Sleep spindle activity and psychotic experiences: the mediating roles of attentional performance and perceptual distortions

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Abstract

Deficits in sleep spindle activity in individuals with psychotic disorders could represent a neurobiological marker of (premorbid) attentional deficits and perceptual distortions that ultimately add to psychotic symptom formation. We analysed the links between sleep spindle activity and psychotic experiences and probed for the mediating roles of attentional performance and self-reported perceptual distortions in a community sample (N=70; mean age 26.33 (SD = 4.84)). Polysomnography was recorded during a 90-minute daytime nap and sleep spindle activity was detected using an automated algorithm. Duration, amplitude, and density from slow (10-13 Hz) and fast (13-16 Hz) sleep spindles were extracted. Attentional performance was assessed via subtests of the Test of Attentional Performance (TAP) and with an antisaccadic eye movement task. Psychotic experiences (i.e., paranoid thoughts; hallucinatory experiences) and perceptual distortions (i.e., anomalous perceptions; sensory gating deficits) were assessed via self-report questionnaires. We conducted sequential mediation analyses with sleep spindle activity as predictor, psychotic experiences as dependent variable, and attentional performance and perceptual distortions as mediators. We found reduced right central slow and fast sleep spindle amplitude to be associated with paranoid thoughts. Increased antisaccadic error rate was associated with anomalous perceptions and perceptual distortions were associated with psychotic experiences. We did not find significant mediation effects. The findings support the notion that reduced sleep spindle activity is involved in psychotic symptom formation and that decreased antisaccadic performance is indicative of perceptual distortions as potential precursors for psychotic experiences. However, further research is needed to corroborate the here proposed mediation hypothesis.
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Abstract

Deficits in sleep spindle activity in individuals with psychotic disorders could represent a neurobiological marker of (premorbid) attentional deficits and perceptual distortions that ultimately add to psychotic symptom formation. We analysed the links between sleep spindle activity and psychotic experiences and probed for the mediating roles of attentional performance and self-reported perceptual distortions in a community sample ($N=70$; mean age $26.33$ ($SD=4.84$)). Polysomnography was recorded during a 90-minute daytime nap and sleep spindle activity was detected using an automated algorithm. Duration, amplitude, and density from slow (10-13 Hz) and fast (13-16 Hz) sleep spindles were extracted. Attentional performance was assessed via subtests of the Test of Attentional Performance (TAP) and with an antisaccadic eye movement task. Psychotic experiences (i.e., paranoid thoughts; hallucinatory experiences) and perceptual distortions (i.e., anomalous perceptions; sensory gating deficits) were assessed via self-report questionnaires. We conducted sequential mediation analyses with sleep spindle activity as predictor, psychotic experiences as dependent variable, and attentional performance and perceptual distortions as mediators. We found reduced right central slow and fast sleep spindle amplitude to be associated with paranoid thoughts. Increased antisaccadic error rate was associated with anomalous perceptions and perceptual distortions were associated with psychotic experiences. We did not find significant mediation effects. The findings support the notion that reduced sleep spindle activity is involved in psychotic symptom formation and that decreased antisaccadic performance is indicative of perceptual distortions as potential precursors for psychotic experiences. However, further research is needed to corroborate the here proposed mediation hypothesis.

Keywords: schizophrenia; neurodevelopmental disorders; thalamus; GABA; risk factor

