

## BNPA MEMBERS' PLATFORM PRESENTATIONS

007 TEMPORAL LOBE EPILEPSY & AFFECTIVE DISORDERS:  
THE ROLE OF THE SUBGENUAL PREFRONTAL CORTEX

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**Objective** To explore the neural substrates of affective psychopathology in temporal lobe epilepsy (TLE), using functional MRI (fMRI).

**Method** A visuo-spatial "n-back" paradigm was used to compare working-memory network activation in 17 TLE patients (Median age: 40, 14 female) with a lifetime diagnosis of depression and/or anxiety with 31 TLE patients (Median age: 38, 17 female) with no formal psychiatric history and 30 healthy controls (Median age: 37, 18 female). There were no significant differences between the TLE groups with respect to age, gender, handedness, epilepsy onset/duration or pre-morbid IQ. All subjects completed the Beck Depression Inventory Fast Screen (BDI-FS) and Beck Anxiety Inventory (BAI) on the day of scanning. Imaging data were analysed using SPM8 software.

**Results** Each group activated the fronto-parietal working memory networks, and deactivated the typical default mode network (DMN) in response to the increasing task demands. Group comparison revealed that TLE patients with a lifetime history of affective disorders showed significantly greater deactivation in bilateral subgenual prefrontal cortex than either the TLE without any psychiatric history and the healthy control groups ( $p < .001$ ). Post-hoc analyses indicated that this main group effect persisted after co-varying for current psychotropic medication and severity of current depressive/anxiety symptoms (all  $p$ -values  $< .001$ ). Correlational analysis revealed that this finding was not driven by differences in task performance ( $r = .49$ ,  $p = .33$ ). There were no significant differences in hippocampal volume or amygdala T2 signal between the TLE groups.

**Conclusion** Hypometabolism of the subgenual prefrontal cortex bilaterally has been reported in association with primary mood disorders (1), and our findings further implicate this region. The subgenual prefrontal cortex shares extensive and reciprocal anatomical connections with areas implicated in emotional and behavioural regulation, such as the posterior orbitofrontal cortex, amygdala, hippocampus and hypothalamus. We hypothesise that altered modulation of this region with increased deactivation may be associated with a predisposition to develop mood disturbance. Our findings suggest that the same neurobiological substrate involved in the pathogenesis of primary mood disorders may also underpin affective psychopathology in TLE.

## REFERENCE

- 1 Drevet WC, *et al.* Subgenual prefrontal cortex abnormalities in mood disorders. *Nature*. 1997 386:824–827