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Individual Sleep Problems Are Associated With an Accelerated Decline in Multiple Cognitive Functions in Older Adults

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Keywords: cognition disorders | cognitive aging | dementia | insomnia | nonlinear mixed models | older adults | prospective cohort study | sleep initiation and maintenance disorders

ABSTRACT

Poor sleep is a known risk factor of cognitive disorders, but the role of individual sleep problems in age-related cognitive changes remains unclear. This study used two complementary statistical models to estimate nonlinear trajectories of decline in four domains of cognitive functioning in the age period between 55 and 100 years depending on the severity of problems with falling asleep, night awakenings, and early morning awakenings, and short/long sleep duration. The sample included 5132 older adults ($M = 67$ years, 48% male) from the Longitudinal Aging Study Amsterdam (LASA), assessed 4–10 times every 2–3 years. Sleep problems were self-reported, and cognitive functioning was measured with the 15-Word test (reflecting episodic memory as immediate and delayed recall), Coding task (information processing speed) and Mini-mental State Examination/MMSE (global cognition). Data were analysed using quadratic and piecewise changepoint mixed models. The piecewise models provided more precise and interpretable findings. Decline in information processing speed accelerated significantly earlier in participants with short sleep duration (regression coefficient (B) = −2.3[95% confidence interval (CI): −3.86; −0.81]; $p < 0.01$) and faster with more severe early morning awakenings ($B = -0.07$ [−0.1; −0.03]; $p < 0.01$). Decline in immediate recall accelerated earlier in those with short sleep ($B = -2.8$ [−4.44; −1.14]; $p < 0.01$) and severe problems with falling asleep ($B = -1.22$ [−2.06; −0.39]; $p = 0.01$). Decline in delayed recall was faster with long sleep ($B = -0.06$ [−0.08;-0.03]; $p < 0.01$). Decline in global cognition accelerated faster in those with short/long sleep duration ($B = -0.07$ [−0.13; −0.01]/−0.10 [−0.18; −0.03]; $p < 0.01$) and severe night awakenings ($B = -0.04$ [−0.07;−0.02]; $p < 0.01$). To conclude, this study showed that some sleep problems can differentially predict earlier acceleration of deterioration in specific cognitive functions in older adults.

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1 | Introduction

Sleep and cognitive disorders often coexist in later life and both pose a high burden for the aging population (Li, Cao, et al. 2022; Wardle-Pinkston et al. 2019; Casagrande et al. 2022). Recent meta-analyses showed that sleep problems, such as difficulties with starting or maintaining sleep and abnormal sleep duration, can predict all-cause dementia (Li, Cao, et al. 2022; Xu et al. 2020). Such interconnection of sleep and cognitive functioning is unsurprising given that healthy sleep plays an essential role in protecting the brain from cognitive decline through waste clearance, synaptic maintenance, and neuroplasticity (Frank and Heller 2019). However, a considerable number of large-scale studies of the elderly in the general population also found no association between sleep problems and different indicators of cognitive functioning, such as memory dysfunction and risk of dementia (Klaming et al. 2017; Sindi et al. 2018; Beydoun et al. 2021; Selbaek-Tungevåg et al. 2023). The associations between individual sleep problems and specific domains of cognitive functioning are even less clear.

Most prior population-based studies focused on dichotomous measures of global cognition based only on two assessments (e.g., poor sleep at baseline predicting the incidence of cognitive disorder at later age (Sindi et al. 2018; Beydoun et al. 2021; Selbaek-Tungevåg et al. 2023; Lai et al. 2022; Damsgaard et al. 2022; Cavaillès et al. 2022; Bokenberger et al. 2017; Sabia et al. 2021; Lysen et al. 2020; Palpatzis et al. 2021; Westwood et al. 2017; Benito-León et al. 2009)) instead of the trajectories of more subtle age-related changes in individual cognitive domains, which may have a different relationship with sleep behaviour (e.g., information processing speed (Zaheed et al. 2023; Cox et al. 2019; Jaarsma et al. 2024), memory (Klaming et al. 2017; Beydoun et al. 2021; Lai et al. 2022; Zaheed et al. 2023; Cox et al. 2019; Hokett et al. 2021; Owusu et al. 2023)). Those that did inspect the trajectories (Cox et al. 2019; Gildner et al. 2019; Ma et al. 2020; Overton et al. 2024), used exclusively linear models to model cognitive changes across ages, even though multiple longitudinal studies showed that decline in global cognitive functioning typically follows a non-linear course (Payton et al. 2022; Liew 2020; Wilson et al. 2020; Wagner et al. 2024), which starts as gradual deterioration in performance that at some point accelerates toward the end of life (Liew 2020; Wilson et al. 2020; Wagner et al. 2024; Capuano and Wagner 2023). Despite the frequent use of quadratic models to model nonlinear trajectories in earlier research, more sophisticated approaches were shown to be more accurate for modelling age-related cognitive change, such as mixed piecewise regression models with a random change point (Wagner et al. 2024). In addition, specific effects of various sleep problems on cognitive functioning remain largely unknown, given that most research only studied the effects of short/long sleep duration (Sindi et al. 2018; Cavaillès et al. 2022; Bokenberger et al. 2017; Sabia et al. 2021; Lysen et al. 2020; Palpatzis et al. 2021; Westwood et al. 2017; Benito-León et al. 2009; Cox et al. 2019; Gildner et al. 2019; Lutsey et al. 2018; Robbins et al. 2021) and general sleep quality (Sindi et al. 2018; Beydoun et al. 2021; Selbaek-Tungevåg et al. 2023; Lai et al. 2022; Damsgaard et al. 2022; Lysen et al. 2018; Benedict et al. 2015), despite some studies showing that certain sleep problems, such as early morning awakenings, may be stronger predictors of cognitive disorders (Sindi et al. 2018; Selbaek-Tungevåg et al. 2023; Lysen et al. 2020; Robbins et al. 2021; Lysen et al. 2018) or even specific cognitive domains (Wardle-Pinkston et al. 2019; Klaming et al. 2017; Lai et al. 2022; Zaheed et al. 2023; Owusu et al. 2023). Therefore, little is known about the trajectories of decline in different domains of cognitive functioning with aging and how this decline's timing and rate of acceleration may differ in those with various sleep problems.

This study aimed to examine how performance in four indicators of cognitive functioning across three domains (i.e., memory function, processing speed, and global cognitive functioning) changes in participants with various sleep problems (problems with falling asleep, night awakenings, early morning awakenings, and abnormal sleep duration) over a large age period spanning from midlife (55 years) to older ages (up to 100 years). Our findings were based on the data from the Longitudinal Aging Study Amsterdam (LASA), a large prospective cohort of older adults covering a broad range of functional domains and with repeated assessments of sleep parameters and cognitive function across 25 years of follow-up. Trajectories of cognitive changes were modelled using advanced (nonlinear) statistical models, which allowed us to identify the point of acceleration (Wagner et al. 2024; Hall et al. 2000) in the two-phase trajectories of age-related cognitive decline.

2 | Methods

2.1 | Study Participants

Longitudinal Aging Study Amsterdam (LASA) is an ongoing, nationally representative, prospective cohort from the Netherlands that investigates determinants, trajectories and consequences of physical, cognitive, emotional and social functioning in older adults (Huisman et al. 2011). The LASA study received approval by the Ethical Review Board of the VU University Medical Center, Amsterdam, and all participants signed a written informed consent. In 1992, participants were randomly selected from 11 municipal registries within three representative geographic regions (in and around Zwolle, Oss and Amsterdam) in the Netherlands, with an oversampling of older individuals and men. The current study used data collected in 1992–1993 (cohort 1, wave B [$n = 3107$], aged 55–85; cooperation rate 89%), 2002 (cohort 2, wave 2B [$n = 1002$], aged 55–65; cooperation rate 62%), and 2012 (cohort 3, wave 3B [$n = 1023$], aged 55–65; cooperation rate 63%), resulting in 5132 participants whose demographic and health characteristics were measured approximately every 3–4 years for up to 25 years (last wave K in 2021–2022). All data were derived from main and medical interviews (that includes several cognitive tests) and self-administered questionnaires (sleep problems). Participation in the main interview was usually the highest (100% at baseline), followed by participation in the medical interview (86%) and questionnaires (79%). A more detailed description of the LASA study is available in three cohort profile publications (Huisman et al. 2011; Hoogendijk et al. 2020; Hoogendijk et al. 2016).

