# Combining two model systems of psychosis: The effects of schizotypy and sleep deprivation on oculomotor control and psychotomimetic states

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## ABSTRACT

Model systems of psychosis, such as schizotypy or sleep deprivation, are valuable in informing our understanding of the etiology of the disorder and aiding the development of new treatments. Schizophrenia patients, high schizotypes, and sleep-deprived subjects are known to share deficits in oculomotor biomarkers. Here, we aimed to further validate the schizotypy and sleep deprivation models and investigated, for the first time, their interactive effects on smooth pursuit eye movements (SPEM), prosaccades, antisaccades, predictive saccades and measures of psychotomimetic states, anxiety, depression, and stress. To do so, *n* = 19 controls and *n* = 17 high positive schizotypes were examined after both a normal sleep night and 24h of sleep deprivation. Schizotypes displayed higher SPEM global position error, catch-up saccade amplitude, and increased psychotomimetic states. Sleep deprivation impaired SPEM, prosaccade, antisaccade, and predictive saccade performance and increased levels of psychotomimetic experiences. Additionally, sleep deprivation reduced SPEM gain in schizotypes but not controls. We conclude that oculomotor impairments are observed in relation to schizotypy and following sleep deprivation, supporting their utility as biomarkers in model systems of psychosis. The combination of these models with oculomotor biomarkers may be particularly fruitful in assisting the development of new antipsychotic or pro-cognitive drugs.

## KEYWORDS

Smooth pursuit, antisaccade, model system, schizotypy, sleep deprivation, schizophrenia

## INTRODUCTION

Model systems of psychosis are important in identifying pathogenic risk factors and providing assays for the evaluation of new pharmacological treatments (Ettinger & Kumari, 2015; Koychev et al., 2011). A popular approach is the use of surrogate populations, such as high schizotypes (Barrantes-Vidal, Grant, & Kwapil, 2015; Mason, 2015). Schizotypy is a multidimensional personality pattern resembling schizophrenia symptoms in an attenuated form (Ettinger, Meyhöfer, Steffens, Wagner, & Koutsouleris, 2014; Nelson, Seal, Pantelis, & Phillips, 2013). Schizotypy can be classified into positive (e.g., unusual perceptual experiences), negative (e.g., anhedonia), and disorganized (e.g., odd speech, eccentric behavior) factors (Raine et al., 1994), similar to the factor structure of schizophrenia (Liddle, 1987). Furthermore, schizotypy is associated with alterations in cognition and brain structure and function that resemble the deficits observed in schizophrenia (Ettinger et al., 2014).

Sleep deprivation has been proposed as a promising experimental model system of psychosis (Ettinger & Kumari, 2015) as it induces cognitive and perceptual aberrations resembling positive, negative, and disorganized symptoms (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007; Luby et al., 1962; West, Janszen, Lester, & Cornelisoon, 1962). Furthermore, sleep deprivation causes cognitive deficits (Lim & Dinges, 2010) overlapping with those observed in schizophrenia (Schaefer, Giangrande, Weinberger, & Dickinson, 2013).

Model systems are of most use when combined with well-validated biomarkers such as oculomotor measures. Oculomotor tasks are short, parameters can be manipulated systematically (Barnes, 2008; Hutton, 2008), measurement is highly reliable (Meyhöfer, Bertsch, Esser, & Ettinger, 2016), their neural mechanisms are well understood (Hutton & Ettinger, 2006; Lencer & Trillenberg, 2008), performance is impacted by antipsychotic drugs (Reilly, Lencer, Bishop, Keedy, & Sweeney, 2008), and deficits in the schizophrenia spectrum have been widely replicated (Calkins, Iacono, & Ones, 2008; Koychev et al., 2016).

The smooth pursuit (SPEM) and antisaccade tasks are amongst the most widely used oculomotor biomarkers. SPEM serves to follow a slowly moving object. SPEM impairments in schizophrenia are well replicated (Levy, Sereno, Gooding, & O'Driscoll, 2010; O'Driscoll & Callahan, 2008). Similar deficits have been reported in overall high schizotypes (Koychev et al., 2016; Meyhöfer et al., 2015), in high positive, negative, and disorganized subgroups (Gooding, Miller, & Kwapil, 2000; Holahan & O'Driscoll, 2005; Lenzenweger & O'Driscoll, 2006; O'Driscoll, Lenzenweger, & Holzman, 1998; Smyrnis et al., 2007), and following sleep deprivation (Meyhöfer, Kumari, Hill, Petrovsky, & Ettinger, 2017).

In the antisaccade task, participants are requested to refrain from looking towards a sudden-onset peripheral target and instead direct their eyes to the opposite direction. Schizophrenia patients show reliable increases in direction errors, whilst performance on the prosaccade task, a visuomotor control condition, is largely unimpaired (Gooding & Basso, 2008). Similarly, high schizotypes demonstrate elevated antisaccade error rates (Ettinger et al., 2005; Gooding, Shea, & Matts, 2005; Holahan & O'Driscoll, 2005; Smyrnis et al., 2003) but normal prosaccade performance (Gooding et al., 2005). Antisaccade impairments following sleep deprivation include elevated errors, increased latencies, and reduced peak velocity (Meyhöfer et al., 2017). In contrast to schizophrenia and schizotypy, sleep deprivation also deteriorates prosaccades (Meyhöfer et al., 2017).

A further oculomotor task that has been studied in schizophrenia is the predictive saccade task (Krebs et al., 2001). Predictive saccades are made towards stimuli that are predictable in time and/or space. Schizophrenia patients demonstrate hypometric amplitudes (Amado, Landgraf, Bourdel, Leonardi, & Krebs, 2008; Hutton et al., 2001) and a higher rate of anticipatory saccades (Karoumi, Ventre-Dominey, & Dalery, 1998). However, no studies have examined predictive saccades in schizotypy or after sleep deprivation.

Here, we aimed to further evaluate the schizotypy and sleep deprivation models using oculomotor measures and to explore, for the first time, interactions between these models. Given the previously articulated limitations of individual model systems (Carhart-Harris, Brugger, Nutt, & Stone, 2013), combinations of trait (e.g., schizotypy) and state (e.g., sleep deprivation) models, might

significantly advance this field. We hypothesized to observe schizophrenia-like deficits in SPEM and antisaccades in schizotypes and following acute sleep deprivation. Given the most consistent antisaccade deficits in the positive schizotypy dimension (Ettinger et al., 2005; Holahan & O'Driscoll, 2005), a high positive schizotypy group was included. To provide a fine-grained analysis of SPEM performance, we used blanking (Thaker, Ross, Buchanan, Adami, & Medoff, 1999) and step-ramp (Clementz & McDowell, 1994) tasks in addition to standard sinusoidal SPEM. Furthermore, we wished to replicate the observed increases in psychotomimetic states after sleep deprivation (Meyhöfer et al., 2017), and we explored effects on state anxiety, depression, and stress to examine the phenomenological specificity of the two models.

## METHOD

### Participant Recruitment and Selection

Volunteers were recruited locally and through social media to complete an online version of the German translation of the Oxford-Liverpool Inventory of Feelings and Experiences short form (O-LIFE; Grant et al., 2013). The positive (Unusual Experiences) subscale was used to identify high schizotypes.

