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Do Older Adults Need Sleep? A Review of Neuroimaging, Sleep, and Aging Studies

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Abstract

Purpose of Review—Sleep habits, sleep physiology, and sleep disorders change with increasing age. However, there is a longstanding debate regarding whether older adults need sleep to maintain health and daily functioning (reduced-sleep-need view). An alternative possibility is that all older adults need sleep, but that many older adults have lost the ability to obtain restorative sleep (reduced-sleep-ability view). Prior research using behavioral and polysomnography outcomes has not definitively disentangled the reduced-sleep-need and reduced-sleep-ability views. Therefore, this review examines the neuroimaging literature to determine whether age-related changes in sleep cause—or are caused by—age-related changes in brain structure, function, and pathology.

Recent Findings—In middle-aged and older adults, poorer sleep quality, greater nighttime hypoxia, and shorter sleep duration related to cortical thinning in frontal regions implicated in slow wave generation, in frontoparietal networks implicated in cognitive control, and in hippocampal regions implicated in memory consolidation. Furthermore, poor sleep quality was associated with higher amyloid burden and decreased connectivity in the default mode network, a network that is disrupted in the pathway to Alzheimer's disease.

Summary—All adults need sleep, but cortical thinning and amyloid deposition with advancing age may weaken the brain's ability to produce restorative sleep. Therefore, sleep in older adults may not always support identical functions for physical, mental, and cognitive health as in young adults.

Keywords

magnetic resonance imaging; cortical thinning; amyloid; obstructive sleep apnea; default mode network; Alzheimer's disease; polysomnography; slow wave sleep; sleep spindles

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Compliance with Ethics Guidelines

Conflict of Interest

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Human and Animal Rights and Informed Consent

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Introduction

One of the most pronounced behavioral and physiological changes with aging is sleep [1]. With increasing age, adults report greater frequency of nighttime awakenings, waking too early without being able to go back to sleep, increased daytime napping, and advanced circadian phase [2]. The effects are most pronounced in older individuals with physical illnesses or psychiatric conditions [3]. Though the proximal causes of awakenings are varied, adults often attribute their sleep disruptions to greater need to use the bathroom during the night (nocturia): 53% of adults ages 55–84 report experiencing nocturia almost every night [4]. Frequency of nocturia confers an increased risk of mortality, though it is unclear whether the mechanism is via sleep disturbances [5].

When measuring sleep using polysomnography (PSG), there are several robust changes across the adult lifespan [6]. The Sao Paulo Epidemiologic Study (EPISONO) conducted nocturnal PSG recordings on over 1,000 adults ranging in age from 20 to 80 [7]. Nearly every PSG variable showed a significant age-related change. They observed age-related increases in N1 sleep, which were likely due to (or the cause of) increased nighttime awakenings. By contrast, chronological age was inversely related to REM duration, N2 duration, and N3 duration (slow wave sleep). Interestingly, in rodent models, older mice show *greater* slow wave activity than younger mice [8], and some human subjects researchers argue that N3 only declines with aging because EEG amplitude declines (guidelines dictate that slow waves be scored only if amplitude is > 75 mV) [1]. However, in studies of aging humans, quantitative EEG analyses have demonstrated that the incidence, frequency, slope, and density of slow waves are altered in older age [9, 10].

PSG variables often correlate with participants' subjective impressions of their sleep quality, at least in young adults [11]. Surprising, such associations may not be observed in older adults [12]. One potential resolution is that sleep *macro*architecture (e.g., sleep stages) is not as sensitive to biological age as is sleep *micro*architecture (e.g., sleep spindles) [13]. For example, in the Sleep and Health in Women study that enrolled 211 women ages 22–71, quantitative EEG analyses of spindle density, K-complex density, and slow wave activity were more strongly associated with advancing age than typical measures of sleep stages [14].