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2.2 | Measurements

2.2.1 | Cognitive Functioning

At each measurement wave, cognitive functioning was assessed during the clinical interview by trained interviewers using standardised tests that are sensitive in recognising the early stages of decline in the corresponding cognitive indicators (Tsatali et al. 2021; Chun et al. 2021).

Memory function was assessed with the 15 Words Test (Kok 2019; Rey 1958), which was shown to be a valid and reliable ($r = 0.7$ for test–retest, $\alpha = 0.80$ for internal consistency) measure of episodic declarative memory (de Sousa Magalhães et al. 2012). During the test, participants needed to memorise 15 words during three trials. The highest score on one of the three trials reflected immediate recall, and the highest score after 20 min reflected delayed recall. Possible practice effects were minimised by switching between two versions of the test at different measurement waves.

Information processing speed, a cognitive ability that reflects the speed by which elementary cognitive operations can be performed, was measured using an adjusted version of the Alphabet Coding Task-15 (Savage 1984), which is, in turn, an adaptation of the Digit Symbol Substitution Test from the Wechsler Adult Intelligence Scale (AUC = 0.9 for validity in discriminating mild cognitive decline and dementia (Tsatali et al. 2021), $r = 0.7$ for test–retest reliability (Pereira et al. 2015)), which was shown to be very sensitive to aging (Kok 2016; Piccinin and Rabbitt 1999). Participants were presented rows of letters along with a key of corresponding symbols. Using this key, participants needed to mentally substitute another row of symbols with letters as fast as possible. The test was conducted in three cycles, each lasting 1 min. Mean scores from three trials ranged between 1 and 51 points, with higher scores indicating faster processing.

Global cognition was assessed with Mini-Mental State Examination (MMSE) (Folstein et al. 1975), a valid ($r = 0.8$ for concurrent validity (Baek et al. 2016)) and reliable ($r = 0.8$ for test–retest, $\alpha = 0.8$ for internal consistency (Kabátová et al. 2016)) measure of the severity of cognitive impairment in different functions, including orientation in time and place, registration and recall of three words, attention and calculation, language, and visual construction (Folstein et al. 1975). In the cases when the MMSE scores based on the subtraction subtest were missing, they were replaced with a similar test based on spelling. Scores ranged between 0 and 30 points, with a standard cut-off score ≤ 23 points indicating probable dementia.

2.2.2 | Sleep Problems

Participants answered four questions about their sleep behaviour at each measurement wave. Problems with falling asleep, night awakenings, and early morning awakenings were assessed on a severity scale of 1–4 points, corresponding to experiencing these problems "almost never," "sometimes," "frequently," or "almost always." Total scores were calculated by summing up the scores on each of these three sleep problems to reflect the severity of the general sleep disturbance that ranged between 1 and 12 points. Sleep duration was reported as average hours spent asleep on a typical night and then classified based on the American Academy of Sleep Medicine and Sleep Research Society recommendations (Consensus Conference Panel 2015) into three categories of normative (7–8 h; the reference level), short (≤ 6 h), and long (≥ 9 h), so that the analyses could account for the nonlinearity commonly reported for the associations between sleep duration and adverse health outcomes, including cognition (Li, Cao, et al. 2022).

2.2.3 | Demographic and General Health Characteristics

Several demographic and general health characteristics measured at baseline were used to describe the study population. Age at each measurement wave and age at death were indicated in years. Status of deceased was registered as "yes" or "no" by the end of wave K on 15 September 2023. Sex was assigned as male or female. Nationality was classified as Dutch or non-Dutch. Marital status was divided into those married, never married, divorced, or widowed. Housing situation was classified as living independently, in a residential home or monastery, and at a hospital or nursing home. Educational attainment was assessed as the number of years of completed education. Employment status was classified as having or not having a paid job at the time of measurement. Symptoms of depression and anxiety at baseline were measured with the Center for Epidemiological Studies Depression (Radloff 1977) questionnaire and Hospital Anxiety and Depression Scale (Zigmond and Snaith 1983), where scores ranged between 0 and 60 points for depression and 0–21 points for anxiety, with higher values indicating higher symptom severity. Use of psychiatric medication at baseline was classified as user and non-user per medication subgroup that included hypnotics, anxiolytics, and antidepressants. Presence of cardiovascular diseases and diabetes mellitus was estimated by LASA (van Schoor 2021; van Schoor 2020) using self-reported data and medical records, and in this study, presence of disease was classified as "yes" (if estimated as "definite") or "no" (if "no," "possible" or "contradictory"). Subjective pain was self-reported by participants on a scale from no to most severe pain (0–5 points).

2.3 | Participants Eligible for the Analysis

All LASA participants with data on cognitive functioning were considered for the analyses, but up to 803 participants were excluded due to lacking the data related to cognitive functioning and/or sleep problems, leaving around 15,332–19,822 observations per analysis (varying per variable combination). Furthermore, the sample size for the second type of analyses was restricted due to the model's requirement of ≥ 4 observations per person, resulting in the sample size of 2078–2703 (11,213–15,478 observations). The average number of observations per participant in the current study was 4 ± 2 depending on the cognitive and sleep characteristic, or 6 ± 2 in the subsample with ≥ 4 observations.

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2.4 | Statistical Analyses

All analyses were performed in R (R Foundation, and for Statistical Computing, R. 2018) (version v4.3.0). Full script is available on the Open Science Framework (https://osf.io/4m29y).

Two different statistical models were used to investigate the effects of four sleep problems (predictors) on the rate of decline in four cognitive domains (outcomes) throughout aging: a quadratic polynomial mixed model and a piecewise mixed model with a random change point and smooth polynomial transition. Both models (1) estimate nonlinear trajectories when measuring the association between cognitive performance and age, (2) account for individual differences in these trajectories, and (3) allow the inclusion of covariates. As follows from the explanation below, these models have notable strengths and limitations, motivating us to apply both.

2.4.1 | Quadratic Polynomial Linear Mixed Models

Models were fit with the *nlme* (Pinheiro et al. 2023) package. Changes in each of the four indicators of cognitive functioning were predicted by age and each of the sleep characteristics in separate models. Before the analyses, age was centered at 55 years (the lowest age in the sample) to allow the interpretation of the intercept. Each model included random effects of the intercept and age, and a quadratic term (age^2) to allow a parabolic relationship between cognitive functioning scores and age. Statistical significance of the quadratic term indicates the nonlinearity of the association, and its estimate reflects the steepness and direction of the curvature: a positive value corresponds to an upward trend and a negative to a downward trend. The rate of change in scores on each domain of cognitive functioning at a particular age can be estimated using derivatives (a detailed explanation can be found in Table S7). Statistical significance of the differences between the trajectories of cognitive decline depending on the levels of the predictor (i.e., sleep problems) was tested using the Wald test from the *car* (Fox and Weisberg 2024) package. Even participants with one observation add information for model estimation, reducing potential bias in model parameter estimates, and the main advantage of this model was that it could make use of all available data. However, this model (a) assumes a parabolic shape, implying that cognitive functioning peaks at a certain age, as opposed to consistent decline, (b) cannot determine the age at which the multi-phase decline accelerates or which part of the trajectory (phase of decline) significantly differs depending on sleep problems, and (c) cannot summarise the rates of decline in 1–2 estimates (as opposed to calculating separate rates of change at each age), which complicates the interpretation of the findings. To address these limitations, the second model was used.

