Journal of the International Neuropsychological Society (2021), **27**, 711–721

doi:10.1017/S1355617720001186

Sleep Mediates Age-Related Executive Function for Older Adults with Limited Cognitive Reserve

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(Received April 1, 2020; Final revision August 25, 2020; Accepted October 21, 2020; First Published Online December 15, 2020)

Abstract

Objective: Sleep quantity and quality are associated with executive function (EF) in experimental studies, and in individuals with sleep disorders. With advancing age, sleep quantity and quality decline, as does the ability to perform EF tasks, suggesting that sleep disruption may contribute to age-related EF declines. This cross-sectional cohort study tested the hypothesis that poorer sleep quality (i.e., the frequency and duration of awakenings) and/or quantity may partly account for age-related EF deficits. **Method:** Community-dwelling older adults ($N = 184$) completed actigraphic sleep monitoring then a range of EF tasks. Two EF factors were extracted using exploratory structural equation modeling. Sleep variables did not mediate the relationship between age and EF factors. *Post hoc* moderated mediation analyses were conducted to test whether cognitive reserve compensates for sleep-related EF deficits, using years of education as a proxy measure of cognitive reserve. **Results:** We found a significant interaction between cognitive reserve and the number and frequency of awakenings, explaining a small (approximately 3%), but significant amount of variance in EF. Specifically, in individuals with fewer than 11 years of education, greater sleep disturbance was associated with poorer EF, but sleep did not impact EF in those with more education. There was no association between age and sleep quantity. **Conclusions:** This study highlights the role of cognitive reserve in the sleep–EF relationship, suggesting individuals with greater cognitive reserve may be able to counter the impact of disturbed sleep on EF. Therefore, improving sleep may confer some protection against EF deficits in vulnerable older adults.

Keywords: Cognition, Aging, Actigraphy, Sleep disturbance

INTRODUCTION

Characteristics of sleep quality such as total sleep time (TST), wake after sleep onset (WASO), sleep onset latency, and sleep efficiency (proportion of time in bed spent asleep) decline with advancing age (Mander, Winer, & Walker, 2017; Ohayan, Carskadon, Guilleminault, & Vitiello, 2004). Sleep quality deficits are concerning given the restorative glymphatic and neuromodulatory functions of sleep for brain performance (Krueger, Frank, Wisor, & Roy, 2016). During wake, the cellular energy turnover rate is high, but this is reversed during slow wave sleep (SWS), allowing for restoration of resources (Krueger et al., 2016). Simultaneously, the brain's glymphatic system removes metabolic waste accumulated during wakefulness, while neural connections are modulated based on the day's experiences (Krueger et al., 2016). Hence, interruptions to these processes due to fragmentation or restriction of sleep impact neural restoration and brain performance. Consequently, deterioration in sleep quantity and quality has been posited as one possible mechanism explaining age-related cognitive deficits (Yaffe, Falvey, & Hoang, 2014). This study's aim, therefore, was to determine if sleep quality and quantity partially mediate age-related variance in cognition, specifically executive function (EF).

EFs are higher-order mental processes required for problem-solving, planning, and organization. EF encompasses up to four domains – inhibition, generativity (speeded word retrieval), working memory, and set-shifting – either

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distinctly (Fisk & Sharp, 2004; Miyake, Friedman, Emerson, Witzki, Howerter, & Wager, 2000) or conjointly (Adrover-Roig, Sesé, Barcelo & Palmer, 2012; Miyake & Friedman, 2012). However, differentiation of EF domains may be influenced by sample characteristics, and the types/complexity of measures used to assess cognitive processes (Adrover-Roig et al., 2012; Cabeza, Anderson, Locantore, & McIntosh, 2002). As EF is predictive of functional independence and mortality among older adults (e.g., Davis, Marra, Najafzadeh, & Liu-Amrbose, 2010), understanding what factors contribute to EF performance may elucidate mechanisms to maintain optimum functioning.

EF and sleep quality both decline with age, hence, it is plausible that sleep quality may contribute to age-related EF variance. However, sleep research has primarily studied young adults or individuals with sleep disorders, or relied on self-report measures, which are less reliable than objective measures, such as polysomnography (PSG) and actigraphy (Bastien et al., 2003; Landry, Best, & Liu-Ambrose, 2015). Further, sleep research involving older adults has tended to focus on TST or WASO without consideration of patterns of sleep interruption. Specifically, WASO does not differentiate (1) sleep that is punctuated by frequent arousals versus and (2) sleep that is marked by fewer but longer awakenings. Obstructive sleep apnea (OSA) and insomnia are extreme examples of these two profiles of sleep disturbance, each associated with different physiological effects and implications for daytime functioning. Thus, WASO, taken in isolation, is problematic, as it limits understanding of the role of sleep disturbance on cognition.