A total of 5006 volunteers completed the questionnaire. O-LIFE mean scores for Unusual Experiences in the whole sample were as follows: females: $M$ = 5.09, $SD$ = 3.07, $n$ = 3354; males: $M$ = 4.51, $SD$ = 3.05, $n$ = 1652. Cronbach's α was 0.78. Schizotypes had to score ≥ 1.25 standard deviations above the same sex mean Unusual Experiences score (female: ≥ 9; male: ≥ 9) and, in order to exclude confounding by high scores on the negative dimension, ≤ 0.50 standard deviations below the same sex mean Introvertive Anhedonia score (female: ≤ 2; male: ≤ 2). Controls were required to score ≤ 0.5 standard deviations below their same sex mean scores on Unusual Experiences (female: ≤ 3; male: ≤ 3) and Introvertive Anhedonia (female: ≤ 2; male: ≤ 2).

Study exclusion criteria included aged < 18 or > 50, first language other than German, any diagnoses of psychotic disorders among first-degree relatives (Neill, 2014), any eye surgery or visual impairment (other than the use of corrective lenses or glasses), any prescription or over-the-counter

medication (except for oral contraceptives and vitamins), any sleep disorder, irregular sleep-wake-rhythm, shift-work, any current Axis I disorder (MINI International Neuropsychiatric Interview; Sheehan et al., 1998), any current or history of psychotic disorders, and current alcohol (ACE AL5500) or drug consumption (Drug-Screen Multi 5T, nal von minden GmbH). The study was approved by the ethics committee of the Department of Psychology, University of Bonn. Participants provided written informed consent and were compensated with €80 or course credits.

**Study Procedure**

The study consisted of a telephone screening and two laboratory sessions, which were conducted one week apart. Following completion of the online questionnaire, participants were contacted via e-mail and, if they agreed, were screened for exclusion criteria via telephone. The laboratory sessions used a randomized counterbalanced within-subjects design. An overview is in Figure 1.

INCLUDE FIGURE 1 ABOUT HERE

In the normal sleep night, participants slept in the laboratory from 11:00pm to 6:55am. Then they were woken and received a light breakfast at 7:30am. After breakfast, they carried out a cognitive and oculomotor test battery (8:00am-12:00am; total duration about 3.5h), following which they provided measures of psychotomimetic states, anxiety, depression, and stress.

In the night of sleep deprivation, participants were administered demographic questionnaires and a measure of verbal intelligence (Mehrfachwahl-Wortschatz-Intelligenztest, Version B; Lehrl, 2005). They stayed awake during the whole night in the presence of an examiner, occupying themselves with reading, watching movies, or playing board games. Participants were not allowed to eat or drink except for water and the examiner took the participants for several 15-minutes walks. The morning was identical to that after the normal sleep night. The participants were dismissed with the caution not to drive or operate heavy machines prior to an adequate rest.

**Eye Movement Measurement and Oculomotor Tasks**

Eye movement measurements (EyeLink 1000, SR Research Ltd., Canada) took place in a quiet and darkened laboratory on an LCD monitor (Viewsonic, 22“, height = 29.5cm, width = 47.5cm, resolution: 1680 x 1050 pixels, 60Hz refresh rate) at a distance of 70cm. Tasks were implemented using ExperimentBuilder (SR Research, version 1.10.1241). Data analysis was performed with purpose-written routines in Matlab R2014a (The MathWorks, Natick, MA). For the prosaccade, antisaccade and predictive saccade tasks, saccades were identified with velocity (≥ 30°/sec), acceleration (≥ 8000°/sec²), and a minimum amplitude (1°) criteria. To distinguish small saccades from pursuit, a more conservative threshold was used for the SPEM tasks (velocity ≥ 22°/sec, acceleration ≥ 3800°/sec²). Task order was randomized and kept constant through the normal sleep and sleep deprivation sessions. A minimum of five valid trials at each of the sessions had to be reached for the data to be included into further analyses.

*SPEM Tasks*

The stimulus consisted of a grey (RGB = 128, 128, 128) circular stimulus (width and height = 15 pixels, diameter = 0.35°, no filling, stroke width = 5 pixels) on a black background (RGB = 0, 0, 0) moving horizontally across the screen. Participants were instructed to follow the stimulus as accurately as possible.

In the sinusoidal task, the stimulus moved in a sinusoidal waveform (± 9.86° from the center) at two different frequencies (0.2Hz, 0.4Hz) with 40.5 half-cycles each. The first half ramp was excluded from further analyses (Lencer & Trillenberg, 2008). Dependent variables were time-weighted and averaged maintenance gain for segments of pursuit in the middle 50% of each half-cycle with a minimum length of 50msec (%; excluding blinks or saccades), root mean square error scores during the entire block (°; excluding blinks), frequency (*N*/sec) and amplitude (°) of catch-up saccades, and frequency (*N*/sec) of anticipatory and leading saccades during the entire block.

Catch-up saccades were defined as saccades in stimulus direction that reduced error of eye position starting and ending behind the stimulus. They were also allowed to start behind and end ahead of the stimulus unless eye position error was decreased by at least 50% (Ross et al., 2002).

Anticipatory saccades were defined as saccades in stimulus direction that increased position error and either started and ended ahead of the stimulus or started behind and ended ahead of the stimulus, whereby the distance ahead of the stimulus had be at least twice the distance the eye spent behind the stimulus (Ross, Olincy, Zerbe, & Radant, 2001). They were followed by a 50msec interval of less than 50% of stimulus velocity (post saccadic slowing) and the amplitude had to be at least 4° of visual angle. Leading saccades were defined in the same way as anticipatory saccades except that their amplitude ranged between 1° to 4° of visual angle (Ross et al., 2001). Saccades that could not be clearly categorized were discarded.

In the blanking task, the stimulus moved in a triangular waveform (± 9.86° from the center) at a constant stimulus velocity of 13°/sec. The stimulus was blanked off in the middle of the half-cycle for 500msec pseudo-randomly during 44 of 89 half-cycles. The duration of one half-cycle was 1500msec. Dependent variables were peak gain and saccade frequency during 400-500msec (= 100msec to stimulus blanking), and residual gain and saccade frequency at 900-1100msec (= 400-600msec after stimulus blanking; Trillenberg et al., 2016). All variables were calculated analogously in blanking and non blanking half cycles. Velocity gain scores (%) and saccade frequencies (*N*/sec) were calculated as above.

In the foveo-petal step-ramp task (Rashbass, 1961), the stimulus started from the center and pseudo-randomly either stepped to the right or left (1.97°) before moving in the opposite direction (± 11.81°) at a constant velocity of 13°/sec. The step was designed so that the center was re-crossed after 150msec (Clementz, Reid, Mcdowell, & Cadenhead, 1995). The task included 40 trials (20 left, 20 right) and the inter-trial interval varied pseudo-randomly between 2000-3000msec (Clementz et al., 1995). Dependent variables were initial eye acceleration (°/sec²), pursuit latency (sec), and early maintenance gain (%; 350-550msec after stimulus step; Lencer et al., 2015). Initial eye acceleration was estimated using the slope in linear regression analysis (RobustFit® in Matlab) of 100msec eye velocity. The beginning of the acceleration time window was defined when eye velocity first exceeded three standard deviations above resting eye velocity (computed from 100msec before to 100msec after the stimulus step) and the eye started to move in the direction of the stimulus (Lencer

et al., 2004, 2015). Pursuit latency was determined as the beginning of the eye acceleration time window which is the intercept of the regression analyses with time (Lencer et al., 2004). Trials with saccades in the resting or acceleration periods were excluded from the analysis of initial eye acceleration and pursuit latency.

