the british psychological society
promoting excellence in psychology

British Journal of Clinical Psychology (2020)

www.wileyonlinelibrary.com

Orginal article

Sleep problems and worrying precede psychotic symptoms during an online intervention for psychosis

Thies Lüdtke*[1,2], Gerit Pfuhl[1], Steffen Moritz[2], Nina Lee Rüegg[3], Thomas Berger[3] and Stefan Westermann[2,4]

[1]Department of Psychology, UiT – The Arctic University of Norway, Tromsø, Norway
[2]Department of Psychiatry and Psychotherapy, University Medical Center Hamburg-Eppendorf, Germany
[3]Department of Clinical Psychology and Psychotherapy, University of Bern, Switzerland
[4]Department of Clinical Psychology and Psychotherapy, MSH Medical School Hamburg, Germany

Objective. Experience sampling assessments (multiple assessments per day for approximately one week) indicate that positive symptoms fluctuate over time in psychosis. Precursors, such as sleep problems or worrying, predict these fluctuations. To date, it remains unclear whether the same precursors predict symptom variability also during treatment in an online intervention for psychosis, using assessments lying temporally further apart.

Methods. Participants completed brief intermediate online self-report assessments on their computers (up to every 7 days during a 2-month waiting period and up to twice every 6 days during a 2-month intervention period) within a randomized controlled trial. We monitored the course of paranoia, auditory verbal hallucinations, and their theory-driven precursors worrying, negative affect, self-esteem, self-reported cognitive biases, and quality of sleep in $n = 124$ participants ($M = 10.32$ assessments per participant; $SD = 6.07$). We tested group differences regarding the course of the composite of precursors, group differences regarding the effect of the composite on subsequent momentary psychotic symptoms, and the effect of each individual precursor on subsequent psychotic symptoms, using (lagged) linear mixed models.

Results. The course composite precursors over time and their lagged effect on subsequent momentary psychotic symptoms did not differ between groups. During the intervention, increased worrying and decreased quality of sleep preceded heightened momentary psychotic symptoms.

**Correspondence should be addressed to Thies Lüdtke, UiT – The Arctic University of Norway, Hansine Hansens veg 18, 9019 Tromsø (e-mail: thies.ludtke@uit.no).*

DOI:10.1111/bjc.12270

Conclusion. The regression-based design does not allow drawing causal conclusions. However, worrying and sleep problems likely represent underlying mechanisms of psychotic symptom variability during online psychosis treatment, indicating that experience sampling findings from everyday life generalize to interventions with assessments lying several days apart.

Practitioner points

- Worrying and sleep problems represent important mechanisms of symptom fluctuations during an online intervention for people with psychosis.
- Our findings further support the notion that worrying and sleep problems are important treatment targets in psychological interventions for people with psychosis.
- Momentary levels of worrying and quality of sleep can signal subsequent fluctuations of psychotic symptom severity so practitioners should monitor these variables during treatment.
- Worrying seems to predict subsequent paranoia specifically during treatment whereas quality of sleep predicts both paranoia and auditory verbal hallucinations

The experience sampling method (ESM; Myin-Germeys et al., 2009) has shed light on the temporal dynamics of positive symptoms in people with psychosis. In ESM studies on psychosis, participants report momentary psychotic symptoms and hypothesized correlates in diary-like self-report assessments repeatedly throughout the day. The longitudinal data enable researchers to predict momentary psychotic symptoms through precursors measured at a previous point in time. Precursors encompass worry and rumination (Hartley, Haddock, Vasconcelos, Emsley, & Barrowclough, 2014), sleep problems (e.g., Kasanova, Hajduk, Thewissen, & Myin-Germeys, 2019; Mulligan, Haddock, Emsley, Neil, & Kyle, 2016), and low self-esteem, defined as negative views about the self, such as being ashamed of oneself (Udachina, Varese, Myin-Germeys, & Bentall, 2014). The 'jumping to conclusions' bias (Dudley, Taylor, Wickham, & Hutton, 2016) precedes subsequent momentary paranoia as well (Lüdtke, Kriston, Schröder, Lincoln, & Moritz, 2017). One of the most consistently found precursors of paranoia is negative affect, a term comprising states such as feeling low, anxious, or lonely (Ben-Zeev, Ellington, Swendsen, & Granholm, 2011; Lüdtke et al., 2017; So et al., 2018). Of note, the association of depression and paranoia seems to rely on the assessment frequency. When measured weeks apart, paranoia predicts depression rather than vice versa (Moritz, Goritz, McLean, Westermann, & Brodbeck, 2017; Moritz et al., 2019). In sum, ESM studies have identified several time-variant precursors of psychotic symptoms in everyday life of people with psychosis.

Although ESM studies do not allow drawing causal conclusions, theoretical models (Freeman & Garety, 2014) and experimental findings (e.g., Reeve, Emsley, Sheaves, & Freeman, 2018) indicate causal relationships between aforementioned precursors and psychotic symptoms. Hence, it appears promising to target precursors of psychotic symptoms in therapeutic interventions, especially because the treatment of symptom precursors seems to coincide well with wishes and needs of participants (Freeman, Taylor, Molodynski, & Waite, 2019; Moritz, Berna, Jaeger, Westermann, & Nagel, 2017). There is initial support for the efficacy of interventions that focus on precursors to alter psychotic symptoms indirectly. For example, Freeman, Dunn, et al. (2015) examined a brief intervention based on cognitive behavioural therapy (CBT) targeting worry. The intervention led to decreased persecutory delusions in people with non-affective psychosis with changes in worry mediating the effect. Findings are less consistent for sleep problems. One trial found that a sleep intervention reduced insomnia, paranoia, and

hallucinations in healthy participants, with insomnia mediating the effect on psychotic symptoms (Freeman et al., 2017). In clinical samples, CBT-based sleep interventions likewise improved sleep but not psychotic symptoms (Freeman, Waite, et al., 2015; Hwang, Nam, & Lee, 2019). It is important to note that psychotic symptoms only served as secondary outcomes in these trials. Hence, the effect of sleep interventions on psychotic symptoms has not yet been adequately tested. CBT-based interventions targeting depression, a key component of the precursor negative affect, are rare (Upthegrove, Marwaha, & Birchwood, 2017). In one trial, a CBT-based online intervention targeting depression improved depressive but not positive symptoms (Moritz et al., 2016). Metacognitive training (Moritz & Woodward, 2007) aims at reducing, inter alia, participants' proneness to cognitive biases, which served as a symptom precursor in one ESM study as well (Lüdtke et al., 2017). One meta-analysis yielded mixed findings (van Oosterhout et al., 2016), but the majority of meta-analyses suggest that the metacognitive training improves psychotic symptoms (Eichner & Berna, 2016; Liu, Tang, Hung, Tsai, & Lin, 2018; Philipp et al., 2019). Taken together, treating precursors of psychotic symptoms can be beneficial for people with psychosis.

Targeting precursors of psychotic symptoms online

Based on aforementioned studies, Westermann et al. (2020) have developed a psychological online intervention (EviBaS) targeting not only psychotic symptoms but also potential precursors of psychosis in guided self-help modules. The authors successfully evaluated EviBaS in a randomized controlled trial. The intervention led to a significant reduction in a composite score of positive symptoms (Westermann et al., 2020). As EviBaS covers the treatment of a wide range of precursors, the aim of the present study was to investigate which of the addressed precursors would predict the course of positive symptoms during the intervention. To do so, we monitored psychotic symptom fluctuations and their presumed precursors similar to the procedure used in ESM trials. Assessments took place every seven days in the waiting period and up to twice per six days during the intervention period. We expected that within-participant changes of the following precursors would represent underlying mechanisms of psychotic symptom variability during the intervention: worrying, negative affect, self-esteem, self-reported cognitive biases, and quality of sleep. Unlike the interventionist-causal model approach (Freeman, Dunn, et al., 2015), our examination of precursors and symptom fluctuations is observational. It enables us to test multiple underlying mechanisms of psychotic symptom variability in parallel while relying on longitudinal data with a relatively high temporal resolution. For the present analyses, we focused on the course of positive symptoms (auditory verbal hallucinations and paranoia) for two reasons. First, the EviBaS intervention targets positive symptoms specifically. Hence, we aimed at identifying precursors of these symptoms throughout the intervention. Second, we selected symptom precursors based on previous ESM studies, which uniformly examined positive symptoms of psychosis (e.g., Hartley et al., 2014; So et al., 2018; Udachina et al., 2014).