Introduction

There is growing evidence for deficits in sleep spindle activity in individuals with psychotic disorders (Zhang, Quiñones, & Ferrarelli, 2020). Sleep spindles – sudden waxing/waning oscillations of 10-16 Hz during non-rapid eye movement sleep (NREM) – are generated in the thalamic reticular nucleus (TRN; Fernandez & Lüthi, 2020) and reflect distinct activity within thalamocortical circuits (Born & Wilhelm, 2012). Deficits in sleep spindle activity have been found in chronic patients with psychotic disorders (Ferrarelli et al., 2010), first-degree relatives (Schilling et al., 2017), adolescent patients with an early onset (Gerstenberg et al., 2020) and antipsychotic-naïve patients (Manoach et al., 2014), suggesting sleep spindle deficits to be unrelated to medication or duration of illness. Additionally, sleep spindle deficits were found to be associated with greater positive symptom severity in psychotic patients (Wamsley et al., 2012), with magical ideation, an index of liability to delusional beliefs, in healthy adults (Lustenberger et al., 2015), and with schizotypal personality traits in healthy adolescents (Kuula et al., 2019). Taken together, sleep spindle deficits are observable across the continuum of psychosis and are associated with (subclinical) psychotic symptoms. Building on these findings, it has been suggested that sleep spindle deficits represent an endophenotype of neurodevelopmental disorders, including psychotic disorders (Manoach & Stickgold, 2019; Steullet, 2020), and reflect abnormal activity in thalamocortical circuits (Baran et al., 2019; Ferrarelli et al., 2010; Ferrarelli & Tononi, 2011; Pinault, 2011; Pratt & Morris, 2015; Vukadinovic, 2011). However, the way in which sleep spindle deficits contribute to psychotic symptom formation is not well understood.

The TRN – a thin inhibitory gamma-aminobutyric-acid (GABA)-ergic shell-like structure that envelops the thalamus – is a key node in the brain’s perceptual and attentional networks (Pinault, 2004; Zikopoulos & Barbas, 2007, 2012). It filters bottom-up sensory information flow from the periphery to the cortex (i.e., sensory gating; Krause, Hoffmann, & Hajós, 2003) and regulates top-down processes in thalamocortical circuits involved in attentional modulation (Chen, Wimmer, Wilson, & Halassa, 2016; Halassa et al., 2014; Halassa & Kastner, 2017; McAlonan, Cavanaugh, & Wurtz, 2006; Wimmer et al., 2015). A TRN-dysfunction – indicated by decreased sleep spindle activity – would lead to an increased and less filtered forwarding of sensory information (i.e., sensory gating deficits) and reduced attentional modulation (i.e., attention deficits). Consequently, perceptual distortions and misconceptions about external events would become more likely,
which in turn could set the foundation for developing hallucinations or delusional beliefs. Thus, it seems reasonable to suggest that attentional deficits and perceptual distortions mediate the relation between sleep spindle deficits and psychotic symptoms.

Most of the postulated links within this mediation hypothesis are backed up by empirical findings, which suggest a causal link between TRN-(spindle)-activity and attentional as well as sensory processes (for reviews, see Behrendt, 2006; Krol, Wimmer, Halassa, & Feng, 2018; Manoach & Stickgold, 2019; Pinault, 2011; Young & Wimmer, 2017). These findings are primarily derived from animal models. Only two studies have explicitly examined the link between sleep spindle activity and attention in humans in the context of psychosis. Forest et al. (2007) found sleep spindle activity to be negatively associated with reaction times in a selective attention task in both patients with psychotic disorders and healthy controls. Similarly, Keshavan, Montrose, Miewald, and Jindal (2011) found reduced sleep spindle activity to be associated with impaired performance in task switching in patients with psychotic disorders. Nonetheless, the assumption that the association between reduced sleep spindle activity and psychotic experiences is mediated through impaired attentional performances and perceptual distortions is yet to be tested.

We investigated whether sleep spindle activity during a nap is associated with psychotic experiences (i.e., paranoid thoughts, hallucinatory experiences) and examined the putative mediating roles of attentional performance in different domains (including antisaccadic eye movement) as well as self-reported perceptual distortions (i.e., phenomena of anomalous perceptions, sensory gating deficits). Based on the continuity of both hallucinations and paranoid beliefs in the general population (Van Os, Linscott, Myin-Germeyys, Delespau, & Krabbendam, 2009), we recruited participants from the general population to test our hypotheses. We chose afternoon naps because they have shown to reliably represent nocturnal sleep spindle activity (Mylonas et al., 2019) and offer a less time-consuming alternative to overnight sleep studies. We expected sleep spindle activity to be negatively associated with psychotic experiences. Furthermore, we examined sequential mediation models including sleep spindle activity as predictor, psychotic experiences as dependent variable, and attentional performance as well as self-reported perceptual distortions as mediators. The proposed mediation model can be seen in Figure 1.