Sleep disorders, such as obstructive sleep apnea, also increase in prevalence with age [15]. In the EPISONO study [7], the apnea-hypopnea index increased from 1.4 breathing events/hour in 20–24 year olds to 21.9 breathing events/hour in 75–80 year olds. Sleep apnea induces hypoxia and sleep fragmentation that, theoretically, should impair neural and cognitive functioning [16]. Nevertheless, some researchers report that sleep apnea incurs few or no behavioral consequences in the elderly [17, 18].

Theories of Whether Older Adults Need Sleep

A common assumption is that age-related changes in sleep have short- and long-term effects on physical, mental, and cognitive health. And, there is good reason to think so: In young adults, sleep restriction/deprivation triggers acute difficulties with cognitive functions such

as attention, mood, memory, and creativity [19, 20]. Sleep loss increases susceptibility to the common cold [21], alters gene expression [22], and affects metabolism [23]. Moreover, in epidemiological studies, sleep disturbances are associated with cardiovascular conditions [24], cancer [25], Type II diabetes [26], and Alzheimer's disease [27]. Because these conditions are more prevalent with advancing age, sleep alterations might explain many deleterious effects generally attributed to aging (hereafter, the *preserved-sleep-need* view).

An alternative view is that the “need” to sleep is reduced with increasing age (for reviews, see 28, 29). The reduced-sleep-need view contends that sleep is important during infancy, childhood, and young adulthood, but is less necessary when older. According to the reduced-sleep-need view, sleep restriction should have no costs and sleep extension should have no benefits to physical, mental, and cognitive health (at least relative to young adults). What is lost in the “sleep need” debate is that sleep may serve a different function in older adults than in young adults (just as there may be different functions from childhood to adulthood [30]), or that neural changes prevent older adults from obtaining restorative sleep (hereafter, the *reduced-sleep-ability* view; see also, functional weakening hypothesis)[28, 31].

Resolving the “sleep need” debate is important to science, but also has implications in clinical settings. According to the preserved-sleep-need view, complaints of disturbed sleep or daytime sleepiness must be addressed, rather than concluding that poor sleep is a normal part of aging. Similarly, according to the reduced-sleep-ability view, practitioners should not only treat the sleep disturbance, but other potential causes of changes in brain structure/function (e.g., cardiovascular dysfunction). By contrast, according to the reduced-sleep-need view, practitioners should not prescribe sedative hypnotics to older adults due to these medications' dangerous side effects such as falls [32].

Despite the scientific and clinical importance of whether sleep need is preserved or reduced with aging, there remains scant agreement amongst the sleep community. Consider, for example, the sleep field's recent consensus papers on what constitutes sufficient sleep *quantity* and *quality* in adults. Sleep experts rated the appropriateness of individual sleep measures (e.g., total sleep time, N3 duration) across the lifespan [33–35]. Despite many points of consensus for children and young adults, the expert panels produced multiple “uncertain” ratings for measures of sleep quality and quantity in older adults (≥ 65 years). For example, the expert panelists disagreed on whether any amount of N3—as short as 5%, and as large as greater than 25%—was bad or good in older adults.

Behavioral and PSG Investigations of Poor Sleep in Older Adults

Given overwhelming changes to sleep physiology with aging, and strong associations between sleep quality and cognitive functioning in young adults, the preserved-sleep-need view predicts that age-related changes in sleep explain age-related changes in cognition. Some studies of sleep and cognitive aging converge with this prediction, but many have not [1, 36, 37]. Therefore, behavioral measures have not incontrovertibly answered whether older adults need sleep.

Some sleep researchers use PSG to examine whether older adults need sleep. These investigations typically find that following sleep deprivation, slow wave rebound is lower in older adults than in young adults [38]. Furthermore, when given 16 hours of bedrest for up to one week, older adults do not sleep as many hours as young adults [39]. Though consistent with the reduced-sleep-need view, these findings also support the reduced-sleep-ability view [13]. For example, if the neural systems supporting sleep initiation and slow wave generation are deficient in older adults, then older adults might still need restorative sleep, but lack the ability to obtain restorative sleep [40, 41].