Aims of the study

The design enabled us to examine several research questions. First, the longitudinal assessments allowed us to monitor the course of precursors over time thus offering insight into moment-to-moment effects of the EviBaS intervention beyond its already established effect on composite positive symptoms (Westermann et al., 2020). Second, we were able

to examine which mechanisms of psychotic symptom fluctuations occurred during the online intervention. The intervention aimed to ameliorate numerous candidate precursors so it was interesting to examine which of these candidates served as actual precursors (i.e., mechanisms of change) of subsequent symptoms, representing variables that are worth monitoring during treatment. Our approach focused on within-participant effects to obtain a more idiographic view, which researchers have called for in psychological and psychotherapy research (Piccirillo & Rodebaugh, 2019). The within-participant approach goes beyond traditional assessments of treatment mechanisms (e.g., group-based mediation analyses) as it uncovers processes within participants over time. To our knowledge, this is the first study to examine within-participant moment-to-moment effects of potential mechanisms of change during an online intervention for psychosis. Third, our analyses provide insights regarding the generalizability of ESM findings to other contexts. Usually, ESM studies examine precursors and outcomes several times per day for a short period, such as one week (e.g., So et al., 2018). The rationale of ESM studies is that within-day associations between symptoms and preceding variables can improve our general understanding of the phenomenology and aetiology of psychopathology (Myin-Germeys et al., 2018). We hypothesized that within-day ESM processes could represent micro-level equivalents of larger-scale mechanisms of symptom fluctuations. To illustrate, momentary levels of worrying vary throughout the day and precede subsequent psychotic symptoms (Hartley et al., 2014). At the same time, worry as a response to a physical assault predicts paranoia four weeks later (Freeman, Thompson, et al., 2013). The underlying process is the same in both cases in that increased worry precedes psychotic symptoms. It is appealing to assume that within-day associations generalize to other contexts as this implies that ESM studies can generate knowledge about larger-scale processes, such as the prediction of relapse or the prediction of symptomatic improvements during treatment, as examined here. In the present study, we tested this assumption by applying the ESM methodology of precursor-symptom associations to an eight-week assessment period (the assessments were several days apart). In addition, the randomized controlled design of the EviBaS trial enabled us to compare associations between precursors and psychotic symptoms in different contexts, namely a waiting condition (i.e., comparable to usual ESM studies) and a treatment condition. We expected that precursors would improve due to the intervention in the treatment condition whereas we expected precursors to fluctuate naturally over time in the waitlist condition. By comparing these conditions, we could investigate whether precursors influence subsequent symptoms only if they vary naturally (as in typical ESM studies) or also when they vary due to a psychological treatment.

We hypothesized that precursors would improve more in the treatment (i.e., immediate access) group compared to the waitlist (i.e., delayed access) group (hypothesis 1), that precursors would predict subsequent psychotic symptoms differently in the two groups (hypothesis 2), and that each individual precursor would predict subsequent psychotic symptoms during participation in the EviBaS intervention across groups (hypothesis 3).

Methods

The study is a registered (https://osf.io/gn8u5) secondary analysis of intermediate assessment data obtained from the EviBaS trial. For results of the main trial, see Westermann et al. (2020). The EviBaS main trial is a pre-registered (NCT02974400, clinicaltrials.gov) multi-centre parallel-group assessor-blind randomized controlled trial

with an allocation ratio of 1:1 evaluating the feasibility and efficacy of a CBT-based psychological online intervention (EviBaS) for people with psychosis. The intervention was guided, meaning that trained and supervised study staff with at least a bachelor's degree in psychology assisted participants using a secure messaging system. The EviBaS intervention encompassed 11 modules in total – one introductory module, one module on relapse prevention and nine modules targeting persecutory delusions, auditory verbal hallucinations, as well as a list of correlates of psychosis, namely worrying, low levels of mindfulness, poor social competence, low self-esteem, depression, sleep problems, and cognitive biases. The intervention's approach was twofold. It addressed psychotic symptoms directly by providing psychological models that explain hallucinations and feelings of persecution while offering exercises to decrease the participant's burden (two modules). On the other hand, the intervention targeted potential precursors of psychosis in order to ameliorate symptoms indirectly (seven modules).

Out of the 11 modules only the introductory module that introduced general CBT concepts, such as the ABC protocol (i.e., activating event, belief, consequences), and the final module on relapse prevention were mandatory. The nine remaining modules were not mandatory and participants could choose the order in which they completed them. Modules contained educational components and exercises conveyed via text, audio, and video files. At the beginning of a module, the texts, illustrations, or video files introduced the module's respective topic usually accompanied by a fictitious case example. Then a CBT-based model explained associations with psychotic symptoms and offered 'leverage points' for interventions. In the following, the module introduced specific interventions (e.g., CBT-based advice on how to improve sleep, such as avoiding meals before going to bed, addressing thoughts that compromise sleep using ABC protocols, or practicing relaxation exercises). Most modules contained worksheets so that participants filled in their own experiences to customize exercises. The module's final page summarized the main points and offered the possibility to give feedback to the guide. Completing a typical module took approximately 30 to 60 minutes. To promote participants' sense of autonomy we did not require participants to complete all modules. Instead, we considered the completion of eight modules over eight weeks as full adherence.

Trial design

At baseline, participants completed a self-report online assessment as well as a diagnostic interview via telephone. After confirming the inclusion criteria, we used a web-based randomization tool (based on random.org, RRID: SCR_008544) to allocate participants to the 'delayed access group' or the 'immediate access group'. We use these terms because waitlist participants received delayed access to the intervention after the waiting period whereas intervention group participants received immediate access. After eight weeks we invited all participants to complete a second self-report online assessment (post-assessment).

Here, we report data from intermediate assessments that we conducted throughout the EviBaS trial, both during the waiting and the intervention period to monitor the course of symptoms and presumed precursors. While using EviBaS, participants completed the assessments whenever they logged in, up to two times in 6 days. When a participant accessed the online intervention using an internet browser the short online questionnaire appeared on the screen. We chose this assessment format because we hoped to minimize the burden for participants by combining the assessments with the intervention. Delayed access participants completed the intermediate assessments once per week (invited via e-

mail) throughout the eight-week period between baseline and post-assessment, and additionally for another eight weeks after receiving access to EviBaS (identical to the immediate access group). For analyses on precursors of momentary symptoms during the intervention (i.e., hypothesis 3), we used data from all participants irrespective of their initial group allocation as we were interested in the processes during the intervention and not in a comparison between groups (see the boxes in Figure 1 with thick outlines).

Recruitment

We recruited participants in Germany and Switzerland. Local ethics committees approved the study (Cantonal Ethics Committee Bern, ID 03/14; German Society for Psychology, ID SM052015_CH). We contacted potential participants from a database listing former participants with schizophrenia spectrum diagnoses who had previously given their consent and we advertised the study online. Furthermore, we reached out to psychiatrists and psychiatric institutions.

All participants provided informed consent prior to participation. Participants were eligible if they were 18 years of age or older, had access to the Internet, showed sufficient command of the German language, had a lifetime diagnosis of a non-affective psychotic disorder (verified in telephone interview), and reported receiving antipsychotic treatment or psychotherapeutic/psychiatric consultations at least monthly. A mandatory emergency plan listed persons that participants could contact in case of an emergency. We excluded participants if they refused to complete the emergency plan, if they reported a diagnosis of a neurological disease, if they displayed acute suicidality, or an acute danger towards others.

For the present secondary analysis, we analysed data from EviBaS participants and additional data from participants who were not part of the EviBaS main trial because they did not meet the positive symptom severity threshold (a PANSS score of 3 or higher on at least one of the following items: delusions, hallucinations, or suspiciousness / persecutory delusions). These 'secondary track' participants completed the trial the same as the main trial participants but with two differences, as depicted on the right-hand side of Figure 1. First, secondary track participants did not complete a telephone interview at post-assessment (which was irrelevant for the present analyses). Second, delayed access participants in the secondary track had to wait 4 months to receive access to the intervention whereas the delayed access participants of the main trial received access directly after completing the post-assessment. The proportion of secondary track participants was equal in the immediate access group and delayed access group (see Table 1).