Materials and Methods

2.1 Participants

For inclusion, participants had to be between 18 and 39 years old, have no history of any neurological condition and not to be taking any prescribed psychiatric drug. Participants were compensated with 40€ or course credits. This study was approved by the local ethics committee and all participants gave their written informed consent beforehand.

2.2 Procedure

First, inclusion criteria were assessed via telephone screening and participants were instructed to refrain from caffeine and nicotine consumption on the day of testing. To increase sleep pressure, participants were asked to go to bed one hour later than usual the day before testing and to get up one hour earlier than usual on the day of testing. On the day of testing, all participants went through a laboratory assessment at the Universität Hamburg (Germany) including self-report questionnaires, which were programmed using Qualtrics software (Qualtrics, Provo, UT), a 6-minutes resting state electroencephalography (EEG; three minutes eyes-open, three minutes eyes-closed)11Not part of this study., a 90-minute daytime napping polysomnography, tests of attentional performance and antisaccadic eye movement. The order of attentional tests was randomized across participants to avoid order effects.

2.3 Polysomnography

Polysomnography was conducted with SOMNO HD (SOMNOmedics GmbH, Randersacker, Germany). EEG was recorded with gold cup electrodes at six locations (F3, F4, C3, C4, O1 and O2) and two channels for the mastoids (M1, M2) according to the standardized 10/20 system. The electromyogram (EMG) and electro-oculogram (EOG) were measured by using gold cup electrodes at three (EMG) and two (EOG) locations.
Additionally, a ground electrode in the middle of the forehead and an online reference (Cz) were used. The sampling rate was 256 Hz and the hardware filters for SOMNO HD were 0.2 - 35 Hz by default. Electrocardiogram (ECG) was measured by using four disposable adhesive electrodes (Ratiomed, megro GmbH, Wesel, Germany), while a finger clip measured blood oxygen level as well as pulse and a respiration belt measured chest breathing. Sleep stages were scored manually using the DOMINO Software (v2.9; SOMNOmedics GmbH, Germany) by two independent raters (including the first author) in 30-seconds epochs according to the American Academy of Sleep Medicine guidelines (v2.4).

2.4 Sleep spindle analyses

Spindles were computationally extracted from NREM sleep (sleep stages N2 and N3) using the methods described by Merikanto et al. (2017) and Ferrarelli et al. (2010). The manually scored sleep stages were extracted as European Data Format (EDF) from the DOMINO software and then further analysed for spindle detection using functions of EEGLab (Version 2019.1; Delorme & Makeig, 2004) running on Matlab R2019a (The Mathworks Inc., USA). To ensure data integrity, 12 participants (N =6 with NREM sleep < 20 minutes; N =6 with overall impedances > 10 k) were removed from sleep spindle analyses. Spindle analyses were conducted in two different frequency bands for slow (10 - 13 Hz) and fast (13 - 16 Hz) spindles. See Supplement A for further information on spindle extraction.

To assess sleep spindle activity, we extracted the average sleep spindle duration (in ms), amplitude (in μV), and density (number of spindles per 30 seconds) for the frontal, central, and occipital derivations. These parameters have repeatedly been shown to be deficient in patients with psychotic disorders (Castelnovo, Graziano, Ferrarelli, & D'Agostino, 2018; Lai et al., 2022). Descriptive data of the sleep spindle parameters of each channel separately for slow and fast sleep spindles are given in Table 1.