Structural Neuroimaging Studies

A promising approach to legislating between the preserved-sleep-need, reduced-sleep-need, and reduced-sleep-ability theoretical viewpoints is to determine whether, and how, sleep correlates with cortical thinning in aging adults. Structural changes should precede behavioral outcomes (potentially explaining why sleep could change in older adults without consistent changes to cognitive ability [1]). Furthermore, the investigation of how poor sleep quantity/quality relates to brain structure should inform which types of behavioral outcomes are most likely to be impacted by poor sleep in older adults.

As detailed in Table 1, several structural neuroimaging studies in healthy older adults have been cross-sectional and used self-report measures of sleep, such as the Pittsburgh Sleep Quality Index (PSQI; [42]) and Epworth Sleepiness Scale (ESS; [43]). The primary findings are illustrated in Figure 1. An early study that used the ESS in middle-aged adults found that greater sleepiness was associated with lower ventromedial prefrontal cortex volume [44], a region that is implicated in emotional regulation and decision making (see Figure 1)[45]. In a recent, large study of older adults (N=1,374), Carvalho et al. [46] found that higher ESS scores not only related to lower cortical thickness in the frontal lobe, but also lower thickness in the parietal, temporal, and occipital lobes. An important question though is whether the daytime sleepiness (ESS) data reflected the presence of sleep apnea, general deficits in sleep quality, or the influence of non-sleep fatiguing factors.

Higher PSQI scores—indicating lower sleep quality—have also been linked to structural neuroimaging measures. In cross-sectional studies, PSQI-defined poor sleepers showed alterations in the right superior frontal gyrus [47], the hippocampus [48], and the insula [49]. These three regions are implicated in cognitive and emotional functioning. The superior frontal gyrus supports cognitive control (including working memory and emotional inhibition)[50–52], the hippocampus facilitates memory encoding and consolidation [53]; and the insula broadly assists with emotional processing, cognitive control, interoception, time perception, and music processing [54].

PSQI scores also relate to longitudinal changes in structural MRI measures. Chee and colleagues [55] reported that, of 66 older adults, worse PSQI-defined sleep quality at baseline was associated with faster ventricular expansion over 2 years, which is interesting because ventricular expansion is associated with preclinical Alzheimer's disease biomarkers [56]. They did not observe any impact of PSQI scores on hippocampal or frontal volume, and so it remains unclear how broadly PSQI scores predict cortical thinning. Nevertheless, it

is clear that cortical thinning strongly affects PSQI scores. In 147 adults ages 20 to 84 who were followed for 3.5 years, Sexton et al. [47] reported that cortical atrophy predicted PSQI sleep quality, with significant clusters spanning the superior frontal gyrus, precuneus, retrosplenial cortex, the lateral and medial frontal cortex, and the cingulate cortex. Note that several of these brain regions compose the brain's default mode network, a network that is active during wakeful rest and mind wandering [57]. The default mode network is disrupted in healthy young adults following acute sleep deprivation [58] and is weakened in preclinical Alzheimer's disease (for elaboration, see amyloid neuroimaging section)[59].

The PSQI and ESS questionnaires indicate that sleep disturbances relate to cortical thinning, but do not inform which sleep disturbance is most important. A popular view is that short sleep underpins declining brain health. In the Coronary Artery Risk Development in Young Adults (CARDIA) study of 613 middle-aged adults, white matter integrity in frontal, parietal, and temporal regions declined over 5 years in participants who reported 6.1–8.0 hours of sleep versus those sleeping 6 or fewer hours at baseline [60]. In the Baltimore Study of Aging of 122 healthy older adults, faster cortical thinning in frontotemporal regions occurred over 8 years in participants reporting sleeping 5 or 6 hours/night relative to participants sleeping 7 hours/night [61]. Interestingly, they also observed faster cortical thinning in participants sleeping 8 or 9 hours/night; this U-shaped pattern is consistent with epidemiological studies in which self-reported short and long sleep duration are associated with poorer health measures [62].

