



Sleep quality and paranoia: The role of alexithymia, negative emotions and perceptual anomalies



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ABSTRACT

Recent evidence suggests that sleep problems are associated with psychotic like experiences including paranoia. However, the mechanisms underpinning this association are not well understood and thus studies modelling hypothesised mediating factors are required. Alexithymia, the inability to recognise and describe emotions within the self may be an important candidate. In two separate studies we sought to investigate factors mediating the relationship between sleep quality and paranoia using a cross-sectional design. Healthy volunteers without a mental health diagnosis were recruited (study 1, N = 401, study 2, N = 402). Participants completed a series of measures assessing paranoia, negative emotions, alexithymia and perceptual anomalies in an online survey. In study 1, regression and mediation analyses showed that the relationship between sleep quality and paranoia was partially mediated by alexithymia, perceptual anomalies and negative affect. In contrast, study 2 found that the relationship between sleep quality and paranoia was fully mediated by negative affect, alexithymia and perceptual anomalies. The link between sleep quality and paranoia is unclear and reasons for discrepant results are discussed. Novel findings in this study include the link between alexithymia and paranoia.

1. Introduction

Paranoia, defined as the unfounded fear that others intend to cause you harm, is a common and distressing experience reported by many individuals with psychosis (Freeman and Garety, 2000). Paranoid thinking is not confined to psychosis, and is reported by up to 30% of the general population (Freeman et al., 2005, Freeman, 2007). However, studying symptoms like paranoia at a sub-clinical level can further inform our knowledge about symptoms at a clinical level in line with psychosis continuum models, as well as identify candidate variables for future clinical research (van Os et al., 2009).

Recent research has identified a robust link between sleep disturbances and paranoia. At the non-clinical level, sleep loss in healthy individuals leads to an increase in paranoid thoughts (Kahn-Greene et al., 2007; Reeve et al., 2017). Another study administered the Oxford Sleep Survey which includes questions on sleep and psychotic like experiences (PLE's) to over 1000 students. Results found links between a number of sleep disorder symptoms such as insomnia, nightmare frequency and nightmare distress and PLE's including paranoia (Sheaves et al., 2016). In a large –scale general population study conducted of 8580 people, there were strong relationships between insomnia and paranoia. Insomnia was associated with an approximately two to

threefold increase in paranoid thinking (Freeman et al., 2010). Prospectively, insomnia was also a significant predictor of new incidence of paranoid thoughts, suggestive of insomnia having a causal role (Freeman et al., 2012).

Furthermore, treating sleep disturbance with Cognitive Behavioural Therapy for Insomnia (CBT-i) leads to improvements in sleep, paranoia, perceptual anomalies and emotional variables, in both clinical and non-clinical samples (Myers et al., 2011; Freeman et al., 2015, 2017).

With the link between sleep and paranoia being established, the next step turns to identifying how sleep and paranoia are related (Reeve et al., 2015). This question can be addressed by mediation analysis. Mediation is a regression based statistical approach that allows us to identify factors that significantly explain the variance between our independent (sleep) and dependent variable (paranoia) (Hayes, 2013). These factors are termed mediators and can be useful in helping us identify targets for treatments, as well as helping refine models of paranoid thinking.

Negative emotions such as anxiety and depression are linked to both sleep and paranoia (Baglioni et al., 2011; Freeman et al., 2012) and have been identified as potential mediators. Indeed, research has shown that negative emotions account largely for the relationship between sleep and paranoia (Freeman et al., 2009, 2010; Mulligan et al., 2016;

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Reeve et al., 2017). Along with negative emotions, another emotion related mediator that has been studied recently is emotion regulation. Grezellschak et al. (2016) found that the effect of insomnia on paranoid ideation was mediated by a frequent use of ineffective strategies such as expressive suppression and an infrequent use of effective strategies such as reappraisal. This suggests there may be a number of emotional processes linking sleep to paranoia.

Along with emotional processes, non-emotional factors such as perceptual anomalies may also mediate the link between sleep and paranoia (Freeman et al., 2010). Sleep deprivation has been known to induce perceptual anomalies such as hallucinations (West et al., 1962). More recently, Sheaves et al. (2016) showed that insomnia was associated with perceptual anomalies (hallucinations), even after controlling for negative emotions. Furthermore, perceptual distortions are considered central to paranoia (Freeman et al., 2002). No direct test of this exists although cannabis use which can induce anomalous experiences partially mediates the relationship between insomnia and paranoia (Freeman et al., 2010).

