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Assessment of cerebral biochemistry in individuals with chronic idiopathic neck pain and healthy individuals:

Evidence for central changes in the presence of chronic neck pain

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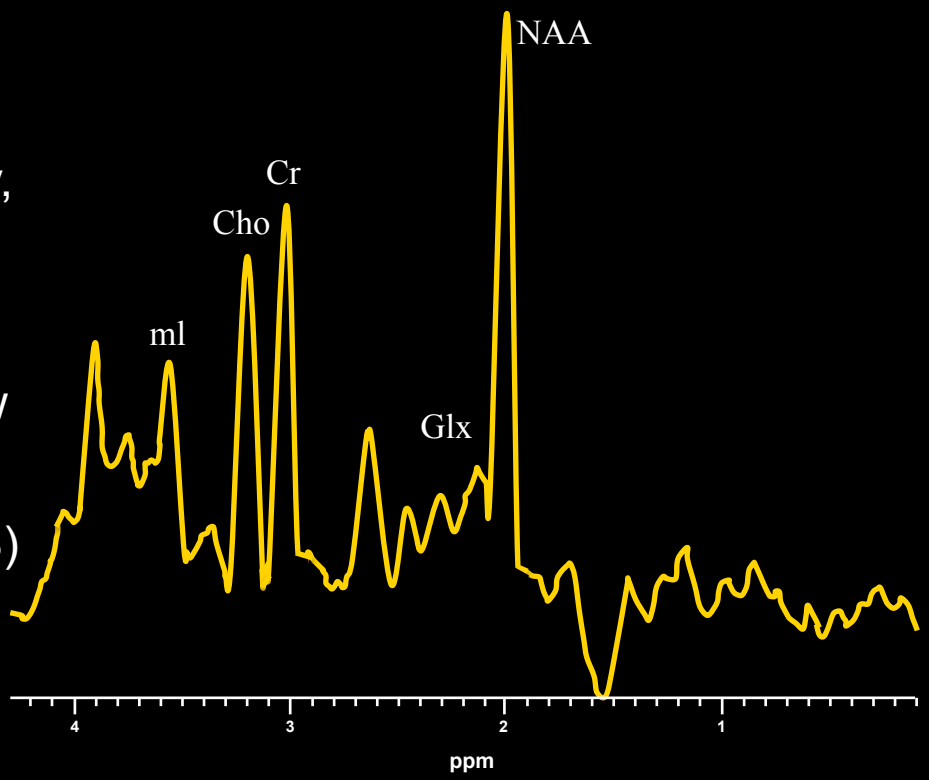
BACKGROUND

Chronic neck pain is disabling and costly, and recurrence is common[1]

Contribution from central adaptations?[2]

Cerebral metabolite concentrations show changes in other pain conditions[3,4]

Magnetic resonance spectroscopy (MRS) quantifies metabolites concentrations



AIM

To determine cerebral biochemistry differences in motor cortex and thalami between individuals with chronic idiopathic neck pain and healthy matched controls

To determine whether cerebral biochemistry correlates with pain intensity

[1]Haldeman et al., Spine 2008;33:S5-7.
[2]Moseley & Vlaeyen, Pain 2015;156:35-8.
[3]Siddall et al., Anesth Analg 2006;102:1164-8.
[4]Stanwell et al., Neuroimage 2010;53:544-52.

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METHODS

Individuals with idiopathic neck pain (> 90 days), with pain ≥ 4/10 that at least 'moderately interferes' with activity

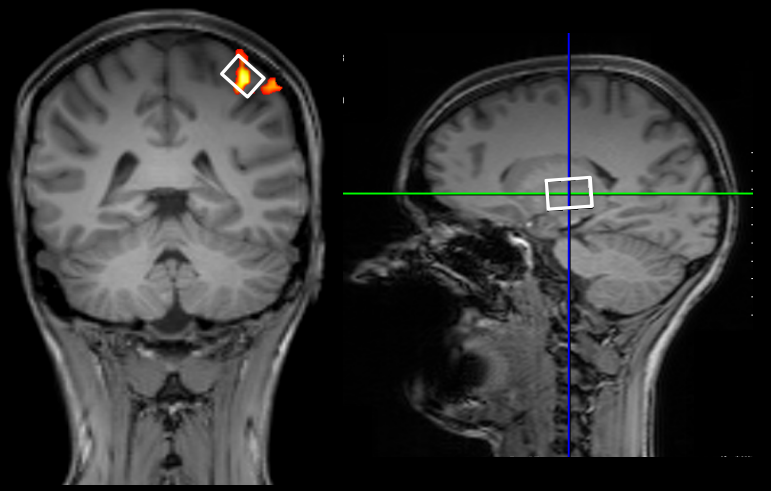
Excluded:

