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Research Article

Sleep Abnormalities in Older Adults with and without Major Depression: Evidence from Polysomnography, Actigraphy and Questionnaires

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Abstract

The link between sleep and depression in older adults is complex and not fully understood. Reports of sleep problems in individuals with a history of depression, but who are no longer symptomatic, are inconsistent. This study aimed to characterize the sleep of 43 older adults (50+ years) with and without a history of clinical depression using questionnaires, actigraphy, and polysomnography. Participants were divided into three groups: current Major Depressive Disorder (MDD; 'Current', n = 10), a history of clinical depression ('Past', n = 14), and no history of depression ('Never', n = 19). Morning salivary cortisol measures confirmed elevated waking cortisol levels in current depression. Examination of sleep measures revealed significantly less Slow Wave Sleep (%SWS) and greater self-reported sleep problems in the current depression group compared to the remaining groups. A past history of depression was not linked to poor sleep relative to those with no history of depression. Across the whole sample, more severe depression was associated with more %REM, less %NREM and %SWS, poorer self-reported sleep, but less time spent Awake After Sleep Onset (WASO). Increased cortisol was linked to more %REM and less %NREM sleep, and shorter actigraphic total sleep time and poorer actigraphic sleep efficiency. Overall, sleep abnormalities were a feature of current MDD, and increased across the sample in a dose-dependent fashion with increasing depression severity and cortisol levels. Results suggest that current depressive symptoms, and cortisol levels, are more important determinants of sleep problems than a past history of depression.

Introduction

Sleep problems occur in up to 50–60% of adults over the age of 65, and included difficulties with sleep onset and duration, and changes in sleep architecture such as reduced slow wave sleep (SWS; or StageN3) and increased Rapid Eye Movement (REM) sleep [1-3]. The presence of depression in this population can exacerbate sleep problems [4]. Indeed, sleep problems are common complaints in patients with depression [4,5] and also cause problems for their carers [6]. However, the association between sleep and depression in older adults is not well understood. A better understanding of the link between sleep and depression in older adults has important clinical implications, as treating sleep problems has the potential to improve depressive symptoms [4].

Evidence for the link between depression and sleep impairment in older adults derives from studies using arrange of different methodologies. For example, questionnaire studies indicate that greater depressive symptom severity is linked to poorer subjective sleep quality in older men and women [7,8]. Furthermore, Naismith et al., [9] found that older adults with a lifetime history of depression (current and past diagnoses) had poorer self-reported sleep in compared to healthy controls. Research using overnight polysomnography (PSG) in older adults is sparse, but tends to support findings from studies in middle-aged adults that show sleep architecture abnormalities such as decreased SWS, reduced REM sleep latency, and increased REM sleep [10-14]. Older adults with clinical depression largely show similar sleep architecture changes (e.g., reductions in REM latency and SWS, and increased REM) to those seen in adults, but with additional age-related decline [15]. For example, Gillin et al., [16] showed that age-related sleep changes are more pronounced in depression and occur earlier than in healthy controls. Similarly, findings from Knowles and MacLean [15] support the idea of 'accelerated ageing' in depression, reporting that the difference between the sleep of depressed and healthy people increases with age.

While PSG findings of sleep abnormalities in older depressed adults are largely convergent, studies using actigraphy recordings are fewer and findings are mixed. Actigraphy has been

recommended by the American Academy of Sleep Medicine to provide accurate assessment of sleep-wake patterns [17].

Actigraphy output is recorded with an 'actiwatch', which allows 24-hour recording while the individual goes about their normal routine. Using actigraphy, Naismith et al., [9] reported that the presence of depression in middle-toold-aged adults (46–86 years) was linked to a greater duration of night-time awakening sand reduced sleep efficiency, compared to age-matched healthycontrols. Incontrast, negative findings were reported by Lieverse [18], who found poorer self-reported sleep quality, as assessed with questionnaires, in older adults (60+years; n=93) with Major Depressive Disorder (MDD), but no differences on any actigraphic sleep variables relative to a group of healthy aged-matched controls (n=74). The reason for the discrepancies between these two studies is not clear, although it is likely that differences in age and clinical characteristics played a role.