Several researchers have contended that short sleep duration is *not* related to cortical thinning [47, 49, 55, 63]. Part of the difficulty in connecting sleep duration to cortical thinning is it remains unresolved whether self-reported sleep duration represents time asleep or time in bed. Furthermore, many demographic and psychological factors can affect how participants respond on sleep questionnaires (e.g., social desirability, mental health)[62]. One recent idea that might reconcile the literature is that reported sleep duration should be considered relative to individuals' perceived sleep need. When Killgore and colleagues [64] subtracted participants' reported sleep need (i.e., minimum duration needed to not be functionally impaired) from the self-reported sleep duration, they found that middle-aged adults who had more sleep than needed also had greater ventromedial prefrontal cortex volume (gyrus rectus, medial orbitofrontal gyrus).

Given the limitations of subjective sleep measures, a seemingly advantageous approach would be to use PSG (Table 1). However, several PSG studies have failed to connect variability in sleep efficiency [65], sleep stages [66], and spindle activity [67] with variability in structural neuroimaging measures in healthy older adults. For example, Cantero and colleagues [66] found that shorter REM duration was associated with cortical thinning in default mode network regions in patients with amnesic mild cognitive impairment, but not in healthy controls (possibly because both REM sleep and the default mode network are impaired in the pathway to Alzheimer's disease [59, 68]). Furthermore, Fogel et al. [67] found that sleep spindle duration and density were associated with cerebellar and hippocampal gray matter volume in young adults, but not in healthy older adults. Perhaps these PSG results indicate an age-related change in how the brain generates sleep or an age-related change in the function that sleep serves for the older brain.

Or, perhaps neuroimaging studies need to look more closely at slow wave activity [13]. Changes in slow wave activity across the adult lifespan constitute critical evidence for the purported decline in sleep need (e.g., older adults show lower slow wave rebound following sleep deprivation than young adults [38]). Walker and colleagues [69] reported that greater slow wave activity was associated with greater medial PFC gray matter volume (see also [70]). Likewise, Carrier and colleagues [71] found that slow wave density was associated with greater cortical thickness in widespread frontal, temporal, and parietal regions. They concluded that age-related cortical atrophy explains age-related changes in slow wave activity. Thus, in contrast to the reduced-sleep-need view, age-related cortical thinning reduces the brain's ability to produce slow wave activity.

Amyloid Neuroimaging Studies

Because self-reported sleep disturbances are common to, and can be predictive of, Alzheimer's disease [72], researchers are investigating whether sleep correlates with beta-amyloid (A β) deposition in *preclinical* adults. This research is important for clinical purposes and for contrasting the reduced-sleep-need and reduced-sleep-ability theories. For example, subtle pathological processes (e.g., amyloid accumulation) may cause sleep quality to decline such that sleep is less deep and less restorative (i.e., indicating reduced sleep ability, not reduced sleep need). Existing studies on sleep and A β in humans have been cross-sectional in design, often using plasma [66] or cerebrospinal fluid [73] measures of amyloid. Given this review's focus on neuroimaging, this section covers studies that used positron emission tomography (PET) to measure A β burden in the brain [74].

Brain regions with greater neuronal activity are most vulnerable to amyloid deposition [75]. Therefore, A β burden is often highest in the default mode network (precuneus, posterior cingulate, medial prefrontal cortex, lateral temporal cortex, hippocampal formation, angular gyrus, retrosplenial cortex)[57]. Interestingly, individuals who report more sleep disturbances also show greater A β burden in several default mode network regions [76]. Which sleep disturbance is most related to A β burden is still debated. One study found that greater PET A β burden was associated with shorter total sleep time [77], whereas two studies found that PET A β burden was associated with longer sleep onset latency [49, 78]. Future studies should control for sleep disorders (e.g., obstructive sleep apnea) that increase risk for Alzheimer's disease [72] and control for psychiatric conditions that are linked to both sleep disturbances and amyloid (e.g., anxiety [79]).