Frequent Awakenings and EF in Clinical and Young Adult Samples

The threshold for waking is lower in older adults and is potentiated by the increased prevalence of arthritic pain, reflux, nocturia, hypertension, and OSA (Crowley, 2011). In reviewing mechanisms of OSA's effect on cognition, Beebe and Gozal (2002) propose that, beyond hypoxia and hypercarbia (excessive carbon dioxide), sleep disruption additionally affects functional homeostasis, suggesting arousals alone could diminish the brain's restorative processes, resulting in poorer EF. In healthy controls, sleep fragmentation similar to that experienced in OSA has been experimentally replicated using auditory tones to produce frequent arousals. Affected individuals subsequently displayed impaired set-shifting (Martin, Engelman, Deary, & Douglas, 1996), suggesting that, irrespective of hypoxia and hypercarbia, sleep fragmentation impacts EF. A systematic review of neurocognitive functions in OSA found deficits in EF compared with controls, which were *somewhat* ameliorated with treatment (Bucks, Olaithe, & Eastwood, 2013). If sleep fragmentation affects EF in clinical and younger adult samples, age-related interruption of restorative sleep may also have implications for cognition among healthy older adults.

Longer Awakenings and EF in Clinical and Young Adult Samples

Delayed resumption of sleep following awakening may suggest impairment of neuroendocrine systems, which limit efficient transition between sleep and wake states (Van Cauter, Leproult, & Kupfer, 1996). An analysis of cerebral metabolism found individuals with insomnia displayed impaired disengagement of the left middle frontal gyrus during sleep, and impaired engagement during wake, which may affect EF (Kay et al., 2016). EF deficits have been found in insomnia, compared to healthy sleepers (Ballesio, 2017; Fernandez-Mendoza et al., 2010). However, other studies have not replicated these findings (Vignola, Lamoureux, Bastien, & Morin, 2000) or have found insomnia-related deficits for only a subset of EF domains (Fortier-Brochu, Beaulileu-Bonneau, Ivers, & Morin, 2012). The chronic *partial deprivation* of sleep, as experienced in insomnia, has been replicated in experimental studies of healthy young adults and found to have equivalent effects on EF to *total sleep deprivation* (Van Dongen, Maislin, Mullington, & Dinges, 2003). Hence, it is plausible that deficits in EF among older adults could be related to impaired metabolic regulation associated with extended awakenings and subsequent impaired neural restoration.

Sleep and EF Among Older Adults

Among older adults, the influence of sleep on EF varies depending on how sleep is measured. Actigraphy research has found greater WASO to predict poorer EF, but no association between EF and TST (Blackwell et al., 2011; Miyata et al., 2013; Wilckens, Woo, Kirk, Erickson, & Wheeler, 2014). Conversely, studies using self-report measures found no relationship between "sleep disruption" and EF (Lo, Loh, Zeng, Sim, & Chee, 2014) and inconsistent findings for TST, with three of four studies finding shorter TST predicted poorer EF (Lo, Groeger, Cheng, Dijk, & Chee, 2016; Lo et al., 2014; Nebes, Buysse, Halligan, Houck, & Monk, 2009; Schmutte et al., 2007).

Methodological Differences

Conflicting findings on the relationship between sleep and EF could be due to methodological differences across studies. Individuals *complaining* of poor sleep were found to underestimate sleep duration and quality on self-report measures, while objective measures indicated no differences in WASO when compared to non-complainers (McCrae et al., 2005). This indicates that individuals may vary in their accuracy when self-reporting sleep and awakening times. Further, complaints of poor sleep may depend on individual expectations of sleep. Thus, the degree of discrepancy between *self-perceived* sleep duration and quality and *actual* sleep duration and quality is a possible explanation for inconsistent findings between sleep and EF. Furthermore, the consistent findings of a relationship between OSA and EF, compared

https://doi.org/10.1017/S1355617720001186 Published online by Cambridge University Press

with equivocal findings for insomnia, may be because OSA is formally diagnosed using PSG, whereas insomnia can be diagnosed from self-reported sleep complaints.

Additional methodological differences include: (1) how EF is measured – specifically whether multiple, well-validated measures of multiple EF domains are included and (2) differences in the covariates included – specifically cognitive reserve and depression (Muzur, Pace-Schott, & Hobson, 2002). Cognitive reserve – the ability to compensate for neural injury – is a strong predictor of late-life cognitive performance and, unless considered in analyses, may obfuscate age-related EF variance (Stern, 2012). Moderate and severe depression has also been associated with EF impairments as well as sleep disturbance. Thus, the unique contribution of sleep quality to EF may be attributed to depression if it is not controlled for in analyses (Lugtenburg et al., 2017).

The present study addressed these methodological limitations by: objectively measuring multiple indices of sleep quality with actigraphy; comprehensively assessing EF using measures of set-shifting, generativity, inhibition, and working memory for a robust representation of EF; and controlling for cognitive reserve and depression. Moreover, we decomposed WASO into two distinct sleep disturbance markers – frequent versus longer awakenings – hypothesized to have separable effects on EF. In summary, this research was the first to test whether sleep quality and quantity partially mediates the relationship between age and EF performance when controlling for cognitive reserve and depression.

