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**Patterns of Sleep Disturbances and Associations with Depressive Symptoms in Autistic
Young Adults**

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Lay Summary

Studies in the general population show that sleep disturbances may have a negative effect on mood. However, this relationship has not been sufficiently studied in autistic adults. More than 80% of autistic young adults in the study sample had sleep disturbances. Adults with certain sleep disturbances reported more depressive symptoms. Understanding the associations between sleep disturbances and depression in autistic adults may offer new avenues for mental health treatments in this population.

Abstract

Autistic individuals are at an increased risk for both sleep disturbances and depression. While studies in the general population and in autistic adults have drawn general links between sleep disturbances and mental health, few studies have examined the extent to which specific sleep problems may be implicated in the extremely high rates of depression among autistic adults. This study aimed to describe the patterns of sleep disturbances in autistic young adults, and their associations with depressive symptoms while controlling for relevant demographic factors. A sample of 304 legally independent adults (age 18 to 35 years old) with a childhood diagnosis of autism spectrum disorder self-reported on their average sleep behaviors during the past week and depressive symptoms on the Beck Depressive Inventory-II. A significant proportion (86.01%) of autistic young adults experienced at least one of the primary sleep disturbances of interest, including short total sleep time (39.59%), poor sleep efficiency (60.07%), and delayed sleep phase (36.18%). Additionally, lower sleep efficiency and delayed sleep phase were both associated with higher depressive symptoms. The associations between sleep and depressive symptoms identified in our study suggest that sleep treatments may hold potential for ameliorating depressive symptoms in autistic adults who also experience sleep problems. Further research using daily sleep diaries and objective measures of sleep behaviors, as well as longitudinal studies, are needed to understand how changes in sleep may relate to changes in depressive symptoms in autistic adults.

Keywords: Sleep, depression, young adults, sleep efficiency, delayed sleep phase.

Introduction

High rates of sleep disturbances have been consistently reported in autistic individuals across the lifespan. Studies of children and adolescents with autism spectrum disorder (ASD) have found evidence of prolonged sleep latency, night waking, delayed sleep phase, decreased total sleep time and sleep efficiency, and parasomnias (Couturier et al., 2005; Krakowiak et al., 2008; Liu et al., 2006; Malow et al., 2006; Mayes & Calhoun, 2009). While sleep has historically been understudied in autistic adults, more recent studies have reported that sleep problems persist into adulthood in this population. One of the most consistently reported sleep disturbances across studies is poor sleep efficiency, as well as related disturbances such as prolonged sleep latency and wake after sleep onset (Baker & Richdale, 2015; Ballester et al., 2019; Hare et al., 2006; Jovevska et al., 2020; Morgan et al., 2020; Øyane & Bjorvatn, 2005; Tani et al., 2003). Moreover, short total sleep time and high rates of insomnia have been reported (Baker & Richdale, 2015; Hohn et al., 2019; Tani et al., 2003). While less frequently studied, there is also evidence for disturbances in circadian rhythm, including indicators of advanced and delayed sleep-wake phase, in autistic adults (Baker & Richdale, 2017; Ballester et al., 2019; Hare et al., 2006).

In addition to high rates of sleep disturbances, autistic adults experience high rates of depression (Hudson et al., 2019; Joshi et al., 2013; Lai et al., 2019), with lifetime depression prevalence estimates between 37% and 77% (Hollocks et al., 2019; Joshi et al., 2013; Zheng et al., 2021). Beginning in childhood, depression symptoms are higher in autistic individuals compared to their typically developing peers (Gotham et al., 2015), and symptoms may increase in severity during adolescence and young adulthood (Gotham et al., 2015; Uljarević et al.,

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2020). Given the high rates of both sleep problems and depression in adults with ASD, improved understanding of the potential links between these constructs may offer new treatment avenues for the growing number of autistic adults experiencing depression.