Very few studies on amyloid imaging have concomitantly used PSG. Mander et al. [70] reported that increased medial PFC A β deposition in older adults was associated with lower slow wave activity (0.6 to 1.0 hz range) over source localized channels (Fz and Cz). The effects were specific to the medial PFC (which included the superior frontal, anterior cingulate, and medial orbitofrontal regions); slow wave activity was not related to A β burden in the dorsolateral PFC, temporal cortex, parietal cortex, or occipital cortex. Thus, many of the PET studies showed associations between sleep difficulties and A β burden in vulnerable default mode network brain regions.

Functional Neuroimaging Studies of Aging

In functional neuroimaging studies, researchers can measure regional glucose metabolism and oxygen consumption during wakeful rest or during cognitive tasks. During wakeful rest, older adults who have mild cognitive impairment show decreased connectivity in the default mode network if they have more sleep disturbances [80, 81]. During cognitive tasks, hippocampal activity has been connected to left-frontal fast sleep spindles [82] and to maintaining consistent circadian rhythm patterns [83]. A tantalizing possibility is that nighttime slow wave activity restores frontal lobe functioning. Though frontal lobe glucose metabolism decreases with aging, Buysse and colleagues [84] found that adults ages 37–61 who showed greater nighttime slow wave activity showed greater whole brain glucose metabolism the following day (adults ages 25–36, however, did not show this association). When controlling for chronological age, greater slow wave activity was associated with increased metabolism the next-day in regions associated with cognitive control (right superior frontal gyrus [50]), language processing (inferior frontal gyrus [85]), and motor functioning/learning (precentral gyrus [86]).

Functional neuroimaging is particularly helpful in addressing one of the fundamental findings used to support the reduced-sleep-need theory. In behavioral studies, experimental sleep deprivation impairs cognition and increases subjective sleepiness in young adults more than in older adults [1]. After sleep deprivation, older adults may even *outperform* young adults on attention-based reaction time tasks [87], an age benefit that is almost never observed when participants are not experimentally sleep deprived [88]. These findings are consistent with the reduced-sleep-need view, but have been challenged because behavioral studies have not ruled out the possibility that older adults are approaching the cognitive tasks differently than the young adults, perhaps due to greater motivation or greater lifetime experience with compensating for sleep deprivation.

Drummond and colleagues [89] tested 28 older adults (59 years old) and 28 younger adults (18–39 years old) on the Go/No-Go attention task while undergoing neuroimaging, once following normal sleep and once following total sleep deprivation. According to the reduced-sleep-need theory, older adults should show no functional changes following sleep deprivation. By contrast, following sleep deprivation, the older adults required significantly greater activation in the frontoparietal attention network [90] to perform the same task as after normal sleep. These patterns indicate less efficient neural processing and/or greater compensatory effort. The elderly brain needs sleep to function efficiently.

Neuroimaging Studies in Patients with Sleep Disordered Breathing

Most neuroimaging studies of patients with sleep disorders have used structural neuroimaging (for coverage of insomnia, see 91, 92). Weng et al. [93] conducted a meta-analysis on eight papers published from 2000 to 2012 comparing obstructive sleep apnea participants to age- and gender-matched participants. They reported that sleep apnea was associated with decreased gray matter in the right superior frontal gyrus (cognitive control [50]), left middle temporal gyrus (semantic processing [94]), and bilateral parahippocampal gyrus (a hub connecting the memory network to the default mode network [95]).