Overall, sleep problems in older adults appear to be more pronounced in the presence of depression, but evidence derives primarily from studies using questionnaires and PSG, with the few existing actigraphy studies yielding contradictory findings. A related domain of interest is in the presence of sleep problems in older adults who have had previous episodes of depression, but who no longer show clinical depressive symptoms. This group is of considerable interest, given the suggestion that persistent sleep abnormalities during remission from depression may act as vulnerability markers for experiencing further clinical episodes [12,19]. In middle-aged adults, evidence exists of SWS and REM changes that persist during remission from depression [11]. A recent review also suggests that shortened REM latency and increased REM sleep in adults have the potential to predict relapse and recurrence [12].

Polysomnography studies in older adults are less common than in middle-aged adults. Lee et al., [20] tested older adults (60+years) with depression (N=15) before and after remission, and found persistent REM sleep changes (shortened REM latency) during remission, suggesting that sleep impairment may not be confined to individuals with current depressive symptoms. Using actigraphy, another longitudinal study supported the idea of enduring sleep problems after successful treatment of depression. Ten older depressed outpatients (60+years) showed persistent sleep impairments on total sleep time, wake after sleep onset (WASO), sleep efficiency, and sleep latency, after 60days of antidepressant treatment [21]. While these studies point to enduring sleep problems in individuals with a past history of depression, they were limited by small sample sizes and the lack of a healthy control group.

An additional limitation of the above studies is the irrelevance on clinical inter views and self-reports for assessing the presence and severity of depression. Interviews and self- reports depend on subjective experiences and the participant's willingness and motivation to report symptoms. Increasingly, studies are including measures such as morning salivary cortisol as a means of detecting biological abnormalities in depression. While cortisol cannot be considered a 'pure' measure of depression as it is linked to other factors such as alcohol consumption [22], research shows that depression is linked to hyperactivity of the Hypothalamic Pituitary Adrenalaxis (HPA), which releases the stress hormone, cortisol [23]. Cortisol, therefore, has been suggested to play an important role in the pathophysiology of depression [24]. Since cortisol levels remain elevated in adults [25,26] and older adults [27] who have recovered from depression, it offers advantages over a clinical diagnosis as it assesses HPA axis activity as a continuous variable. In this way, elevated cortisol is thought to index a biological vulnerability for depression or, atleast, act as a marker of previous episodes [24,28]. Freely circulating cortisol levels also impact on other systems, including sleep architecture and circadian rhythms, as well as human cognition [29-32]. In this study, the effects of cortisol secretion (as a biological index of depression levels) on sleep were examined.

For the first time, this study used multiple methods of sleep (questionnaires, actigraphy and home-PSG) to investigate the sleep of 43 older adults (50+years) with and without a history of MDD. We aimed to:

(i) Provide a comprehensive evaluation of the sleep of older adults with a clinical diagnosis of depression; and

(ii) Compare the sleep characteristics of individuals with current versus past depression with those of a healthy age-matched control group in order to determine whether sleep problems are confined to those with current depression (i.e., state-related), or whether they also occur in those with a past history of depression (i.e., trait-related). Strength of the current study is the use of multiple methods of sleep assessment, which aids in providing a comprehensive profile of sleep in older adults.

Finally, since age [1], gender [33], duration of depression [34], Sleep-Disordered Breathing (SDB) and associated hypoxemia [35] have all been shown to impact on sleep and depression, we examined for the contribution of these variables in the analyses.

Method

Participants

Recruitment of the non-depressed individuals was conducted via advertisements in community centers and mail outs to volunteer groups. Participants with a diagnosis of depression (past/present) were either outpatients from the North Metropolitan Adult Mental Health Older Adults outpatient services at Osborne Park Hospital, Perth, Western Australia, or individuals recruited from the community. Ethics committee approval was granted by both the North Metropolitan Mental Health Service and the University of Western Australia Human Research Ethics Committees.

Inclusion criteria for the depressed groups included a clinical diagnosis of unipolar MDD provided by a mental health professional, and age50–80 years. Exclusion criteria for all participants included: chronic infectious illness; neurological or neurodegenerative conditions; a history of moderate or severe traumatic brain injury; previous loss of consciousness >30 minutes duration; treatment for substance abuse; or any others severe psychiatric disorder. Forty-six participants were recruited. After excluding three participants due to a history of traumatic brain injury and bipolar depression, data were analyzed for 43 participants (12 male, 31 female) aged 50–78 years.