*Pro- and Antisaccades*

Each of the tasks included 32 trials with horizontal amplitude of ± 6° and ± 12° used 16 times each. In each trial, the white (RGB = 255, 255, 255) circular stimulus (width and height = 15 pixels, diameter = 0.35°, no filling, stroke width 5 pixels) appeared first in the center of the black (RGB = 0, 0, 0) screen for a randomized duration of 1000-2000msec and then stepped to the periphery where it remained for 1000msec. The sequence of trials (right/left peripheral stimuli) was pseudo-randomized. For the prosaccade task, participants were instructed to follow the stimulus as fast and accurately as possible. In the antisaccade task, participants had to look at the stimulus whilst it was in the center, but then direct their gaze as fast and accurately as possible to the exact opposite location when the stimulus appeared in the periphery, without glancing at the stimulus first. There were four practice trials to take care of a proper understanding of the antisaccade instructions. Participants always started with the prosaccade task. Saccadic inclusion criteria were the same as in our previous study (Meyhöfer et al., 2017). For each trial, we used only the first saccade after target onset if the starting point was within a range of 100 pixels (2.3°) to the left or the right of the screen center and the minimum latency of the stimulus was at least 80msec in order to avoid anticipatory saccades (Fischer & Ramsperger, 1984). Additionally, no saccades or blinks 50msec prior to peripheral target onset were allowed for the saccade to be included into data analysis. Dependent variables were latency (msec), percentage of amplitude gain, and peak velocity (°/sec) of correct pro- and antisaccades and percentage of direction errors. In data analysis, results of both eccentricities had to be combined to reach an appropriate amount of trials.

*Predictive Saccades*

The task included four conditions to manipulate predictability of horizontal stimulus direction and timing (Allman, Ettinger, Joober, & O'Driscoll, 2012): Completely predictable (direction and time was predictable), spatially predictable (only the direction was predictable), timing predictable (only the timing was predictable), and non predictable (neither direction nor timing was predictable). For completely and timing predictable conditions the duration of the stimulus at each location was 625msec. For spatially and non predictable conditions the duration varied between 500msec and 750msec (average time was 625msec). Each condition consisted of 22 trials and each trial included a stimulus step to the periphery (± 9.86°) and back to the center. The stimulus consisted of a grey (RGB = 128, 128, 128) circular stimulus (width and height = 15 pixels, diameter = 0.35°, no filling, stroke width 5 pixels) on black (RGB = 0, 0, 0) background. Participants were instructed to follow the stimulus as accurately as possible. Only saccades with a minimum amplitude of 3° (Allman et al., 2012) starting from the center of the screen within a range of 100 pixels (2.3°) to the left or right were counted as valid trials. Retrosaccades (saccades from the periphery back to the center) were excluded. Dependent variables for each condition were latency (msec), amplitude gain (%), peak velocity (°/sec), and percentages of predictive (latency < 70msec) and regular (latency > 120msec) saccades (Gagnon, O'Driscoll, Petrides, & Pike, 2002; Smit & Gisbergen, 1989), and direction errors.

**Measures of Psychotomimetic States, Anxiety, Depression, and Stress**

Psychotomimetic states were measured using the Delusional Thinking, Perceptual Distortion, Cognitive Disorganization, Anhedonia, Mania, and Paranoia subscales of the Psychotomimetic States Inventory (PSI; Mason et al., 2008). Anxiety was quantified with the State-Trait Anxiety Inventory State Scale (STAI-S; Laux et al., 1981). Item 8 (feeling rested) was excluded from analyses. Depression was assessed with the Maryland Trait and State Depression State Scale (MTSD-S; Chiappelli et al., 2014). Items 4 (sleep more than usual), 11 (don't sleep enough), 13 (less time for hobbies), and 16 (changes in body weight) were excluded. For state stress, the Perceived Stress Questionnaire (PSQ; Fliege et al., 2005) was applied. Sum scores were used as dependent variables. As missing values were very rare (PSI = 6, STAI-S = 2, MTSD-S = 1, PSQ = 7), no imputation method was applied.

**Statistical Analyses**

Analysis was performed using SPSS 24 (IBM Corporation, USA) and R (R Core Team, 2016). Dependent variables were analyzed using mixed analysis of variance (ANOVA) with *condition* (sleep deprivation, normal sleep) as within-subjects factor and *group* (controls, schizotypes) as between-subjects factor. Additional factors were *time* (PSI, STAI-S, MTSD-S, PSQ: pre night, post night), *frequency* (SPEM: 0.2Hz, 0.4Hz), *blank* (SPEM: blanking, non blanking), *task* (prosaccades, antisaccades), *predictability* (predictive saccade conditions: completely predictable, non predictable, spatially predictable, timing predictable), and *saccade type* (predictive saccades: predictive, regular). For blanking SPEM, a parsimonious model with change scores between peak and residual measures were used to display gain deterioration under blanking.

Only main effects and interactions that were not qualified by further higher-order interactions are reported here. Additionally, possible effects of testing order (sleep deprivation first; normal sleep first) are not investigated here as they are not relevant to the current hypotheses. Such effects may, if required, be explored from the raw data (for raw data and full summaries of ANOVAs see Supplementary Material). All dependent variables were screened for violation of normal distribution in each of the two sessions and each of the two groups by Shapiro-Wilk tests (Shapiro & Wilk, 1965), skewness scores and outliers. Homogeneity of variances were tested using Levene's test (Glass, 1966) and Greenhouse-Geisser correction was applied to adjust when sphericity assumption was violated (Jennings, 1987).

Post hoc tests were reported using Bonferroni-Holm-correction (Holm, 1979). For interactions involving *group* and *condition*, alpha level was corrected separately for each of the groups. Based on previous results (Meyhöfer et al., 2017), significant interactions between *condition* and *time* were followed up by one-tailed t-tests for PSI subscales. For STAI-S, MTSD-S, and PSQ variables, significant interactions for *condition* and *time* were followed up by two-tailed t-tests.

Effect sizes for ANOVAs were estimated using partial eta-squared (Cohen, 1973). Effect sizes for independent post hoc t-tests were given using Cohen's *d* (Cohen, 1988). For repeated measures,

an adapted version was applied (Gibbons, Hedeker, & Davis, 1993). To compute Cohen's d the Cran R package *effsize* (Torchiano, 2016) was used.

## RESULTS

### Data Prescreening

A total of *n* = 19 controls and *n* = 17 positive schizotypes met all inclusion criteria and participated in both laboratory sessions (Table 1). An overview of participant selection is in Supplementary Figure 1.

