BIO-PSYCHO-SOCIAL MODELS of PAIN

Patrick J. McGrath and Anita M. Unruh

“Science Helping Children”
WHO WE ARE?

• Patrick McGrath OC, PhD, FRSC, FCAHS
  • Professor, Psychology, Pediatrics & Psychiatry & Canada Research Chair, Dalhousie University
  • Integrated VP, Research and Innovation, IWK Health Centre and Capital District Health Authority

• Anita Unruh, BScOT, MSW, PhD
  • Professor of OT, Health and Human Performance
  • Associate Dean, Research and Academics, Faculty of Health Professions, Dalhousie University
GOALS OF THIS WORKSHOP

• Understand roles of models or theories in pain
• Understand the models that are currently used
• Be ready to apply these models to clinical practice
DO YOU THINK MODELS OR THEORIES ARE USEFUL TO YOUR PRACTICE?

• If yes, Why?
• If no, Why not?
• What models do you use?
• NOTE: Theory is just an all encompassing model
WHY ARE MODELS OR THEORIES IMPORTANT?

• Facts or observations
  • isolated and don’t mean much on their own
  • Humans hardwired to make sense of facts
  • What facts are observed and how facts are interpreted influenced by models and theories

• Few all encompassing theories anymore
  • Modest attempts at models of specific issues
  • Best models are made to be proven wrong
    • Spur to new thinking
PSYCHOLOGY, BIOLOGY AND SOCIAL FACTORS

Usually not well integrated
PRIMARILY BIOLOGICAL MODELS

• Descartes
• Gate Control Theory
• Descending/Ascending control
• Neuromatrix
• Central sensitization
• Peripheral sensitization
Descartes proposed that pain was a linear transmission of input from the periphery through the spinal cord to the brain.

- Explains what happens if you put your hand on a hot stove element, jump away, shout with pain.
DESCARTE’S MODEL DID NOT EXPLAIN

• Why many minor injuries are identified much later
• Why some serious acute injuries may not be immediately painful
• Why pain may be perceived as occurring in a part of the body that doesn’t exist
• Why pain may be experienced in the absence of any identifiable tissue pathology
• Why non-pharmacological methods provide effective pain relief
GATE CONTROL THEORY

R. Melzack & P.D. Wall

(Adapted from Melzack & Wall, 1965)
GATE CONTROL THEORY OF PAIN – MELZACK & WALL (1965)

- Pain is not a function of one specific part or action of the nervous system but of many parts.
- Pain involves the spinal cord and other parts of the nervous system as a gating mechanism to inhibit or facilitate a noxious stimulus.
GATE CONTROL THEORY CONT’D

• Pain occurs when the number of nocioceptive impulses arriving at neural levels exceed a critical level.

• Gating mechanism can influence nocioceptive impulses before pain is felt.
GATE CONTROL THEORY CONT’D

• Biological, cognitive, behavioural, & affective factors interact to determine whether a stimulus is inhibited or facilitated
• The influence is from the periphery and from the brain
• No mechanisms for psychosocial influence specified
• widely distributed neural network that is genetically predetermined & modified by sensory experience
• Injury, pathology, & chronic stress prompt the neuromatrix & lead to activation of perceptual, homeostatic & behavioral programs
• Specific mechanisms not specified
MODERN CONCEPTUALIZATION OF PAIN

- Complex changes
  - periphery,
  - spinal cord
  - brain
- ascending information
- descending modulation
PAIN AND THE BRAIN

- Pain is seen in multiple areas of the brain
- The primary and secondary somatosensory cortex
- Insula
- Anterior cingulate
- Prefrontal cortices thalamus
  - but also brain stem, amygdala, cerebellum
- Projection into other areas
REORGANIZATION OF THE BRAIN

- The brain is reorganized in chronic pain
- Chronic pain patients light up other areas

Pain Sensitive

Pain Insensitive
DESCARTES FOR THE MODERN AGE

16th Century

Prior experiences
Attention/expectation
Mood (anxiety, depression)

Neurochemical and structural changes
Genetics

Sensitization (Peripheral and Central)

Pain Experience

Noxious stimulus

21st Century

Descending, top down modulation
Ascending, bottom up information
THE COMPONENTS OF THE PAIN SYSTEM