Procedure

Assessments were conducted in the participant's home over two visits. The first visit included: consent forms, questionnaires, clinical and sleep interviews, and the first night of PSG. Participants were also

given an actiwatch for seven days, as well as a sleep diary. The second overnight sleep study was one week later, in order to reduce the 'first night effect' due to unfamiliarity with PSG [36]. Saliva samples to assess cortisol levels were collected by the participant at the second visit, which was a weekday.

Materials

Clinical assessment and classification

A demographics questionnaire included questions about age, gender, occupation, and mental health. The Patient Health Questionnaire (PHQ-9) [37]; is a 9-item questionnaire, assessing levels of depression in the past 2 week sonascale of 0 (not at all), to 3 (nearly every day). ThePHQ-9 has excellent reliability (α=.94 in the current study). The PHQ-9 has diagnostic value as items map onto the DSM-IV criteria for diagnosis of depression. Scores range from 0–24, with higher scores indicating more severe depression. A score of ≥10 (for moderate depression) is recommended to identify clinical levels of depression [38].

The Mini International Neuropsychiatric Interview [39] is a short, diagnostic interview for psychiatric evaluation. This was used to exclude co- morbid psychiatric disorders and to confirm diagnosis of current depression.

Clinical interviews and questionnaire results were used to class participants into groups:

1. Those with a diagnosis of depression by a mental health professional confirmed by the MINI, and a PHQ-9 score of >10 were categorized as currently depressed ('Current');
2. Participants who had a diagnosis of clinical depression by a mental health professional in the past,but who were a symptomatic on interview and in the questionnaire were categorized as 'Past';
3. Those who had never seen a doctor for mental health issues and had a PHQ-9 score of <10 were categorized as 'Never' (Table1).

Salivary cortisol assessment

Three samples were taken immediately after waking, wake +15minutes, and wake +30minutes. Participants were asked to collect their saliva in small vial sand to store the min the freeze run till collection. For each sample, salivary cortisol was assessed in duplicates using a commercial enzyme immunoassay (Salimetrics, LCC). All samples from the same participant were assessed in the same batch. The inter-assay coefficient to variation was 1.63% and 9.61% for high and low quality control standards respectively, indicating consistency between runs. The Area Under the Curve with respect to Ground (AUCG) was calculated from the three samples as an estimate of total cortisol secretion over the first half an hour after awakening (y=0; cortisol levels at time 0). This method is often used in cortisol research where there are repeated measurements overtime [40,41].

Sleep assessment

Sleep electrophysiology: Home-polysomnography (PSG) was conducted using the Compumedics 'Somté' device. It includes Electroencephalogram (EEG), Electro- oculogram (EOG), Electromyogram (EMG), airflow, body position, thoracic and respiratory belts, and blood oxygen saturation. Data were analyzed by a Senior Sleep Technologist (J Maul) and checked over by another Sleep Technologist (A Mellor) according to the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events [42]. Data from the second sleep study are reported. Outcome variables included REM latency (minutes), and the percentage of REM and NREM sleep, and SWS (Stage N3). In order to assess for Sleep-Disordered Breathing (SDB) and associated hypoxia, analyses included the Respiratory Disturbance Index (RDI), and the Oxygen Desaturation Index (ODI-3; no.of times per hour that here is a decrease of >3% in blood oxygen saturation).

Actigraphy: Participants wore an 'actiwatch' a wrist-worn device that non-invasively monitors sleep-wake cycles, for the duration of two weeks to ensure seven nights of viable data were collected. Output measures were mean total sleep time (minutes), sleep efficiency (%), sleep latency (minutes), and minutes spent awake after sleep onset (WASO). Participants kept a sleep diary to enable interpretation of actiwatch data. Actigraphic data were checked against the sleep diary by two researchers to ensure concordance (AM, FW). The Actiwatch Spectrum (Mini Mitter Philips) was used in this study. Seven days of actigraphy recording has been shown to be sufficient to obtain stable measures of domains of sleep quality [43,44] .