- Hx neck trauma, surgery
- Radiculopathy
- Migraine
- Headache/dizziness as primary complaint
- Neuropathic pain (≥10 on S-LANSS)
- Diabetes, peripheral vascular disease

Healthy age/gender matched controls with no neck or back pain for which they sought treatment in the previous 2 years

Excluded:

- Hx neck trauma
- Any musculoskeletal pain in any body area



Magnetic resonance spectroscopy (T3 Tesla)

- LCModel quantified brain metabolites in motor cortex and thalami including
  - Glutamate (Glu): excitatory neurotransmitter
  - Total Creatine (tCr): cellular energy stores

Analysis

- Group comparisons with t-tests
- Pearson's correlations between pain (current and 4-week recall) and metabolite concentrations

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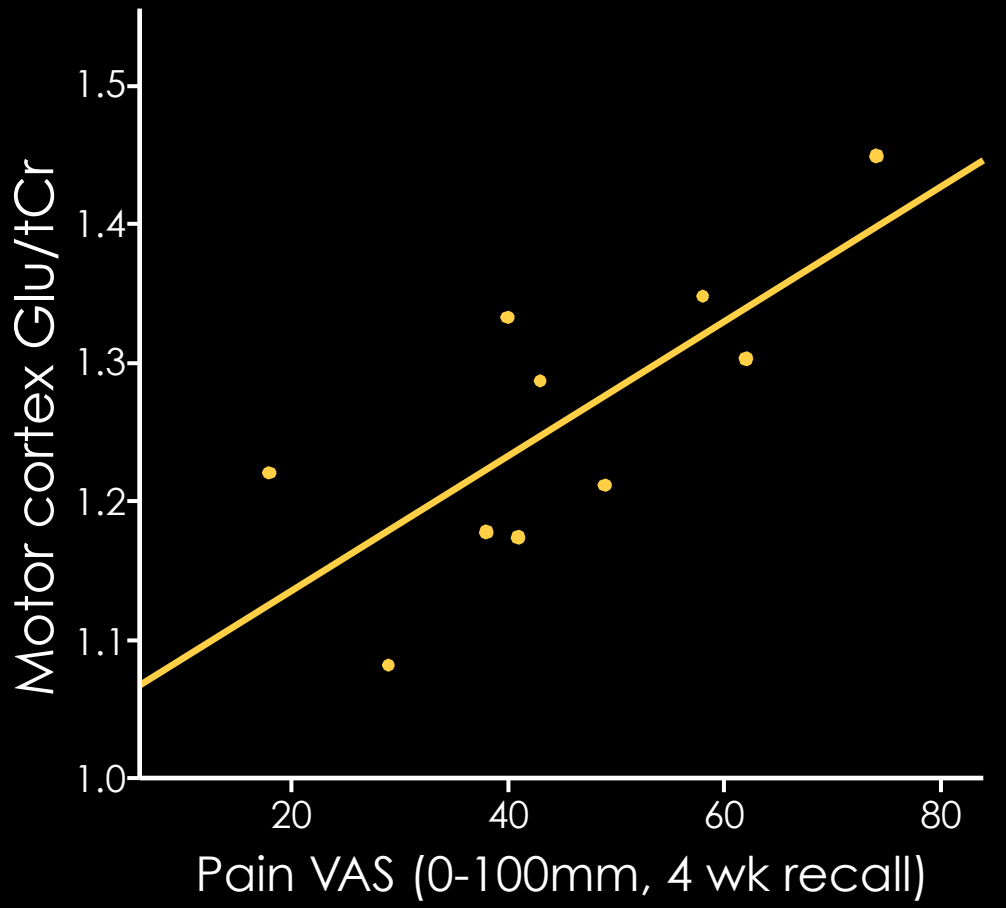
RESULTS

	Pain (n=10)	Healthy (n=10)
Age	32.0 (10.5)	31.9 (11.1)
Gender (male)	8 (80%)	8 (80%)
Average pain previous 4 weeks (VAS 0-100mm)	45.2 (16.3)	

No significant group differences

Higher glutamate to total creatine ratio correlated with greater pain (r = .744, p=.014).



CONCLUSIONS/IMPLICATIONS

Cortical metabolic changes associated with higher levels of chronic idiopathic neck pain

Higher concentrations of the excitatory neurotransmitter glutamate suggest altered neurotransmission in the motor cortex

Preliminary evidence in a small sample for a central cortical contribution to the pain intensity and altered movement patterns observed by clinicians

Suggests treatment should include interventions to address the central control of movement and pain responses

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