In the general population, sleep disturbances are known to have a negative impact on mood and everyday functioning. For example, acute decreased total sleep time has been linked to decreases in positive affect and increases in anxiety (Talbot et al., 2010), as well as impairment of executive functioning (Nilsson et al., 2005). Complaints of poor sleep, poor sleep efficiency, and delayed sleep phase are associated with increased depressive symptoms (Bei, Ong, Rajaratnam, & Manber, 2015; Tsai, Lee, Gordon, Cayanan, & Lee, 2021; Walker, Walton, DeVries, & Nelson, 2020) and suicidal ideation and completion (J. R. Asarnow et al., 2020; Bernert & Joiner, 2007; Goldstein et al., 2008; Rumble et al., 2020; Sabo et al., 1991). There is evidence for a bidirectional link between sleep and depressive symptoms, as sleep disturbance is a common symptom of depression, and depressive episodes may induce or exacerbate sleep disturbances (Franzen & Buysse, 2008). As such, sleep disturbance is often assessed on depression measures. However, recent research suggests that sleep problems may predate depressive symptoms in many cases, and predict depression relapse (L. D. Asarnow & Mirchandaney, 2021; Franzen & Buysse, 2008; Jaussent et al., 2011).

Despite the well-established links between sleep and mood problems in the general population, only a handful of studies have directly investigated these associations in autism. In children and adolescents, general sleep disturbances, as well as number of insomnia symptoms, sleep efficiency, sleep onset latency, and wake time have been associated with affective problems and depression symptoms (Malow et al., 2006; Mayes & Calhoun, 2009; Richdale et al., 2014).

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In autistic adults, there is emerging evidence associating general sleep disturbances with depression (Puzino et al., 2021; Stewart et al., 2020) and psychiatric symptoms (Gustemps et al., 2021). Poor sleep quality and sleep efficiency have also been associated with general mental health and wellbeing (Henderson et al., 2021; Jovevska et al., 2020). Additionally, longitudinal studies have found that sleep disturbances and sleep quality are predictors of later quality of life in autistic adults (Deserno et al., 2019; Lawson et al., 2020). While these recent studies provide preliminary evidence for a link between sleep and mood in autistic adults, more work is needed to understand the extent to which specific types and patterns of sleep disturbances are associated with depressive symptoms in this group. Moreover, autistic adults and other stakeholders report that both depression and sleep are among the most important outcomes for future research to examine (Benevides et al., 2020).

Toward this goal, the current study utilized data from a larger study of autistic young adults recruited from the Simons Foundation Powering Autism Research for Knowledge (SPARK) research match registry (Zheng et al., 2021). Analyses focused on self-reported sleep behaviors and depressive symptoms to answer the following research questions: 1) What are the self-reported patterns of sleep behaviors and disturbances among autistic adults, and 2) what are the associations between specific sleep characteristics and depressive symptoms in autistic adults, after controlling for demographic and life circumstance factors associated with these constructs? Specifically, we selected total sleep time, sleep efficiency, and circadian phase as variables of interest for research question 2 because they have been shown to be associated with depression in the general population (Bei et al., 2015; Tsai et al., 2021; Walker et al., 2020; Zhai

et al., 2015), but have not been extensively explored as correlates of depressive symptoms in autistic adults.

Methods

Participants and Procedures

A sample of 315 autistic young adults were recruited through the SPARK research match registry as part of a larger online survey study on depression and depression service receipt (see Zheng et al 2021 for full sample characteristics). According to the inclusion criteria for the original study, all participants were 18-35 year-old, legally independent adults, who were capable of consenting and self-reporting, had received a childhood diagnosis of ASD, and had completed the background history questionnaire when they registered with SPARK. The Institutional Review Board at the authors' institutions, as well as the Community Advisory Council (CAC) of self-advocates and family members from the autism community organized by the SPARK research match registry, reviewed and approved all study procedures and documents. All study procedures conformed to the standards set by the Declaration of Helsinki.

Of the original sample of 315 adults, the current analysis only included participants who provided information on at least one of the three sleep variables of interest. Thus, we excluded seven individuals who did not complete any sleep questions. Additionally, we excluded four individuals who reported sleep variables outside of possible ranges from the analyses (details in the data cleaning section below). A flowchart of the participant selection for the current analyses can be found in supplementary materials (Figure S1). Demographic characteristics of the analytic sample (N=304) are shown in Table 1 (Table 1).