METHODS

Participants

Community-dwelling older adults aged 55–93 years ($N = 268$) were recruited to the University of Western Australia's (UWA) Healthy Aging Research Program (HARP) *via* referrals from community organizations and the Australian Alzheimer's Research Foundation. Exclusion criteria included history of neurological (e.g., stroke, a loss of consciousness exceeding 30 min) or severe psychiatric (schizophrenia, bipolar affective disorder) disorders that could affect cognitive performance, and probable cognitive impairment based on a score below the normative cutoff given age and education (Iverson, 1998) on the Mini-Mental State Examination (MMSE – Folstein, Folstein, & McHugh, 1975). After exclusions, the sample for initial analyses was 255. A subset of volunteers provided a minimum of five nights of valid actigraphy data ($N = 184$). Twelve volunteers (4.7%) were ineligible for the sleep study due to diagnosed OSA.

Materials

Sleep quality

Actigraphy. Actigraphy (an accelerometer worn on the non-dominant wrist) distinguishes sleep from wake based on algorithms to detect movement (de Souza et al., 2003). The American Academy of Sleep recommends a minimum of five nights' data for reliable estimate of sleep quality for populations where sleep is highly variable, such as older adults (Littner et al., 2003). Participants were asked to wear a GTX3-BT actigraph (ActiGraph, Florida, USA) for seven nights to allow for potential equipment or participant errors and advised to remove the actigraph only for swimming and bathing. All valid nights were included in the analysis. ActiLife software (ActiGraph, Florida, USA) was used to analyze sleep data based on 24-h intervals and 60-s epochs. Data were manually adjusted for self-reported in-bed and out-of-bed times, except when visual inspection indicated self-reported data were discrepant to ActiLife calculations by > 60 min. The Cole–Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992) was employed to calculate TST and the frequency and duration of awakenings. If there was non-wear time immediately preceding sleep onset, the night was excluded from analysis as the veracity of sleep onset could not be assured. Sleep indices for the ≥ 5 nights were averaged in SPSS.

Actigraphy is comparably reliable for assessing sleep parameters against the gold standard, PSG (Ancoli-Israel et al., 2003). The Cole–Kripke algorithm has been validated against PSG (Cole, Kripke, Gruen, Mulaney, & Gillin, 1992) with 88% of all epochs correctly identified as either sleep or wake (*accuracy*). Discrepancies were largely the misclassification of wake as epochs of sleep (*specificity*). Marino et al. (2013) confirmed the validity of the Cole–Kripke algorithm for detecting WASO as age and insomnia severity increased, with moderate concordance against PSG ($r_s = .61$).

Sleep diary. The Consensus Sleep Diary (Carney et al., 2012) documented self-reports of: the time participants got into bed; the time they attempted to go to sleep; estimated time it took them to fall asleep; their final morning awakening time; and periods when the actigraph was not worn. These data were used to cross-reference against actigraphy.

OSA risk. Participants were excluded from sleep analyses if they self-reported diagnosed OSA on a medical history questionnaire, as treatment efficacy could confound associations between sleep disturbance and cognition. To assess for the potential influence of undiagnosed OSA, individuals were categorized as either having high (a positive score on 2 or more domains) or low risk (1 or fewer domains scored as positive) for OSA according to self-reported responses on the Berlin questionnaire (Netzer, Stoohs, Netzer, Clark, & Strohl, 1999).

Executive function

Nine measures of EF were factor analyzed. These measures were informed by Fisk and Sharp's (2004) four-factor model to include comprehensive measurement of inhibition, shifting, working memory, and generativity.

https://doi.org/10.1017/S1355617720001186 Published online by Cambridge University Press

Inhibition. Participants completed the Anti-Saccades and Flanker tests from the NIH Examiner (Kramer et al., 2014). The Anti-Saccades test requires participants to look at the middle of a computer screen as stimulus is presented as moving either to the left or the right. The task is to suppress the reflexive urge to visually track the target and, instead, look in the opposite direction. Correct eye movements (max. 40) were visually scored by the examiner. The Flanker test involves a series of arrows presented in a horizontal line on a computer screen. Participants are instructed to identify the direction of the middle arrow, which points in either the same or the opposite direction as the surrounding arrows (max. 10). Higher scores on both indicate better inhibition.

Set-shifting. Set-shifting was assessed using the NIH Examiner Set-Shifting subtest (Kramer et al., 2014), the Trail Making Test Part B (TMTB – Reitan and Wolfson 1985), and the Verbal Fluency Category Switching subtest from the Delis–Kaplan Executive Function System (DKEFS; Delis, Kaplan, & Kramer, 2001). The Set-shifting Test requires participants to match an image (a square or triangle, shaded either blue or red) to one of two target images on a computer screen based on either a shape or color condition. The stimulus changes with every trial, and the matching command (shape vs. color) is either repeated or altered over trials. The DKEFS category switching test requires participants to name as many animals and instruments as possible within 1 min, alternating between naming an animal and an instrument. A point is awarded for every correct alternation (i.e., switching accuracy). Higher scores on the above tests indicate better set-shifting, while longer times to complete TMTB reflect less efficient switching.