2.5 Assessment of psychotic experiences

The Paranoia Checklist (PCL; Freeman et al., 2005; German: Lincoln, 2017) is a self-report measure of paranoid thoughts (e.g., “I need to be on guard against others”). The frequency subscale of paranoid thoughts was used here. The PCL demonstrated internal consistency and good convergent validity (Freeman et al., 2005). Items range from mild persecutory ideas to more severe paranoid ideations. Cronbach’s alpha was 0.875 here.

The Launay-Slade Hallucinations Scale – Extended (LSHS-E; Larøi, Marczewski, & Van der Linden, 2004; German: Lincoln, Keller, & Rief, 2009) is a self-report measure for assessing the multidimensionality of hallucinatory experiences, tapping into all major sensory modalities (e.g., “I have had the feeling of touching something or being touched and then found that nothing or no one was there”). The LSHS-E has demonstrated reliability and validity in clinical and non-clinical samples (Larøi et al., 2004; Serper, Dill, Chang, Kot, & Elliot, 2005). Cronbach’s alpha was 0.816 here.

2.6 Assessment of mediators

2.6.1 Attentional performance

We used three subtests of the computerized Test of Attentional Performance (TAP; v2.3.1, German version; Zimmermann & Fimm, 2004) to measure different aspects of attentional performance: (1) Divided Attention measures the ability to attend simultaneously ongoing external processes, (2) Visual Scanning measures the ability to explore the visual environment, and (3) Flexibility (set shifting) measures the ability to shift attention to newly relevant aspects of a situation. Outcome measures were T-Scores corrected for age and education.

To measure attention-related oculomotor behaviour, we used an antisaccadic eye movement task following an internationally standardized protocol (Antoniades et al., 2013). For that, participants were instructed to look at a dot in the centre and to look in the opposite direction as fast as they can after a new dot appeared on the left or the right. We recorded eye movements during the task using an RED250 infrared remote eye tracking system (SensoMotoric Instruments, Teltow, Germany) with a high-speed sampling rate.
of 250 Hz, an accuracy of 0.4° visual angle and a spatial resolution of 0.03° visual angle. The stimuli were presented with Experiment Center 3.7 (SensoMotoric Instruments) on a widescreen stimulus monitor with a resolution of 1680 x 1050 pixels and 90 dpi. All raw gaze data were processed and exported using BeGaze 3.7 software (SensoMotoric Instruments). Outcome measures were antisaccadic gain (i.e., spatial accuracy of correct saccades in percentages), latency (i.e., period from appearance of the peripheral dot to the start of a correct saccade), and error rate (i.e., percentages of saccades in the right direction) and were calculated using the Statistical Package for Social Sciences (SPSS; Version 27).

2.6.2 Self-reported perceptual distortions

The Cardiff Anomalous Perception Scale (CAPS; Bell, Halligan, & Ellis, 2006; translated for and used in a study by Stuke, Stuke, Weilnhammer, & Schmack, 2017) is a self-report measure of anomalous perceptual and sensational experiences (e.g., “Do you ever have the sensation that your body, or a part of it, is changing or has changed shape?”). Every experience endorsed is additionally rated on subscales for associated distress, intrusiveness, and frequency. We used the total score for our analyses, which was calculated by adding up all subscale ratings. The CAPS demonstrated good reliability and validity (Bell, Halligan, Pugh, & Freeman, 2011). Cronbach’s alpha of the total score was 0.934 here.

The Sensory Gating Inventory (SGI; Hetrick, Erickson, & Smith, 2012; German version: the SGI was translated into German by the first author and was backtranslated by a British native speaker) is a self-report measure for assessing phenomena related to sensory gating deficits (e.g., “At times I have feelings of being flooded by sounds”). The SGI demonstrated good reliability and validity (Micoulaud-Franchi et al., 2014) and provides a total score as well as subscales for Perceptual Modulation (i.e., modulation of stimulus intensity and perceptual inundation), Over-Inclusion (i.e., anomalies of radial attention as a result of a low threshold of perception), Distractibility (i.e., anomalies of focal attention), and Fatigue-Stress Modulation (i.e., vulnerability of perceptual and attentional anomalies during periods of fatigue and stress). We used the total score for our analyses, which was calculated by adding up all item ratings. Cronbach’s alpha of the total score was 0.951 here.