Measures

Dependent Variable

We measured self-rated depressive symptoms using the *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) where items are scored on a scale of 0 to 3 with higher scores indicating greater symptom-severity. The BDI-II has been shown to have good psychometric properties in autistic adults, comparable to what has been found in the general population (Williams et al., 2020). As previous sleep studies in typically developing populations have used BDI-II scores after excluding item 16 (*changes in sleeping patterns*; Manber et al., 2014; Rosenström et al., 2012) we conducted analyses using the full scale BDI-II total score, and then separately using the BDI-II score excluding item 16. In the current sample, the BDI-II showed excellent internal consistency (Cronbach's Alphas = 0.95 and .94, respectively, for the full scale and excluding the sleep item).

Independent Variables

The *Sleep Questionnaire* consisted of six items asking about the participants' sleep over the past week, adapted from the consensus sleep diary (Carney et al., 2012). The full sleep questionnaire is included in the supplementary materials (Table S1). We calculated standard sleep variables based on the sleep questionnaire (Buysse et al., 2006): *sleep latency* and *wake after sleep onset* (both in minutes) were drawn directly from the sleep questionnaire. We calculated *time in bed* (in minutes) as the time between bedtime and waketime. Then, we calculated *total sleep time* (in minutes) as *time in bed* minus all the time spent awake after getting into bed (*sleep latency*, *wake after sleep onset*, and time between bedtime and lights out time; Table S1). We calculated *sleep efficiency* by dividing *total sleep time* by *time in bed*, and

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multiplying by 100 (to obtain a percentage). Finally, using the sleep onset time criteria from the International Classification of Sleep Disorders, 3rd edition (ICSD-3; American Academy of Sleep Medicine, 2014), as used by Baker & Richdale, 2017 who had a similar sample of autistic adults with no co-occurring intellectual disability, we categorized each individual as having one of three potential circadian phases: delayed sleep phase (sleep onset time was between 1:00am and 6:00am), advanced sleep phase (sleep onset time was prior to 10:00pm), or neither (categorized as “intermediate”). Delayed sleep-wake phase disorder and advanced sleep-wake phase disorder are circadian rhythm sleep disorders, and are characterized by persistent misalignment between the individual’s sleep episode and their desired or necessary sleep-wake schedule (American Academy of Sleep Medicine, 2014). Clinical diagnosis of delayed or advanced sleep-wake phase disorder requires careful clinical judgement based on clinical interviews, sleep diaries, and whenever possible, actigraphy monitoring and biological measurements (American Academy of Sleep Medicine, 2014), Our classification of circadian phase is an indicator for delayed or advanced sleep-wake phase, but should not be equated to a clinical diagnosis of a sleep-wake phase disorder.

Covariates

We included *demographic variables* in the regression model as control variables: age, gender (collapsed to: male, female, and other), relationship status (collapsed to: single vs. in a relationship), children (yes/no), current school status (yes/no) and paid employment status (yes/no).

Data Cleaning

Details about individual cases reviewed for data cleaning can be found in the supplementary materials (Table S2). Three individuals provided sleep variable reports that indicated a time in bed greater than 24 hours, and one individual reported sleep variables that indicated a sleep efficiency of 0%, signaling clear data error (see detailed case description in the supplementary table S3). Out of the 304 individuals in the analytic sample, 11 were missing total sleep time and sleep efficiency data, one was missing gender data, and one was missing school status data.

Statistical Analysis

To address the first aim of the study, we generated descriptive statistics for the sleep characteristics variables of interest (total sleep time, sleep efficiency, and circadian phase). As sleep efficiency is a measure of how much time is spent awake in bed, it may encompass various sleep disturbances, including prolonged sleep latency and increased wake after sleep onset. Though not the primary focus of our study, sleep latency and wake after sleep onset were described and included in follow-up analyses to elucidate sleep efficiency findings. Descriptive statistics are reported in reference to guidelines established by the National Sleep Foundation (NSF; Hirshkowitz et al., 2015; Ohayon et al., 2017). We analyzed total sleep time in minutes for the descriptive analyses and regression models, and reported in hours in the results for interpretability. We examined relationships between the sleep variables by calculating the correlation between total sleep time and sleep efficiency, sleep latency, and wake after sleep onset, and testing group differences across the three circadian phase categories on total sleep time, sleep latency, and wake after sleep onset using one-way ANOVA, and on sleep efficiency

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using Kruskal-Wallis test given the non-normality of the variable. We calculated effect sizes using eta-squared, and conducted post hoc tests for any significant circadian phase group effects, with Tukey's HSD for total sleep time and sleep latency, and Dunn's test for sleep efficiency.