2.4.2 | Piecewise Mixed Models With a Random Change Point and Smooth Polynomial Transition

Models were fit using the *nlive.pmms* function from the *nlive* (Capuano and Wagner 2023) package. This model assumes that cognitive decline is characterised by two different linear phases and estimates the time of onset of accelerated cognitive decline (Comets et al. 2017; Bacon and Watts 1971). The smooth transition at the change point is modelled using a third-degree polynomial function fitted between two straight lines; this transition is more natural than an abrupt change, and the resulting change point estimate corresponds to the age at the start of this smooth transition (Capuano and Wagner 2023; van den Hout et al. 2011). This model estimates the intercept at time zero (in this case, such an anchoring point was the approximate end of life point at 100 years), the age at which the acceleration of cognitive decline starts (*change point*) and two rates of decline before (*preterminal* decline) and after the acceleration occurred (*terminal* decline). When sleep problems are included as covariates, the model shows if the rates of decline and the change point are significantly different (i.e., if the change point happens significantly earlier/later and the rates of preterminal and terminal decline are slower/faster) for different levels of sleep problems. The main advantage of this model is that it assumes a two-phase trajectory of cognitive decline (i.e., slow decline followed by accelerated decline) that is more in line with reality (Wagner et al. 2024) as well as allows estimating the effect of covariates on different parts of this decline. However, this more precise modelling of change a) requires at least 4 observations per participant, which considerably restricts sample size, and (b) lacks interpretable *p* values for main effects of age, which makes it difficult to ascertain whether the difference in rates of decline is significantly different from 0 and whether a clear change point exists.

Effects were considered statistically significant if *p* values were smaller than 0.05. Clinical significance of the effects was assessed based on the magnitude of the differences indicated by regression coefficients (*B*). In the plots, statistical significance of the predicted group differences was approximated by estimated 95% confidence intervals based on fixed effects from the corresponding model. All *p* values were additionally adjusted for multiple testing using the false discovery rate (Benjamini and Hochberg 1995) method.

We compared both models based on (a) visual inspection of plotted marginal effects (i.e., cognitive decline across age), (b) in terms of estimated rates of change and change points, and c) estimated effects of sleep problems on this decline as indicated by effect sizes and *p* values. If trajectories of cognitive decline did not substantially differ between the two models, we assumed that the findings based on the restricted subsample were valid enough to extrapolate to the whole sample.

As additional sensitivity analyses, we investigated whether the effects of the overall severity of sleep problems on cognitive functioning would be stronger than those of individual sleep problems. In addition, we compared the results of the quadratic models on the whole sample with those on the subsample with ≥4 observations.

2.4.3 | Comparison of the Whole Sample to the Subsample With ≥4 Observations

We tested whether the subsample used in the second model was biased by comparing participants with ≥4 observations to

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those with < 4 observations in various demographic, sleep, and cognitive characteristics using *t*-test for continuous variables and *chi*-square for categorical variables. Magnitude of differences was reflected by Cohen's d ($d \leq 0.2$ indicating small, $d = 0.5–0.8$ moderate, and $d > 0.8$ large differences), Cramer's V ($V \leq 0.2$ small, $V = 0.2–0.6$ moderate, and $V \geq 0.6$ large differences), or odds ratio (OR), and statistical significance was indicated by p values. The comparison groups were those with and without ≥ 4 assessments on night awakenings and delayed recall—the smallest sample used in the piecewise regression analysis, which potentially had the largest differences from the whole sample. Additionally, we tested whether some differences between these subgroups remained after controlling for the age at baseline, age at death (a proxy of longer lifespan), and presence of cardiovascular diseases (a proxy of physical health) using multiple linear regression. This approach could approximate bias due to differences between the whole sample and its subsample with ≥ 4 assessments, but these differences could not be directly tested due to the whole and subsamples' interdependency.

3 | Results

3.1 | Sample Characteristics

Across three LASA cohorts, mean age at baseline was 67 ± 9 years, ranging between 55 and 86 years (Table 1). Almost half of the participants were male (48%). At baseline, most participants reported Dutch nationality (99%), attained an average of 10 years of education, lived independently (98%), were married (67%) or widowed (18%), were not in paid employment (69%), and had low depression and anxiety. A small proportion had probable dementia based on their MMSE scores (12%) and used hypnotics, antidepressants, or anxiolytics (2%–4%). Baseline scores on three individual sleep problems were positively and moderately correlated with each other (*rho* = 0.4–0.6), weakly and negatively with sleep duration (*rho* = 0.3–0.4) and very weakly or not correlated (*rho* = 0–0.1) with scores on cognitive functioning (Figure S1).

The subsample with ≥ 4 observations that was used in piecewise models differed from those with < 4 observations (Table S1), as it included participants who were considerably younger ($d = 0.9$) and died at an older age ($d = 0.3$), with correspondingly twice higher chances of being in paid employment (OR = 2.2), almost twice less likely to use hypnotics/anxiolytics (OR = 0.5) or have a cardiovascular disease (OR = 0.6), were less depressed ($d = 0.3$), and less impaired in their cognitive functioning ($d = 0.5–0.7$) and five times less likely to have probable dementia (OR = 0.2). Most of these differences disappeared when controlling the models for age at baseline, except for cognitive functioning that was higher at baseline in the subsample with ≥ 4 observations irrespective of age, even when controlling for age at death and cardiovascular diseases.

3.2 | Changes in Cognitive Functioning Throughout Aging

Visual inspection of the data indicated that in most participants, scores on all four domains of cognitive functioning steadily declined throughout ageing, with an acceleration around the age period of 70–80 years (Figure S2).

3.2.1 | Quadratic Polynomial Mixed Models

For all cognitive indicators, the quadratic term (age^2) was negative and statistically significant (Table S2), indicating acceleration of cognitive decline with older age.

Estimates from this model indicated that performance on immediate and delayed recall somewhat increased before the age of 60 years and after a period of no change, started to decline in the period of 65–70 years with a similar rate that steadily accelerated from 0.1 to 0.3 words per age by 95 years (Figure 1A,C). Information processing speed started to decline the earliest, in the age period of 55–60 years, and accelerated from 0.1 to 0.9 points per year by 95 years (Figure 1E). Global cognition somewhat increased before the age of 65 years and then started to decline in the period between 65 and 70 years, with an accelerating rate from 0.1 to 0.5 points per year by 95 years (Figure 1G). Estimates of the rates of change in cognitive functioning in the period between 55 and 95 years can be found in Table S7.

A considerable proportion of variance was explained by the nested structure in the data (i.e., measurement occasions nested within individuals), with intraclass correlation coefficient values ranging between 0.60 and 0.88. In all four domains, individual intercepts and slopes for age differed from the general trend (Figure S4 and Table S2). Individual trajectories in cognitive decline are illustrated in Figure S3.

3.2.2 | Piecewise Linear Mixed Models

Scores on the immediate recall test slightly decreased with a preterminal rate of −0.02 words per year up to the change point at 74 years, after which the decline accelerated to −0.2 words per year (Figure 1B). Scores on delayed recall did not change ($B = 0.0$) until the age of 73 years, after which the decline occurred by −0.2 words per year (Figure 1D). Information processing speed started to decline from the start point of 55 years ($B = -0.2$) and after 79 years accelerated to −0.7 points per year (Figure 1F). Preterminal decline in global cognition ($B = -0.1$) accelerated to −0.3 points per year after the age of 83 years (Figure 1H). Intercept (i.e., endpoint scores), rates of decline, and age at change point considerably varied between participants (Figure S5). Detailed model output can be found in Table 2.

3.3 | Associations Between Sleep Problems and Cognitive Decline

3.3.1 | Quadratic Polynomial Mixed Models

The trajectories of cognitive decline in immediate recall and delayed recall were significantly different in participants with long (≥ 9 h) as opposed to normative (7–8 h) sleep duration (Figure 2A,C). The trajectories of cognitive decline in information processing speed were significantly different in participants

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TABLE 1 | Sociodemographic, cognitive and sleep characteristics of the participants at baseline in the Longitudinal Aging Study Amsterdam.