Working memory. Digit Span Backwards (DSB) from the Wechsler Adult Intelligence Scale, 3rd Edition (WAIS-III, Wechsler, 1997) and the Dual Task subtest of the Test of Everyday Attention (TEA; Robertson, Ward, Ridgeway, & Nimmo-Smith, 1991) both tap into the central executive component of working memory with simultaneous short-term storage of information in the presence of attentional control (Baddeley, 1996). Higher scores on DSB, and less time taken on the TEA dual task, compared with the single-task condition, indicate better working memory.

Generativity. Generativity was measured using the Controlled Oral Word Association Test (COWAT, Benton, Hamsher, & Sivan, 1994) and the Action Fluency task (Piatt, Fields, Paolo, & Tröster, 1999). The tests operate similarly with participants given 1 min to name as many words as possible that begin with the letter "C" and then repeat for action words. Higher scores indicate more efficient word generation.

Depression and cognitive reserve

Depression. was assessed using eight items of the 9-item self-report Patient Health Questionnaire – 9 (PHQ-9, Kroenke & Spitzer, 2002). We removed Question 3, which involves rating sleep quality, as sleep was objectively assessed in the primary analysis. Higher scores reflected greater depression.

Cognitive reserve. was estimated from total years of full-time education (Ritchie, Bates, Der, Starr, & Deary, 2013).

Study Procedures

Approval was obtained from the UWA Human Research Ethics Committee. Participants provided written informed consent and collected the actigraph and sleep diary and then completed cognitive testing 7 to 11 days later (allowing for a minimum of 7 days' sleep monitoring).

Statistical analysis

Data were checked for normality, homogeneity, and multicollinearity in SPSS and outliers on the cognitive measures were brought within three standard deviations of the mean. Initially, we conducted confirmatory factor analysis (CFA) to isolate independent factors, per Fisk and Sharp (2004). However, due to poor fit, we then conducted exploratory structural equation modeling (ESEM) using MPlus with maximum likelihood robust estimates to allow for relatedness across constructs (per Miyake and Friedman, 2012), in order to determine the factor structure in this sample. We analyzed the broader sample with cognition scores ($N = 255$) to increase power for latent variable modeling. Model fit was ascertained using: the χ^2 statistic, Akaike's information criterion (AIC), Bentler's comparative fit index (CFI), the Root Mean Square Error of Approximation (RMSEA), Tucker Lewis Index (TLI), and the Standardized Root Mean-Squared Residual (SRMR). Factor scores generated by MPlus were reversed for ease of interpretation.

For the sample with sleep data ($N = 184$), we conducted simultaneous regression using Conditional Process Analysis for SPSS Version 3.2 (Hayes, 2013), to test the hypothesis that sleep mediated the relationships between age and EF factor scores, when controlling for cognitive reserve and depression. Bootstrapping was performed to calculate 95% bias-corrected confidence intervals using 5000 bootstrap samples (Preacher & Hayes, 2004). The mediators – awakening length and number of awakenings – were entered at the same time. As there was no association between TST and either age or EF, we did not include TST in the mediation analysis.

Moderated mediation was conducted *post hoc* to identify if cognitive reserve (continuous years of education) ameliorated the mediated effects of age on EF *via* sleep disturbance (see Figure 1). We used Conditional Process Analysis for SPSS Version 3.2 (Hayes, 2013), model 15. Mediators were entered at the same time while controlling for depression, and Johnson–Neyman output was selected. The Johnson–Neyman technique assesses each value of a continuous moderator variable, working backward until it determines when

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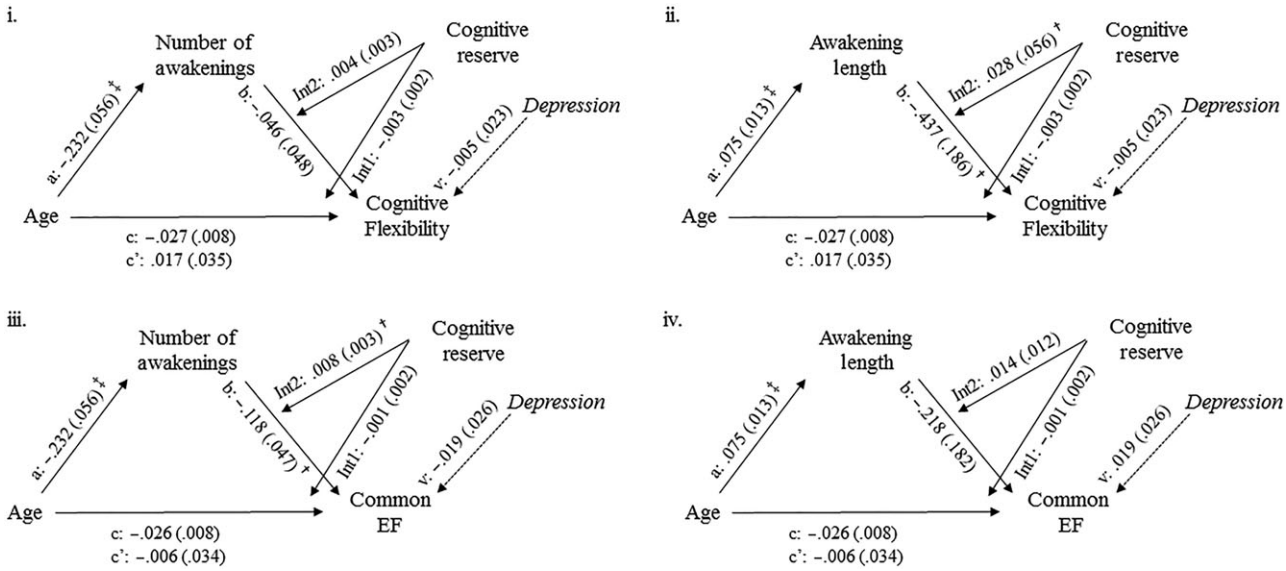