Participants also completed a daily sleep diary to record details such as sleep and wake times, whether they took any naps, and whether the watch was removed for any period of time. This information was used to cross-validate and edit the actigraphy data as needed. Each actigram was visually inspected and compared to the sleep diary in order to identify any major discrepancies. Time in bed was adjusted by the scorer in cases where there was a discrepancy of greater than one hour between the diary and actigraph, so it was consistent with the sleep diary.

The Pittsburgh Sleep Quality Index (PSQI) assesses sleep quality in the previous month. Higher scores indicate poorer sleep. Global scores >5 indicate significant sleep problems. In this study, Cronbach's alpha was .87 for the global score. The three factor model by Cole et al., [45] was applied generating three scores: Sleep Efficiency (comprising PSQI components: sleep efficiency and sleep duration), Perceived Sleep Quality (comprising PSQI components: use of sleep medication, sleep latency, and subjective sleep quality), and Daily Disturbances (comprising PSQI components: day time dysfunction and sleep disturbance), calculated by multiplying the components cores comprising each factor by the factor loadings published by Cole et al., [45] and summing them.

Statistical analysis

Data were analyzed using SPSS Version 20 (IBM). Prior to analysis, data were 'Log10 transformed' where required [46]. Because the self-report data (PSQI) were missing completely at random (Little's MCARp >.05), Estimation maximization was used to replace missing values for self- report data.

In order to investigate differences on demographic and sleep variables between the depression groups, and given that predictions were made a priori, separate one-way Analyses of Variance (ANOVA) were conducted using one-tailed tests. Estimates of effect sizes included partial η2 for continuous data or Cramer's Phi for categorical data. Alpha throughout was .05. Post-hoc pair wise comparisons were used to investigate the nature of any between-

group effects. There was no adjustment for multiple comparisons, given that the relationships were predicted apriority [47]. Gabriel's method was selected for post-hoc comparisons based on the unequal sample size of the groups [46]. Pearson's correlations were conducted to investigate the relationship of depression severity and cortisol and sleep measures. One-tailed tests were used when the direction of the effect was predicted in advance. Variables that were related to the dependent variable (as determined by correlation sort tests) were added as covariates in subsequent Analyses of Covariance (ANCOVA) and partial correlation analyses [48].

While the use of sleep medications and antidepressants was of interest, these variables were not included as covariates in the final analyses to avoid the risk of excluding the critical variance of interest linked to the presence of depression. That is, taking antidepressant medication is somewhat synonymous with depression.

Results

Descriptive statistics for demographic and sleep variables for the three groups are reported in Table1. Apart from the use of antidepressant medication, where the Current group took the most, there were no between-group differences in age, gender, time since first diagnosis of depression, or measures of sleep-disordered breathing (RDI, and ODI-3).

Group differences in sleep variables

On PSG measures, the Current group had significantly less % SWS compared to the Never group (p=.022), whereas the Past group did not differ from the other groups (p>.050). The Past group had significantly longer REM latency than the Never group (p=.004), where as the Current group did not differ from either group (p>.050). These differences remained significant after controlling for age: F (2, 39), = 8.91, p<.001.

Onself-report measures, the Current group had greater PSQIG lobal and Cole's PSQI factor scores (Sleep Efficiency, Perceived Sleep Quality, Daily Disturbances), indicating poorer self-reported sleep quality, compared to the Past and Never groups, who did not differ (Table1). Poorer Sleep Efficiency was associated with age, and SDB (oxygen saturation desaturations below 3%; ODI-3), hence these variables were entered as covariates, but this did not affect the group difference for Cole's Sleep Efficiency, F (2,33),= 11.74, p<.001, partial η2=.42. Age and ODI-3 was not significant covariates of Perceived Sleep Quality or Daily Disturbances.

There were no significant group differences in any actigraphic sleep variables (Table 1).

Correlations with depression severity

In order to examine whether sleep problems increased as a

Table 1: Descriptive statistics for all study variables.