There have been several substantive MRI studies on sleep apnea since Weng et al.'s [93] meta-analysis. Though one study found less cortical thinning in patients with higher apnea-hypopnea index [96], most studies find the opposite pattern. Dusak et al. [97] and Akhan et al. [98] both observed that hippocampal volume was lower in sleep apnea participants than in healthy control participants. The hippocampal reductions related to a greater apnea-hypopnea index [98] and greater subjective sleepiness [46, 97, 99].

Zuurbier et al. [100] conducted the largest, population-based investigation of brain structure alterations in sleep apnea, with their study involving 681 patients who were not using CPAP (age range of 51–95). They controlled for an impressive number of covariates: age, gender, intracranial volume, body mass index, education, smoking, alcohol use, diabetes, myocardial infarction, and interval between MRI and PSG (average of 10 months). Neuroimaging measures did not significantly correlate with arousals, and correlated only weakly with apnea-hypopnea index. However, oxygen desaturations (number of drops by 3% per minute) correlated strongly with smaller hippocampal and parietal white matter estimates. Consistent with these desaturation findings, in the Honolulu-Asia Aging Study, lower oxygen saturation levels predicted post-mortem autopsy findings of microinfarcts, Lewy bodies, and gliosis and neuronal loss by approximately 6 years [101]. These results converge with Yaffe et al.'s [102] finding that oxygen desaturations were predictive of developing mild cognitive impairment or dementia whereas arousals and other measures of sleep fragmentation were not predictive.

Several recent studies have evaluated whether sleep apnea affects neural connectivity. Resting state neuroimaging studies have consistently pointed to disruptions in the default mode network and frontoparietal cognitive control network in sleep apnea patients (for review, see 103). In a related approach, diffusion imaging studies reveal widespread alterations in white matter integrity in sleep apnea patients relative to controls, particularly in regions implicated in sleep/wake control and cognitive functioning (bilateral prefrontal cortex, hippocampus, amygdala, insula, cerebellum, medulla, pons, basal forebrain, basal ganglia, right posterior parietal lobe, and right occipital cortex)[104]. Greater apnea-hypopnea index [105], via acute hypoxic injury [106, 107], worsens the negative effects of sleep apnea on white matter integrity.

Neuroimaging Implications for Cognitive Aging and Alzheimer's Disease

The frontal lobe declines substantially with advancing age and this decline impacts an array of cognitive processes and behaviors [108]. Many scientists and practitioners are therefore interested in identifying modifiable factors that affect frontal functioning (e.g., blood pressure [109]). At least ten studies have reported that sleep quantity/quality relates to cortical thinning in the frontal lobe (Table 1). The frontal lobe is implicated in slow wave generation [70], and slow wave generation is implicated in restoring frontal lobe functioning [110]. Thus, regardless of whether changes in slow wave sleep temporally precede frontal lobe thinning (or vice versa), both factors should eventually be detrimentally impacted if either factor is impaired.

Sleep in advancing age is also associated with alterations in the default mode network. As illustrated in Figure 1, cortical thinning in the precuneus, posterior cingulate, inferior parietal lobe, and medial prefrontal cortex were consistently associated with poor sleep quality. These associations converge with experimental evidence that acute sleep deprivation disrupts connectivity in the default mode network. Furthermore, separate lines of research have shown that disruptions in sleep and disruptions in the default mode network are predictive of Alzheimer's disease [59, 68]. Amyloid neuroimaging and animal model studies indicate that the sleep-amyloid association is bidirectional [27]. Greater amyloid deposition in default mode network regions relates to poorer sleep quality and poorer sleep quality can cause greater amyloid deposition [111, 112].