Fig. 1. Models depicting moderated mediation analyses, whereby the dependent variable is Cognitive Flexibility in models i and ii, and Common EF in models iii and iv. In models i and iii, the mediator is the number of awakenings, and in figures ii and iv the mediator is the length of awakenings. † $p < .05$; ‡ $p < .01$; a = path (*B, SE*) between age and mediator; b = path between mediator and the dependent variable; c = total effect of age on dependent variable, taking account of any covariates; c' = direct effect of age on dependent variable when taking account of covariates and mediators; Int1 = effect of the interaction between age and education on the dependent variable; Int2 = effect of the interaction between cognitive reserve and the mediator on the dependent variable; v = covariate (i.e., the effect of depression on cognitive outcome).

the effect of X on Y ceases to be significant – that is, at what years of education the relationship between sleep quality and EF is not significant (Spiller, Fitzsimmons, Lynch, & McClelland, 2013).

RESULTS

The sleep sample ($N = 184$) did not differ significantly in age ($M = 72.15$ years), education ($M = 14$ years), or gender ratio (62% female) from the complete sample. Sample characteristics are shown in Table 1.

CFA of Fisk and Sharp's (2004) four-factor EF model and ESEM of Miyake and Friedman's (2012) three-factor model were not good fits for our data. Further ESEM analysis produced a two-factor EF model – *Cognitive Flexibility* (set-shifting and inhibition) and *Common EF* (set-shifting, working memory, inhibition, and generativity (χ^2 (32) = 32.28, $p = .07$; AIC = 4838.43; RMSEA = .04 (0–.07); CFI = .97; TLI = .95; SRMR = .04). See Table S1 (Supplemental Material) for standardized factor loadings.

Bivariate correlations of age, sleep, and EF factors have been provided as Supplemental Material (Table S2). Consistent with expectations, older age was associated with poorer Common EF and Cognitive Flexibility, and longer awakening length. Age was not associated with TST, and, against expectations, was associated with fewer awakenings. Sleep indices were generally not correlated with EF, with the exception of greater awakening length predicting poorer Cognitive Flexibility.

After controlling for depression and cognitive reserve, the regression models including sleep indices explained 8.5% of the variance in Cognitive Flexibility, $F(5, 175) = 3.246$, $p < .01$, and 17.4% of the variance in Common EF, $F(5, 175) = 7.376$, $p < .01$. However, indirect paths *via* the number of awakenings and the awakening length were not significant, indicating that sleep disturbance did not appear to mediate the relationship between age and EF factors. See Table S3 in Supplemental Material for mediation analyses. However, *post hoc* moderated mediation analyses indicated differential effects of sleep disturbance patterns on EF when accounting for cognitive reserve, as depicted in Figure 1 and reported in Table 2.

We found a significant interaction between cognitive reserve and awakening length on Cognitive Flexibility, $F(8, 172) = 2.804$, $p < .01$, that accounted for 2.6% of the variance in this EF factor (Table 2). Inspection of the Johnson–Neyman output (see Figure 2) reveals that sleep quality was related to Cognitive Flexibility only in those with 10.9 years of education or fewer (i.e., the 17% of our sample who reported fewer than 11 years of education).

Similarly, there was a mediating role for the number of awakenings on Common EF when cognitive reserve was included in the analysis as a moderator, $F(8, 172) = 5.783$, $p < .01$. The interaction between number of awakenings and cognitive reserve explained 3.0% of the variance in Common EF. Our cohort recorded fewer awakenings with greater age; however, greater awakenings mediated age-related differences in Common EF for those with limited

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Table 1. Demographic, cognitive, and sleep characteristics of the sample

	N	*M* (*SD*)	Range
Sample characteristics			
Complete sample			
Age (years)	255	71.71 (7.66)	55–93
Education	251	13.82 (7.66)	3–27
Depression[a]	255	1.88 (2.42)	0–13
Sleep sample			
Age (years)	184	72.15 (7.38)	55–93
Education	181	13.99 (3.58)	3–27
Depression[a]	184	1.74 (2.32)	0–12
Executive function measures			
Set-shifting	232	7.47 (.81)	4.79–9.21
Trails B (s)	249	76.19 (28.98)	30.00–202.83
Category switching	252	14.86 (3.65)	4–24
Flanker	238	8.32 (.47)	6.74–9.21
Anti-Saccades	243	16.55 (3.43)	5–20
TEA Dual Task	242	1.53 (1.88)	−2.41–9.25
Digits Backward	254	7.19 (2.12)	2–14
COWAT-C	254	16.32 (4.87)	4–33
COWAT-Actions	254	17.83 (5.12)	6–34
EF factors[b]			
Cognitive Flexibility[c]	255	2.72	.00–4.48
Common EF	255	.01 (.85)	−2.38–2.92
Actigraphic sleep indices			
Awakening length (min)	184	3.57 (1.31)	1.27–8.35
Awakening number (per night)	184	12.03 (5.82)	2.00–34.14
Total sleep time (min)	184	418.76 (46.58)	280.20–609.17
Sleep efficiency (%)	184	90.39 (4.71)	68.63–98.01

