MALADAPTIVE NEURAL PLASTICITY AND CHRONIC LOW BACK PAIN

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In 2000 this appeared (Grachev et al. 2000)

Our findings also might explain the low diagnostic value of structural neuroimaging studies (i.e. MRI) of the lumbar spine in this category of subjects. Local disk abnormalities might play a certain role in acute cases of back pain as a trigger of pain mechanisms, and when the process becomes chronic other more central mechanisms driven by or causing changes in brain chemistry may be more important. If this is the case, $^1$H-MRS of the brain, rather than MRI of the lumbar spine, will play a major role in diagnosis of chronic back pain and monitoring of surgical and non-surgical treatments and outcomes. The proper documentation of brain
Particularly Interesting Because

Current treatment for CLBP is not very effective

Maybe we are missing some important part of the problem?

A fair amount of research since then has demonstrate significant alterations in brain structure and function

Maybe the brains contribution to the CLBP experience is the thing we have been missing?
Lots of structural alterations have been shown

**Altered neurochemical profile**
- DLPFC, OFC, ACC, Insula, Thal, S1  

**Altered grey matter density/volume**
- DLPFC, Thal, A/MCC, PPC, S1, midbrain, MTL, MC
- Putamen, Thal, insula  

**Decreased white matter volume**
- Insula, MCC  
  (Buckalew 2008, Gussew 2010)

**Altered white matter connectivity**
- Splenium of corpus callosum  
  (Buckalew 2010) – disabled v non disabled CLBP
Lots of functional differences as well

- Altered sensory representation
  (Flor 1997a, Lloyd 2008)

- Altered response to nociception

- Altered response to non-noxious & verbal stimuli
  (Flor 1997a, Flor 1997b, Lloyd 2008)

- Altered motor representation
  (Tsao 2008, Tsao 2011)

- Altered activation with movement
  (Jacobs 2010)

- Altered corticospinal drive
  (Strutton 2005)

- Altered resting state
  (Baliki 2008, Buckalew 2010, Tagliazucchi 2010, Balenzuela 2010)
There are some problems

- The data are preliminary
- The studies are all small
- Often analysed uncorrected
- Opioids are a confounder
  - Not always controlled
- Patient diversity
- Most data are cross-sectional

- Consequence or Cause?
  - Symptom or disease
- Temporal relationship
- Strength of association
- Consistency
- Experiment
- Other explanations
- Plausibility/Coherence
Enhanced nociceptive efficiency

- Degeneration of antinociceptive areas
  - Implicated in placebo analgesia
- Failed activation of antinociceptive areas

- Local hyperalgesia (Giesbrecht 2005, Kobayashi 2009, Blumenstiel 2011)
  - Not in community CLBP people (Blumenstiel 2011)
- Enhanced remote sensitisation (O’Neill 2007, Kleinbohl 1999)
- Spontaneous pain linked to cortical activation abnormalities
  - Different to peripherally evoked pain (Baliki 2006)
- Multi modal sensitivity (Small 2006, Fann 2005)

- Sensitised system – top down
- Maybe contribute to failure of MT
Psychological problems

- Degeneration frontal areas
- DLPFC activity inversely related to catastrophisation
- DLPFC activity inversely related to unpleasantness

- Emotional decision making poorer (Apkarian 2004)
- Memory and mental flexibility impaired (Weiner 2006, Lorenco 2009, Buckalew 2008)
  - Related to catastrophisation
- Same pain intensity rated as more unpleasant (Kobayashi 2009)
- Left sided LBP more depressed, worse mood than right sided (Wasan 2010)

- Maybe contribute to failure of psychological Rx
Attentional processing

- DLPFC involved in maintaining goal relevant priorities
  - Avoid interference from goal irrelevant information
  - Maintain attentional load and attentional set
  - Prevent attentional capture and interference by painful stimuli
- Reciprocal connections with the cingulate cortex

- Difficulty disengaging from threat pictures (Roelofs 2005)
- Attentional bias to pain related words (Haggman 2010)
- Distraction seems less analgesic in CLBP (Johnson 1997, Goubert 2004)
- Worse at digit span task, working memory, stroop (Gijsen 2011)

- Functioning with pain more difficult
  - Attentional capture greatest when pain intense, novel & threatening
- Failure of Rx approaches emphasise function despite pain
Altered body perception/awareness