Nociceptors
  • respond to tissue-damaging or potentially tissue-damaging stimuli, noxious stimuli
  • In skin, bone, muscle, joint capsules, viscera, blood vessels, meninges, peripheral nerve sheaths
Inhibition of pain

- Endogenous opioids (ex. norepinephrine, serotonin)
- produced by and within nerve cells, prevent noxious stimuli from reaching consciousness
- decreased by prolonged pain, recurrent stress
- increased with brief pain, brief stress, massive trauma, exercise
“CHEMICAL SOUP”

Facilitation of Pain

• Released into tissue from cells because of injury,

• Activate nociceptors & promote sensitization

  • Eg. bradykinin, serotonin, histamine, prostaglandins, substance P, etc.
CHEMICAL SOUP CONT’D

• cause secondary changes unrelated to initial tissue damage

• change membrane permeability

• Change transmission of nerve impulses

• Spread sensitization in peripheral tissues

• signal tissue damage & begin repair
• Nerve fibers collect into peripheral nerves, travel to the spinal cord, synapse at the dorsal horn

• Depending on the type of fiber they divide into pathways to the brain
PAIN PATHWAYS

• Small & large diameter fibers
• Myelinated or unmyelinated
• Diameter and myelination impact transmission of noxious stimuli
BRAIN STRUCTURES INVOLVED IN PAIN

- Brainstem
- hypothalamus,
- thalamus,
- limbic structures,
- basal ganglia,
- cerebral cortex
Central Sensitization

- Persistent pain changes activity in the central nervous system
- Neuroplasticity of the brain means that the brain is responsive and changed by the stimuli it receives
IMAGINE WHAT IS GOING ON IN HIS BRAIN!
PERIPHERAL SENSITIZATION

- Increased activity of neurotransmitters/chemical mediators
- Inactive nociceptors activated (with ↑ spontaneous discharge rates, reduced thresholds, & higher discharge rates)
- Changes in characteristics of nerve fibers
- Facilitates transmission of noxious stimuli
Hyperalgesia – increased response to a stimulus which is normally painful

Primary hyperalgesia – in the zone of tissue damage

Secondary hyperalgesia – in the area beyond the site
HYPERALGESIA

Example

• Inject capsaicin into a hand
• Person experiences mechanical & thermal hyperalgesia in the area around the injection site
• Wider area beyond injection site also becomes sensitized
• Your examples
• Response to tissue damage is to withdraw & protect the area of injury

• primary & secondary hyperalgesia insures protective response to facilitate tissue repair
ALLODYニア

• pain due to a stimulus which is not normally painful
• E.g. light touch, heat
• Your examples...
EXTENT OF TISSUE DAMAGE PREDICTS PAIN

• Similar tissue damage ≠ similar pain
• Variability in peripheral activity is one factor
• Central factors important
• Psychosocial factors important
• Report of pain vs experience of pain
EXPERIENCE DEMONSTRATES:

• A noxious stimulus can originate in the periphery, be sent to the brain, but be blocked by the brain from being perceived as painful (e.g. traumatic injuries)

• The brain can initiate the process & produce pain in a limb even when there is no tissue damage in the limb, indeed no limb (phantom pain)
PAIN AS A WARNING SYSTEM

• Pain is an essential warning system
• Very robust
• Very redundant
• Almost impossible to ablate
PAIN FALSE ALARMS

- Phantom pain has no benefit
- “.. Most backaches, headaches, muscles pains, nerve pains, pelvic pains, & facial pains serve no discernible purpose, are difficult to treat, and are a disaster for the people who suffer them.” (Melzack, 2001, p. 1378)
CONCLUSIONS

• Biological, psychological, and social factors interact to inhibit or facilitate pain
• Infants and young children have a maturing and developing nervous system
• Response to pain is shaped by exposure, and learning, shapes perceptions and emotional response
PSYCHOSOCIAL MODELS OR THEORIES USED IN PAIN

- Biopsychosocial model
- Communication model
- Cognitive model
- Stress model
- Operant model
- Family Therapy
- Attachment
- Fear of pain
- Social modeling
- Catastrophizing
- Social ecological model
BIOPSYCHOSOCIAL MODEL