To address the second aim of the study, we performed multivariate linear regression with BDI-II sum scores as the dependent variable to examine the relationship between sleep variables and depressive symptoms after controlling for the demographic variables in one model. The model included all sleep variables (total sleep time, sleep efficiency, and circadian phase) and covariates that are related to sleep and depression. Specifically, we controlled for age, employment status, school status, gender, relationship status, and whether or not they have children (Dooley et al., 2000; Grandner et al., 2010; Hagen et al., 2013; Hansen et al., 2005; Husain et al., 2000; Inaba et al., 2005; Jovevska et al., 2020; Richdale et al., 2014). The circadian phase variable was contrast coded for the ease of model interpretation: intermediate=0, delayed=-1, advanced=1. As mentioned above, we conducted sensitivity analyses using the BDI-II total score excluding item 16 (*changes in sleeping patterns*). Given that several individuals reported extremely low sleep efficiency values, we also conducted sensitivity analyses with potential outliers for sleep efficiency removed. To determine the cutoff for potential outliers for sleep efficiency, we used the first quartile minus 1.5 x (interquartile range) (Tukey, 1977), which resulted in a cutoff of 40% (first quartile=70%, third quartile=90%). We calculated the Variance Inflation Factor (VIF) for the full model to check for multicollinearity, and the VIFs for all predictors were below 2, indicating no need for correction. Upon identification of significant effects of sleep efficiency, we conducted follow up analyses including sleep latency and wake

after sleep onset as the independent variables in the linear regression to further elucidate the effects of specific aspects of sleep efficiency. All analyses were done using R version 3.6.3.

Results

Sleep patterns and disturbances

Participants in this sample reported an average total sleep time of 7:18 hours (SD=2:07 hours, Range: 0:45-13:00 hours), and a mean sleep efficiency of 77.1% (SD=17.56%, Range: 8-100%). Out of the 293 individuals with all three sleep variables available, 116 (39.59%) had total sleep times below the NSF recommended range (7-9 hours per night; Hirshkowitz et al., 2015) 53 (18.09%) had total sleep times above the recommended range, and 176 (60.07%) had sleep efficiencies below the recommended threshold (85%; Ohayon et al., 2017). Out of the 304 individuals with circadian phase data available, 110 individuals (36.18%) reported sleep onset as occurring between 1:00am-6:00am, and therefore met criteria for delayed sleep phase, while 26 individuals (8.55%) reported sleep onset as occurring earlier than 10:00pm, and met criteria for advanced sleep phase. Average sleep latency was 46 minutes (SD=50 minutes, Range: 0 - 415 minutes), and 122 individuals (41.64%) had sleep latencies greater than the recommended range (0-30 minutes; Ohayon et al., 2017). Average wake after sleep onset was 37 minutes (SD=58 minutes, Range: 0 - 440 minutes), and 123 individuals (41.98%) had wake after sleep onset greater than the recommended range (0-20 minutes; Ohayon et al., 2017).

Out of the 293 individuals, 252 (86.01%) had at least one of the primary sleep disturbances of interest (i.e., total sleep time outside of the NSF recommended range, sleep efficiency below the NSF recommended cutoff, and/or delayed or advanced sleep phase). Less than 14% of the adults (n=41) had none of the three primary sleep disturbances, whereas 89

individuals (30.38% of 293) had one sleep disturbance, 104 individuals (35.49% of 293) had two sleep disturbances, and 59 individuals (20.14% of 293) had all three sleep disturbances. Of the three primary sleep disturbances, total sleep time outside of the recommended range most commonly co-occurred with poor sleep efficiency. For a full summary of co-occurring sleep disturbances, see Figure 1.