Measures

We only describe relevant measures. For a description of all measures included in the EviBaS trial, see Rüegg et al. (2018).

Baseline measures

We report participants' cumulated antipsychotic dosages, indicating the percentage of the maximum dosage of a certain drug because chlorpromazine equivalents have faced criticism (Danivas & Venkatasubramanian, 2013). We administered an adapted version of the Mini International Neuropsychiatric Interview (MINI; Lecrubier et al., 1997) to verify

Table 1. Sample characteristics at baseline (N = 124)

Characteristics	Delayed access (n = 66)	Immediate access (n = 58)	Statistics
Demographics			
Age in years, mean (*SD*)	40.88 (9.84)	42.34 (10.85)	t (122) = 0.789, p = .432
Gender (female/male)	37/29	38/20	χ^2 (1) = 1.155, p = .282
Years of education, mean (*SD*)	11.64 (1.76)	11.93 (1.41)	t (122) = 1.019, p = .310
Clinical variables			
'Secondary track' participants (%)	18 (27%)	15 (26%)	χ^2 (1) = 0.031, p = .859
Diagnosis of current psychotic episode (%)	23 (35%)	28 (48%)	χ^2 (1) = 2.299, p = .129
Diagnosis of current depressive episode (%)	17 (26%)	20 (34%)	χ^2 (1) = 1.123, p = .289
Reported taking antipsychotic medication (%)	59 (89%)	47 (81%)	χ^2 (1) = 1.738, p = .187
Cumulated antipsychotic dosage, mean (*SD*)	42.19 (37.87)	34.58 (36.22)	t (117) = 1.116, p = .267
PANSS total score, mean (*SD*)	50.47 (13.53)	51.67 (14.95)	t (122) = 0.469, p = .640

Note. SD = Standard Deviation; Secondary track participants = number of participants who were not part of the EviBaS main trial because they did not meet the positive symptom severity threshold (a PANSS score of 3 or higher on at least one of the following items: delusions, hallucinations, or suspiciousness/persecutory delusions); diagnoses were assessed using the MINI; all participants had a diagnosis of a previous psychotic episode; cumulated antipsychotic dosage refers to the percentage of the maximum dosage of the antipsychotic drugs that a participant received (not all participants provided information, hence the smaller df).

relevant psychiatric diagnoses and the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) to measure psychotic symptom severity. The PANSS showed good internal consistency in our sample (α = .85). We administered PANSS and MINI via telephone.

Intermediate assessments

Participants completed a 14-item intermediate assessment questionnaire up to once per week during the waiting period and up to two times in six days during the intervention period. Two items (α = .42) captured psychotic symptoms, 'I feel suspicious', adapted from previous ESM trials (Kramer et al., 2014; So et al., 2018) and 'I hear voices that no one else can hear', which was self-generated. We assessed worry with the item 'My worries overwhelm me', adapted from the Penn State Worry Questionnaire-Past Week (Stober & Bittencourt, 1998). We measured negative affect using two items (α = .80). The first item 'I am feeling down, depressed, or hopeless' stems from the Patient Health Questionnaire-9 (Kroenke, Spitzer, & Williams, 2001). We adopted the second item 'I feel anxious' from previous ESM trials (Kasanova et al., 2019; Kramer et al., 2014). We assessed self-esteem using the reverse-coded item 'I am satisfied with myself' adapted from the Rosenberg Self-Esteem scale (Rosenberg, 1965). We included two items to assess self-reported cognitive biases. The item 'When I am certain about something then I must be correct' was inspired

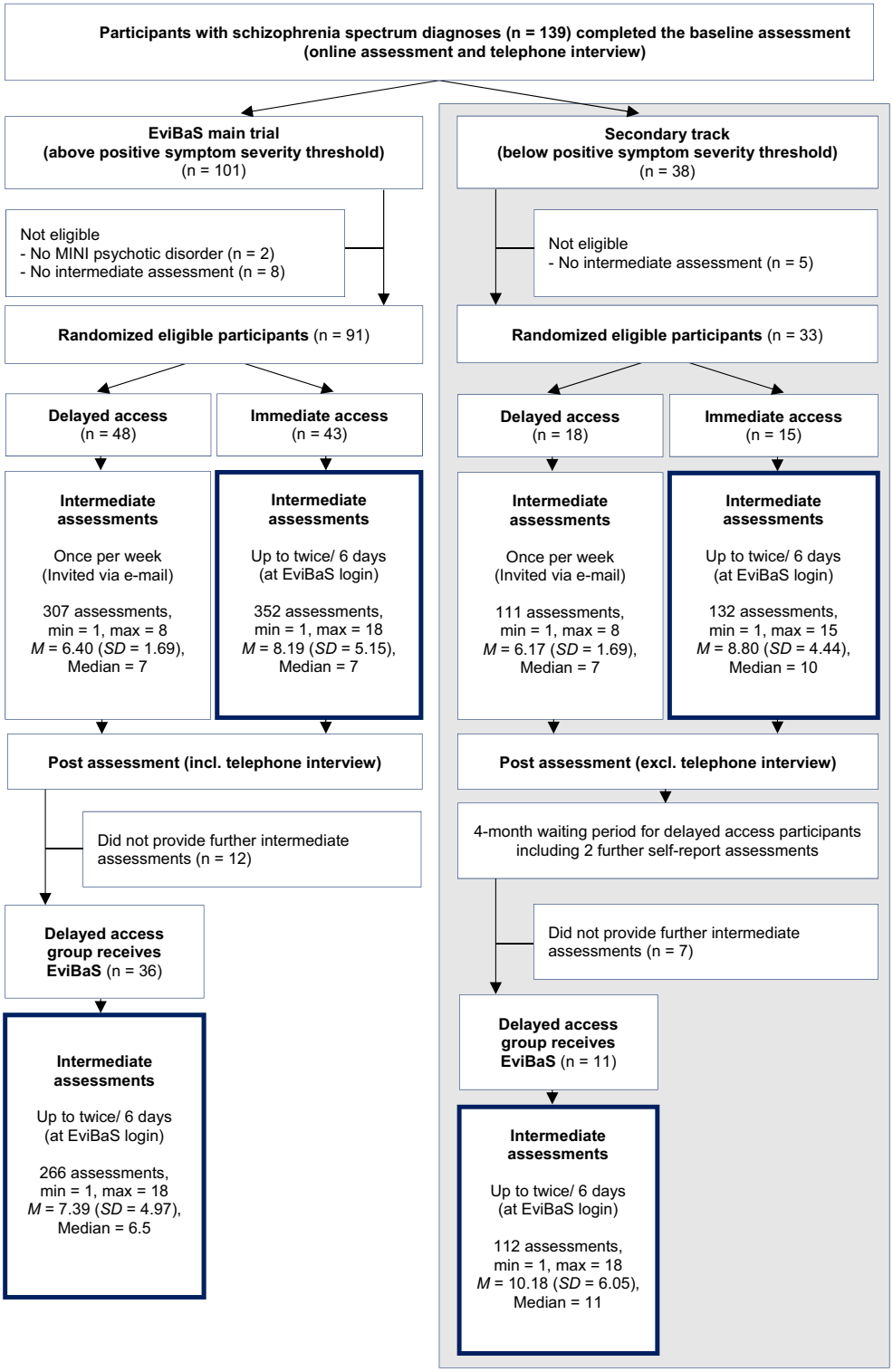

Figure 1. Flow chart (thick outlines highlight intermediate assessments used for hypothesis 3).

by the Beck Cognitive Insight Scale (Beck, Baruch, Balter, Steer, & Warman, 2004). The reverse-coded item 'I consider as much information as possible before I make a decision' aimed at assessing hasty data gathering. The items correlated negatively (after transforming the reverse-coded item), so that we analysed the items separately instead of using the scale ($r = -.277$). We assessed quality of sleep with the item 'The quality of my sleep is good'. We chose this unspecific wording to capture different types of sleep problems (Kasanova et al., 2019). The remaining items of the intermediate assessments were not relevant for the present analyses. Except for sleep, participants rated all items according to how they felt at the current moment. For analyses of hypotheses 1 and 2, we calculated a composite score of precursors defined as the sum of worry, negative affect, self-esteem, self-reported cognitive biases, and quality of sleep ($\alpha = .60$). The rationale of the composite score was to reduce the overall number of confirmatory analyses to maintain sufficient power.