- “The dominant model of disease today is biomedical, and it leaves no room within its framework for the social, psychological, and behavioral dimensions of illness.”
- Major points
  - Biological, social and psychological factors are always important.
  - Each must be considered in every case no matter what the disease
- Not a testable model just says they are important
- Points direction to examining all variables

PSYCHOLOGY, BIOLOGY AND SOCIAL FACTORS

Usually not well integrated
BIOPSYCHOSOCIAL MODEL APPLIED TO PAIN

- Psychosocial factors are important in all types of pain.
- Even pain that has a well delineated physical cause.
- E.g. Needle procedures, Sickle cell disease, Migraine headache, cancer pain.
BIOPSYCHOSOCIAL MODEL

- All pain is physical: pain of every sort is related to brain activity which is beginning to be well mapped.
- All pain is psychological
- All pain is social
ALL PAIN IS BIOPSYCHOLOGICAL

Pain is processed in the brain

Stress, anxiety, depression hopelessness are always important

This is the brain of Patrick McGrath
ALL PAIN IS SOCIAL

• Pain is influenced by social behavior of parents, partners and peers.
• Eg Reassurance and criticism by parents makes pain worse
• Solicitousness
PHARMACOLOGIC INTERVENTIONS

• Are always psychological
• Vehicle for all drugs
  • matrix of social and individual situation
  • cannot be delivered in a vacuum
• vehicle influences pharmacodynamics
• You would never use a good drug with a toxic vehicle, would you?
MODELS AND PEDIATRIC PAIN

Who is involved?

Parents

Friends & sibs

School

Child

Community
A general framework for communication in pain
CAREGIVER MUST BE CONSIDERED

- Pain is a form of communication
- The receiver and the sender must be considered
  - Parent’s reassurance,
  - Spouse solicitousness
COMMUNICATION MODEL

• Alerts us to the role of the environment
• Gives general guidance
• Caregivers, partners, parents important
• A matrix of interactions
• No specific predictions
• No specific treatments
COGNITIVE MODELS OF PAIN

• Based on cognitive models of depression
  • Aaron Beck
• What we think influences our pain
• Chronic pain influences the development of cognitive styles
• The basis of cognitive therapy

Pain ↔ Thoughts
STRESS MODELS IN PAIN

Psychological Stress

Biological changes

Pain
STRESS MODELS
AKA DIATHESIS STRESS MODELS

• Interaction of a vulnerability and stress to produce pain
• Most widely used in recurrent pain especially headache, abdominal pain but also in musculoskeletal pain
• Stress may be psychological
  • E.g. demands at work, interpersonal stress
• Physical stress
  • E.g. Missing meals, bright light, exercise
• Combination of different stressors
EVIDENCE FOR STRESS MODELS

• Life events have a significant but small effect on pain
• Perception of life events somewhat stronger effects
• Specific individuals may be subject to specific stressors e.g. every time John has to do a presentation he gets a tummy ache
OPERANT MODEL

• Bill Fordyce 1923-2009
  • Reinforcement controls behavior
  • Pain behavior can be modified
    • Positive reinforcement
    • Negative reinforcement
    • Punishment
    • Extinction

• Used in conjunction with Applied Analysis of Behavior and more generally
EVIDENCE FOR AN OPERANT COMPONENT

- Parental solicitiousness
  - Widely studied (not always well studied) increases pain response
- Parental reassurance


FAMILY THERAPY

• Salvador Minuchin
• Mothers with children absent from school
  • “over involved”
  • exercise task
  • provided both encouragement and discouragement (Dunn-Geier et al, 1986)

• Few if any trials

MAJOR FEATURES OF ATTACHMENT
(AFTER BOWLBY)
ATTACHMENT MODELS

- Children with more ambivalent or controlling attachment had stronger pain reaction to immunization and everyday pain (Vignette study)
- Attachment models used in some clinics
- No good experimental data