Relationships between sleep variables

There was a strong correlation between sleep efficiency and total sleep time ($r=0.61$). Specifically, there was a moderate correlation between wake after sleep onset and total sleep time (-0.39), and a small correlation between sleep latency and total sleep time (-0.21). There was a significant difference in total sleep time by circadian phase, such that those who had delayed sleep phase had shorter total sleep times than those with advanced or intermediate sleep phase (Table 2). Additionally, there was a significant difference in sleep efficiency by circadian phase, such that those who had delayed sleep phase had lower sleep efficiency than those with advanced or intermediate sleep phase (Table 2). Similarly, there was also a significant difference in sleep latency by circadian phase, such that those who had delayed sleep phase had longer sleep latencies than those with advanced or intermediate sleep phase (Table 2). Wake after sleep onset was not significantly different across the circadian phase groupings.

Depressive Symptoms and the Effect of Sleep Variables

Adults in the current sample reported a wide range of depressive symptoms, with a mean BDI-II score of 18.41 and a standard deviation of 13.85 (Table 1). Controlling for age, employment status, school status, gender, relationship status, and parenting status, sleep efficiency and delayed sleep phase were significantly associated with depressive symptoms, but

total sleep time and advanced sleep phase were not (Table 3). Specifically, lower sleep efficiency and the presence of delayed sleep phase were both associated with higher BDI-II scores.

Sensitivity analysis with the BDI-II score without the sleep change item (Table S4) and with potential outliers for sleep efficiency removed (Table S5) showed similar regression results. The follow up analysis with sleep latency and wake after sleep onset showed that both variables were significantly associated with depressive symptoms.

Discussion

Results of the current study add to a growing literature indicating high rates of sleep disturbances in adulthood for individuals on the autism spectrum, and provide further evidence for associations between specific sleep disturbances (i.e., sleep efficiency and delayed sleep phase) and depressive symptoms in autistic adults. More than a third of our sample slept less than 7 hours and almost 20% slept more than 9 hours per night, with an average sleep efficiency of 77.1%, similar to actigraphy estimates in other adult autism populations (e.g. 80.95%, Baker & Richdale, 2015; and 76%, Øyane & Bjorvatn 2005). Moreover, our sample reported an average sleep latency of 46 minutes and an average wake after sleep onset of 37 minutes, which are above the NSF recommendations (Ohayon et al., 2017). These values are within the range of previously reported values in autistic adults, although estimates vary with measurement method (Baker & Richdale, 2015; Jovevska et al., 2020; Øyane & Bjorvatn, 2005). Finally, while we did not conduct formal diagnosis of delayed or advanced sleep-wake phase disorder in this study, nearly half of our sample was classified as having either a delayed or an advanced sleep phase based on the ICSD-3 (American Academy of Sleep Medicine, 2014) sleep onset criteria used by Baker & Richdale, 2017, compared to less than 10% of adults in the general population meeting

criteria for advanced or delayed sleep phase (Curtis et al., 2019; Micic et al., 2016; Schrader et al., 1993).

Building on previous studies in autistic adults linking general sleep disturbances to depressive symptoms (Puzino et al., 2021; Stewart et al., 2020), the current study is the first to control for demographic and life factors known to influence both sleep and depression in investigating these associations. We found that lower sleep efficiency was associated with higher depressive symptoms on the BDI-II, furthering previous findings on these associations in both autistic adolescents and adults to show the effect of sleep efficiency above and beyond the impact of demographic and life circumstances (Henderson et al., 2021; Richdale et al., 2014). Moreover, the follow-up analysis helps tease apart the effect of sleep efficiency, showing that both sleep latency and wake after sleep onset contributed to the association between sleep efficiency and depressive symptoms. These findings reflect results from autistic adolescents (Richdale et al., 2014) and the general population (Lovato & Gradisar, 2014).