Descriptive results are presented in Tables 2-3 for oculomotor measures and in Table 4 for psychometric measures. Cronbach's $\alpha$ for psychometric measures is in Supplementary Table 2. Due to infrequent SPEM anticipatory and leading saccades, predictive saccade direction errors, and predictive saccades in non predictable and timing predictable saccade conditions, no ANOVAs were computed. Due to low prosaccade error rate, analysis was based only on antisaccade errors.

INCLUDE TABLES 1-4 ABOUT HERE

For sinusoidal SPEM, a control participant had to be removed from analyses due to technical problems. In the blanking SPEM task, five participants had to be excluded due to technical issues (*n* = 2 controls, *n* = 3 schizotypes). For step-ramp SPEM, one control subject had to be excluded due to technical problems. Additionally, for eye acceleration and pursuit latency, six participants were excluded because they failed the trial inclusion criterion (*n* = 1 control, *n* = 5 schizotypes). During pro- and antisaccades, one high schizotype subject had to be excluded due to technical issues and one control subject failed to manage the task instructions. For predictive saccades, one high schizotype subject had to be excluded due to technical issues. Additionally, two schizotypes were excluded because they failed the trial inclusion criterion.

**Oculomotor Measures**

*Effects of Schizotypy*

Main effects of *group* indicated higher sinusoidal SPEM RMSE ($F$[1,33] = 11.34, $p$ = .002, $\eta_p^2$ = 0.26) and catch-up saccade amplitude ($F$[1,32] = 5.46, $p$ = .03, $\eta_p^2$ = 0.15) in schizotypes compared to controls.

*Effects of Sleep Deprivation*

Main effects of *condition* indicated higher sinusoidal SPEM RMSE ($F$[1,33] = 29.22, $p$ < .001, $\eta_p^2$ = 0.47) and catch-up saccade amplitude ($F$[1,32] = 28.48, $p$ < .001, $\eta_p^2$ = 0.47), prolonged step-ramp SPEM latency ($F$[1,27] = 14.46, $p$ = .001, $\eta_p^2$ = 0.35), reduced step-ramp SPEM early gain ($F$[1,33] = 14.67, $p$ = .001, $\eta_p^2$ = 0.31), elevated antisaccade direction error rate ($F$[1,32] = 19.30, $p$ < .001, $\eta_p^2$ = 0.38), higher pro- and antisaccade latencies ($F$[1,32] = 29.03, $p$ < .001, $\eta_p^2$ = 0.48), lower pro- and antisaccade peak velocities ($F$[1,32] = 50.87, $p$ < .001, $\eta_p^2$ = 0.61), and lower predictive saccade gain ($F$[1,31] = 29.11, $p$ < .001, $\eta_p^2$ = 0.48) and peak velocities ($F$[1,31] = 65.42, $p$ < .001, $\eta_p^2$ = 0.68) after sleep deprivation compared to normal sleep. Additionally, for blanking SPEM, an interaction between *condition* and *blank* ($F$[1,29] = 5.74, $p$ = .02, $\eta_p^2$ = 0.17) indicated smaller differences between peak and residual gain after sleep deprivation during blanking ($p$ = .01, $d$ = 0.58) but not during non blanking ($p$ = .90, $d$ = 0.06) half-cycles.

*Interactions between Schizotypy and Sleep Deprivation*

For maintenance gain, an interaction involving *group*, *condition*, and *frequency* ($F$[1,33] = 10.22, $p$ = .003, $\eta_p^2$ = 0.24, Figure 2) indicated no effects at 0.2Hz (controls: normal sleep = sleep deprivation, $p$ = .95, $d$ = 0.01; schizotypes: normal sleep = sleep deprivation, $p$ = .16, $d$ = 0.36) but lower gain at 0.4Hz after sleep deprivation than normal sleep for schizotypes ($p$ = .03, $d$ = 0.67) but not controls ($p$ = .93, $d$ = 0.28). For the blanking SPEM task, an interaction between *condition* and *group* ($F$[1,29] = 5.39, $p$ = .03, $\eta_p^2$ = 0.16) indicated greater differences between peak and residual gain for schizotypes than controls during normal sleep ($p$ = .05, $d$ = 0.67) but not following sleep deprivation ($p$ = 1.00, $d$ = 0.14).

INCLUDE FIGURE 2 ABOUT HERE

*Effects of Tasks*

Main effects of *frequency* showed increased sinusoidal SPEM RMSE ($F$[1,33] = 16.10, $p$ < .001, $\eta_p^2$ = 0.33), catch-up saccade frequency ($F$[1,33] = 274.75, $p$ < .001, $\eta_p^2$ = 0.89), and catch-up saccade amplitude ($F$[1,32] = 61.96, $p$ < .001, $\eta_p^2$ = 0.66) at 0.4Hz. For pro- and antisaccades, main effects of *task* indicated higher latency ($F$[1,32] = 301.59, $p$ < .001, $\eta_p^2$ = 0.90), hypermetric amplitude gain ($F$[1,32] = 9.32, $p$ = .005, $\eta_p^2$ = 0.23), and reduced peak velocity ($F$[1,32] = 60.08, $p$ < .001, $\eta_p^2$ = 0.65) for anti- compared to prosaccades. Main effects of *predictability* suggested differences between predictive saccade conditions with regard to latencies ($F$[3,93] = 97.97, $p$ < .001, ε = 0.64, $\eta_p^2$ = 0.76; completely predictable < spatially predictable, $p$ = .006, $d$ = 0.66; spatially predictable < timing predictable, $p$ = .006, $d$ = 1.85; timing predictable < non predictable, $p$ = .006, $d$ = 0.50), amplitude gain ($F$[3,93] = 55.31, $p$ < .001, ε = 0.73, $\eta_p^2$ = 0.64; non predictable = timing predictable, $p$ = .43, $d$ = 0.16; timing predictable > spatially predictable, $p$ = .006, $d$ = 1.44; spatially predictable = completely predictable, $p$ = .07, $d$ = 0.23), and peak velocities ($F$[3,93] = 26.03, $p$ < .001, ε = 0.79, $\eta_p^2$ = 0.46; non predictable = timing predictable, $p$ = .85, $d$ = 0.17; timing predictable > spatially predictable, $p$ = .006, $d$ = 1.10; spatially predictable = completely predictable, $p$ = .64, $d$ = 0.18). Additionally, for spatially predictable saccades, the main effect of *saccade type* revealed more regular than predictive saccades ($F$[1,31] = 12.26, $p$ = .001, $\eta_p^2$ = 0.28).

**Psychometric Measures**

*Effects of Schizotypy*

Main effects of *group* indicated higher scores for schizotypes than controls on Delusional Thinking ($F$[1,34] = 46.97, $p$ < .001, $\eta_p^2$ = 0.58), Perceptual Distortion ($F$[1,34] = 37.73, $p$ < .001, $\eta_p^2$ = 0.53), Cognitive Disorganization ($F$[1,34] = 29.05, $p$ < .001, $\eta_p^2$ = 0.46), Anhedonia ($F$[1,34] = 16.61, $p$ < .001, $\eta_p^2$ = 0.33), Mania ($F$[1,34] = 15.60, $p$ < .001, $\eta_p^2$ = 0.32), Paranoia ($F$[1,34] = 12.37, $p$ = .001, $\eta_p^2$ = 0.27), STAI-S ($F$[1,34] = 7.41, $p$ = .01, $\eta_p^2$ = 0.18), MTSD-S ($F$[1,34] = 14.89, $p$ < .001, $\eta_p^2$ = 0.31), and PSQ ($F$[1,34] = 27.42, $p$ < .001, $\eta_p^2$ = 0.45).

