

Modulation of BOLD-Response in the hypothalamus by affectively loaded visual stimuli

F. Gerstl^{1,2}, C. Windischberger^{1,2}, K. Å. Karlsson³, and E. Moser^{1,2}

¹MR Center of Excellence, MUW, Vienna, Austria, ²Center for Biomedical Engineering and Physics, MUW, Vienna, Austria, ³Department of biomedical engineering, School of science and engineering, Reykjavik University, Reykjavik, Iceland

Introduction:

The hypothalamus is a small diencephalic structure situated bilaterally between the third ventricle and the thalamus[1], which nevertheless is of pivotal importance for vegetative regulation of the human body. In order to perform its numerous tasks which involve upkeep of homeostasis, feeding and water intake, sexual and mating behavior as well as the regulation of daily physiological cycles and mediation of emotional responses, it has numerous neuronal connections to other parts of the brain and control over the pituitary gland [2]. Recent studies show abnormal activity in the hypothalamus of cataplexy-patients during humor processing * (Schwartz et al 2008) *. Using high-field fMRI, we demonstrate significant modulation of BOLD activation following emotional arousal at the anatomical location of hypocretin neurons in healthy individuals.

Subjects and Methods:

Sixty affectively loaded pictures were used to induce emotional processing in the study group of 21 healthy volunteers (17m, 4f, 26+/- 2.9years). These stimuli were subdivided in 3 groups based on an individual rating of each subject: very funny (10), neutral (20), and very sad (10), while 20 pictures that were rated to be only slightly funny or sad were discarded (10 of each). Stimulus presentation order was randomized and each stimulus was shown for four seconds, with variable inter-stimulus interval ranging from 8 to 12 seconds, during which a white fixation cross was visible. fMRI was performed on a 3T scanner (SIEMENS Tim Trio, Erlangen) using high-resolution gradient-recalled EPI. Twenty axial slices were acquired centered at the hypothalamus (thickness=1.9mm, gap=0.9mm, MA=128x128, TE=40ms, TR=2000ms, parallel imaging (GRAPPA 2)). Preprocessing included slice-timing correction, realignment, normalization to MNI-space and smoothing (Gaussian kernel, FWHM=7mm) in SPM5. Realignment parameters were added as nuisance variables in the design matrix. Repeated measures ANOVA was performed and corrected for multiple comparisons (FDR, p=0.05). Parameter estimates from the amygdalar and hypothalamic activation peaks were extracted bilaterally.

Results

Repeated measures analysis of variance (figure 1) revealed strong bilateral BOLD-modulation in the amygdala (left:F=27.81, right:F=32.48). In addition, significant modulation very funny vs. neutral vs. very sad (see Figure: mean parameter estimates +/- SD) was observed bilaterally in the hypothalamus (left:F=10.53, right:F=6.8). Conclusively, hypothalamus activation experiences strong and specific modulation by emotionally meaningful visual stimuli.

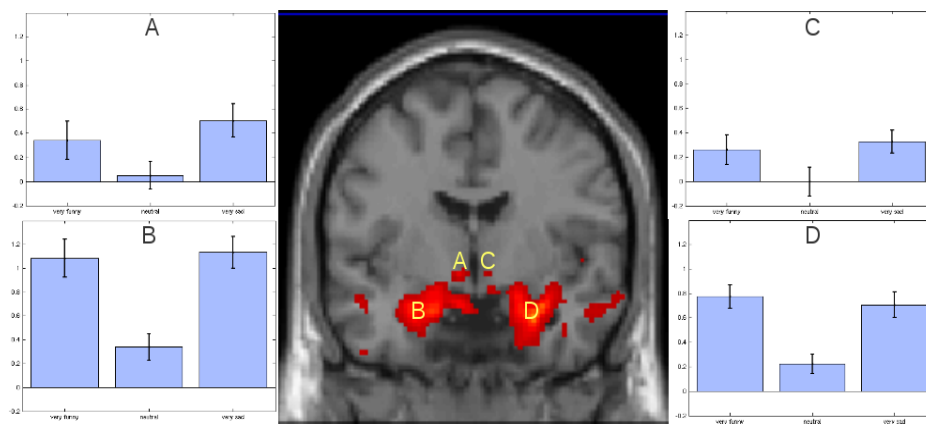


figure 1: ANOVA results on frontal MNI slice (y=4.53) showing bilateral modulation in left (A,B) and right (C,D) hypothalamus and amygdala by very funny, neutral and very sad emotional stimuli. Bar diagrams show parameter estimates in clusters of maximum modulation in left and right hypothalamus, which were found at MNI coordinates -6, -4, -6 (F=10.53; p<0.002) and 8, 0, -6 (F=6.8; p<0.016)

Discussion/Conclusion:

As the first of its kind, this fMRI study demonstrates valence-specific bilateral modulation of hypothalamic activation to emotional stimuli. The observed modulation of activity at the anatomical location of hypocretin neurons supports theories of hypothalamus-mediation of cataplectic attacks following strong emotional arousal in narcoleptic patients. Potential applications of hypothalamic fMRI could also include research of eating disorders and interactions between sleep disturbances and eating disorders.

References:

- [1] P.M.Daniel, J.Clin.Path.Suppl.7,1-7(1976).
- [2] T.Sakurai, Nat.Neurosci.,8,171-181(2007).
- [3]Schwartz, S. et al., 2008. *Brain*: 131(Pt 2), 514-22.