Variable	Never	*n*[1]	Past	*n*[1]	Current	*n*[1]	*p*	Pairwise comparisons	Partial η^2 or Cramer's Phi
			M(SD), range or N(%)						
Age in years	62.32(7.34), 50–79	19	59.07(4.60), 52–68	14	59.80(6.76), 51–74	10	0.32912		0.05
Gender: Males	5(26.3%)	19	2(14.3%)	14	4(40%)	10	0.36112		0.22
Currently employed	12(63.2%)	19	8(57.1)%	13	5(50%)	10	0.78912		0.11
Depression									
PHQ-9 scores[2]	2.21(2.15), 0–8	14	3.64(3.72), 0–9	11	16.60(4.62), 11–24	10	<.001	2>0,1, 1=0	0.78
Time since diagnosis (years)	N/A	0	11.69(12.68), 0–40	13	15.90(16.79), 0–52	10	0.50012		0.02
Taking antidepressants	2(10.5%)	19	7(50%)	14	8(80%)	10	<.001		0.57
Cortisol									
Sample 1 (Wake)	9.28(2.06), 2.07–35.16	16	8.67(1.48), 2.30–18.06	11	14.98(4.45), 3.75–42.69	8	0.157		0.07
Sample 2 (Wake+15)	10.62(1.66), 2.21–24.75	16	11.32(1.96), 1.78–25.06	11	20.72(6.36), 4.36–58.12	8	0.139		0.08
Sample 3 (Wake+30)	13.80(1.95), 2.64–31.41	16	13.82(2.15), 3.19–28.14	11	23.44(5.75), 7.50–48.72	8	0.122		0.09
AUC_G cortisol	332.39(199.28), 82.47–817.36	16	338.54(174.90), 67.77–684.21	11	598.98(469.20), 168.35–1557.42	8	0.109		0.09
ESS (daytime sleepiness)[5]	6.5(4.1), 2–18	19	9.5(4.2), 4–16	13	7.5(4.4), 0–14	10	0.073		0.09
PSQI									
Global score[10]	6.1(3.6), 1–15	19	6.9(3.7), 3–15	14	15.8(3.5), 10–21	10	<.001		0.57
Global > 5	8(42.1%)	19	9(64.3%)	14	10(100%)	10	0.004		0.48
Cole's Sleep Efficiency[10, 11]	1.5(1.5), 0–5	19	1.1(1.4), 0–5	14	3.8(1.5), 1.7–5	10	<.001		0.35
Cole's Perceived Sleep Quality[10, 11]	1.8(1.4), 0–5.1	19	2.2(1.4), 0.7–5.1	14	5(1), 3.8–6	10	<.001		0.52
Cole's Daily Disturbances[10, 11]	1.2(0.5), 0.5–2	19	1.5(0.7), 0.7–2.9	14	2.4(0.5), 1.9–3.6	10	<.001	2>0,1, 1=0	0.47
Use of Sleep Medication component	0.21(0.42), 0-1	19	0.46(0.88), 0-3	14	1.80(1.55), 0-3	10	<.001	2>0,1 1=0	0.34

Actigraphy									
Total sleep time	387.33(61.41), 217.21–458.50	19	431.07(49.74), 339.33–510.05	14	395.10(123.71), 144.19–553.60	10	0.133		0.06
Sleep Latency[6]	12.76(11.44), 1.40–48.14	19	15.07(12.50), 1.27–41.07	14	21.21(16.26), 4.54–60	10	0.13		0.07
Sleep Efficiency (%)[7]	82.25(10.86), 53.84–92.47	19	84.83(3.56), 80–91.45	14	77.12(19.80), 24.36–89.35	10	0.155		0.06
WASO[8]	53.60(26.81), 21.53–118.80	19	52.27(20.05), 11.15–95.13	14	44.74(14.84), 23.77–70	10	0.294		0.03
Polysomnography									
REM Latency[9]	79.13(38.45), 36.50–173.50	19	156.50(98.91), 68.5–412.50	14	114.55(101.34), 41.50–366	10	0.005	1>0, 0,1=2	0.21
%REM	23.31(8.06), 3.30–40.70	19	23.71(6.43), 13–33.50	14	27.71(9.73), 14.50–43.90	10	0.174		0.05
%NREM	76.81(8.09), 59.30–96.70	19	76.29(6.46), 66.50–87	14	72.30(9.74), 56–85.50	10	0.192		0.05