Adults in our study with delayed sleep phase reported significantly higher depressive symptoms compared to adults with intermediate sleep phase. While delayed and advanced sleep phase have not been studied in relation to depressive symptoms in autistic populations, this result is consistent with evidence from the general population linking circadian rhythm disturbances, particularly delayed sleep phase, to depression (Antypa et al., 2016; L. D. Asarnow et al., 2014; Nesbitt, 2018; Walker et al., 2020). Moreover, we also observed that adults with delayed sleep phase reported the lowest sleep efficiency and longest sleep latency, which is consistent with previous studies indicating that delayed sleep phase and insomnia commonly co-occur, and are both associated with worse depressive symptoms in the general population (Alvaro et al., 2014;

Chan et al., 2014; Li et al., 2018). In contrast, total sleep time was not significantly associated with depressive symptoms before ($r=-0.11$) or after controlling for other sleep and demographic variables. Previous studies in the general population report mixed findings on the association between total sleep time and depressive symptoms (L. D. Asarnow et al., 2014; L. D. Asarnow & Mirchandaney, 2021; Zhai et al., 2015). Taken together, these results suggest that the timing and efficiency of sleep may be more important than total sleep time for understanding depression, and possibly other important outcomes such as academic achievement, reactivity to stress, and physical illness (Chiang et al., 2017; Cohen et al., 2009; Eiman et al., 2019; Gruber et al., 2014).

Establishing a relationship between sleep disturbances and depressive symptoms in autistic adults offers a potential opportunity to manage depression in this population through interventions targeting sleep. Furthermore, understanding what specific aspects of sleep are more closely related to depressive symptoms in autistic adults is important for informing treatment goals. While longitudinal research is needed, results from this study suggest that sleep efficiency may be a promising target for intervention in adult autism populations, given the significant associations with depressive symptoms and delayed sleep phase shown in the current study. Since sleep efficiency represents a composite of multiple sleep disturbances, when poor sleep efficiency is found in an individual, in depth examination of what sleep disturbances are driving the poor sleep efficiency is important. More specifically, our results suggest that interventions targeting sleep latency and wake after sleep onset may be useful for depressed autistic individuals. Lovato & Gradisar proposed that prolonged time spent awake in bed without external stimuli may increase the frequency of automatic thoughts and rumination, which may exacerbate depressive symptoms (Lovato & Gradisar, 2014). Interventions such as cognitive

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behavioral therapy for insomnia (CBT-I), which can be used to target ruminative thoughts while attempting to sleep, may be useful for autistic adults with prolonged sleep latency and/or wake after sleep onset who are also suffering from depression. Although the use of CBT to treat sleep problems has not yet been extensively studied in autistic adults, it has been shown to be feasible and effective at reducing sleep disturbances, such as prolonged sleep onset and night waking, in autistic children (Cortesi et al., 2012; McCrae et al., 2021; van Deurs et al., 2021).

Our study did not conduct a full assessment for delayed sleep-wake phase *disorder*. However, we found that delay in sleep onset time was linked to depressive symptoms, as well as poorer sleep efficiency and increased sleep latency. Given these results, clinical assessment for delayed sleep-wake phase disorder, and further intervention, may be warranted in autistic adults presenting with a late bedtime during clinical visits. This may be another promising target for intervention in autistic adults, given the availability of effective depression treatments targeting circadian rhythms in the general population (e.g., bright light therapy, chronotherapy, and behavioral interventions; Harvey et al., 2018; Walker et al., 2020). Additionally, late bedtime in autistic adults may be in part due to the low rates of employment and participation in structured and unstructured activities during the day (Chen et al., 2015; Liptak et al., 2011; Orsmond et al., 2013). This may be particularly problematic in autistic adults as compared to children and adolescents, given that rates of participation in structured events decline after high school exit for autistic individuals (Taylor et al., 2017). Interventions aimed at increasing participation and creating more structured schedules may reduce the likelihood of delayed bedtime in autistic adults. As studies in typically developing populations have shown that sleep disturbances may lessen the benefits of depression treatments (L. D. Asarnow et al., 2019; L. D. Asarnow &

Mirchandaney, 2021), and sleep improvements coincide with improvements in mood (Carney et al., 2017; Manber et al., 2016), future research should examine treatment effects on depressive symptoms when targeting sleep efficiency (particularly sleep latency and wake after sleep onset) and delayed sleep phase in autistic adults.

