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Bodily Sensibility and Anxiety -An fMRI Study-

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I. Introduction

Anxiety disorder including specific phobia, social anxiety disorder, panic disorder and general anxiety disorder, is one of the most popular psychiatric disorder. The disorder causes close related psychological and somatic physiological symptoms and its pathology provide important factors to understand the relationship between subjective emotion and somatic sensation.

The relationship between subjective emotion and associated somatic responses has been the focused and debated long time. William James (1884) proposed that the experience of emotion results from the perception of specific and unique patterns in the somatovisceral response, and researchers have been working on support and not support the statement to date (Schachter and Singer, 1962; Plutchik and Ax, 1967; Damasio, 1994; Rainville et al. 2006). To understand how emotional responses occur, psychological and neural correlates of feeling subjective emotion are studied, and influential hypotheses are suggested such as somatic marker hypothesis (Damasio, 1994). Although it remains unresolved which aspects of the neural and peripheral response fundamentally determine our emotional experiences, the results of recent psychological and brain imaging studies indicate that the perception of bodily signals contribute to and at least partially mediates emotional experience (Bechara et al. 1996; Dunn et al. 2010; Pollatos et al.

2005; Werner et al. 2009; Terasawa et al., in press). According to these studies, the anterior insular cortex is an essential region for this process. The reactivity in this region during anticipation closely related with individual's personality traits such as levels of anxiety and neuroticism. However, psychological mechanisms underlying the relationship have not yet resolved. To elucidate this mechanism, we examined the relationship between the activation in the anterior insular cortex and personality traits while participants evaluate their own emotional and bodily states.

II. Methods

18 undergraduate and graduate students (five male and 13 female) participated in our study (mean 22.9 years \pm 2.11 s.d.). All participants were right-handed, and reported no history of neurological or psychiatric diseases.

Prior to fMRI scanning, participants' anxiety trait, personality and experience of panic attack were assessed by answering some questionnaires. We used following questionnaires for this purpose; Japanese version of State-Trait Anxiety Inventory-JYZ, Japanese version of NEO-FFI (Five Factor Inventory), Japanese version of Panic Disorder Severity Scale, Social Anxiety Disorder Scale. Participants completed all questionnaires by themselves.

In the MRI scanner, participants were required to answer three types of question about; emotional state, bodily state and possessions. We presented a sentence as "I'm happy", "I have a fast pulse" or "I have money", and participants evaluated and selected appropriateness of the sentence as a description for their current state from four options. Bodily sensibility score was calculated using the responses to bodily state condition. We prepared 16 sentences for each three condition.

fMRI scanning employed a 3T Siemens Tim Trio scanner with an 8-channel head coil for data acquisition. Scanning consisted of three experimental functional runs and a high-resolution T1-weighted structural scan (1 mm isotropic resolution 3D MPRAGE). Each functional run consisted of 274 whole-brain T2* weighted single-shot gradient-echo planar imaging (EPI) images, collected in an oblique axial orientation (TR 2.35 s, TE 30 ms, FA 90 degrees, voxel size 3.5 \times 3.5 \times 2 mm, 44 slices (descending), slice gap 1 mm). The first six functional volumes of each session were subsequently

discarded to allow for equilibration of net magnetization. The structural scan was coregistered to the subject's mean EPI image.

Individual data were pre-processed and analyzed using SPM8 software (<http://www.fil.ion.ucl.ac.uk/spm/software/spm8/>). Functional time images from each participant were spatially corrected for head movement, and temporally corrected for slice timing (using the middle slice acquired in time as a reference), spatially normalized to the Montreal Neurological Institute (MNI) template with a resample voxel size of $1 \times 1 \times 1$ mm and spatially smoothed with a three-dimensional Gaussian filter (8 mm full width half maximum; FWHM). In addition, high-pass temporal filtering with a cut-off of 128 s was applied to remove low-frequency drifts in signal, and global changes were removed by proportional scaling.

III. Results

The intersection of contrast images for the body trials and the emotion trials identified the commonly activated regions. Those were bilateral anterior insular cortex, bilateral temporoparietal junction, the right inferior frontal cortex, and bilateral lingual gyrus.

In the emotion trials, there was significant correlation between the right anterior insular activation and scores of Neuroticism, and social fear (Fig. 1). Also, there were negative correlation between its activation and scores of Extraversion, Openness to Experience and Agreeableness. Similar pattern was observed for the left anterior insular cortex.

Multiple regression analysis revealed that the activation in left thalamus (somatosensory region), precuneus and supramarginal gyrus, during emotion trials correlated with bodily sensibility scores ($p < .001$, uncorrected). To disentangle the relationship between bodily sensibility and anxiety trait, mediation analyses were conducted with the levels of social fear and the activity of these regions and the insular cortex. The result indicated that the right anterior insular cortex mediates the activation of left thalamus and levels of social fear ($p < .03$).

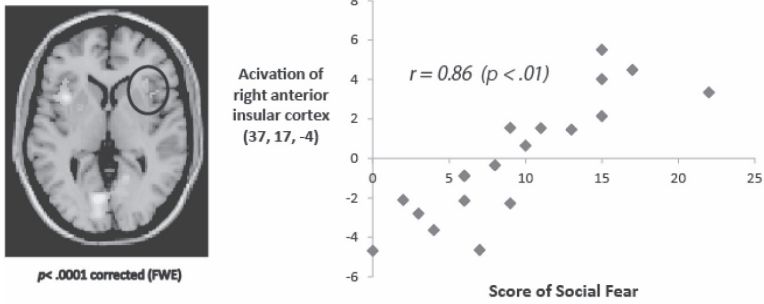


Figure 1. The activation of right anterior insular cortex and the score of social fear.

IV. Discussion

This study sought to disentangle the relationship between interoception and the subjective experience of emotion by examining the neural mechanisms underlying the evaluation of emotional and bodily states. To address this question, we tested which neural areas were commonly activated between both processes, and which areas were closely related with individual personality and anxiety traits.

Our bodily state changes depending on the emotional impact of subsequent events when we make a prediction. Our findings provide direct evidence that high anxiety people tend to be too sensitive to bodily signals, and the greater attention to our own bodily state possibly contributes to development of anxiety disorder.

In addition, our results are in accord with clinical observations indicating the involvement of the anterior insular cortex in some psychiatric symptoms involving disrupted subjective feelings, such as anosognosia (Orfei et al. 2007), alexithymia (Kano et al. 2003), anxiety disorder (Paulus and Stein, 2006), depersonalized disorder (Phillips et al. 2001) and schizophrenia (Makris et al. 2006). Taken together, these findings imply that the way in which we perceive and interpret physiological changes inside the body can strongly impact our health. To clarify the relationship between interoception, emotional experience and underlying neural mechanisms in patients with psychiatric disorders could improve understandings about the mechanisms underlying these clinical symptoms, and lead to the development of effective

treatment. Future studies with psychiatric patients should thus be conducted to provide further insight into emotional experience and its psychological and neural mechanisms.

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