We sought to replicate and build on some of these findings. The main aim of our paper is to investigate the role of alexithymia as a potential mediator. Alexithymia is an aspect of emotion that is concerned with an individual's ability to recognise, and verbalise their emotions (Bagby et al., 1994). Individuals with high alexithymia experience difficulties in identifying their emotions, describing their feelings, and exhibit an externally orientated style of thinking which can be accompanied with arousal (Bagby et al., 1994). Alexithymia is a relevant candidate as it linked to both sleep and paranoia. One study reported that those with low levels of emotional awareness (high alexithymia) reported higher suspiciousness (Boden and Berenbaum, 2007). Higher levels of alexithymia in people with psychosis have also been reported in comparison to controls (Van-Wout et al., 2007; Kimhy et al., 2012). Furthermore, alexithymia is linked to a range of sleep disturbances including insomnia, daytime sleepiness and nightmares (Bauermann et al., 2008). It has also been shown that under conditions of sleep deprivation there is a decrease in emotional intelligence- a trait similar to alexithymia (Killgore et al., 2008). More specifically, this study reported a decrease in the intrapersonal functioning component of emotional intelligence which is concerned with knowing and understanding our feelings (Killgore et al., 2008).

Therefore, we predicted that alexithymia mediates the relationship between sleep quality and paranoia, in addition to negative emotional states such as anxiety and depression.

A secondary aim of our study was to test whether perceptual anomalies mediates the relationship between sleep and paranoia. To do so, we included a comprehensive measure of perceptual anomalies- the Cardiff Anomalous Perceptions Scale (Bell et al., 2006).

In study 1 we hypothesised that; 1) sleep quality, negative emotions, alexithymia and perceptual anomalies predict paranoid thoughts and 2) that the relationship between sleep quality and paranoia will be mediated by negative emotions, alexithymia and perceptual anomalies. Study 2 was conducted to try and replicate the mediation model we developed in study.

2. Study 1: methods

2.1. Participants and procedures

In order to recruit a large sample, and provide a safe, environment for volunteers to disclose paranoid thoughts, an anonymous online survey was set up using Qualtrics (Provo, UT) (2017). The sample was recruited using the University of Glasgow's School of Psychology's online subject pool. Participants were excluded from the study if they reported a diagnosed psychiatric/physical condition, traumatic brain injury, recreational drug use and were aged under 16. The online survey was available online from 2012 to 2013. The study was approved by the University of Glasgow, School of Psychology Ethics

committee.

2.2. Measures

2.2.1. Positive and negative affect schedule (Watson et al., 1988, PANAS)

The PANAS has two ten-item subscales of positive (PA) and negative (NA) affect. The questionnaire has been psychometrically evaluated in non-clinical groups (Watson et al., 1988). The range of scores for each sub-scale ranges from 10 to 50, with higher scores meaning higher negative or positive affect. The PANAS can be used to assess affect on various time scales by altering the instructions. For the present study 'current moment' assessment was used. Internal consistency in the study was ($\alpha = 0.84$).

2.2.2. Hospital anxiety and depression scale (Zigmond and Snaith, 1983, HADS)

The HADS is a self-report rating scale designed to measure both anxiety and depression over the past week. It consists of two subscales, each containing seven items on a 4-point Likert scale (ranging from 0 to 3). It is scored by summing the ratings for the 7 items of each subscale to yield separate scores for anxiety and depression. Internal consistency in the study was ($\alpha = 0.82$). The questionnaire has been psychometrically evaluated in non-clinical groups (Crawford et al., 2001)

2.2.3. Toronto alexithymia scale (Bagby et al., 1994, TAS-20)

The TAS – 20 has a 3-factor structure. Factor 1 assesses difficulty in identifying feelings. Factor 2 assesses difficulty in describing feelings. Factor 3 assesses externally oriented thinking. Scores range from 20 to 100 with higher scores reflect higher alexithymia. There is no timescale for alexithymia as it is measured as a trait factor. Internal consistency for the TAS-20 is high $\alpha = 0.81$ (Bagby et al., 1994). The questionnaire has been psychometrically evaluated in non-clinical groups (Bagby et al., 1994).

2.2.4. Pittsburgh sleep quality index (Buysse et al., 1989, PSQI)

The PSQI provides a reliable, valid, and standardized measure of sleep quality. Scores range from 0 to 21 with higher scores representing poorer sleep. A PSQI global score > 5 indicates that a subject is having severe difficulty in at least two areas, or moderate difficulty in more than three areas of sleep quality in the past month. Internal consistency of the PSQI is high $\alpha = 0.83$ (Buysse et al., 1989). The questionnaire has been psychometrically evaluated in non-clinical groups (Mollaveva et al., 2016).