Note: [1] Sample size varies due to missing data; PHQ-9 = Patient Health Questionnaire; MINI = Mini International Neuropsychiatric Interview; AUC_G Area Under the Curve with reference to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen Desaturation Index below 3%; [2]Higher scores indicate increased depression severity; [3]Higher scores indicate increased Respiratory Disturbance; [4]Higher scores indicate greater oxygen desaturations; ESS = Epworth Sleepiness Scale; MEQ = Morningness Eveningness Questionnaire; [5]Higher scores indicate more sleepiness; [6]Higher scores indicate longer sleep latency; [7]Higher scores indicate better sleep efficiency; WASO = Wake After Sleep Onset (mins); REM = Rapid Eye Movement sleep; NREM = Non Rapid Eye Movement sleep; SWS = Slow Wave Sleep; [8]Higher scores indicate greater minutes spent awake after sleep onset; [9]Higher scores indicate longer REM latency; PSQI = Pittsburgh Sleep Quality Index; [10]Higher scores indicate poorer sleep quality; (2>0,1, 1=0) The Current group had higher scores than the Never and Past groups, who did not differ; (2>0, 1=0,2) The Current group had higher values than the Never group, the Past group did not differ from the remaining groups; (1<0, 0,1=2) The Past group had lower values than the Never group, the current group did not differ from the remaining groups; (0>2, 1=0,2) The Never group had higher values than the Current group, the Past group did not differ from the remaining groups; [11]Cole et al. [46]; [12] two-tailed. All tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.

Table 2: Correlation matrix for all study variables.

	Cortisol AUC_G	PSQI Global	PSQI Cole's Sleep Efficiency	PSQI Cole's Perceived Sleep Quality	PSQI Cole's Daily Disturbances	Acti TST	Acti Sleep Latency	Acti Sleep Efficiency	Acti WASO	PSG REM latency	PSG %REM	PSG %NREM	PSG %SWS
Cortisol AUC_G	-	0.27	0.25	0.22	0.18	-0.19	0.13	-0.26	0.1	-0.15	.33†	-.33†	-0.06
Age	-0.221	-0.23	-.29†	-0.2	-0.07	-.35†	0.17	-.28†	0.2	.30†	-.27†	.28†	-0.12
PSQI Use of Sleep Medication	.39‡	.72‡	.42‡	.68‡	.65‡	0.03	0.15	-0.01	-0.02	-0.16	.38‡	-.38‡	-0.19
PHQ-9	.22†	.88‡	.72‡	.87‡	.68‡	-0.02	0.27	-0.08	-.32†	-0.07	.46‡	-.46‡	-.34†
RDI	-0.021	-0.08	-0.2	-0.05	0.05	-.36†	0.18	-.33†	0.13	0.25	-0.14	0.14	-0.07
ODI-3	-0.201	-0.21	-.37†	-0.14	0.05	-.36†	0.14	-.32†	0.17	0.25	-.31†	.30†	-0.04
MEQ	-0.251	-0.021	0.091	-0.061	-0.061	0.091	-0.111	0.091	0.121	-0.221	-0.131	0.121	0.221
Time since diagnosis (years)	0.381	0.181	0.221	0.151	-0.121	-0.321	0.061	-0.161	0.221	-0.361	0.141	-0.131	0.411

Note: AUC_G= Area Under the curve with respect to ground; PHQ-9 = Patient Health Questionnaire; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen desaturation Index, < 3%; MEQ = Morningness-Eveningness Questionnaire; PSQI = Pittsburgh Sleep Quality Index; Acti = Actigraphy; TST = Total sleep time; WASO = Wake after sleep onset; PSG = Polysomnography; REM = Rapid eye movement sleep; NREM = Non Rapid eye movement sleep; SWS = Slow wave sleep; †$p<$ .05, ‡$p<$ .01; [1] two-tailed: all tests were one-tailed, based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.

function of depression severity regardless of group, correlations were conducted between PHQ-9 scores and sleep variables across the whole sample. This had the benefit of maximizing sample size and accounting for the possible impact of any subclinical depression. After controlling for age and ODI-3, more severe depression was associated with greater %REM (r=.42, p=.009) and lower %NREM (r=-.42, p=.010). In addition, more severe depression was correlated with lower % SWS (Table2). Depression severity was associated with less WASO (Table2), which was an unexpected result. Onself-report, results were consistent with the group comparisons and revealed that greater depression severity was associated with poorer sleep quality on PSQI global score, Cole's Sleep Efficiency, Cole's Perceived Sleep Quality and Cole's Daily Disturbances (Table 2). After controlling for ageandODI-3, the correlation for PHQ-9 and Cole's Sleep Efficiency remained significant, r (29) =.71, p<.001.