Additionally, we screened for insomnia using the Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001) and participants provided information about their age, gender, education, occupation, napping habits, whether or not they had a physical or mental disorder (including psychoses), as well as their alcohol, caffeine, nicotine, and drug use during the preceding four weeks.

Strategy of data analyses

We used RStudio (Version 4.2.1) and SPSS (Version 27) for all statistical analyses. Prior to the analyses, all variables were visually inspected for normality and outliers (defined as values above or below the interquartile range multiplied by three). For mediation analyses, we used the sem function from the lavaan package for R (Rosseel, 2012). We observed missing values in slow sleep spindles (n = 23), fast sleep spindles (n = 18), TAP (n = 3), and antisaccadic variables (n = 3). Because Little’s test (Little, 1988) indicated that these values were missing completely at random (MCAR), we estimated models by full information maximum likelihood (FIML). First, we conducted structural equation modelling including all z-standardized manifest variables as indicators of our hypothesized latent factors (i.e., sleep spindle activity, attentional performance, perceptual distortions, and psychotic experiences), separately for slow and fast sleep spindles (see Supplement B for a schematic representation). As these models did not converge – most likely due to the large number of free parameters – we used principal component analyses (PCA) with Promax rotation (oblique; set to one-factor solutions), to reduce the number of free parameters. We performed PCA separately for slow and fast sleep spindle variables (including duration, amplitude, and frequency for each derivation) as well as for attention variables (including TAP and antisaccadic variables) and thereby selected those variables with the highest factor loadings for the sequential mediation analyses. Finally, we conducted separate regression-based path analyses (k = 8), including sleep spindle activity as predictor (slow vs fast), psychotic experiences as dependent variable (paranoid thoughts vs hallucinatory experiences), and attentional performance as well as self-reported perceptual distortions (CAPS vs SGI) as mediators (see Figure 1). We report standardized
beta estimates and standard errors next to bias-corrected 95% bootstrap confidence intervals (BCa 95% CI) based on 5,000 bootstrap samples to account for the non-normality of indirect effects. We considered estimates significant when the BCa 95% CI did not include zero.

Results

3.1 Sample characteristics

As can be seen in Table 2, the mean age was 26.33 (SD = 4.84), mean sleep duration was 63.26 minutes (SD = 17.14), and the mean NREM sleep duration was 49.33 minutes (SD = 15.70). Thirty-six (51.4%) were female, 46 (65.7%) were students, seven (10%) reported to have a physical illness, six (8.6%) showed scores indicative of moderate clinical insomnia, and one (1.4%) reported to have a mental disorder (i.e., eating disorder). See Supplementary C and D for descriptive statistics of the self-report questionnaires as well as attentional and antisaccadic eye movement performance.

3.2 Principal component analyses

Principal component analyses for sleep spindle activity indicated right central (C4) amplitude to show the highest factor loadings for both, slow and fast sleep spindle activity. For attentional performance variables, antisaccadic error rate showed the highest factor loading (see Supplement E for PCA results). Thus, these variables were used as predictors and first mediator in subsequent mediation analyses.