- Degeneration S1, MC
- Reorganisation of S1
- Reorganisation of M1
- Altered biochemistry S1

- Decreased sensory acuity
  (Luomajoki 2011, Moseley 2008, Wand 2010)

- Poorer graphaesthesia
  (Wand 2010)
  - Sensory thresholds intact

- Altered proprioception

- Poorer laterality recognition
  (Bray 2011)
Altered body perception/awareness

- Smaller than really is
- Midline shift
- Miss bits out (Moseley 2008)

- Neglect that side
  - Body and space (Moseley under review)

- Certainly move differently
- Altered muscle recruitment
  - Motor control related to acuity (Luomajoki 2011)

- Qualitative study (Smith & Osborn 2008)
  - ‘Not part of me’
  - ‘Not controlled automatically’
  - ‘Doesn’t belong’
Quantitative assessment of self-reported disturbances in body perception - FreBAQ

<table>
<thead>
<tr>
<th>Item</th>
<th>Never</th>
<th>Rarely</th>
<th>Occasionally</th>
<th>Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not part of the rest of my body</td>
<td>56</td>
<td>15</td>
<td>19</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Focus all my attention on my back to make it move</td>
<td>22</td>
<td>22</td>
<td>30</td>
<td>19</td>
<td>7</td>
</tr>
<tr>
<td>Back moves involuntarily, without my control</td>
<td>48</td>
<td>19</td>
<td>19</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>I don’t know how my back is moving</td>
<td>44</td>
<td>22</td>
<td>11</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>I don’t know where my back is in space</td>
<td>52</td>
<td>33</td>
<td>4</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>I can’t perceive the exact outline of my back</td>
<td>41</td>
<td>30</td>
<td>19</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>My back feels like it is swollen</td>
<td>26</td>
<td>22</td>
<td>22</td>
<td>22</td>
<td>7</td>
</tr>
<tr>
<td>My back feels like it has shrunk</td>
<td>78</td>
<td>11</td>
<td>7</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>My back feels lopsided (asymmetrical)</td>
<td>19</td>
<td>19</td>
<td>26</td>
<td>22</td>
<td>15</td>
</tr>
</tbody>
</table>
Mislocalisation of sensory information

- 24 CLBP patients and 24 healthy controls
- 672 stimulations in total per group
- Referred and Mislocalised sensations

**Referred sensations**
- 5 patients - 3 controls
- 26 stimulations for patients - 5 for controls

**Mislocalisation**
- 16 patients - 5 controls
- 41 stimulations for patients – 8 for controls
Altered body perception might influence the clinical condition in a number of ways

**Might cause pain**
- Virtual back ≠ Real back
- Sensori-motor incongruence  
  - Discordance generates pain
- Some experimental support
- All movement patterns faulty if the brain doesn't know what happening

**Might affect tissue health**
- Disrupt homeostasis
- Cooling with disownership
- Perception changes swelling

**Likely to change way move**
- Reason for motor problems
  - Another pain generator
- Extra challenge for Retraining

**Might contribute to fear/worry**
- Feels wrong/strange/peculiar
- Unexpected mvts and sensations
- Loss of control
- Extra challenge for cognitive treatments
Is chronic LBP a problem of perception of the back?

Maladaptive cognitive perception
- Amount degenerated
- Fragility/robustness
- Reversibility of the problem
- Controllability of problem
- Prognosis of the problem

Maladaptive self perception
- How the back feels
- The size
- The outline
- When it is and isn’t moving
- How much moving
- Position in space
- Ownership and control

And these two issues are likely to be strongly interactive and mutually reinforcing
The maladaptive perceptions model

**Episode of Low Back**

- **Likely to be enhanced by**
  - High pain intensity
  - Psychological distress
  - Somatisation
  - Pathoanatomical Dx
  - Previous back pain

- **Maladaptive perception of the problem**
  - Uncontrollable
  - Irreversible
  - Unlikely to resolve
  - Fragile
  - Indicative of a serious structural problem

- **Increased threat value attached to nociceptive information**

- **Adoption of movement strategies**
  - Limit spinal movement, increase rigidity, and decrease flexibility and variability of motor responses