COWAT-C = Controlled Oral Word Association Test letter C; EF = executive function; *SD* = standard deviation; TEA = Test of Everyday Attention.
[a] The depression rating is the PHQ-8, the PHQ-9 recalculated to exclude the influence of sleep (Question 3).
[b] Higher scores reflect better performance.
[c] Cognitive Flexibility factor scores were reversed for ease of interpretation.

cognitive reserve, specifically, when education was 10.4 years or fewer (see Figure 3).

The size and significance of the effects did not change when OSA risk was added as a covariate. In both instances, the direct effect of age was non-significant when the moderator was added to the model. Regardless of cognitive reserve, the number of awakenings did not mediate the relationship between age and Cognitive Flexibility, and awakening length did not mediate the relationship between age and Common EF.

DISCUSSION

The present study aimed to evaluate the inter-relationships between age, sleep, and EF. Specifically, whether age-related differences in EF could be partially accounted for by more frequent awakenings and/or longer awakening duration. The present findings supported the hypothesis that sleep disruption (i.e., greater number and length of awakenings), mediated age-related differences in EF – but only when individual differences in cognitive reserve were considered.

The interaction of cognitive reserve and the length of awakenings explained a small, but significant 2.6% of the variance of age-related Cognitive Flexibility. Similarly, the interaction between the number of awakenings and cognitive reserve explained 3.0% of age-related variance of Common EF. Increasing duration of awakenings was associated with poorer Cognitive Flexibility, and increasing frequency of awakenings was associated with poorer Common EF, but only for older adults with approximately 11 years of education or fewer. Individuals with higher levels of education were not affected by poorer sleep quality.

These findings suggest improving sleep could confer a protective effect against EF declines for those with limited cognitive reserve. Notably, the different indices of sleep fragmentation – duration of awakenings and number of awakenings – singularly affected a specific factor of EF, but not both. Thus, these different patterns of sleep disturbance appeared to have differential effects on the two factors of EF. In our older adult sample, there was an inverse relationship between the number of awakenings and wake length, such that those with more awakenings had shorter awakening durations (similar to that found with OSA), while those with fewer awakenings were awake for longer periods (similar to insomnia). This reinforces the need to assess different components of EF, as well as the subcomponents of WASO individually.

Arguably, these differential effects could be associated with subtle differences in the organization of sleep. Sleep fragmentation is more widely distributed across SWS than other stages (Anderson & Horne, 2003). SWS is purported to facilitate elimination of metabolic waste and regulation of metabolic homeostasis, thus, disruptions to these processes may confer more global deficits (Scullin & Bliwise, 2015). Longer wake periods may reflect poorer regulation of the hypothalamic pituitary adrenal axis, which affects the robustness of sleep–wake transitions and engagement of specific areas of the frontal lobes (Dijk, Duffy, Silva, Shanahan, Boivin, & Czeisler, 2012; Kay et al., 2016). It could be postulated that Cognitive Flexibility is more mentally demanding, while also more specific in nature, and thus, more sensitive to declining amplitudes in specific patterns of neural activity. Longer awakenings but not number of awakenings mediated the relationship between age and prospective memory, which is cognitively demanding because it recruits both EF and retrospective memory (Fine et al., 2019). Thus, threshold effects on cognitive resources may explain the different outcomes.

Unlike previous research, WASO, the product of both frequency and duration of awakenings, was not associated with scores on either EF factor. This divergence with past research is plausibly due to sample differences. Our sample experienced less sleep disturbance than other samples, with 41 min of WASO, compared to 65 to 78 min reported

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Table 2. Moderated mediation analysis of the interactive effects of cognitive reserve and sleep disturbance on EF when controlling for depression (N = 181[a])

