Brain Activity for Osteoarthritis Pain : an fMRI Study

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INTRODUCTION

We have shown that the brain of chronic pain patients is functionally and morphologically affected by the chronic condition (Apkarian et al. 2004). In this study, we investigate brain activity of knee osteoarthritis (OA) patients in response to a painful pressure stimulus.

Contrasting brain activity underlying nociception in these patients with that of normal subjects aid in unraveling the dynamics of the major brain areas encoding chronic pain and provide a history of the progression and pathology of the disease.

METHODS

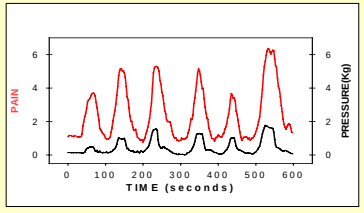
- 14 patients with knee OA participated in this study. Prior to scanning, all patients were trained to use a finger-span device to rate magnitudes.
- In the scanner , patients rated their on-line pain. Each volunteer performed 4 pain scans (each knee was stimulated twice) and one visual control scan.
- The WOMAC pain questionnaire were also collected on all the patients
- The signals for pain and control scans are used to search for the BOLD signal and to control for various contaminants. First and higher level analysis were performed using FSL software (fmrib, Smith et al. 2001). The end result was a COPE image for each subject that accounted for both pain runs and the visual motor control. These cope images were used to generate the average activity maps (panel 3) and covariate analysis for pain intensity (panel 4).

1 Experimental Set Up for Knee Stimulation

An air propelled plastic cylinder is used to exert painful pressure on the knee, while the patients rate online their pain using the a finger span device shown here fixed to the thumb and index fingers. Time courses of both pain and pressure are generated as shown in panel 2.

A visual feedback of the patient's rating is projected on a computer screen with a scale from 0 (no pain) to 10 (worst imaginable pain)

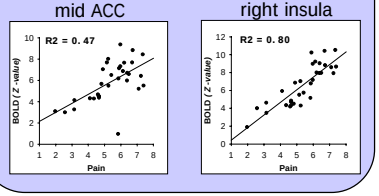
2 On-line pain rating



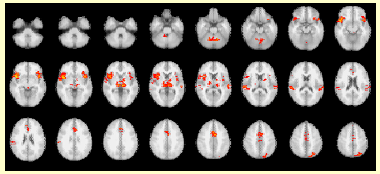
Shown here is an example pain rating (red trace) generated by one subject with corresponding pressure time course (black).

The pain rating is convolved with a canonical hemodynamic function and used to look for brain components of OA pain shown in panel 3.

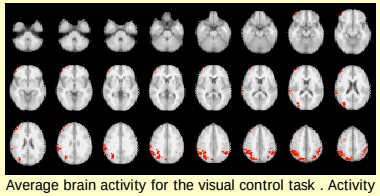
Pain Intensity vs brain activity (normals)



3 Brain Activity for OA Pain and Visual Control

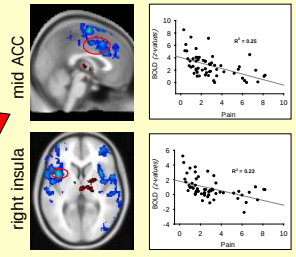


Average brain activity for OA pain after subtraction of the visual control task (shown below). Brain regions that are activated include bilateral thalamus, insula, and SII, in addition to cerebellum, left striatum, midbrain regions and cingulate cortex (BA 24/32). These same areas are also commonly seen in acute pain studies in normal subjects.



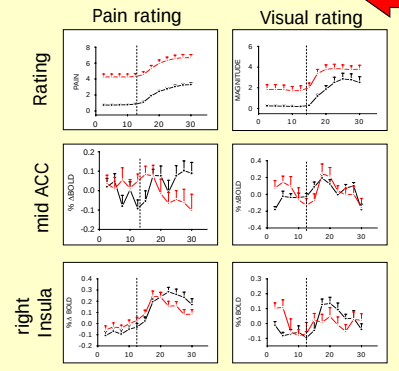
Average brain activity for the visual control task . Activity is mainly seen in the attention areas in the bilateral parietal cortices.

4 OA Pain Intensity vs brain activity



OA pain intensity calculated as the average of the ratings (such as shown in red in panel 2) correlate positively only with thalamus and striatum activity. However, the correlations are negative with bilateral insula, SII, ACC (24/32), and lateral prefrontal areas. The latter areas usually correlate positively with pain intensity in acute pain in normal subjects (Red arrow).

5 BOLD Signal Analysis



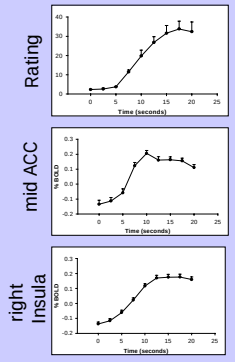
Time windows where pain (left) or visual ratings (right) were increasing (see panel 2) were averaged within runs and across subjects and then separated into high pain (red trace) and low pain (black trace).

Corresponding BOLD signal was extracted from the ROIs obtained in the pain intensity covariate map.

The temporal pattern of the BOLD signal in ACC (BA24) and right insula shows an opposite pattern to the pain rating.

The same ROIs do not show this pattern in the control task (red arrow).

BOLD Signal Analysis (normals)



CONCLUSION

- Overall, Brain activity for knee stimulation in OA is similar to that of acute pain in normal subjects, including bilateral insula, thalamus, SII and mid ACC and SMA.
- However, unlike acute pain in normal subjects, activity in these brain areas exhibit a negative correlation with pain intensity (mainly insula and mid ACC).
- The difference in pain intensity encoding is dependent on the level of baseline pain, suggesting a unique interaction between nociception and cortical processing for chronic OA pain.
- The results are consistent with the companion poster indicating that cortical activity is only a reaction to nociceptive inputs to subcortical areas, and that this response is unique to OA pain.

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