The current study is one of the largest to date examining the relationship between specific sleep disturbances and depressive symptoms in autistic adults. However, our study is not without limitations. First, we used self-report data and a modified sleep questionnaire. While self-report data provides an accessible estimate of sleep variables, previous studies in the general population suggest that people may overestimate their total sleep time on questionnaires as compared to more objective measures, such as polysomnography (Matthews et al., 2018). Additionally, our sleep questionnaire asked participants to rate their average sleep for the past week as opposed to the gold-standard daily sleep diary. This method may miss daily variability in sleep within individuals, and limits our ability to examine factors such as social jetlag, which may manifest differently in autistic populations. Thus, future studies should include daily sleep diaries and objective measures of sleep. Additionally, we did not include time in bed after waking in the morning in our calculation of sleep efficiency, and thus we may be overestimating our sample's sleep efficiency, and underestimating the rates of poor sleep efficiency. Circadian phase classification in our study was based only upon ICSD-3 sleep onset time criteria (American Academy of Sleep Medicine, 2014) derived from the sleep questionnaire, rather than sleep diary monitoring, actigraphy, and gold standard biological measurements such as dim light melatonin onset (Lewy et al., 1999). Thus, the rates of reported delayed/advanced sleep phase are by no means representative of the actual prevalence of disordered sleep phase in autistic adults.

Data used in the current study were collected as part of a larger survey, so we lack detailed information on the demographic variables. Future research may want to further examine how individual demographic variables, particularly relationship status given that it was significantly associated with depressive symptoms in our study, may impact the relationship between sleep disturbances and depressive symptoms. Finally, due to the cross-sectional nature of the data, the relationships between sleep characteristics and depressive symptoms were associations, not causations. While some longitudinal and retrospective studies suggest that the relationship between sleep and depression may be bidirectional (Alvaro et al., 2013; L. D. Asarnow & Mirchandaney, 2021), longitudinal studies in autism are needed to better understand how changes in various aspects of sleep are related to changes in depressive symptoms (and vice versa). Further, although sleep problems may predate depressive symptoms in some cases (L. D. Asarnow & Mirchandaney, 2021; Franzen & Buysse, 2008; Jaussent et al., 2011), sleep disturbances are also a symptom of depression and may increase during depressive episodes. Longitudinal studies are required to determine if sleep disturbances should be utilized as an early intervention target to prevent depression, or if sleep disturbances after depression onset should be treated in conjunction with other symptoms to improve outcomes.

Conclusions

Our findings suggest that a large proportion of autistic adults experience sleep disturbances, and lower sleep efficiency and delayed sleep phase are associated with increased depressive symptoms, even after controlling for demographic characteristics not considered in previous studies of individuals with ASD. Given the relationship between sleep and depressive symptoms shown by our findings, and previous treatment evidence in typically developing

populations (Baglioni et al., 2011; Gee et al., 2019; Howland, 2011; Wehr, 1990), sleep treatments may hold potential for ameliorating depressive symptoms in autistic adults.

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SLEEP AND DEPRESSION IN AUTISTIC ADULTS

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Tables

Table 1.

Sample Demographics

| Variable | | N | % or Mean (Standard Deviation) | Range |
|---|--------------------------------|---------|--------------------------------|-------|
| Age (years) | [continuous] | 30 4 | 26.35 (4.69) | 18-35 |
| Gender | Male | 15 6 | 51.32% | - |
| | Female | 12 9 | 42.43% | - |
| | Other [†] | 18 | 5.92% | - |
| | Missing | 1 | 0.33% | - |
| Paid employment status | Not working | 14 6 | 48.03% | - |
| | Working | 15 8 | 51.97% | - |
| School status | Not in school | 21 4 | 70.39% | - |
| | In school | 89 | 29.28% | - |
| | Missing | 1 | 0.33% | - |
| In school and employed | - | 41 | 13.49% | - |
| Relationship status | Single | 19 1 | 62.83% | - |
| | In a relationship [‡] | 11 3 | 37.17% | - |
| Children | No children | 26 8 | 88.16% | - |
| | One or more children | 36 | 11.84% | - |
| Lifetime depression diagnosis | No lifetime diagnosis | 10 5 | 34.54% | - |
| | Lifetime diagnosis | 19 9 | 65.46% | - |
| Depressive symptoms (BDI-II sum scores) | [continuous] | 30 4 | 18.41 (13.85) | 0-62 |

Note.

Sample demographics for the full analytic sample of N=304.