*Effects of Sleep Deprivation*

Effects of sleep deprivation were detected by interactions between *condition* and *time* in Perceptual Distortion ($F$[1,34] = 5.07, $p$ = .03, $\eta_p^2$ = 0.13), Cognitive Disorganization ($F$[1,34] = 20.68, $p$ < .001, $\eta_p^2$ = 0.38), Anhedonia ($F$[1,34] = 7.49, $p$ = .01, $\eta_p^2$ = 0.18), STAI-S ($F$[1,34] = 7.77, $p$ = .009, $\eta_p^2$ = 0.19), and MTSD-S ($F$[1,34] = 16.57, $p$ < .001, $\eta_p^2$ = 0.33). Thus, mean scores were higher in the morning after sleep deprivation than normal sleep (Perceptual Distortion: $p$ = .04, $d$ = 0.40; Cognitive Disorganization: $p$ = .004, $d$ = 0.82; Anhedonia: $p$ = .02, $d$ = 0.46; STAI-S: $p$ = .004, $d$ = 0.96; MTSD-S: $p$ = .004, $d$ = 0.80) and, additionally, Cognitive Disorganization and MTSD-S scores were higher in the morning after sleep deprivation compared to the evening before (Perceptual Distortion: $p$ = .11, $d$ = 0.31; Cognitive Disorganization: $p$ = .004, $d$ = 0.66; Anhedonia: $p$ = .11, $d$ = 0.29; STAI-S: $p$ = .21, $d$ = 0.28; MTSD-S: $p$ = .02, $d$ = 0.48).

*Effects of Time of Day*

Main effects of *time* suggested lower scores in the morning than the evening for Delusional Thinking ($F$[1,34] = 24.41, $p$ < .001, $\eta_p^2$ = 0.42) and Paranoia ($F$[1,34] = 46.90, $p$ < .001, $\eta_p^2$ = 0.58).

## DISCUSSION

The current study aimed to further evaluate the schizotypy and sleep deprivation models using oculomotor biomarkers and, for the first time, to reveal their potential interactions. In short, schizotypes were found to show poorer SPEM performance and higher levels of psychosis-like experiences than controls. Generally, sleep deprivation deteriorated oculomotor control and increased psychotomimetic states across all participants. Most importantly, sleep deprivation impaired SPEM gain at 0.4Hz in schizotypes but not controls.

*Effects of Schizotypy.* Schizotypes displayed higher position error and catch-up saccade amplitude than controls during closed-loop SPEM, consistent with previous evidence from schizophrenia (O'Driscoll & Callahan, 2008) and schizotypy (Gooding et al., 2000). Unlike previous results (Kattoulas et al., 2011), predictive SPEM was not found to be worse in schizotypes compared to controls. Schizophrenia patients tend to have impaired predictive SPEM (Ivleva et al., 2014;

Trillenberg et al., 2016; however see Sprenger, Trillenberg, Nagel, Sweeney, & Lencer, 2013) but effect sizes are rather low (O'Driscoll & Callahan, 2008). Unexpectedly (Ettinger et al., 2005; Koychev et al., 2016), there were no schizotypy related differences in antisaccade performance. Possible reasons include less strict schizotypy inclusion criteria (Gooding, 1999; Holahan & O'Driscoll, 2005) and generally lower error rates in this study compared to prior research (Koychev et al., 2016).

In accordance with previous studies, schizotypes scored higher on psychotomimetic states, anxiety, depression, and stress than controls, consistent with previous research in schizotypy (Barrantes-Vidal et al., 2013; Barrantes-Vidal, Ros-Morente, & Kwapil, 2009; Preti, Bonventre, Ledda, Petretto, & Masala, 2007) and schizophrenia (Buckley, Miller, Lehrer, & Castle, 2009), overall supporting the idea of common underlying mechanisms and that schizophrenia mirrors the extreme end of the schizotypy continuum (Ettinger et al., 2014; Nelson et al., 2013).

*Effects of Sleep Deprivation.* Sleep deprivation impaired SPEM, increased pro- and antisaccade latencies and antisaccade direction error rate, and reduced pro- and antisaccade peak velocities and predictive saccade gain and peak velocities. These findings partly replicate our and other sleep deprivation studies (Lee, Manousakis, Fielding, & Anderson, 2015; Meyhöfer et al., 2017; Tong, Maruta, Heaton, Maule, & Ghajar, 2014) and resemble findings from schizophrenia (Amado et al., 2008; Gooding & Basso, 2008; O'Driscoll & Callahan, 2008). Whilst hypometric saccade gain has been discussed as an adverse effect of antipsychotic medication (Keedy et al., 2014), impairments have also been found in antipsychotic-free (Krebs et al., 2001) and antipsychotic-naïve (Hutton et al., 2001) patients. Overall, these findings thus support the sleep deprivation model of schizophrenia.

The observed effects on pursuit latency, prosaccades, and saccade peak velocities suggest somewhat reduced specificity of the sleep deprivation model, given that these parameters are unaffected in most (Broerse, Crawford, & den Boer, 2001; Lencer et al., 2004) but not all (Clementz & Sweeney, 1990; Haraldsson et al., 2008) studies of schizophrenia. Prolonged latencies and reduced peak velocities might be the results of the sedative impact by the lack of sleep (Reilly et al., 2008). However, sedative effects are not presumed to cause psychosis-like states and, therefore, cannot explain the psychotomimetic results.

Sleep deprivation also led to increased psychotomimetic states and state levels of depression and anxiety. These findings largely replicate our previous study (Meyhöfer et al., 2017; Petrovsky et al., 2014) and broadly agree with previous self-reports of hallucinations and negative symptoms after sleep deprivation (Killgore et al., 2008; Luby et al., 1962; West et al., 1962). Schizophrenia patients also demonstrate higher state levels of depression (Chiappelli et al., 2014), further supporting the validity of the sleep deprivation model.

*Interactions between Schizotypy and Sleep Deprivation*. In addition to these effects of schizotypy and sleep deprivation, the two models interacted on oculomotor performance. Schizotypes but not controls demonstrated sleep deprivation induced reduction in SPEM maintenance gain. Reduced gain is a key measure of SPEM impairments in schizophrenia spectrum disorders, having been reported in schizotypy (Gooding et al., 2000; Holahan & O'Driscoll, 2005; Lenzenweger & O'Driscoll, 2006), after sleep deprivation (Fransson et al., 2008; Tong et al., 2014), in pharmacological models (Steffens et al., 2016), and in patients with schizophrenia (O'Driscoll & Callahan, 2008). Overall, SPEM gain emerged as the most sensitive biomarker that detected common and interactive effects of the two model systems.