	B	*SE*	*t*	*p*	LLCI	ULCI
a paths						
Age › No. Awakenings	−.232	.056	−4.127	<.001	−.342	−.121
Age › Wake Length	.075	.013	5.989	<.001	.050	.100
b paths						
No. Awakenings › Cognitive Flexibility	−.046	.048	−.953	.342	−.141	.049
Wake Length › Cognitive Flexibility	.437	.186	2.347	.020	.069	.804
No. Awakenings › Common EF	−.118	.047	−2.496	.014	−.211	−.025
Wake Length › Common EF	−.218	.182	−1.198	.232	−.577	.141
c paths						
Age › Cognitive Flexibility	−.027	.008	−3.355	.001	−.043	−.011
Age › Common EF	−.026	.008	−3.426	.001	−.041	−.010
c' paths						
Age › Cognitive Flexibility	.017	.035	−.483	.630	−5.539	4.523
Age › Common EF	−.006	.034	−.175	.861	−.073	.061
Index of moderated mediation						
Age › No Awakenings › Cognitive Flexibility	−.001	.001	–	>.05	−.003	.000
Age › No Awakenings › Common EF	−.002	.001	–	<.05	−.004	−.000
Age › Awakening Length › Cognitive Flexibility	.002	.001	–	<.05	.000	.004
Age › Awakening Length › Common EF	.001	.001	–	>.05	−.001	.003
Interactions predicting Cognitive Flexibility						
Age × Education	.003	.002	1.161	.247	−.002	.008
No. Awakenings × Education	.004	.003	1.125	.262	−.003	.010
Wake Length × Education	.028	.013	2.252	.026	.004	.053
Interactions predicting Common EF						
Age × Education	−.001	.002	−.566	.572	−.006	.003
No. Awakenings × Education	.008	.003	2.569	.011	.002	.014
Wake Length × Education	.014	.012	1.149	.252	−.010	.038
Common EF	−.006	.034	−.175	.861	−.073	.061

EF = executive function; LLCI = Lower Limit Confidence Interval; *SE* = Standard Error; ULCI = Upper Limit Confidence Interval; INT = interaction between.
[a] Three participants did not provide their years of education, reducing the sample size to 181.

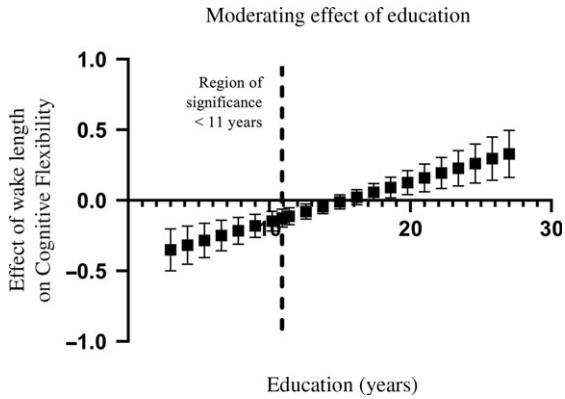

Fig. 2. The effect (as per Johnson–Neyman output) of awakening length on Cognitive Flexibility at different levels of cognitive reserve, whereby years of education is a proxy measure of cognitive reserve.

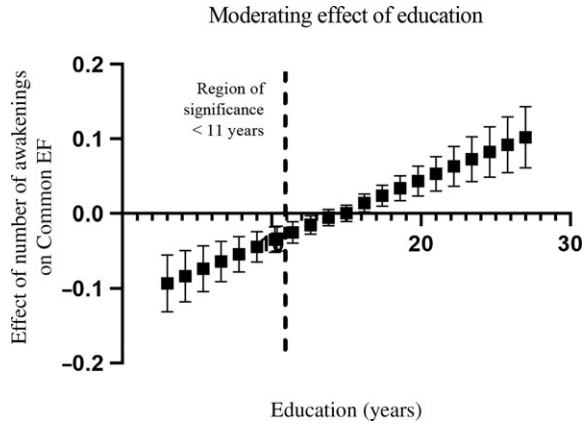

Fig. 3. The effect (as per Johnson–Neyman output) of the number of awakenings on Common EF at different levels of cognitive reserve, whereby years of education is a proxy measure of cognitive reserve.

elsewhere (Blackwell et al., 2006, 2011; Wilckens et al., 2014). These differences in sleep disturbance may be related to health factors. Further, Blackwell et al.'s sample (2006) was less educated (*Mean* = 12.9 years) and older (*Mean* = 83.5 years) than ours and potentially more likely to include individuals with mild cognitive impairment (MCI). Greater WASO predicted poorer EF among a sample with MCI with less average WASO than the aforementioned

https://doi.org/10.1017/S1355617720001186 Published online by Cambridge University Press

studies at 62 min (Naismith et al., 2010). Thus, cognitive status, as well as cognitive reserve, may further explain differences between our study and those described by Blackwell and colleagues.

Sleep disturbance is elevated among MCI and Alzheimer's disease (AD) populations, whereby a bidirectional relationship has been proposed as potentiating declines in both sleep quality and cognition (Landry & Liu-Ambrose, 2014; Naismith, et al., 2010). As cognitive reserve confers protection against neural insult (Stern, 2012), it is plausible that the interaction between sleep quality and cognitive reserve is of a greater magnitude among more cognitively (MCI and AD) and sleep impaired (insomnia and OSA) samples. Likewise, as our sample was better educated than the general population – 68% completing high school and 42% holding tertiary qualifications compared to the national average of 58% and 30%, respectively (Organisation for Economic Co-operation and Development, 2012) – there may be more individuals in the general population at risk of poor cognitive outcomes associated with poor sleep, than in this sample.