[†]The authors combined the following possible answer choices into the “Other” category due to a small number of responses: Agender, Bigender, Non-binary, and Other. [#]The authors combined the following answer choices into the “In a relationship” category: Married to first spouse, Remarried, Dating someone, and Civil partnership. Additionally, all individuals who responded “Other” to the relationship status questions answered an open text question and described either “Engaged,” “Domestic partner,” or “Long-distance,” and were all added to the “In a relationship” category.

Table 2.*Total Sleep Time, Sleep Efficiency, Sleep Latency, and Wake After Sleep Onset by Circadian Phase*

| Sleep Variable | Delayed (N=104) (mean (standard deviation), range) | Intermediate (N=164) (mean (standard deviation), range) | Advanced (N=25) (mean (standard deviation), range) | Group comparisons | | | | |
|-----------------------------|---|--|---|-------------------|----|----------|--------|--|
| | | | | F or χ^2 | Df | η^2 | p | Post hoc |
| Total sleep time (h:mm) | 6:46 (2:26), 0:45-12:40 | 7:31 (1:52), 1:10-13:00 | 8:01 (1:57), 1:00-11:55 | 5.758 | 2 | 0.038 | 0.004 | delayed<advanced delayed<intermediate |
| Sleep efficiency (%) | 68.63 (19.71), 8.00-99.00 | 81.45 (13.56), 17.00-100.00 | 83.72 (18.63), 8.00-100.00 | 41.918 | 2 | 0.133 | <0.001 | delayed<advanced delayed<intermediate |
| Sleep latency (mm) | 67 (63), 4-415 | 35 (34), 0-230 | 35 (47), 0-240 | 15.18 | 2 | 0.095 | <0.001 | delayed>advanced delayed>intermediate |
| Wake after sleep onset (mm) | 43 (64), 0-360 | 33 (56), 0-440 | 32 (42), 0-139 | 1.114 | 2 | 0.008 | 0.33 | - |

SLEEP AND DEPRESSION IN AUTISTIC ADULTS

Table 3.*Results from the Regression Model with Demographic and Sleep Variables Predicting**Depressive Symptoms*

| | Variable | | Regression model (N=291) | |
|------------------------------------|-------------------------------|----------------------|---------------------------|----------|
| | | | <i>B</i> (standard error) | β |
| Independent Variables | Total sleep time (in Minutes) | [continuous] | 0.01 (0.008) | 0.11 |
| | Sleep efficiency | [continuous] | -0.24 (0.06)*** | -0.30*** |
| | Circadian Phase | Intermediate | -- | -- |
| | | Delayed | 5.68 (1.72)** | 0.41** |
| Advanced | | -3.33 (2.77) | -0.24 | |
| Covariates | Employment status | Not working | -- | -- |
| | | Working | -2.03 (1.56) | -0.15 |
| | School status | Not in school | -- | -- |
| | | In school | -0.10 (1.76) | -0.007 |
| | Relationship status | Single | -- | -- |
| | | In a relationship | 4.65 (1.65)** | 0.34** |
| | Children | No children | -- | -- |
| | | One or more children | -0.70 (2.61) | -0.05 |
| | Gender | Male | -- | -- |
| | | Female | 0.48 (1.59) | 0.034 |
| Other | | 5.86 (3.35) | 0.42 | |
| Age | [continuous] | 0.16 (0.18) | 0.05 | |
| Overall Variances Explained | R ² | | 0.21 | |
| | Adjusted R ² | | 0.18 | |

Note: * indicates $p < 0.05$, ** for $p < 0.01$, and *** for $p < 0.001$; with a sample size of 291 in the model, we are sufficiently powered to conduct the linear regression analysis.

Figure Legends

Figure 1. Occurrence of sleep disturbances. **TST:** Disrupted total sleep time, **SE:** Disrupted sleep efficiency, **CP:** Delayed or advanced circadian phase. From the sample with all sleep variables available (N=293), 13.99% were determined to have none of the three primary sleep disturbances, 30.38% had one sleep disturbance (6.14% CP, 12.29% SE and 11.95% TST), 35.49% had two sleep disturbances (9.90% SE & CP, 7.85% TST & CP, and 17.75% TST & SE), and 20.14% had all three sleep disturbances.