- **Excessive attention / hypervigilance to sensory information**

**Changes in cortical and subcortical areas that subserve nociception, attention and sensorimotor control of the back**

- **Enhanced nociceptive efficiency**
  - Enhanced pain response
  - Hyperalgesia
  - Allodynia
  - Spontaneous pain

- **Disturbed perception of the back**
  - Loss of sensory acuity
  - Difficulty delineating the outline and size of the back
  - Loss of proprioceptive acuity
  - Disownership, neglect
  - Loss of control
  - Foreignness and peculiarity
  - No visual information to correct
    - Altered motor control
    - Altered homeostatic control
    - Decreased function

- **Failure of attentional processes**
  - Distraction ineffective
  - Difficulty with dual task performance
  - Difficulty functioning with pain
  - Cognitive dysfunction
  - Problems with working memory

**Further reinforcement of maladaptive perception of the problem**
What does this mean for management

Biopsychosocial rehabilitation which has normalisation of cognitive perception and self perception as its goal
Cognitive perception

- Provide a coherent understanding of the problem
  - Explains all of what they feel, including treatment failure
- Outline a clear path to resolution
  - Reduce anxiety about condition and future course
- Disavow lumbar pathoanatomical basis
- Emphasise robustness of the spine
- Emphasis controllable, reversible, amenable to treatment
- Plausible reasons to engage in activities been avoiding
- Additional reasons why fearful and avoidant

I think a cortical perspective makes these things easier

What about self perception?
Treatment of other pain problems directed towards self perception has helped

- MVF for PLP and CRPS
- GMIP for CRPS and PLP
- Sensory discrimination for PLP and CRPS
- Magnifying hand made worse - minifying it made better in CRPS
- Simulated walking for SCI
A lot of what we currently do may positively influence self perception of the back

- Massage, mobilisation, dry needling, TENS
  - Sensory awareness

- Specific muscle activation training
  - Motor awareness
  - Often without movement so may minimise incongruence

- Dissociation training
  - Sharpen up delineation between areas
  - Again without moving

- And these things are a bit helpful
- Maybe better if done with explicit aim of improving perception
MIRROR VISUAL FEEDBACK
Cross-over experiment
Standardised range, speed and reps – 60 reps moving with vision v without vision

<table>
<thead>
<tr>
<th></th>
<th>With visual feedback mean (SD)</th>
<th>Without visual feedback mean (SD)</th>
<th>Mean difference (SD)</th>
<th>95% CI</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pain intensity</td>
<td>7.75 (11.92)</td>
<td>17.00 (14.61)</td>
<td><strong>9.25 (18.50)</strong></td>
<td>1.44-17.06</td>
<td>.022</td>
</tr>
<tr>
<td>Time to ease</td>
<td>48.50 (56.09)</td>
<td>97.38 (80.17)</td>
<td><strong>48.88 (69.50)</strong></td>
<td>19.53-78.22</td>
<td>.002</td>
</tr>
</tbody>
</table>
Graded cortical retraining

Progressive engagement of sensory areas

- Localisation of stimulus
- Localisation and type of stimulus
- Graphaesthesia training
  - Letters
  - Words
  - Sums
    - Size
    - Orientation
    - Speed
    - Laterality
    - Overlap
Graded cortical retraining

Progressive engagement of motor areas

- Laterality recognition
  - Recognise©
- Imagined movements
  - Small range then large
- Isometric local muscle activation
  - Inner unit contraction
  - with dissociation exercises
- Movements with feedback optimised
  - Small range movements
  - Large range movements
Meta-analysis rTMS: Pain short term

Corrected n=267

High frequency
≥5Hz

SMD = -0.32
95% CI -0.51 to -0.13 (p=0.0009)

I² = 71%

Low frequency <5Hz
Meta-analysis rTMS: Pain short term: Subgroup: High frequency motor cortex stimulation

Single dose studies

SMD = -0.40
95% CI -0.54 to -0.26
(p=0.00001)

I^2 = 36%

On a 0-100mm VAS scale:

9.3mm (95% CI 6.2 to 12.5mm)
15% (95% CI 10% to 20%)

Corrected n = 184
Meta-analysis tDCS

All studies

SMD -0.59
95% CI -1.10 to -0.08
p=0.02
I² = 45%
n = 83

Subgroup: Motor cortex studies

n = 73
Thank You

Questions?

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