Statistical analyses

We registered analyses prior to accessing the relevant data (https://osf.io/gn8u5). All analyses represent variants of linear mixed models. Mixed models allow accounting for the 'nested' structure of measurements clustered within participants, which is characteristic for longitudinal data. We did not impute missing values because mixed models are flexible in handling missing data (Twisk, 2019, p. 150).

For hypothesis 1 we tested whether the composite score of precursors (worry, negative affect, self-esteem, self-reported cognitive biases, and quality of sleep) improved more in the immediate access group compared to the delayed access group. The analysis relied on intermediate assessment data obtained between baseline and post-assessment. The statistical model included time, group (immediate vs. delayed access), and the time x group interaction. The composite score of precursors served as the outcome. Intermediate assessments took place at different points in time during the intervention compared to the waiting period (upon EviBaS login vs. once per week). To obtain a comparable number of assessments in both groups, we aggregated the assessments by averaging the respective outcome scores from the same week, resulting in one outcome value per week per participant. We aggregated scores only for hypothesis 1.

For hypothesis 2, we examined if the composite score of precursors (t-1) predicted subsequent psychotic symptoms (t0) differently in the two groups (immediate vs. delayed access). As for hypothesis 1, we only considered data from the first eight weeks. We conducted a lagged linear mixed model analysis with momentary psychotic symptoms (t0) as the outcome, the composite score of precursors (t-1), group (immediate vs. delayed access), and the group x composite score (t-1) interaction as predictors, controlling for psychotic symptoms at t-1.

For hypothesis 3, we examined if each individual precursor predicted subsequent psychotic symptoms during the EviBaS intervention. We conducted separate lagged linear mixed model analyses with each precursor (t-1) as the predictor variable (e.g., worry) and momentary psychotic symptoms as the outcome, while controlling for previous psychotic symptoms (t-1). We considered data from all participants taking part in the intervention, both immediate access participants and delayed access participants. As the reliability analysis revealed a negative correlation between the two self-reported cognitive bias items, we excluded the scale from the analyses for hypothesis 3 and examined the items separately in exploratory analyses. This was a deviation from the analysis plan.

All models included a random intercept but no random slope. For hypotheses 2 and 3, we person-mean-centred all time-variant predictors and covariates to obtain within-participant effects rather than between-participant effects. We calculated the within-participant mean and subtracted it from each value. One unforeseen issue was the distance between two consecutive measurements in lagged analyses (hypotheses 2 and 3). During the intervention, it was possible that two assessments from the same participant took place only minutes but also several weeks apart. Post hoc we set the minimum distance between two consecutive measurements to 24 hours because we expected the underlying mechanisms of symptom variability to require at least 24 hours to take place. We defined the upper limit as 252 hours (approximately 1.5 weeks) so that it would overlap with the one-week interval in the waiting period. If a value lied outside the range of 24 to 252 hours, we defined it as missing in lagged analyses (8.3%).

We used two-sided tests and conventional p-values of .05. We applied the Benjamini and Hochberg correction to control for the false discovery rate due to multiple tests (Benjamini & Hochberg, 1995). We conducted separate corrections for analyses 1 and 2 (two tests) and for analyses 3a to 3d (four tests). We used SPSS 25® (RRID: SCR_002865).

Results

Sample characteristics and adherence

Table 1 displays sample characteristics at baseline. Participants provided 1,280 data points in total. Each participant completed $M = 10.32$ ($SD = 6.07$) intermediate assessments on average (range = 1 to 25; median = 9.5). The aggregated data set that we used to compare the course of precursors between groups throughout the first eight weeks of the trial consisted of 600 assessments (hypothesis 1). With PANSS total scores of roughly 50, participants were mildly ill (Leucht et al., 2005). Ordered completion rates of EviBaS modules for the whole sample ($n = 124$) were as follows: Introduction (72%), self-esteem (43%), social competence (39%), mindfulness (36%), cognitive biases (35%), depression (34%), auditory hallucinations (32%), worrying (32%), persecutory delusions (30%), relapse (30%), and sleep (30%).

Hypothesis tests

Group comparisons

For hypothesis 1, we examined if using the EviBaS intervention led to an improvement of composite precursors (i.e., a sum score of negative affect, worrying, etc.) over time. We used aggregated intermediate assessment data from the first eight weeks of the trial to compare the temporal course between groups. Contrary to our hypothesis the group x time interaction was non-significant, indicating that the course of precursors over time did not differ between groups ($b = -0.043$, $SE = .096$, $t = 0.449$, $p = .653$). Hence the intervention did not improve the composite score of precursors compared to the waitlist condition. In fact, the main effect of time was non-significant both in the immediate access group ($b = -0.001$, $SE = .069$, $t = 0.013$, $p = .989$) and the delayed access group ($b = 0.041$, $SE = .067$, $t = 0.613$, $p = .540$), indicating that – across groups – precursors did not improve. For hypothesis 2 we examined if the composite score of precursors predicted subsequent momentary psychotic symptoms differently in the immediate access group compared to the delayed access group. The group x composite precursors

Table 2. Coefficients of lagged linear mixed model analyses within participants using EviBaS intervention (n = 105)

Precursor (t-1)	Unstandardized coefficient (b)	*SE*	*t*	*p*	*FDR*
Worry	0.156	0.064	2.438	.015	.030
Negative affect	0.046	0.034	1.350	.177	.237
Self-esteem	−0.007	0.069	0.106	.916	.916
Quality of sleep	−0.198	0.059	3.359	.001	.003

Note. Outcome = momentary psychotic symptoms (t0) defined as the sum of self-reported suspiciousness and auditory verbal hallucinations; all precursors are participant-mean-centred; all models contain participant-mean-centred psychotic symptoms at t-1 as covariates; we do not present coefficients for self-reported cognitive biases because of the scale's inconsistency; SE = Standard Error; FDR = False Discovery Rate-corrected values based on 4 tests, according to Benjamini and Hochberg (1995).

interaction was non-significant, indicating that the composite score of precursors did not predict subsequent momentary psychotic symptoms differently in the two groups (b = 0.031, SE = .032, t = 0.945, p = .345).

Analyses within the intervention (across groups)

For hypotheses 3 we conducted separate analyses to evaluate if each individual precursor (worry, negative affect, self-esteem, and quality of sleep) predicted subsequent momentary psychotic symptoms within the intervention only. We used data from all participants who completed intermediate assessments while using EviBaS irrespective of their initial group allocation. Please note that we conducted the analyses despite the insufficient internal consistency of the psychotic symptom scale because we wanted to adhere to the registered analysis plan. We addressed the problem in exploratory analyses, in which we analysed paranoia and auditory hallucinations as separate outcomes. Table 2 displays the model coefficients for each precursor. The effects of worry and quality of sleep on subsequent momentary psychotic symptoms remained significant after applying the Benjamini and Hochberg correction. The results indicate that more momentary worry, compared to a participant's average level of worrying, preceded more momentary psychotic symptoms at the following intermediate assessment throughout the intervention (b = 0.156, p_{FDR} = .030). Higher momentary quality of sleep, compared to a participant's average quality of sleep, preceded less momentary psychotic symptoms (b = −0.198, p_{FDR} = .003).

Exploratory analyses

First, the self-reported cognitive bias scale consisted of two items, which correlated negatively. Hence, we analysed each item separately to examine if it preceded subsequent momentary psychotic symptoms in the EviBaS intervention (equivalent to analyses for hypotheses 3). Neither item predicted subsequent momentary psychotic symptoms (p's ≥ .359). Second, the 'psychotic symptoms' scale (auditory verbal hallucinations and paranoia) displayed insufficient internal consistency. Exploratory analyses with auditory verbal hallucinations and paranoia as separate outcomes indicated that the effect of worry consisted mainly of an effect on paranoia (b = 0.116, SE = .044, t = 2.608, p = .009) rather than auditory verbal hallucinations (b = 0.035, SE = .034, t = 1.031, p = .303).

