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## Research report

# Behavioral activation can normalize neural hypoactivation in subthreshold depression during a monetary incentive delay task



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#### ABSTRACT

*Background:* Late adolescents are under increased risk of developing depressive symptoms. Behavioral activation is an effective treatment for subthreshold depression, which can prevent the development of subthreshold depression into a major depressive disorder. However, the neural mechanisms underlying the efficacy of behavioral activation have not been clearly understood. We investigated neural responses during reward processing by individuals with subthreshold depression to clarify the neural mechanisms of behavioral activation.

Methods: Late adolescent university students with subthreshold depression (n=15, age 18–19 years) as indicated by a high score on the Beck's Depression Inventory-II (BDI-II) and 15 age-matched controls with a low BDI-II score participated in functional magnetic resonance imaging scanning conducted during a monetary incentive delay task on two occasions. The Individuals in the subthreshold depression group received five, weekly behavioral activation sessions between the two scanning sessions. Moreover, they did not receive any medication until the study was completed.

Results: Behavioral activation significantly reduced depressive symptoms. Moreover, compared to the changes in brain functions in the control group, the behavioral activation group showed functional changes during loss anticipation in brain structures that mediates cognitive and emotional regulation, including the left ventrolateral prefrontal cortex and angular gyrus.

Limitations: Replication of the study with a larger sample size is required to increase the generalizability of these results.

*Conclusions:* Behavioral activation results in improved functioning of the fronto-parietal region during loss anticipation. These results increase our understanding of the mechanisms underlying specific psychotherapies.

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## 1. Introduction

Recently, there has been considerable interest in the clinical significance of subthreshold depression in late adolescence (Georgiades et al., 2006). The high prevalence of subthreshold depressive symptoms among 18–20 years old, and the significant health problem caused by it have been identified (Bertha and Balazs, 2013). It has been reported that 20.7% of college freshman have experienced a major depressive episode within the past one year due to stressful situations they experience, such as environmental changes (Tomoda et al., 2000). Previous studies have

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suggested that subthreshold depression is associated with severe impairments (Keenan et al., 2008) and difficulties in emotional regulation (Hughes et al., 2011). Subthreshold depression is also considered to be a precursor to MDD (Fergusson et al., 2005; Gonzalez-Tejera et al., 2005; Johnson et al., 2009; Rohde et al., 2009).

A recent systematic review (Wesselhoeft et al., 2013) has indicated that subthreshold depression has common clinical features with MDD and equally poor outcome. The clinical characteristics of both conditions are depressed mood, suicidal ideation and high comorbidity. However, little attention has been given to neurofunctional changes in subthreshold depression.

A recent study of subthreshold depression in the elderly ( $66.5 \pm 5.7$  years) using the resting state functional magnetic resonance imaging (fMRI) has reported lower regional homogeneity in the right orbitofrontal cortex, left dorsolateral prefrontal cortex

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(DLPFC), and the left middle frontal cortex, as well as higher regional homogeneity in the bilateral insula and right DLPFC (Ma et al., 2013). Another study has suggested that the medial frontal gyrus, anterior cingulate cortex, insula, and amygdala (key regions in the experience of affective states) are important for discriminating between individuals with subthreshold depression and controls (Modinos et al., 2013). Moreover, hyperactivity of the parietal cortex during linear-order reasoning has been reported in individuals with subthreshold depression (Hinton et al., 2014).

The National Institute for Health and Care Excellence guidelines recommend low intensity psychological interventions for individuals with subthreshold depression (NCCMH, 2010). Moreover, because of increasing concerns about the adverse effects of medication in youth, such as suicidal ideations and behaviors (Hammad et al., 2006), cognitive behavior therapy (CBT) is considered an important treatment for this age group. Behavioral activation (BA) is a type of cognitive behavioral therapy that focuses directly on changing behaviors by increasing opportunities for obtaining positive reinforcements from the environment (Jacobson et al., 2001). The rationale of BA is simpler than CBT in general, and therefore, BA has been focused for its feasibility (Ekers et al., 2011).

Expecting a reward, or a loss, is considered an important psychological mechanism of behavioral activation. The goal of BA is to help patients expand access to positive reinforcing activities, in order to increase the rate of response-contingent positive reinforcement available to them (Martell et al., 2001). In BA, patients conduct behavioral experiments and reviewing activities that make them feel rewarding and give a feeling of accomplishment. As a result, patients become able to create reasonable expectations about positively reinforcing activities. We focused on this anticipation phase and attempted to identify neural mechanisms of BA. The monetary incentive delay (MID) task has been useful for probing neurocircuitry related to incentive-motivation. This task enables us to separate anticipatory reward processing from consummatory reward processing. Changes in the neurocircuitry underlying reward processing in MDD has been investigated using neuroimaging approaches (Knutson et al., 2008; Pizzagalli et al., 2009; Schiller et al., 2013; Stoy et al., 2012). A recent meta-analysis on neural correlates of reward processing in MDD (Zhang et al., 2013) has suggested a reduced caudate response, and increased middle frontal gyrus activation during monetary reward anticipation. However, there are no consistent findings regarding loss anticipation in MDD.