2.2.5. Green paranoid thoughts scale part B (Green et al., 2008, GPTS)

The G-PTS is a 16-item measure of paranoia, assessing ideas of persecution over the past month. Scores range from 16 to 80 with higher scores indicating greater levels of paranoid thinking. The questionnaire includes subscales for conviction, preoccupation, and distress. The questionnaire has been psychometrically evaluated in clinical and non-clinical populations. Internal consistency in the study was ($\alpha = 0.92$).

2.2.6. Cardiff anomalous perceptions scale (Bell et al., 2006, CAPS)

The CAPS is a 32-item questionnaire, developed in both non-clinical and psychosis groups, and assesses perceptual anomalies such as changes in levels of sensory intensity, distortion of the external world, sensory flooding and hallucinations. Scores range from 0 to 32, higher scores mean higher endorsement of perceptual anomalies. For the present study the time scale was changed to the 'past month' in order to provide a more current assessment of perceptual anomalies. Internal consistency in the study was ($\alpha = 0.88$). The questionnaire has been psychometrically evaluated in non-clinical groups (Bell et al., 2006).

2.3. Data analysis and preparation

The GPTS showed high levels of skewness, which was expected as paranoia is thought to lie on a quasi-continuum, whereby ‘many endorse few paranoid thoughts and few endorse many paranoid thoughts’ (Verdoux and van Os, 2002; Freeman et al., 2005). To address this we dichotomised the variable. Subjects with paranoia scores at the 75th percentile or above were classed as the ‘high paranoid’ group and those below the 75th percentile as the ‘low-paranoid’ group (methodology adopted from Stopa and Clark, 2001). The high paranoid group in our sample included a range of scores from 20 to 73, with some of the scores reaching levels that are reported in clinical groups (Green et al., 2008). However, it is normal for non-clinical samples to have some overlap with clinical samples (Green et al., 2008).

Next, linear regression models were run to test which variables significantly predicted paranoid group membership. Our dependent variable was paranoid group (0 = low paranoia, 1 = high paranoia). Forced entry and backward stepwise models were run to ensure reliability of results.

Mediation analysis was conducted using the SPSS macro PROCESS (model 4) which uses a bootstrapping approach to mediation (Hayes, 2013). Bootstrapping involves creating a repeated series of representations of the population by resampling from the current sample to replicate the original sampling procedure (Hayes, 2013). For the current study, we chose to set the number of bootstrapping samples to 10,000. In turn, these 10,000 bootstrapping samples were used to generate a 95% confidence interval for the mediation effect. The mediation effect is statistically significant if the confidence interval does not contain the value of zero (Hayes, 2013). For ease of interpretation, standardised values are presented unless otherwise stated.

3. Study 1: results

3.1. Descriptive statistics and correlations

Table 1 presents the samples demographics. A total of 401 individuals ($n = 93$ males, 308 females) completed the survey with a mean age of 24 ($SD = 8.1$, range 16–70). The correlations between all the variables are shown in Table 2. Age was correlated with several of our predictors including sleep quality, alexithymia and perceptual anomalies, therefore we included it as a co-variate in further analysis. A series of Mann-Whitney U tests revealed gender was not related to any of our variables except anxiety and depression. Anxiety scores were significantly higher in females ($Mdn = 8$) than males ($Mdn = 7$), $U = 11985.5$, $p = 0.01$ and depression scores were significantly higher in males ($Mdn = 5$) than females ($Mdn = 4$), $U = 12340.5$, $p = 0.04$. As these differences are small, we chose not to include gender as a covariate in further analysis.

Table 1
Sample demographics for study 1 ($N = 401$).

	Mean	SD	Median	Range
Age	24.08	8.15	22.00	16–70
PSQI	6.56	3.27	6.00	0–21
PA	24.73	8.15	24.00	11–50
NA	15.87	6.44	14.00	10–40
HADS-A	8.15	3.91	8.00	0–20
HADS-D	4.59	3.28	4.00	0–19
TAS-20	48.08	11.55	48.00	23–79
GPTS	20.03	8.43	17.00	16–70
CAPS	3.73	4.73	2.00	0–28

Abbreviations: PSQI- Pittsburgh Sleep Quality Index, PA and NA- Positive and Negative Affect, HADS-A and D Hospital Anxiety and Depression Scale respectively, TAS-20 – 20-item Toronto Alexithymia Scale, GPTS- paranoia subscale of the Green Paranoid Thoughts Scale, CAPS- Cardiff Anomalous Perceptions Scale.

Table 2
Spearman rho correlations for study 1.