We confirmed previous findings that the older an individual is, the longer it takes them to return to sleep, and added further support to the evidence that *objective* TST does not decline with age past late mid-life (Blackwell et al., 2006, 2011; Ohayon et al., 2004; Wilckens et al., 2014). The perception that sleep duration declines with older age stems from early research that used self-report measures of sleep; however, self-reported distress around sleep, expectations of what is an "acceptable" amount of sleep, depression, and cognitive ability are all associated with discrepancies between subjective and objective measures of sleep (Alapin et al., 2000; Landry et al., 2015; Vitiello, Larsen, & Moe, 2004).

Perhaps more important than TST is the duration and quality of different sleep stages, particularly rapid eye movement (REM) sleep and SWS, which both decline with age (Yaffe, Falvey, & Hoang, 2014). Thus, variation in sleep architecture, rather than overall sleep duration, could be critical to cognitive performance, and potentially only for more resource-intensive EFs, given that older adults appear to recruit compensatory mental processes to perform tasks (Cabeza, Anderson, Locantore, & McIntosh, 2002). Future studies should explore specific stages of sleep as potential mechanisms for variance in EF, while considering the contributions of cognitive reserve and the changing nature of EF with older age.

Strengths and Limitations

The present study has multiple strengths, including a relatively large sample of older adults. We also measured sleep objectively over a minimum of five consecutive nights, closely preceding cognitive assessment. Actigraphy as a measure of sleep is advantageous because it is unobtrusive and less expensive than PSG; however, accelerometer technology has been found to overestimate TST, while underestimating WASO compared with PSG data (Marino et al., 2013). WASO underestimates are particularly evident when WASO > 30 min as actigraphy assumes resumption of sleep when there is an absence of movement. Our index of wakening duration, and the effect of the interaction with cognitive reserve, is plausibly underestimated, as 62% of our sample experienced WASO > 30 min.

The comprehensive measurement of the most commonly identified EF domains, and the extraction of two EF factors, has psychometric advantages over the simple scores provided by each EF measure. However, the measures used in our study to assess the "working memory" domain may be more reflective of complex attention which, although incorporated in working memory processes, may be less cognitively demanding.

We note that we did not adjust for multiple comparisons in the *post hoc* analyses, and if we had done so, the significance of the interaction between cognitive reserve and awakening length on Cognitive Flexibility would have been at the trend level ($p = .026$), just failing to meet the Bonferoni-adjusted p value of .025. As these analyses were planned as exploratory, we chose a less conservative approach (Perneger, 1998). The results of these analyses were consistent with those determined using a more stringent cutoff and are also consistent with the pattern of results reported by Fine et al. (2019) for a related cognitive domain (prospective memory).

Another limitation of the study is that recruitment through community organizations may have attracted more educated and healthier individuals than is representative of the general population. As health influences sleep, it is plausible that general population levels of sleep disturbance are underrepresented in this cohort. Another limitation is that our use of education as a proxy measure of cognitive reserve may be an underestimate of actual cognitive reserve for individuals with less formal education but substantial lifelong learning gained through professional and social pursuits. Furthermore, as we relied on self-report to exclude individuals with OSA, our sample may have included people with undiagnosed OSA, potentially limiting inference of the effects of sleep fragmentation on EF.

Future research should assess the *longitudinal* effects of sleep disturbance on EF, extend research into samples with MCI, in which the contribution of sleep for cognition may play a greater role, and assess for the effect of intra-individual variability in sleep disturbance on EF, as it has been suggested that the degree of day-to-day inconsistency between sleep duration and quality may be a more sensitive predictor of both insomnia complaints and health burden than mean sleep indices (Baron et al., 2017; Bei Wiley, Trinder, & Manber, 2016). In addition, research on sleep intervention is warranted as a more controlled methodological approach to assessing the causality of sleep disturbance for deficits of EF.

Summary and Conclusions

While previous research has found associations between parameters of sleep and EF, none has sought to distinguish

https://doi.org/10.1017/S1355617720001186 Published online by Cambridge University Press

if sleep mediates age-related differences in EF, or if sleep and EF are both independently influenced by age. Further, this was the first study to investigate specific subcomponents of WASO separately. This is important, as we propose awakening length and number of awakenings may have differential effects on EF.

Among a cohort of relatively healthy, community-dwelling older adults, without MCI, the relationship between age and EF was partially explained by an interaction between sleep quality and cognitive reserve when controlling for depression. In our study, older adults with below 11 years of education were vulnerable to the effects of more frequent awakenings when drawing on Common EF and more vulnerable to the effects of longer awakenings when executing Cognitive Flexibility. There was no evidence that TST influenced EF, and the frequency of awakenings declined with age in this cohort. Health factors, rather than age, may explain fewer awakenings among the old-old in this cohort; something that warrants further investigation. It is plausible that sleep-related deficits in cognition are related to disrupted REM and SWS, and future studies should examine sleep architecture as a potential underlying mechanism. Imperatively, all future research should account for differences in cognitive reserve.

ACKNOWLEDGEMENT

We would like to thank fellow researchers and participants of the Healthy Ageing Research Program for their important contributions, and Manja Laws and Shaun Markovic of the Australian Alzheimer's Research Foundation for their assistance with participant recruitment.

FINANCIAL SUPPORT

There are no grant funding or external funding disclosures to report.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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