	Overall ($N = 5132$)
Age	
Mean ± SD [min; max]	66.6 ± 8.8 [54.8;85.6]
Sex	
Male	2477 (48.3%)
Female	2655 (51.7%)
Deceased[a]	
No	1885 (36.7%)
Yes	3228 (62.9%)
Missing	19 (0.4%)
Age at death[b]	
Mean ± SD [min; max]	83.3 ± 8.6 [55.6;106.3]
Missing	1904 (37.1%)
Nationality	
Dutch	5069 (98.8%)
Non-Dutch	63 (1.2%)
Marital status	
Never married	373 (7.3%)
Married or registered partnership	3445 (67.1%)
Divorced	380 (7.4%)
Widowhood	934 (18.2%)
Housing situation	
Independent	5002 (97.5%)
Residential home or monastery	104 (2.0%)
Hospital or nursing home	25 (0.5%)
Missing	1 (0.0%)
Educational attainment (years)	
Mean ± SD [min; max]	9.6 ± 3.6 [5.0;18.0]
Missing	8 (0.2%)
Employment status	
No	3550 (69.2%)
Yes	1406 (27.4%)
Missing	176 (3.4%)
Immediate recall, max score (15 Word Test) (1–15 words)	
Mean ± SD [min; max]	8.3 ± 2.6 [0.0;15.0]
Missing	730 (14.2%)
Delayed recall (15 Word Test) (0–15 words)	
Mean ± SD [min; max]	5.7 ± 2.9 [0.0;15.0]
Missing	751 (14.6%)
Information processing speed (Coding task) (1–51 points)	
Mean ± SD [min; max]	26.3 ± 7.6 [3.0;51.3]
Missing	766 (14.9%)
Global cognition (Mini-Mental State Examination) (1–30 points)	
Mean ± SD [min; max]	26.8 ± 3.1 [2.0;30.0]
Missing	21 (0.4%)
Sleep duration	
7–8 h	2671 (52.0%)
≤ 6 h	717 (14.0%)
≥ 9 h	601 (11.7%)
Missing	1143 (22.3%)
Problems falling asleep	
Almost never	1582 (30.8%)
Sometimes	1803 (35.1%)
Frequently	495 (9.6%)
Almost always	188 (3.7%)
Missing	1064 (20.7%)
Night awakenings	
Almost never	1402 (27.3%)
Sometimes	1713 (33.4%)
Frequently	666 (13.0%)
Almost always	283 (5.5%)
Missing	1068 (20.8%)
Early morning awakenings	
Almost never	1347 (26.2%)
Sometimes	1672 (32.6%)
Frequently	722 (14.1%)
Almost always	325 (6.3%)
Missing	1066 (20.8%)
Depression	
Mean ± SD [min; max]	7.8 ± 7.7 [0.0;54.0]
Missing	65 (1.3%)

(Continues)

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TABLE 1 | (Continued)

	Overall ($N = 5132$)
Anxiety	
Mean ± SD [min; max]	2.6 ± 3.3 [0.0;21.0]
Missing	1197 (23.3%)
Use of hypnotics (benzodiazepines)	
No	4200 (81.8%)
Yes	279 (5.4%)
Missing	653 (12.7%)
Use of anxiolytics (benzodiazepines)	
No	4251 (82.8%)
Yes	228 (4.4%)
Missing	653 (12.7%)
Use of antidepressants	
No	4328 (84.3%)
Yes	151 (2.9%)
Missing	653 (12.7%)
Probable dementia	
No	4505 (87.8%)
Yes	606 (11.8%)
Missing	21 (0.4%)
Cardiovascular disease	
No	2827 (55.1%)
Yes	162 (3.2%)
Missing	2143 (41.8%)
Diabetes mellitus	
No	5035 (98.1%)
Yes	82 (1.6%)
Missing	15 (0.3%)
Subjective pain	
Mean ± SD [min; max]	0.7 ± 1.3 [0.0;5.0]
Missing	3090 (60.2%)

Note: For continuous variables, the estimates represent the mean ± standard deviation (SD) and the minimum and maximum values as [min; max]. For categorical variables, the estimates represent the absolute number and proportion of the sample (in parentheses).
Abbreviation: N = sample size.
[a]Status of being deceased by August 15, 2021 (Wave K).
[b]These data concern 63% of all LASA participants who died before the last measurement wave used in this study.

with long sleep duration and more severe problems with falling asleep, night awakenings, and early morning awakenings (Figures 2–5E). The trajectories of cognitive decline in global cognition were significantly different in participants with short (≤ 6 h) sleep duration and more severe problems with falling asleep, night awakenings, and early morning awakenings (Figures 2–5G). The results from the analyses on the sample with ≥ 4 observations were similar, except the effects of sleep duration on immediate recall and global cognition were no longer significant. Detailed model output and the estimated rates of change can be found in Tables S3–S7.

3.3.2 | Piecewise Linear Mixed Models

Compared to participants with normative sleep duration, the decline in immediate recall accelerated significantly earlier in participants with short sleep duration ($B = -2.79$; $p < 0.01$) and was generally faster in those with long sleep duration, reaching a significantly lower score at 100 years ($B = -1.08$; $p = 0.02$). In addition, the decline in immediate recall accelerated significantly earlier (B = −1.22; $p = 0.01$), but occurred somewhat slower ($B = 0.02$; $p = 0.01$), reaching a higher end score at 100 years in participants with more severe problems with falling asleep compared to those with no or less severe problems ($B = 0.36$; $p = 0.02$).

The decline in delayed recall started significantly faster in participants with long sleep duration ($B = -0.06$; $p < 0.01$), and despite somewhat later acceleration ($B = 4.29$; $p < 0.01$), reached significantly lower scores at 100 years ($B = -1.39$; $p = 0.02$) than in those with normative sleep duration.

The decline in information processing speed accelerated faster ($B = -2.34$; $p = 0.01$) in participants with short sleep duration and somewhat earlier in participants with more severe night awakenings ($B = -0.81$; $p = 0.02$). In participants with more severe early morning awakenings, information processing speed occurred somewhat slower in the preterminal phase ($B = 0.02$; $p = 0.03$) but faster in the terminal phase ($B = -0.07$; $p < 0.01$), reaching lower scores at 100 years ($B = -2.05$; $p < 0.01$).

The rate of terminal decline in global cognition was faster in participants with short ($B = -0.07$; $p = 0.02$) and long ($B = -0.10$; $p = 0.01$) sleep duration, reaching lower scores at 100 years in those with both short ($B = -1.41$; $p < 0.01$) and long ($B = 2.22$; $p < 0.01$) sleep duration. Terminal decline in global cognition was also slightly faster in those with more severe problems with night awakenings ($B = -0.04$; $p < 0.01$), reaching lower scores at 100 years ($B = -0.83$; $p < 0.01$) (Figure 4H). In addition, global cognition at 100 years was significantly worse for those with more severe early morning awakenings ($B = -0.55$; $p < 0.01$).

Detailed model output can be found in Tables 3–6. All associations are visualised in Figures 2–5.

3.3.3 | Additional Analyses

The trajectories of decline in information processing speed and global cognition were significantly different in the quadratic model in participants with higher cumulative sleep problems. Piecewise models similarly showed a faster decline in these two indicators that reached lower end point scores ($B = -0.4$; $p = 0.02$ and $B = -0.3$; $p < 0.01$, respectively; Figure S6 and Tables S8 and S9), but only the effect on global cognition in the quadratic model was somewhat stronger than that of individual sleep problems.