Quality of sleep preceded both paranoia ($b = -0.104$, SE = .041, $t = 2.529$, $p = .012$) and auditory verbal hallucinations ($b = -0.087$, $SE = .032$, $t = 2.736$, $p = .006$). Negative affect, which failed to predict composite psychotic symptoms, predicted paranoia ($b = 0.058$, $SE = .023$, $t = 2.484$, $p = .013$), while there was no effect on auditory verbal hallucinations ($b = -0.011$, $SE = .017$, $t = 0.651$, $p = .516$). Third, we addressed the poor internal consistency of the composite score of precursors (negative affect, worry, self-esteem, self-reported cognitive biases, and quality of sleep). Note that we summarized these precursors to reduce the number of confirmatory tests and we did expect poor internal consistency. We repeated the analyses using single precursors instead of the composite score in separate models. No group x time interactions reached significance (all p's $\geq .411$) replicating our initial findings that group allocation did not affect the course of precursors. Following up hypothesis 2, we examined if each individual precursor (instead of the composite score) preceded subsequent momentary psychotic symptoms differently when compared between groups (immediate vs. delayed access). None of the group x precursor interactions reached significance (all p's $\geq .084$), replicating our initial findings that the effect of precursors on subsequent psychotic symptoms did not differ between groups.

Discussion

We conducted registered longitudinal mixed model analyses on data obtained from a randomized controlled trial (Rüegg, Moritz, Berger, Lüdtke, & Westermann, 2018) and a secondary track of the trial to identify mechanisms of psychotic symptom fluctuations during a psychological online intervention for people with psychosis. By analysing brief intermediate assessments, we were able to compare the course of presumed symptom precursors (worry, negative affect, self-esteem, self-reported cognitive biases, and quality of sleep) between groups, and to examine each precursor's association with subsequent psychotic symptoms. There were no group differences, neither regarding the course of precursors, nor regarding their effect on subsequent psychotic symptoms. However, during the EviBaS intervention we found that momentary worry and quality of sleep predicted subsequent psychotic symptoms (auditory verbal hallucinations and paranoia) across the entire sample. When participants experienced more worry or worse sleep than usual while using the EviBaS intervention, they reported more severe psychotic symptoms upon the next assessment. Exploratory analyses with separate outcomes (due to the poor internal consistency of psychotic symptoms) replicated these findings, showing that fluctuations of quality of sleep preceded both auditory hallucinations and paranoia whereas fluctuations of worry preceded paranoia. Within-participant effects during the intervention in the absence of group effects indicate that the intervention did not improve worry or quality of sleep consistently and persistently across participants, but when fluctuations of worry or quality of sleep occurred, these fluctuations preceded subsequent symptoms. We cannot make the causal inference that worry or sleep led to momentary psychotic symptoms during the intervention. Further, we cannot conclude that the effectiveness of the EviBaS intervention relied on its capacity to reduce worry or sleep problems. We can gain confidence, though, that worry and quality of sleep represent important mechanisms of symptom fluctuations and hence treatment targets in interventions for people with psychosis. It seems worthwhile to monitor worrying and quality of sleep during treatment as these variables could indicate upcoming change of psychotic symptoms. Interestingly, the modules addressing worrying and sleep problems

had lower completion rates compared to other modules of the intervention. It is possible that this finding hints at a discrepancy between the importance of these variables as symptom precursors and their perceived importance by patients (and or perceived changeability).

The effects of worry and quality of sleep on psychotic symptoms occurred although assessments were several days apart over a period of 8 weeks. Hence, one can speculate that worry and quality of sleep, established precursors in previous ESM trials (Hartley et al., 2014; Kasanova et al., 2019), generalize to longer assessment periods. This finding can help to expand our knowledge about the temporal reach of ESM effects. Whereas effects of momentary negative affect or momentary self-esteem could be limited to few hours, worrying and sleep problems could be candidate processes in the prediction of events lying further away, such as relapse or treatment response.

The absence of group differences regarding the course of composite precursors over time was unexpected given the intervention's focus on improving these precursors. This null result was particularly surprising in the light of the intervention's overall effectiveness in the reduction of positive symptoms of psychosis (Westermann et al., 2020). One might argue that the intervention's effect on psychotic symptoms was hence independent of within-participant improvements of precursor symptoms, which would partly contradict the rationale of the intervention. Possibly, the intervention's efficacy mainly relied on directly targeting psychotic symptoms.

Another unexpected finding was that neither negative affect, nor self-esteem, nor self-reported cognitive biases served as precursors of subsequent psychotic symptoms during the intervention despite being well-established precursors of psychotic symptoms in ESM studies (Lüdtke et al., 2017; So et al., 2018; Udachina et al., 2014). The null effect of self-reported cognitive biases on subsequent psychotic symptoms should be interpreted with great caution as our two-item self-report scale was not internally consistent. However, even if it had been, the scale would nonetheless differ from experimental paradigms such as the beads task (Ross, McKay, Coltheart, & Langdon, 2015). Even established self-report scales show no correlation (CBQp; Peters et al., 2014) or only moderate correlation with the beads task (DACOBS; van der Gaag et al., 2013). This inconsistency of self-report and objective measures could be due to the lack of metacognitive awareness of cognitive deficits and biases in psychosis (Peters et al., 2014). An alternative explanation would be that certain experimental tasks measure something other than self-report scales as indicated by research on objective versus subjective effort in schizophrenia (Kreis, Moritz, & Pfuhl, 2020).

The non-significant association of negative affect with subsequent psychotic symptoms was surprising as well, considering the body of research consistently showing lagged effects of negative affect on psychotic symptoms within the same day (Ben-Zeev et al., 2011; Lüdtke et al., 2017; So et al., 2018). This finding is again subject to caution due to the insufficient internal consistency of the outcome variable 'psychotic symptoms'. Exploratory analyses indicated that negative affect might predict paranoia but not auditory verbal hallucinations, which coincides with findings that negative affect relates to paranoid thinking specifically (Freeman, Dunn, et al., 2013). Despite this methodological concern, it is possible that current negative affect influences psychotic symptoms only within the same day – not across several days. This interpretation fits the empirical finding that negative emotions fluctuate intensely throughout the day in people with psychosis (Myin-Germeys, Delespaul, & deVries, 2000). Consequently, effects on subsequent psychotic symptoms are likely to be short-term and transient due to the temporal variability of negative affect. In contrast, worry could be more stable, meaning

that it fluctuates at a slower rate thus making it a better precursor of treatment progress during interventions such as EviBaS. Conceptually, worrying represents both a momentary action as well as a more trait-like thinking style (i.e., a 'worry thinking style'; Freeman & Garety, 2014). This could explain why worrying functions as a precursor both of psychotic symptoms within the same day (Hartley et al., 2014) and of symptom fluctuations during treatment assessed several days later.

Finally, momentary self-esteem did not precede psychotic symptoms, contradicting findings by Udachina et al. (2014). Comparable to negative affect, momentary self-esteem could be highly unstable in people with psychosis as suggested by the revised Attribution–Self-Representation Cycle model of paranoia (as reviewed by Murphy, Bentall, Freeman, O'Rourke, & Hutton, 2018), which would predict only short-term associations with subsequent symptoms. The model proposes that instead of restoring stable self-esteem, externalizing attributions lead to highly unstable self-esteem. Consistent with these predictions the moment-to-moment variability of self-esteem predicts trait paranoia more than the overall level of self-esteem (Thewissen, Bentall, Lecomte, van Os, & Myin-Germeys, 2008). Preliminarily, our findings suggest that self-esteem is not suited as a precursor of infrequently monitored treatment progress, possibly due to its instability impeding far-reaching predictions.

