimulation of receptors does not mark the beginning of a pain process. Rather stimulation produces neural signals that enter an active nervous system which is already the substrate of past experience, culture, anxiety and so forth. These brain processes actively participate in the selection, abstraction and synthesis of information from the total sensory input. Pain is not the end product of a linear sensory transmission system. Rather it is a dynamic process that involves continuous interactions among complex ascending and descending systems.”

- R. Melzack 1986
Pain = perception

- A *perceptual* phenomenon that requires a conscious being
- Complex neural events modulate efferent and afferent phenomena
- The report of pain includes suffering: loss of control, helplessness and intolerability
<table>
<thead>
<tr>
<th>Factors that increase pain perception</th>
<th>Sensory factors</th>
<th>Cognitive factors</th>
<th>Emotional factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injury, inactivity, body mechanics</td>
<td>Focus on pain, worrying, negative thoughts</td>
<td>Depression, anger, anxiety, stress, frustration</td>
<td></td>
</tr>
</tbody>
</table>
the pain matrix

- Widely distributed
- Creates a multidimensional experience of pain
- Is accessible by noxious and non-noxious stimuli
**insula**
- Regulates emotions, cognition, memory, regulation of ANS
- Involved in empathy, hypnotically induced pain & pain recall

**pfc**
- Regulates cognitive evaluation / behavioral expression of pain
- In successful reappraisal dLPFC suppresses amgydala

**amygdala**
- Fear conditioning and aggression
- Responsible for intense emotions
- **emerging info**
medial and lateral systems

**Medial system**
- **Emotional aspect of pain, outcome evaluation**
  - Anterior insula, PFC, amygdala, PAG, OFC, ACC
  - Lamina 1, 5, deep lamina

**Lateral system**
- **Sensory aspect, cognitive control and discrimination**
  - S1, SII, posterior insula
  - Lamina 1 and 5
emotions and pain

**Negative**: depression, anxiety, anger, fear
- Predicts chronicity and intensity of pain
- Hippocampus, amygdala and insula

**Positive**: Resilience during chronic pain
- Facilitates cognition, coping, flexibility, problem solving.
- Insula, Para-hippocampal gyrus, thalamus, amygdala
Sensitive subjects - robust pain induced activation of 1° somato-sensory cortex, ACC and PFC than the “less sensitive patient” Coghill 2003

N = 17
Reinterpreting significance of adverse events helps control negative emotions.

Perceived control changes emotional appraisal of pain. → *attenuates* pain and pain-directed anxiety.
self vs. external control of pain

greater activation during self-control vs. with external control

greater activation during voluntary reappraisal

N= 12  

Wiech, Katja et al. 2006
Anticipation and pain

- Anticipation of pain activates anterior insula, posterior cerebellum and prefrontal cortex.

- Pain activates mid insula, anterior cerebellum and anterior cingulate cortex.

N=12  Ploghaus et al 1999
Activation in bilateral ACC, insular cortex, brainstem, and cerebellum correlates with empathy scores.

Singer et al 2004 Science
placebo effect

- pain = physiology + psychology
- involves high order cognitive networks (altering expectations and beliefs) endogenous opioid systems (higher levels of endogenous opioids)
- hypothalamus, amygdala, PAG and brainstem play a role in placebo analgesia.
- dLPFC initiates placebo analgesia
- Opioid and placebo analgesia show increase activity in the rACC.

Petrovic et al 2002, 2010
genetics and pain

Variant gene encoding *Catechol-O-methyl transferase* → **MORE PAIN**

- Greater perception of pain
- Decreased release of endogenous opioids.

Variant gene encoding *Melanocortin 1 receptor* = → **LESS PAIN**

- Higher pain threshold
- Increased analgesia from opioids

Quantitative trait locus (QTL) mapping localizes genes responsible for variability in nociception.
summary

• There is **incongruence between sensation and perception**.

• There is **context dependence** between feeling and reporting pain, influenced by emotions, motivation and behaviors.

• The complex **pain matrix** helps us understand connections between sensory and affective components of pain and explains the multidimensionality of pain.

• We must move beyond simple spatial representation of pain to better understand the dynamic nature of the pain experience.
Understand pain as perception.

Highlight the anatomic connections between the sensory and affective components of pain.