Measure	1	2	3	4	5	6	7	8
Age								
PA	0.13*							
NA	–0.12*	–0.02						
HADS- A	0.00	–0.11*	0.47*					
HADS-D	0.01	–0.29*	0.42*	0.46*				
PSQI	–0.13*	–0.23*	0.24*	0.36*	0.35*			
TAS	–0.18*	–0.28*	0.27*	0.43*	0.41*	0.33*		
GPTS	–0.13*	0.09	0.28*	0.24*	0.20*	0.19*	0.30*	
CAPS	–0.18*	–0.05	0.21*	0.29*	0.25*	0.23*	–0.29	0.39*

Abbreviations: PSQI- Pittsburgh Sleep Quality Index, PA and NA- Positive and Negative Affect, HADS-A and D Hospital Anxiety and Depression Scale respectively, TAS-20 – 20-item Toronto Alexithymia Scale, GPTS- paranoia subscale of the Green Paranoid Thoughts Scale, CAPS- Cardiff Anomalous Perceptions Scale.

* $p < 0.05$.

3.2. Logistic regression

Hypothesis 1: Sleep quality, negative emotions, alexithymia and perceptual anomalies predict paranoia

Descriptive statistics of the low and high paranoid group are presented in Table 3. Mann-Whitney U tests were used to test the validity of our grouping. Validity of our grouping was confirmed as the high paranoid group reported higher levels of negative affect, anxiety, depression, alexithymia, perceptual anomalies and paranoia. There were no differences in positive affect or gender.

Next, we ran logistic regressions with the predictors entered being age, negative affect, sleep quality, anxiety, depression, perceptual anomalies and alexithymia. The result of both logistic regressions is shown in Table 4. A test of the full model against a constant only model (no predictors) was statistically significant, indicating that the predictors reliably distinguished between the paranoid and non-paranoid groups ($\chi^2 = 69.078$, $p < 0.001$). The Wald test was used to determine what predictors are significant. Significant predictors of paranoid group membership included negative affect, sleep quality, alexithymia and perceptual anomalies. Age, anxiety and depression did not significantly predict paranoid group membership. The log odds (Exp B) column in Table 4 is an indicator of the log odds of being in the paranoid group due to a one point increase in the predictor variable. For example, a one unit increase in negative affect increases the odds of being in the paranoid group by 1.05, 95%CI [1.00–1.09]. Nagelkerke's R^2 of 0.231 indicated that 23% of the variance was explained by the significant

Table 3
Descriptive information of the high and low paranoid groups for study 1.

	Low Paranoia ($n = 295$) Mean (SD)	High Paranoia ($n = 106$) Mean (SD)	Test Statistic (DF)	P Value
Gender	69:226	24:82	$\chi^2(1) = 0.02$	0.49
Age	24.59(8.66)	22.67(6.37)	$U = 13,569$	0.04
PSQI	6.11(3.04)	7.81(3.57)	$U = 11,073$	< .001
PA	24.84(8.08)	24.42(8.39)	$U = 15,039$	0.56
NA	14.86(5.72)	18.67(7.46)	$U = 10,768$	< .001
HADS-A	7.65(3.81)	9.55(3.87)	$U = 11,334$	< .001
HADS-D	4.30(3.16)	5.38(3.50)	$U = 12,642$	< .001
TAS-20	46.23(11.56)	53.25(9.85)	$U = 10,092$	< .001
GPTS	16.60(9.95)	29.60(11.95)	$U = 000$	< .001
CAPS	2.77(3.53)	6.40(6.37)	$U = 9121$	< .001

Abbreviations: PSQI- Pittsburgh Sleep Quality Index, PA and NA- Positive and Negative Affect, HADS-A and D Hospital Anxiety and Depression Scale respectively, TAS-20 – 20-item Toronto Alexithymia Scale, GPTS- paranoia subscale of the Green Paranoid Thoughts Scale, CAPS- Cardiff Anomalous Perceptions Scale.

Table 4
Logistic regression of variables that predict membership of being in the Paranoid Group in study 1.

Model	B(SE)	Wald	Sig	Exp(B)	95% CI	
					Lower	Upper
Forced Entry model						
Constant	-3.85(0.82)	21.81	0.00	n/a		
Age	-0.22(0.01)	1.34	0.24	0.97	0.94	1.01
NA	0.04(0.02)	4.91	0.02	1.05	1.00	1.09
HADS-A	0.01(0.04)	0.10	0.74	1.01	0.93	1.09
HADS-D	-0.05(0.04)	0.72	0.27	0.95	0.86	1.04
PSQI	0.08(0.04)	4.39	0.03	1.09	1.00	1.18
TAS-20	0.03(0.01)	6.24	0.01	1.03	1.00	1.05
CAPS	0.10(0.02)	14.00	0.00	1.11	1.05	1.17
Stepwise model (Backward)						
Constant	-4.31(0.64)	44.90	0.00	n/a		
NA	0.04(0.01)	5.30	0.02	1.04	1.00	1.08
PSQI	0.07(0.04)	3.86	0.04	1.08	1.00	1.17
TAS-20	0.03(0.01)	6.96	0.00	1.03	1 0.00	1.05
CAPS	0.10(0.02)	14.36	0.00	1.11	1.05	1.17