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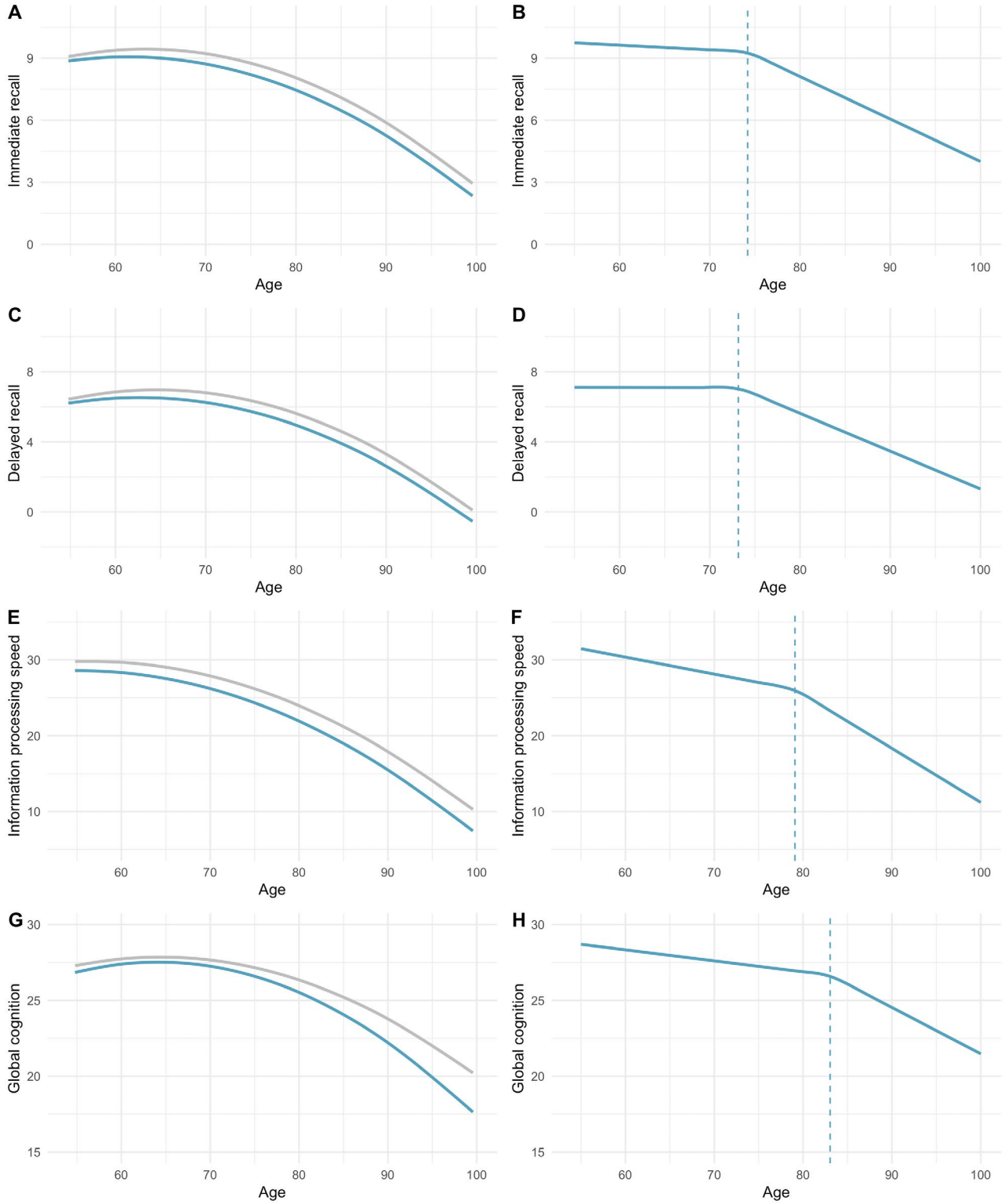

FIGURE 1 | Changes in cognitive functioning throughout aging. Coloured lines show predicted rates of decline in the scores on immediate recall (A, B), delayed recall (C, D), information processing speed (E, F), and global cognition (G, G) as estimated by quadratic polynomial mixed models (on the left) and piecewise mixed models with a random changepoint (on the right). Grey lines show the quadratic trajectories in the subsample with ≥ 4 observations per participant that was used for the piecewise models. Vertical dashed lines show the age at the changepoint.

3.3.4 | Comparison of the Two Models

The changes in cognitive functioning over time overall agreed when estimated by piecewise and quadratic models (as compared in Figures 1–4), both showing a progressive, accelerating decline in performance over time. Most cognitive trajectories were affected by sleep problems in both models, but the way these models estimated the shapes of these trajectories considerably differed. Although both models seemed to differentiate two phases of cognitive decline, the division into phases was

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TABLE 2 | Changes in cognitive functioning throughout aging as estimated by piecewise mixed models with a random changepoint.

	Immediate recall	Delayed recall	Information processing speed	Global cognition
Subjects	2285	2263	2265	2703
Observations	12,592	12,424	12,476	15,478
Fixed effects				
Score at 100 years	4.01 [3.77; 4.24]	1.31 [1.04; 1.58]	11.21 [10.6; 11.81]	21.48 [21.14; 21.82]
Preterminal decline	−0.02 [−0.03; −0.01]	0 [−0.01; 0.01]	−0.22 [−0.23; −0.21]	−0.07 [−0.08; −0.07]
Terminal decline	−0.21 [−0.22; −0.19]	−0.22 [−0.23; −0.2]	−0.71 [−0.75; −0.68]	−0.31 [−0.33; −0.28]
Change point	−25.8 [−26.41; −25.2]	−26.84 [−27.39; −26.29]	−20.92 [−21.43; −20.41]	−16.96 [−17.26; −16.66]
Error	1.63	1.90	2.52	1.74
Random effects				
$\omega 2_{\text{Score at 100 years}}$	2.42 [1.96; 2.87]	3.02 [2.41; 3.63]	24.42 [20.74; 28.09]	1.57 [0.86; 2.27]
$\omega 2_{\text{Preterminal decline}}$	0 [0; 0.01]	0.01 [0.01; 0.01]	0.03 [0.02; 0.03]	0.02 [0.01; 0.02]
$\omega 2_{\text{Terminal decline}}$	0 [0; 0]	0 [0; 0.01]	0.05 [0.04; 0.06]	0.03 [0.02; 0.03]
$\omega 2_{\text{Change point}}$	15.68 [10.7; 20.66]	16.37 [12.11; 20.62]	26.17 [21.86; 30.47]	3.29 [1.77; 4.81]
$\text{Covariance}_{\text{Preterminal decline/Terminal decline}}$	0 [0; 0]	−0.01 [−0.01; 0]	−0.03 [−0.04; −0.03]	−0.02 [−0.02; −0.02]

Note: The numbers represent regression coefficients (for fixed effects, *B*) or variance (for random effects, $\omega 2$) with the corresponding 95% confidence intervals (in brackets) for all model terms (rows) in each of the four statistical models (columns).