FEAR/AVOIDANCE OF PAIN

Disuse
Disability
Depression

Avoidance/Escape, hypervigilance

Fear of movement, re/injury, pain

Catastrophising

Injury

Pain experience

Low Fear

Recovery

Exposure

Intervention points
PEDiatric Fear-Avoidance Model

Asmundson, Noel, Petter, Parkerson, Pediatric Fear-Avoidance Model of Chronic Pain: Foundation, Application, and Future Directions, in press, Pain Research and Management
INTERPERSONAL FEAR AVOIDANCE MODEL OF PAIN

Goubert & Simons, 2012
FEAR OF PAIN

• One of the most popular models in pain
• Many studies validating separate components
• Hard to validate total model
• Can be very useful clinically and in research
• Several different variations
FEAR OF PAIN

• Cognitive features:
  • Anxiety, catastrophizing

• Behavioral
  • Inactivity and resultant deconditioning
  • Avoidance of feared events

• Biological
  • Not explicit in model
    • Muscle tension
    • HPA axis activation
    • Stress hormones
FEAR OF PAIN

• Theory suggests several foci for intervention
  • Catastrophizing
  • Fear of injury/pain
  • Avoidance of what might cause pain

• Intervention at targets
  • Education about pain
  • Cognitive interventions
  • Systematic desensitization via Exposure
  • Operant approaches

• Major outcomes are:
  • social function e.g. school, work, peer relations
  • physical function e.g. walking
PAIN CATASTROPHIZING, SULLIVAN, BISHOP AND PIVIK 1995

• Rumination,
• Magnification
• Helplessness
• Related to outcomes in several studies
  • Predicts pain, disability
  • Robust findings in children, adolescents and adults
  • Across studies and diseases
“I don’t think you are too interested in this, you think that the real action is somewhere else, and anyway all this research psychology is Ivory Tower stuff, I saw Andre Sourander yawning. You will probably start walking out before I am finished. I don’t know anything, really, they’ll probably fire me from the IWK and maybe even from the university when my president learns of this. She will learn how I botched this workshop. Then my wife will be fed up with me and leave. I’ll begin drinking and my liver will go. I’ll end up homeless in the gutter and die miserably and go to hell too.”
PAIN CATASTROPHIZING

• Rumination
  • I keep thinking about how much it hurts.
  • I keep thinking about how badly I want the pain to stop

• Magnification
  • I become afraid that the pain will get worse.
  • I wonder whether something serious may happen.

• Helplessness
  • I feel I can’t go on.
  • It’s terrible and I think it’s never going to get any better
# PAIN CATASTROPHIZING SCALE

<table>
<thead>
<tr>
<th>Clinical Symptoms</th>
<th>Sub-Scales</th>
</tr>
</thead>
<tbody>
<tr>
<td>I worry all the time about whether the pain will end.</td>
<td>Rumination</td>
</tr>
<tr>
<td>I feel I can’t go on.</td>
<td>Magnification</td>
</tr>
<tr>
<td>It’s terrible and I think it’s never going to get any better</td>
<td>Helplessness</td>
</tr>
<tr>
<td>It’s awful and I feel that it overwhelms me.</td>
<td></td>
</tr>
<tr>
<td>I feel I can’t stand it anymore</td>
<td></td>
</tr>
<tr>
<td>I become anxious that the pain will get worse.</td>
<td></td>
</tr>
<tr>
<td>I keep thinking of other painful events</td>
<td></td>
</tr>
<tr>
<td>I anxiously want the pain to go away</td>
<td></td>
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<tr>
<td>I can’t seem to keep it out of my mind</td>
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<tr>
<td>I keep thinking about how badly I want the pain to stop</td>
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<tr>
<td>There’s nothing I can do to reduce the intensity of the pain</td>
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<tr>
<td>I wonder whether something serious may happen.</td>
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URIE BRONFENBRENNER’S SOCIAL ECOLOGICAL MODEL

- Ecological child development
  - Microsystem, mesosystem, exosystem, macrosystem
- Not applied to pediatric pain
  - A general framework
BRONFENBRENNER’S SOCIAL ECOLOGICAL MODEL OF CHILD

Areas with little or no attention
MODELS OR THEORIES

• We all use them
• Determines
  • What we look for
  • Our explanations
  • Our interventions
• Better to be explicit
• Use of multiple models
• Biological and psychosocial models not well integrated
Thanks to students, colleagues

And you for being so attentive