To our knowledge, there are two studies that have focused on BA-related brain changes in MDD. The first study reported that BA increased activation of the right caudate nucleus, inferior temporal cortex, as well as paracingulate and orbitofrontal gyri during reward processing in a wheel of fortune task (Dichter et al., 2009). In the second study, the same investigators evaluated BA-related changes in neural correlates of cognitive control in an affective context with the same participants (Dichter et al., 2010). These studies found that BA resulted in decreased activation of the prefrontal structure, including the right frontal pole, orbitofrontal cortex, and left paracingulate gyrus, in response to stimuli related with cognitive control in the sad context.

However, neural response related to loss anticipation has not been evaluated to date and remain ambiguous. Furthermore, the potential of behavioral activation treatment to change neurobiological responses in subthreshold depression has not been evaluated and the accurate identification of these changes remains a major challenge.

The main goal of this investigation was to evaluate neural effects of behavioral activation treatment on subthreshold depression. The second goal of the study was to extend research on neural changes in subthreshold depression during gain/loss anticipation. We investigated neural responses of participants

with subthreshold depression using the MID task, before and after treatment with BA. We hypothesized that the hypoactivation of striatum and altered activation of PFC during reward and loss anticipation would normalize after successful intervention with BA.

#### 2. Methods

### 2.1. Participants

The BDI-II is routinely administered yearly to all freshmen, as a part of the medical examination conducted by the Health Service Center of Hiroshima University. Freshman attending Hiroshima University were recruited for the randomized controlled trial of behavioral activation over a two-year period by using their BDI-II scores. Of 5106 freshmen between 2013 and 2014, 826 students, with the exception of part time students and those over 20 years of age, we randomly selected approximately half of 826 students and contacted them through the university e-mail system. Of these, 209 students with BDI-ll scores of 10 or more responded to our email. These students were assigned as participants in the study, after they signed a written informed consent form. These individuals responded to a structured clinical interview conducted by telephone, using the Japanese version of the Composite International Diagnostic Interview (CIDI). We then sent self-report scales to 209 participants through the internet. The scales included the Japanese version of BDI-ll (Kojima and Furukawa, 2003), BADS (Takagaki et al., 2013) and EROS (Kunisato et al., 2011). Of these participants, 205 completed and returned the self-report scales. Then, 118 individuals with subthreshold depression were selected. based on the criteria described below, and randomly allocated to an intervention (n=62), or to a non-treatment control group (n=56). Participants in the intervention group took part in a randomized controlled trial of behavioral activation consisting of five, weekly BA treatment sessions. The Inclusion criteria in the BA program were 18-19 years old freshman students at screening, and a BDI-ll score of 10 or more, participants who did not have a major depressive episode within the past one year, participants who gave their informed consent, and participants who were not undergoing psychopharmacological or psychological treatment and the willingness to give informed consent. Exclusion criteria were a diagnosis of MDD during the past year, a lifetime history of bipolar disorder, a lifetime history of taking psychiatric medications or undergoing psychotherapy, possibility of acute suicide attempts, difficulty in understanding the purpose of the study, or difficulty in completing the self-report scales due to serious mental condition, or severe physical disease. We used the CIDI to assess participants for 12-month history of MDD, Generalized Anxiety Disorder, Panic Disorder, Social phobia, and Posttraumatic Stress Disorder. Because of high comorbidity rate for depression and anxiety (GAD, PD, SP and PTSD) reported in previous research (Hirschfeld, 2001), we addressed GAD, PD, SP, and PTSD with special care.

Among the 62 participants allocated to the BA treatment group, 16 participants were randomly selected and enrolled in the fMRI study because of the limited availability of fMRI equipment for this study. The control group of the fMRI study included 16 participants with low BDI-Il scores of less than 10, who were also recruited from Hiroshima University. They also completed self-report scales and were assessed by using the CIDI. The inclusion criteria for control participants were 18–19 years of age, and a BDI-II score less than 10, whereas the exclusion criteria were identical to that of the BA group. Moreover, contraindication to magnetic resonance was an additional common exclusion criteria for both groups. We checked that no participants had smoking behavior, or

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