predictors. Similar results were obtained in the stepwise logistic regression which also found that negative affect, sleep quality, alexithymia and perceptual anomalies predicted paranoid group membership and this model explained 22% of the variance (Nagelkerke's R² of 0.222). In sum, two separate regression models showed that negative affect, alexithymia, perceptual anomalies and sleep quality predicted membership of the paranoid group.

3.3. Main analysis: mediation

We next tested hypothesis 2 that the relationship between sleep quality and paranoid thinking is mediated by negative emotion, by alexithymia and by levels of perceptual anomalies. As, anxiety and depression were not significant predictors of paranoia in the logistic regressions, we did not include these variables in mediation analysis. Paranoid group (high and low) was entered as the dependent variable, sleep quality as the predictor variable, age as a co-variate and alexithymia, negative mood and perceptual anomalies were entered as mediators. This model was significant as revealed by the bootstrapped confidence intervals all being above 0.

The indirect (mediation) pathway from PSQI to paranoia via negative affect was significant $b = 0.06$, 95% CI [0.009, 0.143], the indirect (mediation) pathway from sleep quality to paranoia via alexithymia was significant $b = 0.11$, 95% CI [0.033, 0.216], the indirect (mediation) pathway from sleep quality to paranoia via perceptual anomalies was significant, $b = 0.13$, 95% CI [0.03, 0.21]. Finally, the direct pathway from sleep quality to paranoia was significant, $b = 0.26$, 95% CI [0.009, 0.527]. Together the results support our hypothesis and suggest that the relationship between sleep quality and paranoia is partially mediated by levels of negative affect, alexithymia and perceptual anomalies. As a precaution, we re-ran analysis including gender as a co-variate. This did not alter the results and the effects remained the same size.

4. Study 2

In study 2 we aimed to replicate the mediation model in a second, independently collected data set. We hypothesised that the relationship between sleep quality and paranoia would be partially mediated by levels of negative affect, perceptual anomalies and alexithymia.

4.1. Participants procedure and measures

The recruitment procedures, questionnaires and data analytical techniques used were identical to study 1. A separate Qualtrics survey

Table 5
Sample Demographics for study 2 (N = 402).

	Mean	SD	Median	Range
Age	24.75	10.64	20.00	17–77
PSQI	6.24	3.01	6.00	0–16
PA	25.43	7.80	25.00	10–49
NA	15.09	5.87	13.00	10–42
HADS-A	7.96	3.83	8.00	0–20
HADS-D	4.19	3.04	4.00	0–17
TAS-20	48.20	11.70	48.00	22–81
GPTS	23.29	11.70	18.00	16–80
CAPS	3.56	4.50	2.00	0–24

Abbreviations: PSQI- Pittsburgh Sleep Quality Index, PA and NA- Positive and Negative Affect, HADS-A and D Hospital Anxiety and Depression Scale respectively, TAS-20 – 20-item Toronto Alexithymia Scale, GPTS- paranoia subscale of the Green Paranoid Thoughts Scale, CAPS- Cardiff Anomalous Perceptions Scale.

was available online 2013–2015. Cronbach alphas were conducted for the following questionnaires: TAS-20 (α 0.85), GPTS (α 0.95), PANAS (α 0.84), CAPS (α 0.86), HADS D (α 0.74) and HADS A (α 0.80).

5. Results

5.1. Descriptive statistics and correlations

Table 5 presents the samples demographics. A total of 402 ($n = 114$ males, 288 females) individuals completed the survey with a mean age of 24 ($SD = 10.8$, range 16–77). Overall, the sample from study 2 was similar to the sample in study 1. This was confirmed by a series of Mann-Whitney U tests, which revealed no significant differences between the samples in study 1 and 2 on anxiety, depression, positive affect, negative affect, sleep quality, alexithymia or perceptual anomalies ($p > 0.05$). However, sample 2 ($Mdn = 18$) reported slightly higher levels of paranoia compared to sample 1 ($Mdn = 17$), $U = 62283$, $p = < 0.001$ and sample 2 were slightly younger ($Mdn = 20$) than sample 1 ($Mdn = 22$), $U = 70386$, $p = 0.002$. The correlations between all the variables are shown in Table 6. Age was correlated with several our predictors including sleep quality, alexithymia and perceptual anomalies; therefore, we included it as a co-variate in further analysis. A series of Mann-Whitney U tests revealed that gender was not associated with any of the variables in this study, all $p > 0.05$.