3.3 Sequential mediation with attentional performance (Mediator 1) and perceptual distortions (Mediator 2)

Tables 3 and 4 depict the results of the sequential mediation analyses. We observed significant negative direct effects of sleep spindle amplitude on paranoid thoughts in two models (path cp; Model 1 and Model 2). Sleep spindle amplitude did not predict attentional performance (path a1) nor perceptual distortions (path a2) in any of the models. Likewise, attentional performance did not predict psychotic experiences (path b1). Attentional performance significantly predicted perceptual distortions (path d) only in models measuring perceptual distortions via CAPS (Models 1, 2, 5, and 6). Perceptual distortions significantly predicted paranoid thoughts and hallucinatory experiences (path b2) across all models. Probing for mediation effects, none of the partial or total indirect effects were significant (Model 1: R² = 24.90; Model 2: R² = 25.11; Model 3: R² = 24.58; Model 4: R² = 24.92; Model 5: R² = 40.04; Model 6: R² = 38.09; Model 7: R² = 43.08; Model 8: R² = 43.45).

Discussion

This study aimed to examine the relation between sleep spindle activity and psychotic experiences and to determine whether this relation is mediated through attentional performance and self-reported perceptual distortions in a community sample.

Expanding on previous findings, we found decreased slow and fast right central sleep spindle amplitude to be significantly associated with paranoid thoughts (path cp). Thus, this study contributes to the existing literature by showing that besides magical ideation (Lustenberger et al., 2015) and schizotypal personality traits (Kuula et al., 2019), sleep spindle amplitude is also associated with (subclinical) paranoid thoughts in healthy individuals. However, this effect was not robust across all models with paranoid thoughts as dependent variable. We did not find significant associations between right central sleep spindle amplitude and hallucinations. As our sample exhibited relatively low ratings in paranoid thoughts and hallucinations, constraints in variance could have compromised the statistical power to detect an effect. Moreover, heterogenous survey methods to measure psychotic experiences between studies could explain our unexpected findings.

In line with basic theoretical assumptions (Nuechterlein, Dawson, & Green, 1994) and recent reviews (Javitt & Freedman, 2015), we found antisaccadic error rate to be associated with anomalous perceptions (path d) and anomalous perceptions as well as sensory gating deficits to be associated with psychotic experiences (path b2). Corroborating the notion of a potential pathway from aberrant TRN-mediated thalamocortical activity (i.e., reduced sleep spindle activity) to psychotic symptoms through aberrant sensory information processes
(Krol et al., 2018; Manoach & Stickgold, 2019), there is growing evidence for increased thalamic connectivity with somatosensory cortices (i.e., hyperconnectivity) and decreased connectivity with prefrontal cortices (i.e., hypoconnectivity) in individuals with psychotic disorders (Giraldo-Chica & Woodward, 2017; Zhang et al., 2021) and those with an elevated risk (Anticevic et al., 2015). Importantly, thalamic-somatosensory hyperconnectivity is also associated with decreased sleep spindle activity (Baran et al., 2019) and increased psychotic symptom severity (Ferri et al., 2018). Against this background, a thalamic-somatosensory hyperconnectivity would indicate an increased liability to perceptual distortions, while a thalamicprefrontal hypoconnectivity would indicate decreased attentional control, which both should mediate the relation between reduced sleep spindle activity and psychotic experiences.

We did not find significant associations between sleep spindle amplitude and antisaccadic error rate as well as perceptual distortions (paths a1 and a2). To the best of our knowledge, this is the first study to examine these associations. Given that so far research only showed associations between sleep spindle activity and task switching or selective attention (Forest et al., 2007; Keshavan et al., 2011), one could speculate that antisaccadic eye movement performance is not as relevant here. However, thalamic activity was found to be involved in volitional eye movements (Ostendorf, Liebermann, & Ploner, 2013; Tanaka & Kunimatsu, 2011), indicating neural regions associated with sleep spindle activity to be relevant for antisaccadic eye movements. Because our hypothesis of a relation between sleep spindle activity and perceptual distortions (path a2) was mainly derived from animal studies that utilised optogenetic techniques and measured single-cell activity (e.g., Young & Wimmer, 2017), one could argue that using only self-report measures to assess perceptual distortions is a shortcoming. It could be worthwhile to include psychophysiological measures indicative of perceptual distortions, such as prepulse inhibition (Swerdlov et al., 2018) and mismatch negativity (Umbricht & Kriljesb, 2005) in future studies.

