



Season-independent cognitive deficits in seasonal affective disorder and their relation to depressive symptoms



Liv Vadskjær Hjordt^{a,b,c}, Dea Siggaard Stenbæk^{a,b}, Brice Ozenne^{a,d}, Brenda Mc Mahon^{a,b},
Ida Hageman^e, Steen Gregers Hasselbalch^{a,b,c}, Gitte Moos Knudsen^{a,b,c,*}

^a Neurobiology Research Unit, Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark

^b Center for Integrated Molecular Brain Imaging, the Neuroscience Centre, Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark

^c Faculty of Health and Medical Sciences, University of Copenhagen, Blegdamsvej 3B, 2200 Copenhagen, Denmark

^d Department of Public Health, Section of Biostatistics, University of Copenhagen, 5 Øster Farimagsgade, Entrance B, 2nd floor, 1014 Copenhagen, Denmark

^e Psychiatric Center Copenhagen, Copenhagen University Hospital, 10 Edell Sauntes Allé, 2100 Copenhagen, Denmark

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ABSTRACT

Although cognitive impairments are common in depressed individuals, it is unclear which aspects of cognition are affected and whether they represent state or trait features of depression. We here exploited a naturalistic model, namely the seasonal fluctuations in depressed status in individuals with Seasonal Affective Disorder (SAD), to study depression-related cognition, longitudinally. Twenty-nine medication-free individuals diagnosed with winter-SAD and 30 demographically matched healthy controls with no seasonality symptoms completed the Letter-number Sequencing task (LNS), the Symbol Digit Modalities Test (SDMT) and the Simple Reaction Time (SRT) twice; in summer and in winter. Compared to controls, SAD individuals showed significant season-independent impairments in tasks measuring working memory (LNS), cognitive processing speed (SDMT) and motor speed (SRT). In SAD individuals, cognitive processing speed was significantly negatively associated with the seasonal change in SAD depressive symptoms. We present novel evidence that in SAD individuals, working memory, cognitive processing- and motor speed is not only impaired in the winter but also in the summer. This suggests that certain cognitive impairments are SAD traits. Furthermore, impairments in cognitive processing speed appear to be related to depressive symptoms in SAD. Reduced processing speed may thus constitute a SAD vulnerability trait marker.

1. Introduction

Seasonal affective disorder (SAD), is a subtype of major depression disorder (MDD) and is typically characterised by recurrent major depression episodes (MDE) in autumn or winter and full remission the following spring or summer (American Psychiatric Association, 1994; Rosenthal et al., 1984). In countries at Northern latitudes, SAD is frequent and approximately 12% of the Copenhagen population reports that they have manifest symptoms of SAD, and another 5% report sub-syndromal symptoms of SAD (Dam et al., 1998). Additionally, among subtypes of major depression, SAD has the highest incidence of recurrence on an almost yearly basis, and increasing number of MDEs seems to be associated with increasing risk of recurrence, increasing symptomatic severity of episodes and decreasing threshold for developing episodes (Kessing and Andersen, 2017), emphasizing the importance of studying vulnerability to SAD relapses. Besides affective symptoms

(American Psychiatric Association, 1994), SAD is associated with marked and clinically significant cognitive impairments compared to healthy controls (Drake et al., 1996; Harmer et al., 2012; Hjordt et al., 2017; Jensen et al., 2015; Michalon et al., 1997; O'Brien et al., 1993). Such impairments may influence regulation of depressed affect (Bless and Fiedler, 2012) as well as social and professional functioning (Evans et al., 2014; Pendse et al., 2003; Schlager et al., 1995). However, while general knowledge about SAD is accumulating, a critical gap exists in our knowledge of cognitive impairments in individuals with SAD and their implications to the development of depressive symptoms. For example, knowledge of whether cognitive impairments observed during symptomatic phase disappear or remain during remitted phase has implications for targeted therapeutic interventions and for identifying vulnerability for SAD. Current cognitive models of SAD are mainly based on studies examining cognitive functions in MDD. However, such studies are hampered by several methodological limitations, such as

* Corresponding author at: Neurobiology Research Unit, Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark.

E-mail addresses: hansen@nru.dk (L.V. Hjordt), dea.stenbaek@nru.dk (D.S. Stenbæk), brice.ozenne@nru.dk (B. Ozenne), brenda.mcmahon@nru.dk (B. Mc Mahon), ida.hageman.pedersen@regionh.dk (I. Hageman), steen.gregers.hasselbalch@nru.dk (S.G. Hasselbalch), gmk@nru.dk (G.M. Knudsen).

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insufficient definitions of remission and confounding effects of psychotropic drugs, which affect cognitive functions (Rosenblat et al., 2015; Wingo et al., 2009). These issues also complicate separating persisting traits from transient state-markers. However, SAD populations allow us to study distinctively characterised seasonal fluctuations in affectivity, which makes it a “natural” and powerful model for disentangling traits from state-dependent cognitive features of major depression.

Two components of information processing have been shown to be especially affected in MDD: working memory and processing speed. Whereas working memory deficits in currently depressed individuals are reported in most (Austin et al., 1999; Elliott et al., 1996; Hammar and Ardal, 2009; Harvey et al., 2004; Landro et al., 2001; Nebes et al., 2000; Porter et al., 2003; Rose and Ebmeier, 2006; Taylor Tavares et al., 2007) but not all studies (Elderkin-Thompson et al., 2003; Purcell et al., 1997; Sweeney et al., 2000), it is less clear whether these deficits disappear, improve, or remain, in the remitted phase. Two studies have reported that clinically remitted depressed individuals experience residual deficits in working memory (Marcos et al., 1994; Weiland-Fiedler et al., 2004). However, these studies employed cross-sectional designs, rendering it difficult to determine whether these impairments were in fact present during the symptomatic phase and to what degree, or whether they represent cognitive scars in the aftermath of major depression. We have previously reported that individuals with SAD exhibit a significantly larger decrease in recall of positive words during symptomatic phase in winter compared to healthy controls (Jensen et al., 2015) and that this may reflect specific deficits within working memory related to emotional information. However, to our knowledge, no other studies have examined working memory functions in SAD.