Worry and quality of sleep as potential mechanisms of symptom fluctuations

Worry is associated with paranoia in experimental (Martinelli, Cavanagh, & Dudley, 2013) and ESM trials (Hartley et al., 2014). Further, treating worry leads to improved persecutory delusions in people with psychosis mediated by change in worrying (Freeman, Dunn, et al., 2015). Freeman, Dunn, et al. (2015) discuss that their mediator analysis cannot determine the direction of associations beyond doubt. Our longitudinal design supports the hypothesis that alterations of worry precede paranoia during psychological interventions. Freeman, Dunn, et al. (2015) assume a causal relationship of worry with paranoia in particular. Albeit exploratory, our results support this notion inasmuch as we found associations of worry with subsequent paranoia only but not auditory verbal hallucinations. From a theoretical point of view, worrying is conceptually very similar to paranoia. As Freeman and Garety phrased it 'worry brings implausible ideas to mind, keeps them there, and increases the distress that they cause' (Freeman & Garety, 2014, p. 1180).

Quality of sleep was another significant predictor of subsequent momentary psychotic symptoms in our study preceding both paranoia and auditory verbal hallucinations in exploratory analyses. As for worry, we cannot conclude that quality of sleep causally led to subsequent symptoms but, again, we gain confidence that quality of sleep represents a worthwhile treatment target as it indicates upcoming symptom change. This conclusion coincides with cross-sectional (Koyanagi & Stickley, 2015), experimental (Petrovsky et al., 2014; Reeve et al., 2018), ESM- (Kasanova et al., 2019; Mulligan et al., 2016), and interventional trials in healthy participants (Freeman et al., 2017). So far, preliminary clinical trials in patients have not yielded significant effects of sleep interventions on psychotic symptoms as secondary outcomes (Freeman, Waite, et al., 2015; Hwang et al., 2019) so that effects of sleep interventions on clinical psychotic symptoms await to be established. How sleep contributes to psychotic symptoms is not entirely clear. Negative affect could mediate the effect of quality of sleep (Kasanova et al., 2019; Rehman, Gumley, & Biello, 2018) or induced sleep loss (Reeve et al., 2018) on paranoia. Further, sleep influences several neural processes (Krause et al., 2017) but also psychological outcomes,

such as quality of life, emotion regulation, or cognitive functioning (Faiola et al., 2018; Grezellschak, Jansen, & Westermann, 2017; Harvey, 2009). In sum, sleep is consistently associated with psychotic symptoms but potential pathways are manifold and await further elaboration.

The effects of worry and sleep on psychotic symptoms are not exclusive to people with psychosis. Harvey (2008) describes insomnia as a transdiagnostic process, and both worry (Bell & O'Driscoll, 2018) and sleep (Hennig & Lincoln, 2018) are associated with paranoia in general population samples, indicating that associations between worry/sleep and subclinical psychotic symptoms could be universal. Furthermore, 30% of our sample fulfilled the criteria of a current depressive disorder, corroborating findings that comorbid depression is highly prevalent in people with schizophrenia (Buckley, Miller, Lehrer, & Castle, 2009). Future studies should examine whether comorbid depression moderates the association between psychotic symptoms and worrying or quality of sleep.

Limitations

First, the regression-based design does not permit causal interpretations. Second, the validity of the self-reported cognitive biases assessment was insufficient. Third, analyses in this study were nomothetic despite appearing idiographic at first sight (Piccirillo & Rodebaugh, 2019). Although our focus lied on within-participant effects, we examined 'fixed effects' across participants instead of individual symptom trajectories. Fourth, the proportion of female participants in our sample (60%) was higher than in comparable face-to-face psychotherapy trials for people with psychosis (e.g., 44%; Lincoln et al., 2012). However, the unequal gender distribution is not surprising when considering that online interventions for other disorders, such as depression, seem to attract female users particularly (e.g., 67% female participants in Karyotaki et al., 2018). Nonetheless, the gender distribution limits the generalizability of our findings beyond online interventions.

Conclusions

Worry and quality of sleep are potential mechanisms of symptom variability and hence treatment targets in the context of online interventions for psychosis. While effects of worry might be limited to paranoia, quality of sleep appears to affect auditory verbal hallucinations as well. From a methodological point of view, our results show how participant-mean-centred longitudinal analyses represent an informative addition to group comparisons in randomized controlled trials. For clinicians, it seems promising to monitor worrying and sleep problems during treatment as indicators of both positive and negative treatment progression. Future studies should examine the effects of worrying and quality of sleep on symptoms other than auditory hallucinations and paranoia, such as negative symptoms.

Acknowledgements

We would like to thank all participants, J. Ahlf-Schumacher, F. Alt, F. Baumann, L. Blunier, L.-E. Braunschneider, L. Bücker, S. Christen, M. Denzer, L. Henkel, N. Kreienbühl, A. Paulus, H. Platow, M. Rahmede, K. Rauprich, J. Ricciardi, M. Stojisavlevic, N. Werkle, and R. Zürcher. The EviBaS main trial was funded by the Swiss National Science Foundation (project number: #159384) and the German Research Foundation (project number: DFG Mo 969/17-1); the

secondary study presented here did not receive additional funding. Gerit Pfuhl was funded by the Norwegian Research Council (grant number: 262338/F20).

Conflict of interest

All authors declare no conflict of interest.

Author contributions

Thies Lüdtke (Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Visualization; Writing – original draft); Gerit Pfuhl (Conceptualization; Investigation; Methodology; Project administration; Supervision; Writing – review & editing); Steffen Moritz (Conceptualization; Funding acquisition; Investigation; Methodology; Project administration; Resources; Supervision; Validation; Writing – review & editing); Nina Lee Rüegg (Conceptualization; Data curation; Investigation; Project administration; Writing – review & editing); Thomas Berger (Conceptualization; Funding acquisition; Investigation; Project administration; Resources; Supervision; Writing – review & editing); Stefan Westermann (Conceptualization; Funding acquisition; Investigation; Methodology; Project administration; Resources; Supervision; Writing – review & editing).

Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

References

Beck, A.T., Baruch, E., Balter, J.M., Steer, R.A., & Warman, D.M. (2004). A new instrument for measuring insight: the Beck Cognitive Insight Scale. *Schizophrenia Research*, *68*, 319–329. https://doi.org/10.1016/S0920-9964(03)00189-0

Bell, V., & O'Driscoll, C. (2018). The network structure of paranoia in the general population. *Social Psychiatry and Psychiatric Epidemiology*, *53*, 737–744. https://doi.org/10.1007/s00127-018-1487-0

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: a practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B (Methodological)*, *57*(1), 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x

Ben-Zeev, D., Ellington, K., Swendsen, J., & Granholm, E. (2011). Examining a cognitive model of persecutory ideation in the daily life of people with schizophrenia: a computerized experience sampling study. *Schizophrenia Bulletin*, *37*, 1248–1256. https://doi.org/10.1093/schbul/sbq041

Buckley, P.F., Miller, B.J., Lehrer, D.S., & Castle, D.J. (2009). Psychiatric comorbidities and schizophrenia. *Schizophrenia Bulletin*, *35*, 383–402. https://doi.org/10.1093/schbul/sbn135

Danivas, V., & Venkatasubramanian, G. (2013). Current perspectives on chlorpromazine equivalents: Comparing apples and oranges!. *Indian J Psychiatry*, *55*, 207–208. https://doi.org/10.4103/0019-5545.111475

Dudley, R., Taylor, P., Wickham, S., & Hutton, P. (2016). Psychosis, delusions and the "Jumping to conclusions" reasoning bias: A systematic review and meta-analysis. *Schizophrenia Bulletin*, *42*, 652–665. https://doi.org/10.1093/schbul/sbv150

Eichner, C., & Berna, F. (2016). Acceptance and efficacy of metacognitive training (MCT) on positive symptoms and delusions in patients with schizophrenia: a meta-analysis taking into account important moderators. *Schizophrenia Bulletin*, *42*, 952–962. https://doi.org/10.1093/schbul/sbv225

Faiola, E., Meyhofer, I., Steffens, M., Kasparbauer, A.M., Kumari, V., & Ettinger, U. (2018). Combining trait and state model systems of psychosis: The effect of sleep deprivation on cognitive functions in schizotypal individuals. *Psychiatry Research*, *270*, 639–648. https://doi.org/10.1016/j.psychres.2018.10.033