Generally, sleep deprived schizotypes showed the largest phenomenological overlap with schizophrenia: Independent of sleep condition and time of day, they demonstrated higher psychosis-like experiences and, additionally, these experiences were worsened by sleep deprivation.

*Limitations and Conclusions.* A limitation of this study concerns the small number of males. Hence, we were not able to investigate gender differences in sensitivity to sleep deprivation. Secondly, we had to combine target eccentricities in the pro- and antisaccade task to achieve an appropriately amount of trials. However, due to known relationships between saccade amplitude and other measures, e.g., peak velocity (Bahill, Clark, & Stark, 1975), further studies should analyze eccentricities separately. A third limitation refers to different methodological approaches in schizotypy research such as extreme groups using mean deviation criteria (Gooding et al., 2000) or absolute questionnaire scores (Koychev et al., 2016), median splits (Klein, Brügner, Foerster, Müller, & Schweickhardt, 2000), and correlative designs (Ettinger et al., 2005). Our inclusion criterion for

high schizotypes was stricter than some (van Kampen & Deijen, 2009) but less strict than other studies (Holahan & O'Driscoll, 2005) and thus may even underestimate existing effects. Lastly, as negative and disorganized schizotypy have previously also been linked to deficits in oculomotor performance (Gooding et al., 2005; Holahan & O'Driscoll, 2005; Smyrnis et al., 2007), further studies might profit from the inclusion of additional subgroups.

In conclusion, our results demonstrate that both schizotypy and sleep deprivation represent replicable and valid model systems of psychosis with regards to self-report and oculomotor biomarkers. Evidence of interactions between these two models suggests that the closest approximation to psychosis is observed in high schizotypy after sleep deprivation. The combination of these systems appears a promising tool for the investigation of underlying mechanisms of schizophrenia and in assisting the development of new antipsychotic and pro-cognitive drugs.

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**Table 1.** Overview of Demographic Variables by Group.

| | **Controls** | **Schizotypes** | Statistics |
|---|---|---|---|
| ***N*** | 19 | 17 | |
| **Mean Age** (*SD*) | 24.63 (5.42) | 26.82 (7.91) | $T(34) = 0.98$, $p = .33$ |
| **Gender** (*n* female/male) | 12/7 | 12/5 | $\chi^2(1) = 0.22$, $p = .64$ |
| **Order** (*n* normal sleep first/sleep deprivation first) | 9/10 | 8/9 | $\chi^2(1) = 0.00$, $p = .99$ |
| **Mean Years of Education** (*SD*) | 16.37 (4.06) | 17.18 (2.88) | $T(34) = 0.68$, $p = .50$ |
| **Mean Verbal IQ** (*SD*) | 30.05 (3.37) | 29.65 (3.74) | $T(34) = 0.34$, $p = .73$ |
| **Mean Unusual Experiences** (*SD*) | 1.79 (0.98) | 9.94 (0.97) | $T(34) = 25.13$, $p < .001$ |
| **Mean Introvertive Anhedonia** (*SD*) | 0.68 (0.48) | 1.12 (0.93) | $T(23.32) = 1.73$, $p = .10$ |

**Table 2.** Descriptive Statistics of SPEM Measures.

| **Sinusoidal SPEM 0.2Hz** | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes |
| | **Maintenance Gain (%)** | | **RMSE (°)** | | **CUS Amplitude[1] (°)** | | **CUS (*N*/sec)** | | **Anticipatory (*N*/sec)** | | **Leading (*N*/sec)** | |
| Sleep | 95.27 (4.48) | 93.21 (4.76) | 0.78 (0.34) | 1.22 (0.60) | 1.22 (0.12) | 1.40 (0.23) | 0.17 (0.14) | 0.17 (0.14) | 0.00 (0.01) | 0.00 (0.01) | 0.00 (0.00) | 0.00 (0.01) |
| Wake | 95.34 (5.66) | 95.49 (5.78) | 1.37 (0.81) | 2.17 (1.19) | 1.58 (0.32) | 1.74 (0.40) | 0.26 (0.16) | 0.31 (0.17) | 0.00 (0.01) | 0.00 (0.01) | 0.00 (0.00) | 0.00 (0.01) |
| **Sinusoidal SPEM 0.4Hz** | | | | | | | | | | | | |
| Sleep | 89.15 (9.52) | 87.90 (6.82) | 1.11 (0.83) | 1.31 (0.69) | 1.63 (0.23) | 1.69 (0.28) | 0.95 (0.39) | 0.92 (0.30) | 0.03 (0.09) | 0.02 (0.03) | 0.00 (0.01) | 0.00 (0.01) |
| Wake | 87.83 (9.42) | 81.98 (8.97) | 1.58 (0.97) | 2.52 (1.10) | 1.90 (0.46) | 2.16 (0.46) | 0.87 (0.39) | 1.02 (0.30) | 0.01 (0.01) | 0.01 (0.02) | 0.01 (0.02) | 0.02 (0.02) |

| **Blanking SPEM** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Gain (%)** | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes |
| | **Peak Blanking** | | **Peak Non Blanking** | | **Residual Blanking** | | **Residual Non Blanking** | |
| Sleep | 94.09 (8.34) | 91.80 (12.37) | 93.89 (9.60) | 93.38 (10.07) | 50.53 (20.76) | 35.48 (14.32) | 83.56 (12.74) | 78.26 (14.01) |
| Wake | 98.24 (12.24) | 94.23 (15.21) | 100.47 (16.94) | 91.64 (10.58) | 58.56 (17.99) | 54.20 (24.40) | 87.90 (8.47) | 79.92 (14.41) |
| **Saccades (*N*/sec)** | | | | | | | | |
| Sleep | 1.32 (0.76) | 1.37 (0.63) | 1.05 (0.68) | 1.63 (0.81) | 1.65 (0.64) | 1.68 (0.82) | 1.23 (0.87) | 1.58 (0.49) |
| Wake | 1.32 (0.70) | 1.25 (0.80) | 1.06 (0.63) | 1.74 (0.55) | 1.50 (0.69) | 1.40 (0.81) | 1.13 (0.43) | 1.52 (0.57) |

| **Step-ramp SPEM** | | | | | | |
|---|---|---|---|---|---|---|
| | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes |
| | **Initial Eye Acceleration (°/sec²)** | | **Latency (sec)** | | **Early Gain (%)** | |
| Sleep | 64.54 (22.71) | 57.31 (17.19) | 0.21 (0.02) | 0.22 (0.03) | 91.46 (7.98) | 84.53 (11.97) |
| trials included | 22 (12-38) | 17 (6-37) | 22 (12-38) | 17 (6-37) | | |
| Wake | 57.31 (22.21) | 51.41 (22.46) | 0.23 (0.03) | 0.23 (0.03) | 82.87 (12.62) | 80.37 (8.75) |
| trials included | 11 (6-30) | 12 (5-34) | 11 (6-30) | 12 (5-34) | | |

*Note*: Data represent untransformed means (standard deviations). *SPEM* = smooth pursuit eye movements. *Sleep* = normal sleep night. *Wake* = sleep deprivation night. Sinusoidal SPEM: $n_{controls}$ = 18. $n_{schizotypes}$ = 17. [1]For one control participant catch-up saccade frequency in the 0.2Hz sleep condition was zero and, therefore, he was excluded from catch-up saccade amplitude analysis. Blanking SPEM: $n_{controls}$ = 17. $n_{schizotypes}$ = 14. Step-ramp SPEM initial eye acceleration and latency: $n_{controls}$ = 17. $n_{schizotypes}$ = 12. Step-ramp SPEM early gain: $n_{controls}$ = 18. $n_{schizotypes}$ = 17.

