Sensation Seeking and Aversive Stimulation - A functional MRI Study at 3 Tesla

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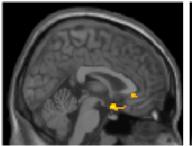
Introduction: Sensation Seeking (SENS) is a personality trait characterized by the need for varied and complex sensations even at the cost of personal risks for such experiences (1). People with high and low SENS scores differ in their tendency to avoid or seek threatening stimuli or situations as well as in their anxiety reactivity (2, 3). Subjects with high SENS score experience a positive reward in response to intense, aversive stimuli, whereas people with low score avoid intense and dangerous sensory stimulations. We examined the association between Sensation Seeking and the neurobiological mechanisms of fear processing with fMRI. It is suggested that the personality trait SENS correlates with neuronal responses to fear-stimuli.

Methods: In this ongoing study, a fear-conditioning paradigm was presented to 16 healthy volunteers (8 f; mean age, 24.9 y) in fMRI at 3 T (Gyroscan Intera 3.0 T, Philips, Best, NL) with whole head EPI (TR/TE 2s/30ms, isotropic voxels 3.6 mm edge length). Pictures of two neutral faces (NimStim; (4)) served as conditioned stimuli (CS+, CS-). The experiment consisted of 4 phases: Habituation, Acquisition (twice), and Extinction. In the Acquisition phases 25% of the CS+ where paired with an acoustic startle as unconditioned stimulus (UCS) in an event-related design. Following each phase participants rated valence (higher value = more pleasant) and arousal for the CSs with a 5-point scale (SAM; (5)). SENS-scores were assessed individually with an appropriate scale (SSS-V; (6)). FMRI data were analyzed with SPM5.

Results: *Technical:* EPI data were sufficiently undistorted to allow reliable evaluation even of sensitive areas as the amygdala.

Behavioral data: Ratings of valence and arousal reflected successful conditioning (Fig. 1): conditioned faces scored lower (unpleasant) valence even without startle ('unpaired').

Activation of classical fear circuits (CS+unpaired > CS-): In Acquisition phase 1 enhanced activation was found in fear related areas, mainly in anterior cingulated cortex but also right insula. In Phase 2 known fear-network structures, among them bilateral amygdala, insula, thalamus, and striatum were activated.



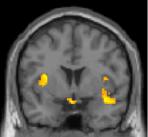


Fig.2 (whole brain analysis, p < 0.005, uncorrected for multiple comparisons, contiguity threshold ≥ 10 voxels)

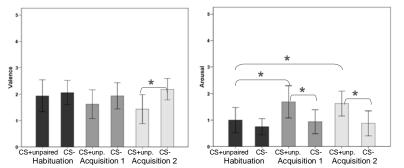


Fig.1 (*p < 0.05, ** p < 0.01)

Correlation: For BOLD response vs. SSS-V (SENS-Score) only during Acquisition phase 1 a significant negative correlation was detected for bilateral insula and subgenual cingulated cortex (Fig. 2).

Discussion: FMRI data demonstrated enhanced activation in fear-related areas like insula and anterior cingulated cortex during fear conditioning. Interestingly, significant negative correlations between Sensation Seeking and BOLD-signals only emerged in the first acquisition phase including left insula and subgenual cingulated cortex. The results point to the ACC's key role in processing of conditioned stimuli (7) and its involvement in integrating affective and cognitive information. Insula and ACC might convey a cortical representation of fear to the Amygdala (8), which seems to be

stronger for subjects more sensitive to threat. Individuals scoring low on SENS are not only more anxiously reactive to direct presentation of threatening stimuli (2) but also to anticipation of fear stimuli. The personality trait SENS thus shows a negative correlation of neurobiological reactions with conditioned fear, i.e. for high sensation seekers these reactions are reduced.

References: (1) Zuckerman M, Psychiatry Res 1979; 1:255-264 (2) Lissek S, Powers AS, Biol Psych 2003; 63:179-197 (3) De Pascalis V et al., International Journal of Psychophysiology 2007; 63:16-24 (4) Tottenham N et al., Psychiatry Research, in press (5) Bradley MM, Lang PJ, J Behav Ther Exp Psychiatry 1994; 26:49-59 (6) Beauducel A et al., Diagnostica 2003; 49:61-72 (7) Gallinat J et al., NeuroImage 2007; 36:671-678 (8) Phelps EA et al., Nature Neuroscience 2001; 4:775-776