5.2. Mediation analysis replication

Paranoid group was entered as the dependent variable, sleep quality as the predictor variable, age as a co-variate and alexithymia, negative mood and perceptual anomalies were entered as mediators. This model was significant as revealed by the bootstrapped confidence intervals all

Table 6
Spearman rho correlations for study 2.

Measure	1	2	3	4	5	6	7	8
Age								
PA	0.17*							
NA	-0.13*	-0.09						
HADS- A	-0.10*	-0.17*	0.48*					
HADS-D	0.00	-0.33*	0.29*	0.47*				
PSQI	-0.07	-0.16*	0.24*	0.38*	0.33*			
TAS	-0.17*	-0.22*	0.25*	0.45*	0.51*	0.29*		
GPTS	-0.17*	0.04	0.31*	0.39*	0.26*	0.17*	0.41*	
CAPS	-0.19*	-0.02	0.18*	0.29*	0.27*	0.13*	-0.26*	0.45*

Abbreviations: PSQI- Pittsburgh Sleep Quality Index, PA and NA- Positive and Negative Affect, HADS-A and D Hospital Anxiety and Depression Scale respectively, TAS-20 – 20-item Toronto Alexithymia Scale, GPTS- paranoia subscale of the Green Paranoid Thoughts Scale, CAPS- Cardiff Anomalous Perceptions Scale.

* $p < 0.05$.

being above 0. The indirect (mediation) pathway to paranoia via negative affect was significant $b = 0.06$, 95% CI [0.012, 0.1450], the indirect (mediation) pathway from sleep quality to paranoia via alexithymia was significant $b = 0.08$, 95% CI [0.012, 0.200], the indirect (mediation) pathway from sleep quality to paranoia via perceptual anomalies was significant, $b = 0.08$, 95% CI [0.012, 0.200]. However, the direct pathway from sleep quality to paranoia was non-significant, $b = 0.10$, 95% CI [-0.1630, 0.3813]. In sum, the relationship between sleep quality and paranoia was fully mediated by levels of negative affect, alexithymia and perceptual anomalies. This contrasts with study 1 which found partial mediation.

5.3. Post Hoc- Analysis

As the mediation models were different in study 1 and 2, we sought to explore the data in study 2 further by running post-hoc regression analyses to see whether we could replicate the regression results found in study 1.

The predictors entered were age, negative affect, anxiety, depression, perceptual anomalies, sleep quality and alexithymia. A test of the full model against a constant only model (no predictors) was statistically significant, indicating that the predictors reliably distinguished between the paranoid and non-paranoid groups ($\chi^2 = 94.331$, $p < 0.0001$). The Wald test was used to determine what predictors are significant. Significant predictors of paranoid group membership included anxiety, alexithymia and perceptual anomalies. Sleep quality, age, negative affect and depression did not significantly predict paranoid group membership. The log odds (Exp B) is an indicator of the log odds of being in the paranoid group due to a one point increase in the predictor variable. For example, a one unit increase in anxiety increases the odds of being in the paranoid group by 1.09, 95%CI [1.00, 1.19]. Nagelkerke's R^2 of 0.314 indicated that 31% of the variance was explained by the significant predictors. Similar results were obtained in the stepwise logistic regression which also found that anxiety, alexithymia and perceptual anomalies predicted paranoid group membership and this model explained 30% of the variance (Nagelkerke's R^2 of 0.30). In sum, two separate regression models showed that anxiety, alexithymia and perceptual anomalies predicted membership of the paranoid group. Similar to the mediation results, we found sleep quality did not predict paranoia. However, we also found that anxiety predicted paranoia rather than negative affect, as in study 1.

6. Discussion

Previous research has found that sleep is related to paranoia and that this relationship is mostly mediated by negative emotions (Freeman et al., 2009, 2010; Reeve et al., 2017). Non-emotional factors such as perceptual anomalies have also been proposed as potential mediators (Freeman et al., 2010). We sought to add to this previous work by testing whether alexithymia is related to paranoia and whether it mediates the relationship between sleep quality and paranoia. We also sought to test the role of perceptual anomalies as a mediating factor.