**Table 3.** Descriptive Statistics of Saccade Measures.

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Prosaccades** | | | | | | | | | | |
| | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes |
| | **Latency (msec)** | | **Amplitude Gain (%)** | | **Peak Velocity (°/sec)** | | **Direction Errors (%)** | | **Trials Included** | |
| Sleep | 157.51 (14.12) | 166.44 (12.78) | 94.18 (5.65) | 91.43 (7.28) | 306.16 (45.61) | 297.15 (52.61) | 0.00 (0.00) | 0.00 (0.00) | 30 (22-32) | 29 (23-32) |
| Wake | 171.68 (21.31) | 179.72 (19.61) | 85.69 (6.93) | 88.78 (9.20) | 259.90 (37.89) | 261.29 (44.87) | 0.24 (1.02) | 0.54 (1.49) | 26 (18-32) | 24 (10-32) |
| **Antisaccades** | | | | | | | | | | |
| Sleep | 248.45 (33.26) | 254.09 (33.11) | 105.95 (43.49) | 111.25 (23.71) | 261.70 (63.34) | 258.84 (57.93) | 22.98 (19.13) | 27.01 (21.51) | 30 (21-32) | 29 (23-32) |
| Wake | 269.54 (61.20) | 285.98 (33.60) | 102.37 (39.64) | 110.52 (34.93) | 214.37 (47.70) | 218.18 (48.32) | 35.33 (22.33) | 41.38 (26.70) | 23 (7-32) | 22 (7-32) |
| **Predictive Saccades: Completely Predictable** | | | | | | | | | | |
| | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes | Controls | Schizotypes |
| | **Latency (msec)** | | **Amplitude Gain (%)** | | **Peak Velocity (°/sec)** | | **Predictive Saccades (%)** | | **Regular Saccades (%)** | | **Direction Errors (%)** | |
| Sleep | 27.30 | 28.44 | 85.02 | 83.02 | 305.52 | 301.03 | 47.13 | 44.23 | 33.44 | 35.84 | 0.00 | 0.00 |
| | (69.96) | (78.81) | (8.03) | (9.04) | (59.26) | (51.91) | (23.96) | (22.90) | (19.20) | (23.39) | (0.00) | (0.00) |
| Wake | 38.37 | 43.54 | 82.00 | 79.17 | 270.88 | 262.31 | 44.03 | 45.88 | 39.39 | 40.38 | 0.00 | 0.00 |
| | (71.84) | (83.01) | (9.00) | (8.29) | (48.44) | (44.79) | (26.32) | (25.74) | (23.42) | (20.93) | (0.00) | (0.00) |
| Trials Included: Controls: Sleep = 21 (17-22), Wake = 16 (6-22); Schizotypes: Sleep = 20 (11-22), Wake = 16 (11-21) | | | | | | | | | | | | |
| **Predictive Saccades: Non Predictable** | | | | | | | | | | | | |
| Sleep | 153.21 | 158.11 | 94.83 | 93.98 | 332.16 | 331.08 | 2.82 | 4.24 | 91.79 | 88.72 | 0.07 | 0.10 |
| | (20.10) | (21.70) | (5.00) | (5.60) | (51.93) | (53.43) | (5.51) | (6.28) | (10.35) | (11.65) | (0.09) | (0.09) |
| Wake | 172.59 | 164.24 | 91.10 | 90.16 | 291.28 | 296.82 | 1.33 | 7.25 | 93.79 | 87.68 | 0.03 | 0.09 |
| | (24.05) | (27.77) | (10.02) | (4.56) | (55.61) | (40.78) | (3.33) | (7.64) | (9.34) | (9.59) | (0.03) | (0.08) |
| Trials Included: Controls: Sleep = 21 (15-22), Wake = 17 (10-22); Schizotypes: Sleep = 21 (16-22), Wake = 16 (9-22) | | | | | | | | | | | | |
| **Predictive Saccades: Spatially Predictable** | | | | | | | | | | | | |
| Sleep | 74.71 | 65.69 | 88.92 | 85.44 | 311.16 | 306.87 | 29.68 | 30.80 | 51.71 | 50.20 | 0.00 | 0.00 |
| | (63.55) | (72.70) | (7.58) | (5.94) | (58.74) | (56.20) | (24.53) | (22.99) | (26.06) | (26.89) | (0.00) | (0.00) |
| Wake | 81.44 | 75.01 | 82.48 | 80.13 | 270.46 | 269.10 | 31.28 | 30.08 | 54.15 | 49.49 | 0.01 | 0.00 |
| | (63.30) | (45.20) | (9.88) | (7.76) | (53.14) | (51.93) | (21.30) | (17.23) | (25.57) | (14.56) | (0.03) | (0.00) |
| Trials Included: Controls: Sleep = 21 (19-22), Wake = 16 (8-22); Schizotypes: Sleep = 20 (11-22), Wake = 14 (6-22) | | | | | | | | | | | | |
| **Predictive Saccades: Timing Predictable** | | | | | | | | | | | | |
| Sleep | 140.65 | 150.04 | 94.06 | 93.02 | 335.90 | 331.86 | 7.16 | 7.52 | 83.29 | 84.34 | 0.06 | 0.11 |
| | (38.59) | (33.13) | (7.26) | (3.13) | (55.44) | (53.56) | (12.52) | (10.43) | (15.32) | (13.11) | (0.07) | (0.12) |
| Wake | 149.60 | 154.67 | 90.94 | 89.82 | 291.22 | 288.58 | 7.25 | 7.37 | 86.34 | 83.34 | 0.08 | 0.11 |
| | (27.60) | (33.23) | (7.03) | (4.44) | (45.20) | (46.94) | (7.93) | (9.31) | (13.40) | (12.76) | (0.09) | (0.11) |
| Trials Included: Controls: Sleep = 21 (20-22), Wake = 18 (8-22); Schizotypes: Sleep = 21 (18-22), Wake = 16 (7-22) | | | | | | | | | | | | |

*Note*: Data represent untransformed means (standard deviations). *Sleep* = normal sleep night. *Wake* = sleep deprivation night. Pro- and antisaccades: $n_{controls}$ = 18. $n_{schizotypes}$ = 16. Predictive saccades: $n_{controls}$ = 19. $n_{schizotypes}$ = 14.