The association between sleep quality and cortical thinning informs some of the cognitive changes observed in "normal" aging (i.e., cognitive changes in the absence of dementia)[1]. For example, as shown in Figure 1, sleep quality was consistently related to thinning in the frontoparietal attention and cognitive control networks [90], including the superior frontal gyrus [50–52, 113], the inferior frontal gyrus [85, 114], and the superior parietal lobe [115, 116]. Deficits in these regions may explain why aging adults who sleep poorly tend to perform worse on some executive functioning and memory tasks [1]. Furthermore, the hippocampus, which is largely responsible for memory encoding and consolidation [53, 117], is susceptible to many health conditions (e.g., ischemia, chronic stress, seizures) [118]. In animal models, sleep loss diminishes hippocampal functioning [119], and in human studies (Table 1), this association was especially evident in patients with obstructive sleep apnea [97–100]. Thus, age-related changes in both the hippocampus and sleep physiology combine to explain why older adults show less memory consolidation than young adults [120].

Neuroimaging Implications for Whether Older Adults Need Sleep

The reduced-sleep-need view contends that neither sleep restriction nor sleep extension impair or improve physical, mental, or cognitive health in older adults (at least relative to young adults). The sleep and neuroimaging correlations reviewed herein disfavor the reduced-sleep-need view, regardless of whether sleep loss causes neural changes [60, 61] or whether neural changes cause sleep loss [47, 71]. Rather than concluding that older adults do not need sleep, if cortical thinning or amyloid deposition are the cause of older adults' poor sleep then a more fitting interpretation is that they have lost the ability to sleep in a restorative manner.

It is also possible that a third variable explains both poor sleep and neural changes with aging (e.g., education, depression, anxiety, cardiovascular dysfunction [121]); however, several neuroimaging studies have controlled for likely third variables and still observed correlations between sleep and cortical thinning. Future studies should disentangle causal direction by treating sleep disturbances in middle age (cognitive-behavioral therapy, continuous positive airway pressure, and/or pharmacological treatments) and assessing whether preserving sleep quality preserves brain health. But, the current literature seems sufficiently strong to produce this take-home message: The "need" for sleep persists into older age.

Conclusion: Are Sleep Changes with Aging Inevitable?

Sleep need persists into older age, but this conclusion may be irrelevant if age effects on sleep quality, quantity, and physiology are inevitable. There are three general points of evidence that adults can sleep well despite advancing age. First, self-report studies show that many older adults report their sleep quality to be good, and sometimes even better than in middle age [122]. Second, in population-based studies, many older adults remain free from sleep disorders such as obstructive sleep apnea [123, 124]. Third, in studies that followed adults aged 61–71 who had excellent physical and mental health, polysomnography features were generally stable over a three-year time period [125]. To definitively answer what percentage of adults demonstrate no marked changes in sleep physiology, a multi-decade longitudinal polysomnographic investigation would be required.

The Global Council on Brain Health recently concluded that “People, at any age, can change their behavior to improve their sleep” [126]. Positive behavior changes include obtaining more daytime light, waking up at the same time *seven* days a week, and consulting a sleep physician if consistently feeling sleepy during the day. Behaviors to avoid include bright light in the evening (e.g., from technology devices), caffeine use after lunch time, too much fluid intake three hours before bedtime, and unlimited use of over-the-counter or prescribed sleep medications. Beyond these recommendations for sleep hygiene, taking care of one’s body through diet [127], exercise [128], and mental engagement [129] helps preserve brain functioning, which would help to preserve sleep quality. Thus, poor sleep quality is not an inevitable consequence of aging, older adults can modify their behaviors to sleep better, and better sleep can have a positive impact on physical, mental, and cognitive health.

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Measurement Abbreviations

ALE	activation likelihood estimation meta-analysis
ESS	Epworth Sleepiness Scale
FA	fractional anisotropy
MD	mean diffusivity
PSQI	Pittsburgh Sleep Quality Index
MCI	Mild Cognitive Impairment
OA	Older adult
OSA	Obstructive Sleep Apnea. SBM: surface-based analysis
VBM	voxel-based morphometry
YA	young adults

FP	frontal pole
GM	gray matter
HC	hippocampus
IFG	inferior frontal gyrus
IOG	inferior occipital gyrus
IOS	inferior occipital sulcus
IPL	inferior parietal lobe
L	left hemisphere
mPFC	medial prefrontal cortex
ITG	inferior temporal gyrus
MFG	middle frontal gyrus
MTG	middle temporal gyrus
OFG	orbitofrontal gyrus
PHG	parahippocampal gyrus
PCC	posterior cingulate cortex
PCUN	precuneus
post-CG	postcentral gyrus
pre-CG	precentral gyrus
R	right hemisphere
SFG	superior frontal gyrus
SFS	superior frontal sulcus
STL	superior temporal lobe
vmPFC	ventromedial prefrontal cortex
WM	white matter

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Figure 1.