Freeman, D., Dunn, G., Fowler, D., Bebbington, P., Kuipers, E., Emsley, R., ... Garety, P. (2013). Current paranoid thinking in patients with delusions: the presence of cognitive-affective biases. *Schizophrenia Bulletin*, *39*, 1281–1287. https://doi.org/10.1093/schbul/sbs145

Freeman, D., Dunn, G., Startup, H., Pugh, K., Cordwell, J., Mander, H., ... Kingdon, D. (2015). Effects of cognitive behaviour therapy for worry on persecutory delusions in patients with psychosis (WIT): a parallel, single-blind, randomised controlled trial with a mediation analysis. *Lancet Psychiatry*, *2*, 305–313. https://doi.org/10.1016/S2215-0366(15)00039-5

Freeman, D., & Garety, P. (2014). Advances in understanding and treating persecutory delusions: a review. *Social Psychiatry and Psychiatric Epidemiology*, *49*, 1179–1189. https://doi.org/10.1007/s00127-014-0928-7

Freeman, D., Sheaves, B., Goodwin, G.M., Yu, L.M., Nickless, A., Harrison, P.J., ... Espie, C.A. (2017). The effects of improving sleep on mental health (OASIS): a randomised controlled trial with mediation analysis. *Lancet Psychiatry*, *4*, 749–758. https://doi.org/10.1016/S2215-0366(17)30328-0

Freeman, D., Taylor, K.M., Molodynski, A., & Waite, F. (2019). Treatable clinical intervention targets for patients with schizophrenia. *Schizophrenia Research*, *211*, 44–50. https://doi.org/10.1016/j.schres.2019.07.016

Freeman, D., Thompson, C., Vorontsova, N., Dunn, G., Carter, L.A., Garety, P., ... Ehlers, A. (2013). Paranoia and post-traumatic stress disorder in the months after a physical assault: a longitudinal study examining shared and differential predictors. *Psychological Medicine*, *43*, 2673–2684. https://doi.org/10.1017/S003329171300038X

Freeman, D., Waite, F., Startup, H., Myers, E., Lister, R., McInerney, J., ... Yu, L.M. (2015). Efficacy of cognitive behavioural therapy for sleep improvement in patients with persistent delusions and hallucinations (BEST): a prospective, assessor-blind, randomised controlled pilot trial. *Lancet Psychiatry*, *2*, 975–983. https://doi.org/10.1016/S2215-0366(15)00314-4

Grezellschak, S., Jansen, A., & Westermann, S. (2017). Emotion regulation in patients with psychosis: A link between insomnia and paranoid ideation? *Journal of Behavior Therapy and Experimental Psychiatry*, *56*, 27–32. https://doi.org/10.1016/j.jbtep.2016.08.001

Hartley, S., Haddock, G., Vasconcelos, E.S.D., Emsley, R., & Barrowclough, C. (2014). An experience sampling study of worry and rumination in psychosis. *Psychological Medicine*, *44*, 1605–1614. https://doi.org/10.1017/S0033291713002080

Harvey, A.G. (2008). Insomnia, psychiatric disorders, and the transdiagnostic perspective. *Current Directions in Psychological Science*, *17*, 299–303. https://doi.org/10.1111/j.1467-8721.2008.00594.x

Harvey, A.G. (2009). A transdiagnostic approach to treating sleep disturbance in psychiatric disorders. *Cognitive Behaviour Therapy*, *38*(Suppl 1), 35–42. https://doi.org/10.1080/16506070903033825

Hennig, T., & Lincoln, T.M. (2018). Sleeping paranoia away? An actigraphy and experience-sampling study with adolescents. *Child Psychiatry and Human Development*, *49*(1), 63–72. https://doi.org/10.1007/s10578-017-0729-9

Hwang, D.K., Nam, M., & Lee, Y.G. (2019). The effect of cognitive behavioral therapy for insomnia in schizophrenia patients with sleep Disturbance: A non-randomized, assessor-blind trial. *Psychiatry Research*, *274*, 182–188. https://doi.org/10.1016/j.psychres.2019.02.002

Karyotaki, E., Kemmeren, L., Riper, H., Twisk, J., Hoogendoorn, A., Kleiboer, A., ... Cuijpers, P. (2018). Is self-guided internet-based cognitive behavioural therapy (iCBT) harmful? An

individual participant data meta-analysis. *Psychological Medicine*, *48*, 2456–2466. https://doi.org/10.1017/S0033291718000648

Kasanova, Z., Hajduk, M., Thewissen, V., & Myin-Germeys, I. (2019). Temporal associations between sleep quality and paranoia across the paranoia continuum: An experience sampling study. *Journal of Abnormal Psychology*, *129*(1), 122–130. https://doi.org/10.1037/abn0000453

Kay, S.R., Fiszbein, A., & Opler, L.A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*, 261–276. https://doi.org/10.1093/schbul/13.2.261

Koyanagi, A., & Stickley, A. (2015). The association between sleep problems and psychotic symptoms in the general population: a global perspective. *Sleep*, *38*, 1875–1885. https://doi.org/10.5665/sleep.5232

Kramer, I., Simons, C.J., Wigman, J.T., Collip, D., Jacobs, N., Derom, C., . . . Wichers, M. (2014). Time-lagged moment-to-moment interplay between negative affect and paranoia: new insights in the affective pathway to psychosis. *Schizophrenia Bulletin*, *40*, 278–286. https://doi.org/10.1093/schbul/sbs194

Krause, A.J., Simon, E.B., Mander, B.A., Greer, S.M., Saletin, J.M., Goldstein-Piekarski, A.N., & Walker, M.P. (2017). The sleep-deprived human brain. *Nature Reviews Neuroscience*, *18*, 404–418. https://doi.org/10.1038/nrn.2017.55

Kreis, I., Moritz, S., & Pfuhl, G. (2020). Objective versus subjective effort in schizophrenia. *Frontiers in Psychology*, *11*, 1469. https://doi.org/10.3389/fpsyg.2020.01469

Kroenke, K., Spitzer, R.L., & Williams, J.B. (2001). The PHQ-9: validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*, 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Lecrubier, Y., Sheehan, D.V., Weiller, E., Amorim, P., Bonora, I., Sheehan, K.H., . . . Dunbar, G.C. (1997). The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: Reliability and validity according to the CIDI. *European Psychiatry*, *12*, 224–231. https://doi.org/10.1016/S0924-9338(97)83296-8

Leucht, S., Kane, J.M., Kissling, W., Hamann, J., Etschel, E., & Engel, R.R. (2005). What does the PANSS mean? *Schizophrenia Research*, *79*, 231–238. https://doi.org/10.1016/j.schres.2005.04.008

Lincoln, T.M., Ziegler, M., Mehl, S., Kesting, M.L., Lullmann, E., Westermann, S., & Rief, W. (2012). Moving from efficacy to effectiveness in cognitive behavioral therapy for psychosis: a randomized clinical practice trial. *Journal of Consulting and Clinical Psychology*, *80*, 674–686. https://doi.org/10.1037/a0028665

Liu, Y.C., Tang, C.C., Hung, T.T., Tsai, P.C., & Lin, M.F. (2018). The efficacy of metacognitive training for delusions in patients with Schizophrenia: a meta-analysis of randomized controlled trials informs evidence-based practice. *Worldviews on Evidence-Based Nursing*, *15*, 130–139. https://doi.org/10.1111/wvn.12282

Lüdtke, T., Kriston, L., Schröder, J., Lincoln, T.M., & Moritz, S. (2017). Negative affect and a fluctuating jumping to conclusions bias predict subsequent paranoia in daily life: An online experience sampling study. *Journal of Behavior Therapy and Experimental Psychiatry*, *56*, 106–112. https://doi.org/10.1016/j.jbtep.2016.08.014

Martinelli, C., Cavanagh, K., & Dudley, R.E. (2013). The impact of rumination on state paranoid ideation in a nonclinical sample. *Behavior Therapy*, *44*, 385–394. https://doi.org/10.1016/j.beth.2013.02.002