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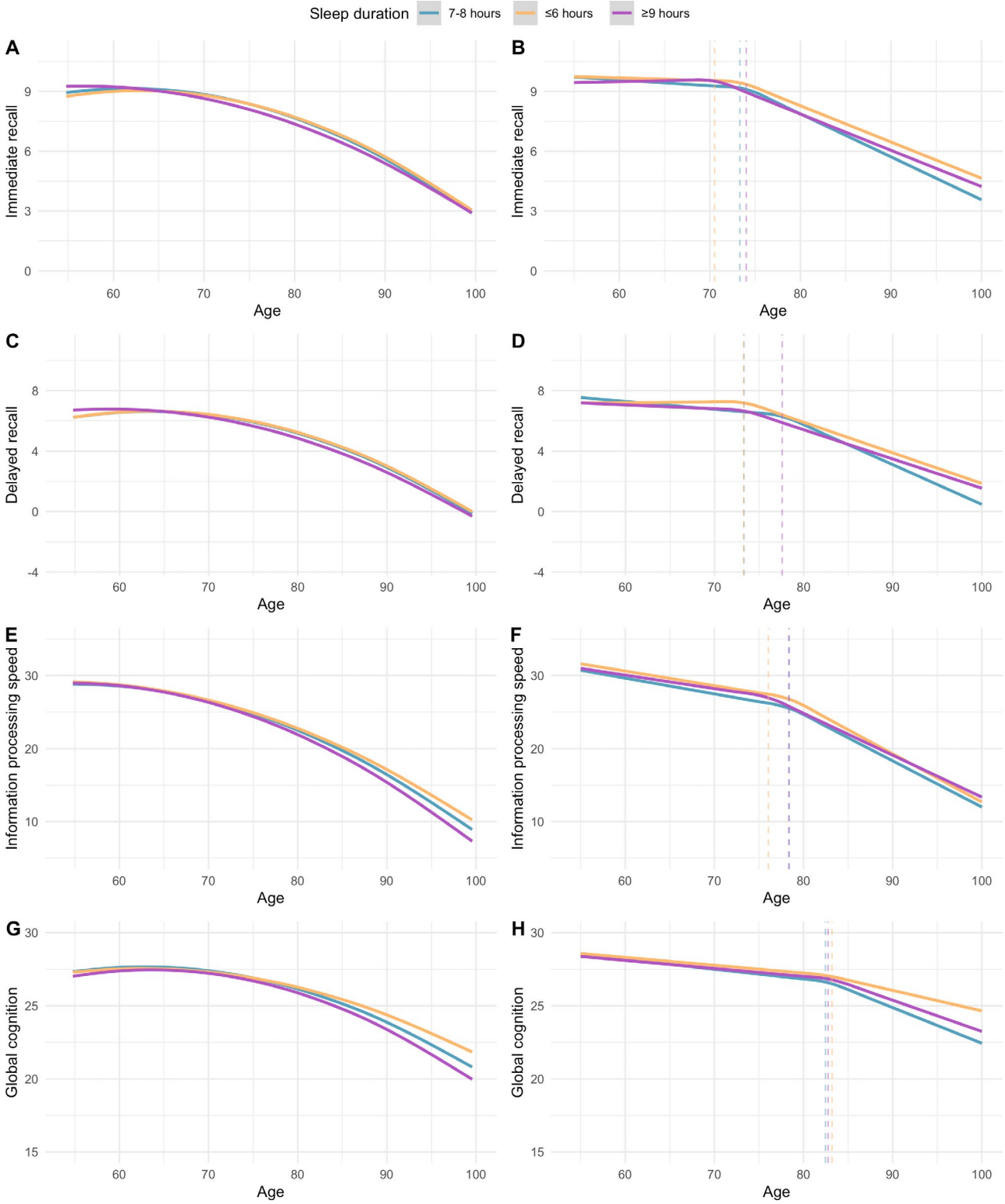

FIGURE 2 | Changes in cognitive functioning throughout aging depending on sleep durationColoured lines show predicted rates of decline in the scores on immediate recall (A, B), delayed recall (C, D), information processing speed (E, F), and global cognition (G, H) as estimated by quadratic polynomial mixed models (on the left) and piecewise mixed models with a random changepoint (on the right). Colour of the line represents corresponding categories as indicated by the legend. Vertical dashed lines show the age at the change point.

constrained by the parabolic shape of the quadratic model, not allowing the independent decline trajectories during the earlier and later phases of aging as estimated in the piecewise model, which was the likely reason for the reversal of the association (crossing of the lines) in the later phase, such as the shift to a faster decline in information processing speed

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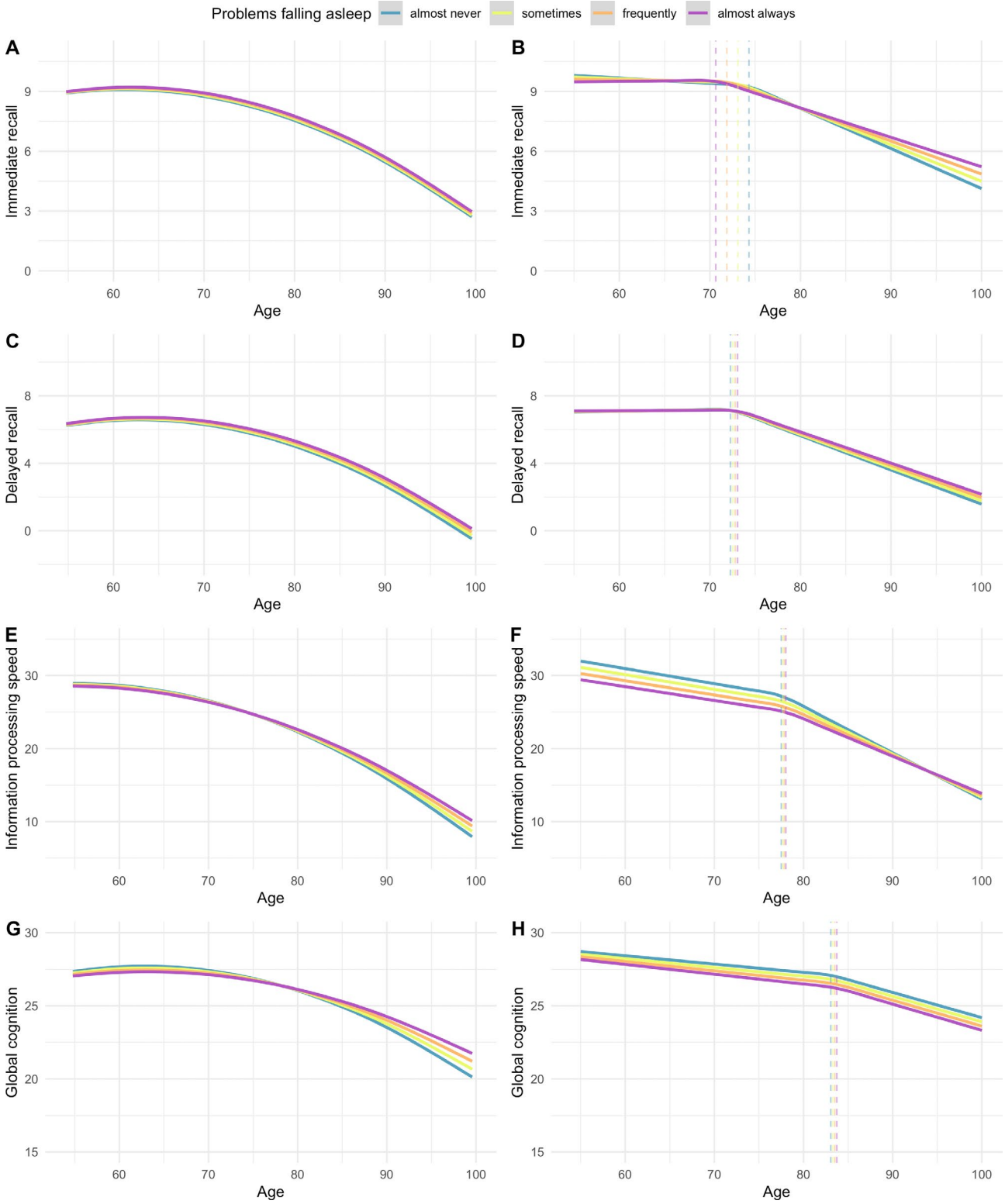

FIGURE 3 | Changes in cognitive functioning throughout ageing depending on problems with falling asleep. Coloured lines show predicted rates of decline in the scores on immediate recall (A, B), delayed recall (C, D), information processing speed (E, F), and global cognition (G, H) as estimated by quadratic polynomial mixed models (on the left) and piecewise mixed models with a random changepoint (on the right). Colour of the line represents corresponding categories as indicated by the legend. Vertical dashed lines show the age at change point.

and global cognition in those with *less* severe sleep problems. Furthermore, the initial plateau (absence of decrease) before a steep decline in the terminal phase was estimated as temporary improvement in performance, which is inconsistent with the natural course of cognitive changes in the elderly (Payton et al. 2022; Liew 2020; Wilson et al. 2020; Wagner et al. 2024). Furthermore, piecewise change point models enforced a change point even when the decline accelerated