Analyses of cortisol

When the three subgroups were compared, there were no significant differences for morning cortisol measures (AUCG or means). However, further analyses revealed that when the Past and Never groups were collapsed, individuals with Current depression

showed significantly elevated cortisol readings: t (33) = -1.81, p=.040, partial η2=.09. Across the whole sample, cortisol AUCG levels increased in a dose- dependent manner with PHQ-9scores (Table 2).

Whole sample correlations also revealed a relationship between higher cortisol levels (AUCG) and greater % REM and less% NREM (Table2). These relationships remained significant after controlling for age and ODI-3. After controlling forage, RDI and ODI-3, a negative association of cortisol (AUCG) and actigraphy TST was revealed (r=-.35, p=.039); and a negative association of cortisol (AUCG) and actigraphy Sleep Efficiency was likewise revealed (r=-.39, p=.021). However, cortisol was not linked to self-reported sleep quality.

Discussion

This study aimed to characterize the sleep of older adults with current MDD compared to those with a past history, and no history of depression, using PSG, actigraphy recording, and questionnaires. In summary, results indicate that individuals with current symptoms of depression (Current group) had the lowest percentage of SWS sleep and poorer self-reported sleep quality. Depression symptom severity was also associated with greater sleep architecture abnormalities (more %REM, less %NREM and %SWS) and poorer self-reported sleep quality across the whole sample. Finally, increased cortisol levels were significantly associated with greater sleep abnormalities, including greater %REM, less %NREM, and shorter total sleep time and sleep efficiency. The results are discussed in turn.

Group comparisons

The group comparisons revealed that the currently depressed individuals exhibited the most sleep abnormalities, including lower% SWS and greater self-reported sleep problems. These findings are consistent with previous studies reporting reduced SW Sin middle-aged and older adults with depression [10], as well as increased subjective reports of poor sleep [18].

An examination of the profile of those individuals classed as having a past history of depression ('Past') revealed a puzzling finding– that of longer REM latency compared to the Never group. This contrasts with studies showing shorter REM latency in depression [20]. One possibility for this unexpected result is that a significant proportion of the Past group was taking antidepressants. In support, studies show that the use of antidepressants is linked to increased REM latency [49], and group comparisons in this sample using individuals taking antidepressants versus whose not taking antidepressants (results not presented) confirm this suggestion (p<.001). Altogether, the Past group had longer REM latency relative to the Never group, which was linked to the use of antidepressants. On the basis of these analyses, our findings show that sleep abnormalities were a particular feature of those with current MDD. Sleep stages and self-reported sleep quality were the most sensitive to depression status, and sleep-wake patterns were the least sensitive. Group comparisons made on the basis of diagnosis, however, do not take into consideration the continuous nature of depressive symptoms, so our next step was to examine the performance of patients based on severity of symptoms.

Symptom severity correlations across the whole sample

Correlation analyses of sleep with depressive symptom severity (PHQ-9) revealed the expected pattern of findings for sleep architecture. Specifically, depression has been linked to sleep architecture abnormalities concerning REM and SWS sleep [14]. In the current study, higher depression levels across the whole sample were linked to significantly more% REM, and less %NREM and% SWS, as well as greater subjective reports of poor sleep. These findings underscore the findings in middle-aged and older adults [7,8,14] and suggest a dose-dependent relationship, where by more severe depression is linked to greater sleep problems. One reason why %REM and %NREM did not show group differences, but showed an association with symptom severity might be related to our sample size. Group comparisons contained relatively small groups of participants– due to the demanding nature of PSG studies–whereas whole sample correlation analyses comprised a larger sample size and, therefore, had greater statistical power.