6.1. Sleep quality and paranoia

Our hypothesised mediation model proposed that the relationship between sleep quality and paranoia is mediated by alexithymia, negative emotions and perceptual anomalies. While this mediation model was found in both studies, we found discrepant results such that there was partial mediation in study 1 and full mediation in study 2. The discrepancy can be ruled out due to sample differences between study 1 and 2 as they did not significantly differ in levels of sleep quality. Indeed, our post-hoc regression analysis further confirmed that sleep quality was not a significant predictor of paranoia. This is surprising as several studies have reported links between a range of sleep

disturbances and paranoia (Kahn-Greene et al., 2007; Sheaves et al., 2016; Reeve et al., 2017).

The discrepancy could be due to our chosen measure of sleep. Sleep quality is a broad term and the PSQI measures a wide range of sleep disturbances rather than focus on a specific disturbance (Buysse et al., 1989). Research suggests that the relationship between sleep and paranoia is strongest for insomnia. Insomnia has been found to predict paranoia in cross-sectional studies (Grezzelschak et al., 2016) but also predict the development of paranoid thoughts in a longitudinal study (Freeman et al., 2012). We propose that while sleep quality scores did not differ between study 1 and 2, the nature of the sleep disturbance may have. The participants in sample 1 may have experienced more insomnia in comparison to sample 2. This may have resulted in significant results in study 1 but not in study 2. Future research would benefit from focusing on specific sleep disturbances.

6.2. Alexithymia and paranoia

A novel finding in our study is the link between alexithymia and paranoia. Across both studies regression analyses showed that alexithymia predicted paranoia, independently of negative emotions, perceptual anomalies and sleep. Furthermore, we found some preliminary evidence that alexithymia may mediate the relationship between sleep quality and paranoia. This suggests that along with negative emotions, alexithymia is an additional emotional facet to consider when studying paranoia. Our emotions provide us with information on how we are feeling at a given moment. These emotions then help guide and direct us to make sense of the world, to make judgements about others behaviours, intentions and emotions (Boden and Berenbaum, 2010; Clore and Huntsinger, 2007). In alexithymia, this ability is diminished and this may make such individuals prone to faulty judgements and beliefs about others (including paranoid beliefs). This fits with a study reporting that low levels of emotional awareness (high alexithymia) was linked to higher levels of suspiciousness (Boden and Berenbaum, 2007). Another line of evidence for our claim comes from research looking at alexithymia and social dysfunction. One study found that individuals with difficulties identifying and describing feelings had significantly lower levels of social contacts, fewer acquaintances, and were more often unmarried (Kauhanen et al., 1993).

The relationship between alexithymia and paranoia may have clinical implications. Individuals with psychosis who also experience alexithymia may be at risk of experiencing greater paranoia but struggle to convey their concerns to others. They may also experience lower levels of trust towards their clinicians. As such, their responses to treatment may be poorer (Gumley, 2011). Assessing alexithymia may be useful in clinical practice to identify individuals who may require support to understand and regulate emotions. This could be achieved in clinical practice by including an assessment of alexithymia by using questionnaires such as the Toronto Alexithymia Scale. When individuals with alexithymia are identified, a focus on their emotional experiences could be incorporated into their CBT approach to sleep.

6.3. Negative emotions and paranoia

Across both studies we found that negative emotions predicted paranoia and mediated the relationship between sleep quality and paranoia. Negative emotions have been linked to paranoia in both cross-sectional and longitudinal studies (Freeman et al., 2010, 2012). In particular, anxiety has a strong association with paranoia as they share many features such as the anticipation of threat and a worry reasoning style (Freeman et al. 2008)

However, we found discrepant results in regards to what negative emotion was significant, with study 1 finding that negative affect predicted paranoia whilst in study 2 it was anxiety. Depression did not predict paranoia in either study. One of the reasons for this may be because the samples in our studies had low levels of depression overall.

The HADS provides scoring criteria, with scores of 0–7 being normal, 8–10, being borderline and more than 11 being clinically significant levels (Zigmond and Snaith, 1983). In both samples, the mean depression scores in the low and high paranoid groups fell in the normal range. This may be why depression was not a significant predictor in our regression analyses. In contrast, our measure of negative affect was measured with the PANAS, which was developed to measure a range of affective states (Watson, 1988). The negative affect component covers a range of states such as jittery, guilt and ashamed and is a measure of overall distress rather than a specific type of negative emotion (Watson, 1988). These differences between measures may have contributed to the discrepant results. It is also worth noting that the timeframe of the questionnaires differed and may be another reason behind the different results. The HADS timeframe is the past two weeks, whereas the PANAS was measured at the “current” level. The current timeframe captures momentary states which would experience more fluctuation than fortnightly levels.