Moritz, S., Berna, F., Jaeger, S., Westermann, S., & Nagel, M. (2017). The customer is always right? Subjective target symptoms and treatment preferences in patients with psychosis. *European Archives of Psychiatry and Clinical Neuroscience*, *267*, 335–339. https://doi.org/10.1007/s00406-016-0694-5

Moritz, S., Goritz, A.S., McLean, B., Westermann, S., & Brodbeck, J. (2017). Do depressive symptoms predict paranoia or vice versa? *Journal of Behavior Therapy and Experimental Psychiatry*, *56*, 113–121. https://doi.org/10.1016/j.jbtep.2016.10.002

Moritz, S., Schmidt, S.J., Lüdtke, T., Braunschneider, L.E., Manske, A., Schneider, B.C., & Veckstenstedt, R. (2019). Post-psychotic depression: Paranoia and the damage done. *Schizophrenia Research*, *211*, 79–85. https://doi.org/10.1016/j.schres.2019.06.022

Moritz, S., Schröder, J., Klein, J.P., Lincoln, T.M., Andreou, C., Fischer, A., & Arlt, S. (2016). Effects of online intervention for depression on mood and positive symptoms in schizophrenia. *Schizophrenia Research*, *175*, 216–222. https://doi.org/10.1016/j.schres.2016.04.033

Moritz, S., & Woodward, T.S. (2007). Metacognitive training in schizophrenia: from basic research to knowledge translation and intervention. *Current Opinion in Psychiatry*, *20*, 619–625. https://doi.org/10.1097/YCO.0b013e3282f0b8ed

Mulligan, L.D., Haddock, G., Emsley, R., Neil, S.T., & Kyle, S.D. (2016). High resolution examination of the role of sleep disturbance in predicting functioning and psychotic symptoms in schizophrenia: A novel experience sampling study. *Journal of Abnormal Psychology*, *125*, 788–797. https://doi.org/10.1037/abn0000180

Murphy, P., Bentall, R.P., Freeman, D., O'Rourke, S., & Hutton, P. (2018). The paranoia as defence model of persecutory delusions: a systematic review and meta-analysis. *Lancet Psychiatry*, *5*, 913–929. https://doi.org/10.1016/S2215-0366(18)30339-0

Myin-Germeys, I., Delespaul, P.A.e.g., & deVries, M.W. (2000). Schizophrenia patients are more emotionally active than is assumed based on their behavior. *Schizophrenia Bulletin*, *26*, 847–854. https://doi.org/10.1093/oxfordjournals.schbul.a033499

Myin-Germeys, I., Kasanova, Z., Vaessen, T., Vachon, H., Kirtley, O., Viechtbauer, W., & Reininghaus, U. (2018). Experience sampling methodology in mental health research: new insights and technical developments. *World Psychiatry*, *17*, 123–132. https://doi.org/10.1002/wps.20513

Myin-Germeys, I., Oorschot, M., Collip, D., Lataster, J., Delespaul, P., & van Os, J. (2009). Experience sampling research in psychopathology: opening the black box of daily life. *Psychological Medicine*, *39*, 1533–1547. https://doi.org/10.1017/S0033291708004947

Peters, E.R., Moritz, S., Schwannauer, M., Wiseman, Z., Greenwood, K.E., Scott, J., . . . Garety, P.A. (2014). Cognitive Biases Questionnaire for psychosis. *Schizophrenia Bulletin*, *40*, 300–313. https://doi.org/10.1093/schbul/sbs199

Petrovsky, N., Ettinger, U., Hill, A., Frenzel, L., Meyhofer, I., Wagner, M., . . . Kumari, V. (2014). Sleep deprivation disrupts prepulse inhibition and induces psychosis-like symptoms in healthy humans. *Journal of Neuroscience*, *34*, 9134–9140. https://doi.org/10.1523/JNEUROSCI.0904-14.2014

Philipp, R., Kriston, L., Lanio, J., Kuhne, F., Harter, M., Moritz, S., & Meister, R. (2019). Effectiveness of metacognitive interventions for mental disorders in adults-A systematic review and meta-analysis (METACOG). *Clinical Psychology & Psychotherapy*, *26*(2), 227–240. https://doi.org/10.1002/cpp.2345

Piccirillo, M.L., & Rodebaugh, T.L. (2019). Foundations of idiographic methods in psychology and applications for psychotherapy. *Clinical Psychology Review*, *71*, 90–100. https://doi.org/10.1016/j.cpr.2019.01.002

Reeve, S., Emsley, R., Sheaves, B., & Freeman, D. (2018). Disrupting sleep: the effects of sleep loss on psychotic experiences tested in an experimental study with mediation analysis. *Schizophrenia Bulletin*, *44*, 662–671. https://doi.org/10.1093/schbul/sbx103

Rehman, A., Gumley, A., & Biello, S. (2018). Sleep quality and paranoia: The role of alexithymia, negative emotions and perceptual anomalies. *Psychiatry Research*, *259*, 216–222. https://doi.org/10.1016/j.psychres.2017.09.066

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.

Ross, R.M., McKay, R., Coltheart, M., & Langdon, R. (2015). Jumping to conclusions about the beads task? A meta-analysis of delusional ideation and data-gathering. *Schizophrenia Bulletin*, *41*(5), 1183–1191. https://doi.org/10.1093/schbul/sbu187

Rüegg, N., Moritz, S., Berger, T., Lüdtke, T., & Westermann, S. (2018). An internet-based intervention for people with psychosis (EviBaS): study protocol for a randomized controlled trial. *BMC Psychiatry*, *18*(1), 102. https://doi.org/10.1186/s12888-018-1644-8

So, S.H.W., Chau, A.K.C., Peters, E.R., Swendsen, J., Garety, P.A., & Kapur, S. (2018). Moment-to-moment associations between negative affect, aberrant salience, and paranoia. *Cognitive Neuropsychiatry*, *23*, 299–306. https://doi.org/10.1080/13546805.2018.1503080

Stober, J., & Bittencourt, J. (1998). Weekly assessment of worry: an adaptation of the Penn State Worry Questionnaire for monitoring changes during treatment. *Behavior Research and Therapy*, *36*, 645–656. https://doi.org/10.1016/s0005-7967(98)00031-x

Thewissen, V., Bentall, R.P., Lecomte, T., van Os, J., & Myin-Germeys, I. (2008). Fluctuations in self-esteem and paranoia in the context of daily life. *Journal of Abnormal Psychology*, *117*(1), 143–153. https://doi.org/10.1037/0021-843x.117.1.143

Twisk, J.W.R. (2019). *Applied mixed model analysis: a practical guide* (2nd ed.). Cambridge, UK: Cambridge University Press.

Udachina, A., Varese, F., Myin-Germeys, I., & Bentall, R.P. (2014). The role of experiential avoidance in paranoid delusions: an experience sampling study. *British Journal of Clinical Psychology*, *53*, 422–432. https://doi.org/10.1111/bjc.12054

Upthegrove, R., Marwaha, S., & Birchwood, M. (2017). Depression and schizophrenia: cause, consequence, or trans-diagnostic Issue? *Schizophrenia Bulletin*, *43*, 240–244. https://doi.org/10.1093/schbul/sbw097

van der Gaag, M., Schutz, C., ten Napel, A., Landa, Y., Delespaul, P., Bak, M., . . . de Hert, M. (2013). Development of the davos assessment of cognitive biases scale (DACOBS). *Schizophrenia Research*, *144*(1–3), 63–71. https://doi.org/10.1016/j.schres.2012.12.010

van Oosterhout, B., Smit, F., Krabbendam, L., Castelein, S., Staring, A.B., & van der Gaag, M. (2016). Metacognitive training for schizophrenia spectrum patients: a meta-analysis on outcome studies. *Psychological Medicine*, *46*(1), 47–57. https://doi.org/10.1017/S0033291715001105

Westermann Stefan, Rüegg Nina, Lüdtke Thies, Moritz Steffen, Berger Thomas (2020). Internet-based self-help for psychosis: Findings from a randomized controlled trial.. *Journal of Consulting and Clinical Psychology*, *88*(10), 937–950. http://dx.doi.org/10.1037/ccp0000602.

Received 29 July 2020; revised version received 3 November 2020