P02-330

FOOD-CUE EVOKED ACTIVATION OF REWARD PATHWAYS IS MODULATED BY THE SATIETY FACTOR LEPTIN: AN FMRI STUDY IN OBESE AND NORMAL WEIGHT SUBJECTS

M. Grosshans<sup>1</sup>, C. Vollmert<sup>1</sup>, S. Vollstaedt-Klein<sup>1</sup>, S. Leber<sup>1</sup>, P. Bach<sup>1</sup>, M. Buehler<sup>1</sup>, C. von der Goltz<sup>1</sup>, K. Wiedemann<sup>2</sup>, F. Kiefer<sup>1</sup>

<sup>1</sup>Department of Addiction Medicine, Central Institute of Mental Health, University of Heidelberg, Mannheim, <sup>2</sup>Department of Psychiatry, University of Hamburg, Hamburg, Germany

Introduction: Mechanisms contributing to the development and maintenance of obesity remain to be elucidated especially regarding the interaction between appetite regulating hormones and mesolimbic reward circuits. Leptin was recently suggested to attenuate dopamine release in mesolimbic reward pathways. We now test the functional relevance assessing whether leptin plasma concentration affects the BOLD-response following the presentation of food cues.

Methods: 21 obese and 23 normal weight subjects were investigated. Visual food cues and neutral stimuli were presented in a block design during fMRI. Blood-samples were collected immediately prior to the scan to assess plasma leptin concentration. Using linear regression analyses, the association between BOLD response to food cues and the body mass index (BMI) as well as plasma leptin concentration was examined.

Results: Food-cues elicited activation of large cortical and subcortical networks, whereas only in obese patients food cues activated the left ventral and right dorsal striatum. Mean plasma concentration of leptin was significantly increased in obese subjects compared to normal weight controls. We found a significant positive correlation between the food cueinduced BOLD signal change in the ventral striatum and leptin plasma concentration. Furthermore, ventral and dorsal striatum BOLD response to food cues was significantly positive associated with the body mass index (BMI).

Conclusions: These findings suggest a physiological role of the satiety factor leptin in modulating responsivity of reward pathways towards food cues. Altered homeostatic feedback regulation of the mesolimbic brain reward circuit might explain the inability of obese patients to adapt their food intake according to physiologically needs.