With regard to actigraphy, our correlational analyses revealed an association between greater depression severity and less WASO. This finding is counter intuitive as we would expect poorer sleep quality (i.e., greater WASO) in depression. We explored the possibility that WASO might be linked to demographic or clinical characteristics (e.g., antidepressant use), but our analyses did not yield any significant associations. Other actigraphy studies in older adults with depression reported greater WASO [9], so this contradictory finding remains unexplained.

Cortisol readings

Elevated morning cortisol was linked to more% REM, and less %NREM in the whole sample. These results are largely consistent with those demonstrated above using correlational analyses of symptom severity–both increased cortisol and depression severity were associated with more %REM and less %NREM. On actigraphy measures, cortisol was linked to shorter total sleep time and poorer sleep efficiency across the whole sample.

Overall, the current findings stand in contrast to studies that report enduring sleep problems and elevated cortisol during remission from depression. One reason for this discrepancy could be due to differences in the characteristics of the Past group. However, the most likely possibility is that definitions of 'remission' are not uniform across studies. For example, Lee et al., [20] tested participants after an average of 38 weeks since the last episode–where as our study was reliant simply on a 'previous' diagnosis without exclusion criteria regarding the date of previous diagnosis. The possibility exists, therefore, that our participants had been in remission for too long.

Although we collected information regarding the time of the first diagnosis of depression, the time since last depressive episode was not known. However, it is not worthy that a significant percentage of the Past group was taking antidepressants, which argues against the idea that the Past group was incomplete remission. Therefore, future research must clearly delineate the period of remission.

Another interesting observation was that depression status and severity (assessed with diagnoses, interviews, and self-reports) were largely only associated with sleep architecture indices and self-reports. By contrast, cortisol levels were associated with actigraphy monitoring and sleep staging. This suggests that different methods of assessing depression yield different profiles of associations with sleep assessment methods.

Limitations

Given that we do not know how much time had passed since the last depressive episode in the Past group, nor information about the use of different medications, we must be cautious in drawing firm conclusions about the Past group. We were also limited by power, as our sample was small. For example, anapriori sample size calculation revealed that an overall sample of 114 would be needed to detect a significant effect on key actigraphy sleep measures such as sleep latency (effect size=0.26 at .05 significance and .80 power, one-tailed), and an overall sample size of 130 would be needed to detect an effect of sleep efficiency (effect size= 0.25 at .05 significance and .80 power, one-tailed) [50]. However, because PSG is costly, onerous and time consuming, PSG studies are commonly limited by issues of power, and our sample size is not greatly different from other studies in older adults. Despite being limited by power, it is worth noting the substantial magnitudes of the effect sizes (partial η2) (Tables 1 and 2), which range from medium to large [51].

In summary, results indicate that current MDD was associated with arrange of sleep abnormalities. This finding was confirmed across three different metrics of depression: group comparisons based on depression diagnosis; depression symptom severity; and cortisol measures. Our examination of different methods of sleep assessment is a notable strength of this study. This enabled a comprehensive evaluation of sleep, recognizing it as a multifaceted process, and yielded valuable information about assessing sleep abnormalities in healthy and depressed populations. Results revealed that the association of depression and sleep differed depending on the method of sleep assessment. Firstly, depression was not reliably associated with sleep-wake pattern disturbance as assessed with actigraphy. Secondly, self-reported sleep measures, while associated with current depression, did not detect evidence of sleep disturbance in individuals with a past history of depression, nor were they correlated with depression severity across the whole sample. Finally, sleep architecture abnormalities were associated with current depression and depressive symptoms across the entire sample. Due to limitations of the current study (as discussed above), we must avoid making firm conclusions regarding the influence of a past history of depression on sleep. However, in view of these findings, clinicians may wish to consider screening for sleep architecture abnormalities (using PSG) as potential vulnerability markers of depression. This is of clinical importance given that persistent sleep abnormalities during remission from depression could represent vulnerability to experiencing further clinical episodes. Identification of sleep problems during remission allows for the possibility of early intervention. Infact, research has shown that treating sleep problems can reduce the risk of future clinical depression [4]. For example, therapies aimed at treating SDB, such as Continuous Positive Airways Pressure (CPAP) have been shown to improve depressive symptoms [52], and Cognitive Behavioral Therapy for Insomnia (CBTI) has been associated with a higher rate of remission from depression [53].

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