**Table 4.** Descriptive Statistics of Psychometric Measures.

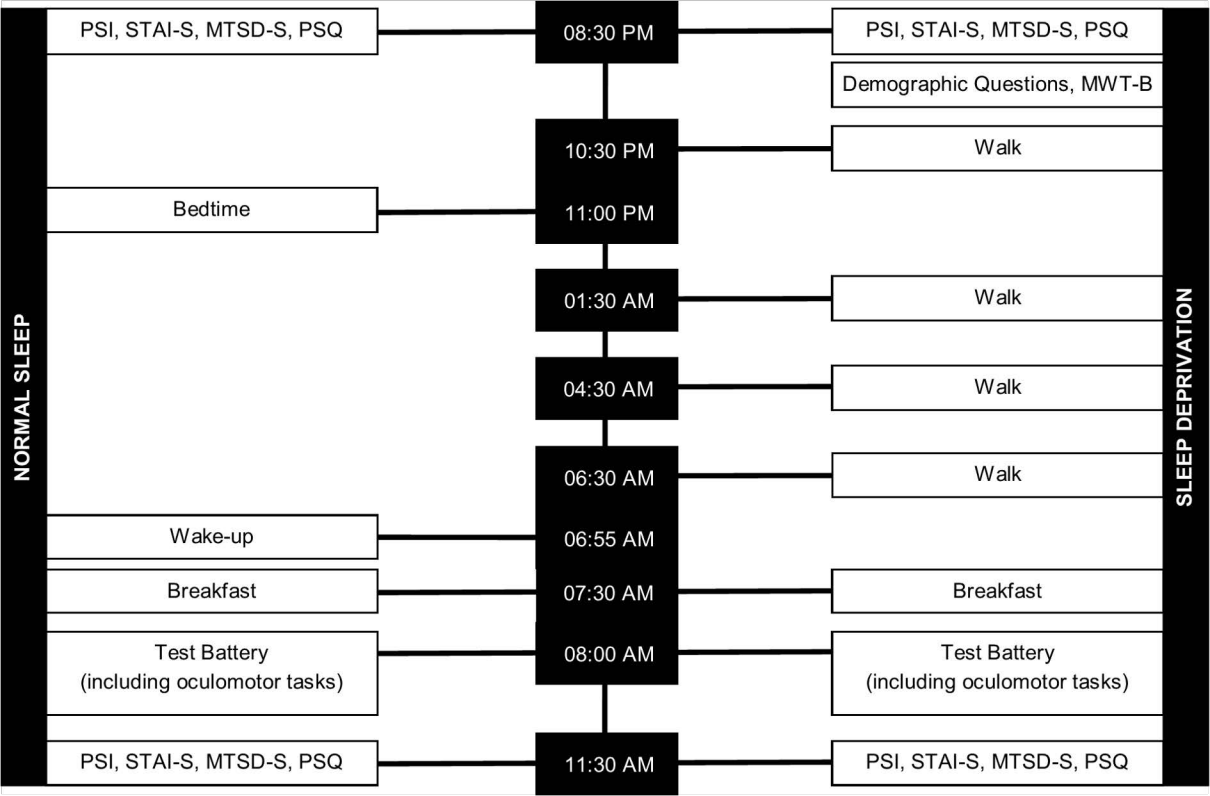
| | Normal Sleep | | | | Sleep Deprivation | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre Night | | Post Night | | Pre Night | | Post Night | |
| | **Controls** | **Schizotypes** | **Controls** | **Schizotypes** | **Controls** | **Schizotypes** | **Controls** | **Schizotypes** |
| **Delusional Thinking** | 1.32 (1.80) | 7.94 (3.78) | 0.89 (1.76) | 5.88 (4.28) | 1.42 (2.80) | 7.47 (4.60) | 0.89 (2.77) | 6.59 (5.41) |
| **Perceptual Distortion** | 1.11 (2.26) | 5.76 (4.68) | 1.00 (2.00) | 4.76 (3.29) | 1.05 (1.96) | 6.12 (4.51) | 1.32 (2.45) | 7.94 (4.70) |
| **Cognitive Disorganization** | 3.74 (3.97) | 10.85 (6.15) | 2.84 (3.13) | 9.47 (4.74) | 3.63 (4.10) | 9.94 (6.07) | 5.42 (5.24) | 14.76 (4.72) |
| **Anhedonia** | 2.55 (2.72) | 5.53 (2.58) | 1.87 (2.38) | 4.76 (2.39) | 2.32 (2.14) | 4.71 (2.87) | 3.05 (3.44) | 6.41 (3.57) |
| **Mania** | 2.47 (1.81) | 6.06 (3.38) | 2.05 (1.47) | 4.47 (2.85) | 2.47 (2.12) | 5.74 (3.64) | 2.00 (1.63) | 5.53 (3.54) |
| **Paranoia** | 2.00 (3.23) | 5.59 (4.40) | 0.89 (1.52) | 2.76 (3.91) | 2.16 (3.04) | 4.97 (3.70) | 1.05 (2.09) | 3.35 (2.91) |
| **STAI-S** | 28.68 (5.53) | 36.18 (9.32) | 28.00 (4.53) | 33.24 (8.47) | 31.89 (4.97) | 35.12 (8.47) | 32.68 (6.39) | 39.53 (10.75) |
| **MTSD-S** | 2.63 (4.61) | 10.71 (7.64) | 2.05 (2.55) | 7.41 (7.39) | 3.32 (4.67) | 8.53 (6.12) | 4.95 (4.52) | 12.06 (8.04) |
| **PSQ** | 34.11 (8.10) | 50.18 (12.01) | 32.11 (7.98) | 48.00 (10.63) | 34.74 (6.23) | 48.29 (10.73) | 34.79 (7.76) | 48.53 (9.42) |

*Notes.* Data represent untransformed means (standard deviations). *STAI* = State-Trait Anxiety Inventory, State Scale. *MTSD-S* = Maryland Trait and State Depression, State Scale. *PSQ* = Perceived Stress Questionnaire. $n_{controls} = 19$. $n_{schizotypes} = 17$.

**Figure Legends**

**Figure 1.** Overview of the laboratory sessions. *PSI* = Psychotomimetic States Inventory. *STAI* = State-Trait Anxiety Inventory, State Scale. *MTSD-S* = Maryland Trait and State Depression, State Scale. *PSQ* = Perceived Stress Questionnaire. *MWT-B* = Mehrfachwahl-Wortschatz-Intelligenztest, Version B.

**Figure 2.** Untransformed mean scores (± standard error of the mean) of sinusoidal SPEM maintenance gain. *SPEM* = smooth pursuit eye movements. $n_{controls} = 18$. $n_{schizotypes} = 17$. Significant post hoc effects are flagged and effect sizes are given (Gibbons et al., 1993).


NORMAL SLEEP
SLEEP DEPRIVATION
PSI, STAI-S, MTSD-S, PSQ
08:30 PM
PSI, STAI-S, MTSD-S, PSQ
Demographic Questions, MWT-B
10:30 PM
Walk
Bedtime
11:00 PM
01:30 AM
Walk
04:30 AM
Walk
06:30 AM
Walk
Wake-up
06:55 AM
Breakfast
07:30 AM
Breakfast
Test Battery
(including oculomotor tasks)
08:00 AM
Test Battery
(including oculomotor tasks)
PSI, STAI-S, MTSD-S, PSQ
11:30 AM
PSI, STAI-S, MTSD-S, PSQ