Illustration of brain regions that were associated with sleep duration or sleep quality by at least two structural neuroimaging studies. The figure was created using Mango 4.0.1 software (see original sources listed in Table 1 for specific Talairach coordinates). Regions depicted include the hippocampus (red), superior frontal gyrus (green), ventromedial prefrontal cortex (blue), inferior frontal gyrus (orange), precuneus (turquoise), posterior cingulate (pink), inferior parietal lobe (purple), and superior parietal lobe (yellow).

Table 1

Structural neuroimaging studies in relation to sleep measures in adults with a mean age 30 years old. Unless otherwise noted, sleep disturbances are associated with lower volumetric estimates bilaterally.

Paper	Sample		Measurements		Brain Regions Associated with Sleep			
	N	Age Means	MRI	Sleep	Design	Frontal	Parietal	Temporal
Killgore [44]	36	M=30	VBM	ESS	Cross-Sectional	vmPFC		
Carvalho [46]	1,374	M=72	SBA	ESS	Cross-Sectional	Total Frontal	Total Parietal	HC, Total Temporal
Sexton [47]	147	M=54	SBA	PSQI	Cross-Sectional & Longitudinal (3.5 years)	Cluster, R-SFG	Cluster, L-PCUN	Cluster
Branger [49]	50	M=64	VBM	PSQI	Cross-Sectional	R-Insula, R-Putamen		
Lo [55]	66	M=67	SBA	PSQI, TST	Longitudinal (2 years)	Ventricle size increase		
Yaffe [60]	631	M=50	VBM, MD, FA	TST	Longitudinal (5 years)	WM-MD	WM-MD	WM-MD
Spira [61]	122	M=67	SBA	TST	Longitudinal (8 years)	SFG, L-SFS, L-IFG, L-MFG		L-STL
Weber [64]	55	M=31	VBM	TST	Cross-Sectional	vmPFC		
Lauriola [65]	70	M=64	SBA	Home PSG	Cross-Sectional			
Sanchez-Espinosa [66]	42	M=68	SBA	PSG	MCI Case Control	L-PCC, L-PCUN, post-CG		
Fogel [67]	N _{YA} =13, N _{OA} =15	M _{YA} =24 M _{OA} =62	VBM	PSG	Cross-Sectional			
Mander [69]	N _{YA} =16 N _{OA} = 14	M _{YA} =20 M _{OA} = 72	VBM	PSG	Cross-Sectional	mPFC		
Dubé [71]	N _{YA} =30, N _{OA} =33	M _{YA} =23 M _{OA} =60	SBA	PSG	Cross-Sectional	R-MFG	R-IPLe, R-SPL	R-STL, R-ITG
Weng [93]	408	M=47.4		OSA Case-Control ALE meta-analysis on VBM studies		R-SFG		L-MTG, PHG
Baril [96]	71	M=65	VBM, SBA	PSG	OSA Case - Control			
Dusak [97]	42	M=45	Manual	PSG, ESS	OSA Case - Control			HC
Akhan [98]	209	M=52	Manual	PSG, ESS	OSA Case - Control			HC
Sforza [99]	232	M=75	SBA	Home PSG, ESS	OSA Cross-Sectional			HC
Zuurbier [100]	681	M=62	SBA	Home PSG	OSA Cross-Sectional		GM/WM parietal	HC