Processing speed has been shown to be slowed in currently depressed (Den Hartog et al., 2003; Kertzman et al., 2010; Sobin and Sackeim, 1997) and remitted depressed individuals (Beats et al., 1996; Hasselbalch et al., 2011; Weiland-Fiedler et al., 2004). Two studies have reported that slowing of processing speed is partly responsible for working memory deficits observed in MDD (Nebes et al., 2000; Salthouse, 1996), supporting the critical role of lower-order processing speed for higher-order cognitive functions (e.g. working memory). Moreover, many studies have stressed that individuals with MDD show slower reaction times than do healthy control subjects (Bennabi et al., 2013a; Marazziti et al., 2010). Given that most studies have evaluated processing speed in MDD using tasks requiring both cognitive and motor processes, little is known about the relative contribution of slowed motor processes to slowed cognitive processes. Thus, studies that wish to address cognitive processing impairments, also need to adjust for the potential confounding effects of a motor slowing on cognitive processing speed.

One small prospective study reported that individuals with SAD showed significantly slower responses to a spatial recognition test in summer and winter as compared to controls (O'Brien et al., 1993). The authors interpreted these findings as a generally slowed cognitive processing capacity rather than simply slower motor response, supported by another study in which motor speed was the same in SAD and controls (Michalon et al., 1997). Consequently, more studies are needed to address patterns of cognitive processing- and motor speed deficits as a function of season in SAD.

In a longitudinal study design, we aim to examine trait-like and state-dependent deficits in working memory, cognitive processing- and motor speed in individuals diagnosed with SAD and age-, gender- and education-matched healthy controls. We further aim to examine whether any such impairments in working memory in individuals with SAD arises from slowed cognitive processing speed. Finally, we aim to examine whether any trait-like or state-dependent deficits in cognitive functions are associated with change in depressive symptom severity in SAD. Based on the existing SAD and MDD literature, we hypothesise that:

1. In the winter, individuals with SAD exhibit impairments in working memory compared to controls, whereas working memory performances in SAD normalizes in the summer.
2. In winter and summer, individuals with SAD exhibit impairments in cognitive processing speed compared to controls.
3. Trait-like or state-dependent impairments within working memory, cognitive processing- and motor speed are associated with seasonal change in severity of SAD symptoms.

2. Methods

2.1. Recruitment

Recruitment procedures are similar to those described previously (Hjordt et al., 2017). Volunteers were recruited from the community via websites, newspaper advertisements, and through bulletin boards at libraries. Exclusion criteria were age < 18 or > 45 years, current or lifetime history of neurological, significant somatic or psychiatric illness, other than SAD for potential individuals with SAD, as determined by physical exam, routine blood tests and history, severe head trauma, visual or hearing impairment, known retinal pathology, smoking or use of illegal drugs including cannabis within the last week or more than 10 times in lifetime (cannabis was allowed up to 50 times in lifetime), alcohol abuse, current or planned pregnancy, night shift work or travelling to destinations with a different climate 6 months prior to test sessions. Additionally, individuals eligible for the SAD group were not allowed to have taken psychotropic drugs or received bright light therapy within the past year.

2.2. Screening procedures

Subsequent to initial screening, eligible individuals completed a Danish version of the Seasonal Pattern Affective Questionnaire (SPAQ) (Rosenthal et al., 1987); a self-administered instrument evaluating seasonal variations in sleep, social activity, appetite, mood, body weight and energy. The score of each item is summed to obtain the Global Seasonality Score [GSS; range from 0 to 24]. Healthy volunteers were required to have a GSS ≤ 10 and reporting no problems with seasonality, whereas SAD candidates were required to have a GSS ≥ 11, and a seasonality problem rating of at least moderate (Kasper et al., 1989).

Trained psychiatrists interviewed SAD candidates in winter and in summer to ensure that they presented with SAD during winter (November-February) and full remission in summer (May-July). The SAD diagnosis was confirmed when individuals met the ICD-10 diagnostic criteria for a major depression (WHO, 1994) and the SAD criteria suggested by Rosenthal et al. (1984). To exclude any other axis I and axis II illnesses before final inclusion, all referred candidates underwent a Schedules for Clinical Assessment in Neuropsychiatry interview (SCAN V. 2.1) (Wing et al., 1990). Symptom severity in summer and winter was assessed using The Structured Interview Guide for the Hamilton Rating Scale for Depression-Seasonal Affective Disorder (SIGH-SAD) (Williams, 1988). The SIGH-SAD combines the 21 items from the Hamilton Depression Rating Scale (Hamilton, 1960) with eight additional items to assess atypical depressive symptoms associated with SAD. Remission from depression was defined by a SIGH-SAD score ≤ 8 (Eastman et al., 1998; Terman et al., 1998), and by the clinical evaluation of the psychiatrists. For all individuals, the subjective ratings of depressive symptoms in summer and winter, were assessed with the Major Depression Inventory (MDI) (Bech et al., 2001). Controls with a MDI score > 21 in summer or in winter, indicating depressed mood, were excluded from the study.

2.3. Participants

The screening procedure resulted in a total of 44 SAD candidates

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