Furthermore, despite marked deficits on a wide range of attentional functions (Kim et al., 2012; Luck, Leonard, Hahn, & Gold, 2019) and in antisaccadic eye movement performance (Myles, Rossell, Phillipou, Thomas, & Gurvich, 2017; Obyedkov et al., 2019) in individuals with psychotic disorders, we did not find a significant relation between antisaccadic error rate and psychotic experiences (path b1). Even though antisaccadic performance deficits are considered to be a phenotype for psychotic disorders (Clementz et al., 2022), studies on their relation to psychotic symptoms are relatively scarce (Subramaniam et al., 2018; Wan, Thomas, Pisipati, Jarvis, & Boutros, 2017) and potential associations may not be as pronounced at the lower end of the spectrum (i.e., low error rate, low symptom severity).

Finally, we did not find attentional performance and perceptual distortions to mediate the relation between sleep spindle activity and psychotic experiences. Given that we did not observe a significant indirect effect of sleep spindle amplitude on psychotic experiences (path cp), which often is viewed as the prerequisite for mediation effects (Preacher & Hayes, 2004), our models did not provide enough of an effect to be mediated.

Strengths of our study include the proposition and testing of a novel mechanistic model potentially underlying the links between sleep spindle deficits and psychotic symptoms. Further, our study offers a framework which is based on a solid theoretical, evidence-based, and interdisciplinary background that can be built upon in future studies. As to limitations, our study design was cross-sectional. The community sample comprised many well-educated students, but was nevertheless comparable to other studies in terms of psychotic experiences (Kammerer, Bub, & Lincoln, 2021), sleep spindle activity (Lustenberger et al., 2015; Manoach et al., 2014), and attention (TAP-scores were within the average range; Zimmermann & Fimm, 2004). Nonetheless, ratings of psychotic experiences, antisaccadic error rate and perceptual distortions were relatively low in variances and positively skewed (see Supplement F), which could have decreased the probability of detecting significant effects. Additionally, as our models did not converge, we decided to reduce the number of variables via PCA. Although it enabled us to conduct more parsimonious path analyses which were appropriate for our sample size, this strategy might have resulted in disregarding relevant predictor or mediator variables. To corroborate the mediation hypothesis we proposed, (longitudinal) replication studies with larger samples of clinical and at-risk participants are needed. Further, although the method we used for spindle detection is widely applied, there is no agreed-upon approach for quantifying sleep spindle activity, which limits the
comparability between studies. Along the same line, we support the idea of standardized and transparent methods to improve reproducibility and scientific progress (Poldrack et al., 2017; Steegen, Tuerlinckx, Gelman, & Vanpaemel, 2016).

Conclusion
To conclude, our findings add to the notion that decreased sleep spindle activity is involved in psychotic symptom formation in young adults and could represent a potential readout for aberrant (GABA-ergic) TRN-mediated thalamocortical activity. However, further studies are needed to determine the proposed mediating effect of aberrant sensory information processes in patients and healthy adults. Apart from that, our study showed antisaccadic error rate and anomalous perceptions to be related and that anomalous perceptions as well as sensory gating deficits are associated with psychotic experiences. This implies that early prevention strategies might benefit from targeting attentional processes (e.g., Attention Training Technique; ATT; Knowles, Foden, El-Deredy, & Wells, 2016) to increase attentional flexibility and control, and thereby decrease potential sensory overloads. This could be combined with addressing maladaptive interpretations of anomalous sensory experiences. For example, the technique of normalizing anomalous experiences, as is proposed in cognitive behavioural interventions for psychosis (Morrison & Barratt, 2010), aims at reducing the distress that arises from catastrophic interpretations of said experiences.

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