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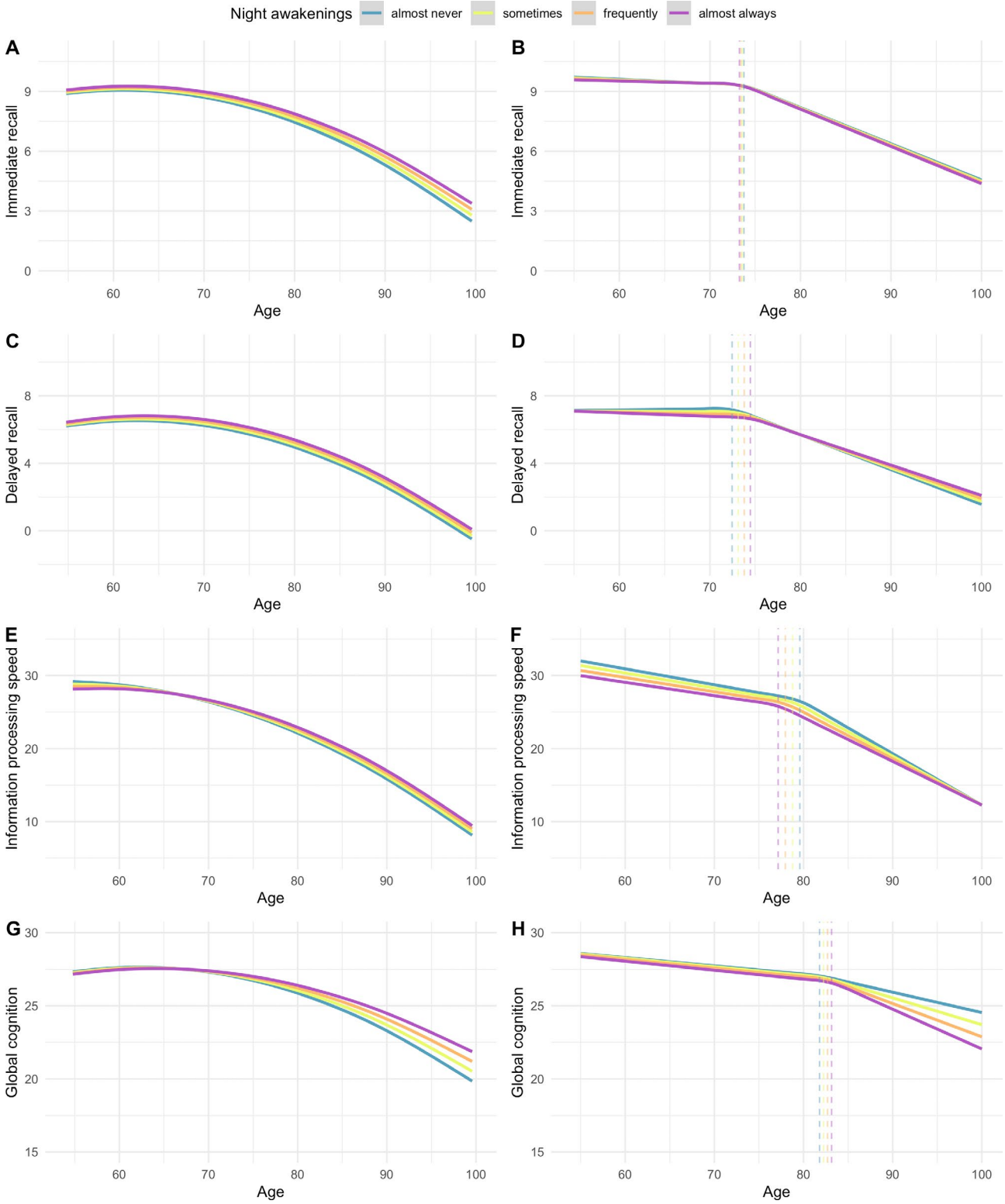

FIGURE 4 | Changes in cognitive functioning throughout aging depending on problems with night awakenings. Coloured lines show predicted rates of decline in the scores on immediate recall (A, B), delayed recall (C, D), information processing speed (E, F), and global cognition (G, H) as estimated by quadratic polynomial mixed models (on the left) and piecewise mixed models with a random changepoint (on the right). Colour of the line represents corresponding categories as indicated by the legend. Vertical dashed lines show the age at change point.

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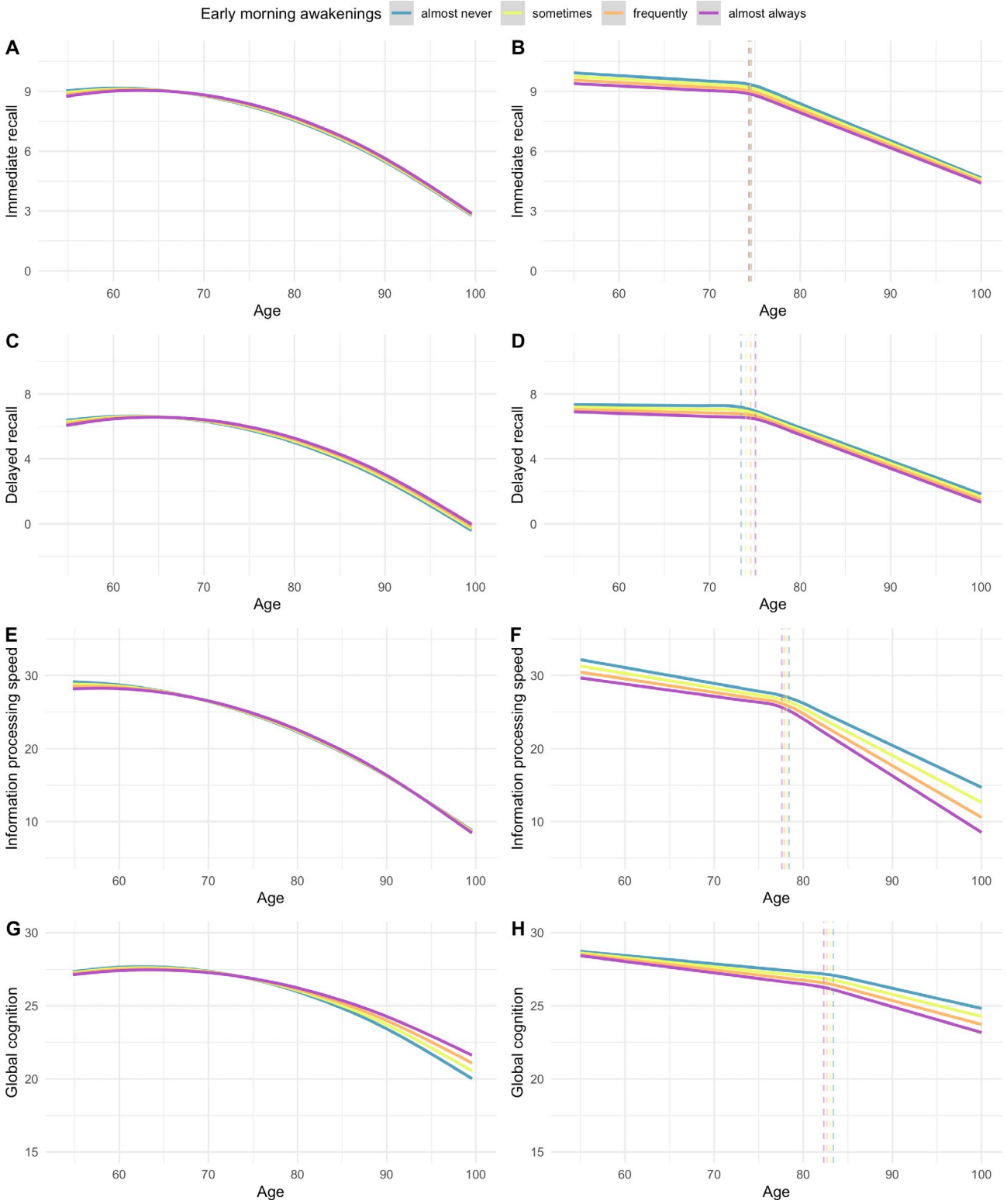

FIGURE 5 | Changes in cognitive functioning throughout aging depending on problems with early morning awakenings. Coloured lines show predicted rates of decline in the scores on immediate recall (A, B), delayed recall (C, D), information processing speed (E, F), and global cognition (G, H) as estimated by quadratic polynomial mixed models (on the left) and piecewise mixed models with a random changepoint (on the right). Colour of the line represents corresponding categories as indicated by the legend. Vertical dashed lines show the age at change point.

