Possible Clinical Application of Decoded Neurofeedback to Treatment of Obsessive-compulsive Disorder

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Abstract:

Obsessive-compulsive disorder (OCD) is a common neuropsychiatric disorder with a lifetime prevalence of 2–3% (1), which is characterized by persistent intrusive thoughts (obsessions), repetitive actions (compulsions), and excessive anxiety. OCD is the sixth leading psychiatric disorder in terms of the total number of years lived with disability and accounts for 2.5% of global years lost because of disability (2). Although selective serotonin reuptake inhibitors and behavioral therapy (e.g. exposure and response prevention) are first-line treatments for OCD, response rates of these therapies are assumed to be 40-60%. Therefore, there is an urgent need to develop the novel therapy.

Although prevalent pathophysiological model suggests that OCD is associated with fronto-striatal circuitry (3), there have not been noninvasive methods which can modulate the brain activity precisely. Decoded neurofeedback (DecNef) is a novel method of controlling distributed activity patterns of multiple voxels within a circumscribed regions of interest (ROIs) (4), thereby greatly increasing spatial precision over the previous method of regulation of localized activity in coarsely defined ROIs. We are trying to develop a novel therapy of OCD by controlling the activity patterns within fronto-striatal circuitry using DecNef.

We conducted DecNef interventions to 3 subclinical subjects and 7 patients with OCD. In decoding experiments, the exposure paradigm using symptom related stimuli was conducted to decode the brain activity pattern related with the evoked symptoms. The detected pattern was used to instruct subjects to manipulate their own brain activity in consecutive neurofeedback sessions for 5 days. The outcomes were evaluated by the clinical scale (Yale-Brown Obsessive-Compulsive Scale (Y-BOCS)) and recently developed anxiety neuromarker calculated using pre- and post- resting state fMRI (5) (details of anxiety neuromarker will be presented in the poster session by Yu Takagi).

The most of selected voxels related with evoked symptoms were in the orbitofrontal cortex consistent with previous studies (6), although we conducted decoding analysis within much more broad brain regions. As preliminary clinical effects of the DecNef intervention, the mean scores of Y-BOCS and anxiety neuromarker were improved in both groups. However, the current approach only showed the insufficient effects in patient group from the viewpoint of clinical application.

Our preliminary application of DecNef to OCD therapy showed the improvement both in the clinical scale and the neural substrate of anxiety. Further development will be needed to increase the effect size for the clinical application.

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