6.4. Perceptual anomalies and paranoia

A consistent finding across both studies was that perceptual anomalies predicted paranoia and mediated the relationship between sleep quality and paranoia. It has been long proposed that delusions serve as explanations for odd or strange internal experience (Maher, 1974). Furthermore, perceptual anomalies are incorporated within a cognitive model of paranoid delusions, where paranoid beliefs arise from an attempt to explain anomalous and odd internal experiences (Freeman et al., 2002). Indeed, research has shown that perceptual anomalies increase the risk for the development of delusional ideas (Krabbendam et al., 2004) and anomalies of experience caused by illegal drug use have also been linked to delusional ideation (D'Souza et al., 2004). Our mediation model suggests that one route that people with sleep problems may experience paranoia is via perceptual anomalies.

6.5. Summary of findings

Taking all the mediation and regression results together, we can conclude that alexithymia, perceptual anomalies and negative emotions predict paranoid thoughts. The role of sleep quality is not clear, and our mediation model requires replication. In regards to effect size, our results were significant but small. For example, our regression models explained between 22–31% of the variance and many of the mediation effects could be considered small as they lie between 0.10 and 0.20 (Cohen, 1992). Although small, we feel they are clinically significant. A recent trial has found that improving sleep disturbance with CBT-I in a student sample was associated with medium to large, sustained improvements in paranoia (Freeman et al., 2017). Furthermore, another study has found that the relationship between sleep loss (as found in insomnia) is associated with moderate to large effect size increases in paranoia and negative emotions. This study also found that negative affect accounted for 90% of the increase in paranoia after sleep loss (Reeve et al., 2017). This highlights the usefulness of studying sleep and paranoia in non-clinical samples, and that effects can be detected in non-clinical samples. This is complemented by work in clinical samples where CBT-I is associated with large effect size reductions in sleep disturbance (Freeman et al., 2015) and in paranoia, perceptual anomalies and mood variables (Myers et al., 2011). One potential reason for our small effect sizes in comparison to these papers is that they focused on insomnia, whereas we looked at sleep quality. It may also be the case that the effects we found in our mediation analysis would be larger and more exaggerated in a clinical sample who experience more severe sleep and mood disturbance.

6.6. Limitations

Several limitations of our study should also be noted when considering the results. Our study was cross-sectional and it is highly likely that the mediation model pathways proposed are interacting and bi-directional over time. Furthermore, the timeframe of our questionnaires also differed. The PANAS was measured at the moment level, whilst sleep quality and paranoia at the monthly level. Therefore, hypothesised causal links between variables should be interpreted with caution. To tease out causal effects experimental and randomised control trials are called for. Another way to look at causal effects could be to conduct experience sampling studies (Myin-Germeys et al., 2009). This approach allows temporal mapping of relationships and questions of directionality can be addressed. A recent study using this methodology found that the relationship between a range of sleep parameters and paranoia was mediated by negative affect (Mulligan et al., 2016).

The sample population was predominantly university students that may not be representative of the general population. However, students are one population that may experience higher levels of psychotic like experiences such as paranoia (Lincoln and Keller, 2008) suggesting that this is a useful population to study psychotic experiences in. The gender ratio of the sample was skewed with the majority of the sample being females. This is a finding that has been noted in other online survey studies of paranoia (Freeman et al., 2005). However, as there is some research to suggest that males may experience more alexithymia (Levant et al., 2009), future studies should aim for more gender-balanced studies. All our variables were skewed which could have reduced power and the ability to detect certain relationships (Wilcox and Keselman, 2003). However, we dealt with the skewed data in a number of ways including bootstrapping making our results more reliable and robust. Another limitation is that all our measures were self-report and required the individual to accurately report their responses. The questionnaire assessment of paranoia has the additional limitation that it may capture paranoid thoughts that are justified suspicions to real threats. Nonetheless, laboratory based virtual reality experiments where unfounded paranoia can be tested have reached the same conclusions as questionnaire studies of paranoid thinking in non-clinical populations (Freeman et al., 2003).

We used the same recruitment method in sample 1 and 2 and have no way to know whether some participants completed the survey in both study 1 and 2. However, given that there is no incentive to complete the survey and that the survey is long and extensive, we feel it is unlikely that someone would complete the survey twice.

Finally, a concern may be whether the findings in non-clinical samples are generalizable to a clinical sample. However, it is now widely accepted that psychotic symptoms lie on a continuum with normal experience, that non-clinical and clinical symptoms share the same risk factors and the presence of non-clinical experiences increase the risk of clinical disorder (van Os et al., 2009; Johns et al., 2004).

6.7. Conclusions

In conclusion, we found inconclusive evidence for a link between sleep quality and paranoia. The current research also emphasises that alexithymia and paranoia are related. Alexithymia can easily be assessed and should be considered in studies of paranoid thinking and emotion.

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