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TABLE 3 | Changes in cognitive functioning throughout aging depending on sleep duration as estimated by piecewise mixed models with a random changepoint.

	Immediate recall	Delayed recall	Information processing speed	Global cognition
Subjects	2095	2079	2086	2300
Observations	11,341	11,213	11,301	13,028
Fixed effects				
Score at 100 years	4.64 [4.34; 4.95]	1.86 [1.51; 2.21]	12.69 [11.95; 13.43]	24.65 [24.23; 25.07]
Short sleep (Score at 100 years)	−0.42 [−1.03; 0.2] $^{0.25}$	−0.32 [−1.09; 0.45] $^{0.54}$	0.67 [−0.86; 2.2] $^{0.57}$	**−1.41 [−2.29; −0.53]** $^{<0.01}$
Long sleep (Score at 100 years)	**−1.08 [−1.86; −0.3]** $^{0.02}$	**−1.39 [−2.47; −0.31]** $^{0.02}$	−0.69 [−2.58; 1.19] $^{0.57}$	**−2.22 [−3.27; −1.17]** $^{<0.01}$
Preterminal decline	−0.01 [−0.02; 0]	0 [−0.01; 0.02]	−0.2 [−0.21; −0.18]	−0.05 [−0.06; −0.05]
Short sleep (Preterminal decline)	0.02 [−0.01; 0.05] $^{0.19}$	−0.03 [−0.06; 0] $^{0.05}$	0.01 [−0.02; 0.05] $^{0.57}$	0 [−0.02; 0.02] $^{0.92}$
Long sleep (Preterminal decline)	−0.02 [−0.05; 0.01] $^{0.34}$	**−0.06 [−0.08; −0.03]** $^{<0.01}$	−0.02 [−0.06; 0.03] $^{0.57}$	−0.01 [−0.03; 0.01] $^{0.26}$
Terminal decline	−0.18 [−0.2; −0.17]	−0.2 [−0.22; −0.19]	−0.66 [−0.7; −0.62]	−0.14 [−0.17; −0.11]
Short sleep (Terminal decline)	0 [−0.03; 0.03] $^{1.00}$	0.01 [−0.03; 0.04] $^{0.69}$	0.09 [0.01; 0.16] $^{0.06}$	**−0.07 [−0.13; −0.01]** $^{0.02}$
Long sleep (Terminal decline)	−0.03 [−0.07; 0] $^{0.13}$	−0.06 [−0.12; 0] $^{0.07}$	0.03 [−0.08; 0.13] $^{0.66}$	**−0.1 [−0.18; −0.03]** $^{0.01}$
Change point	−26.69 [−27.48; −25.91]	−26.71 [−27.45; −25.97]	−21.61 [−22.29; −20.92]	−17.54 [−18.04; −17.04]
Short sleep (Change point)	**−2.79 [−4.44; −1.14]** $^{<0.01}$	−0.02 [−1.89; 1.85] $^{0.98}$	**−2.34 [−3.86; −0.81]** $^{0.01}$	0.73 [−0.31; 1.78] $^{0.22}$
Long sleep (Change point)	0.71 [−1.25; 2.66] $^{0.52}$	**4.29 [2.18; 6.4]** $^{<0.01}$	−0.05 [−1.81; 1.71] $^{0.96}$	0.3 [−0.9; 1.49] $^{0.68}$
Error	1.63	1.88	2.48	1.59
Random effects				
$\omega 2_{\text{Score at 100 years}}$	2.28 [1.82; 2.74]	2.91 [2.26; 3.57]	24.86 [21.27; 28.44]	1.09 [0.53; 1.66]
$\omega 2_{\text{Preterminal decline}}$	0 [0; 0.01]	0 [0; 0.01]	0.03 [0.02; 0.03]	0.01 [0.01; 0.01]
$\omega 2_{\text{Terminal decline}}$	0 [0; 0]	0 [0; 0.01]	0.03 [0.02; 0.04]	0.02 [0.01; 0.02]
$\omega 2_{\text{Change point}}$	13.47 [8.09; 18.84]	16.96 [11.59; 22.34]	30.92 [25.68; 36.15]	11.22 [7.92; 14.53]
$\text{Covariance}_{\text{Preterminal decline/Terminal decline}}$	0 [0; 0]	0 [0; 0]	−0.03 [−0.03; −0.02]	−0.01 [−0.02; −0.01]

Note: The numbers represent regression coefficients (for fixed effects, *B*) or variance (for random effects, $\omega 2$) with the corresponding 95% confidence intervals (in brackets) and *p* values, adjusted for multiple testing (in superscript), for all model terms (rows) in each of the four statistical models (columns). Statistically significant effects are highlighted in bold.

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TABLE 4 | Changes in cognitive functioning throughout ageing depending on problems with falling asleep as estimated by piecewise mixed models with a random changepoint.

	Immediate recall	Delayed recall	Information processing speed	Global cognition
Subjects	2123	2106	2114	2333
Observations	11,526	11,394	11,482	13,246
Fixed effects				
Score at 100 years	3.76 [3.13; 4.4]	1.38 [0.66; 2.1]	12.83 [11.3; 14.37]	24.47 [23.56; 25.39]
Problems with falling asleep (Score at 100 years)	**0.36 [0.06; 0.67]$^{0.02}$**	0.2 [−0.16; 0.55]$^{0.57}$	0.25 [−0.52; 1.02]$^{0.59}$	−0.29 [−0.73; 0.16]$^{0.32}$
Preterminal decline	−0.04 [−0.06; −0.02]	0.01 [−0.01; 0.04]	−0.21 [−0.25; −0.18]	−0.05 [−0.07; −0.04]
Problems with falling asleep (Preterminal decline)	0.01 [0; 0.02]$^{0.09}$	0 [−0.02; 0.01]$^{0.71}$	0.01 [−0.01; 0.02]$^{0.59}$	0 [−0.01; 0.01]$^{0.50}$
Terminal decline	−0.22 [−0.25; −0.19]	−0.21 [−0.24; −0.18]	−0.67 [−0.76; −0.59]	−0.17 [−0.23; −0.11]
Problems with falling asleep (Terminal decline)	**0.02 [0; 0.03]$^{0.02}$**	0.01 [−0.01; 0.02]$^{0.58}$	0.04 [0; 0.08]$^{0.08}$	0 [−0.03; 0.03]$^{0.89}$
Change point	−24.47 [−26.09; −22.84]	−27.99 [−29.46; −26.52]	−22.67 [−24.06; −21.28]	−17.17 [−18.13; −16.2]
Problems with falling asleep (Change point)	**−1.22 [−2.06; −0.39]$^{0.01}$**	0.26 [−0.51; 1.03]$^{0.58}$	0.17 [−0.56; 0.9]$^{0.65}$	0.22 [−0.25; 0.7]$^{0.48}$
Error	1.63	1.87	2.49	1.60
Random effects				
$\omega 2_{\text{Score at 100 years}}$	2.18 [1.72; 2.63]	3.04 [2.4; 3.67]	24.28 [20.73; 27.83]	1.13 [0.51; 1.75]
$\omega 2_{\text{Preterminal decline}}$	0 [0; 0.01]	0.01 [0; 0.01]	0.03 [0.03; 0.04]	0.01 [0.01; 0.01]
$\omega 2_{\text{Terminal decline}}$	0 [0; 0]	0 [0; 0.01]	0.04 [0.03; 0.05]	0.02 [0.02; 0.02]
$\omega 2_{\text{Change point}}$	15.02 [9.45; 20.58]	14.89 [10.24; 19.54]	23.27 [18.55; 28]	9.71 [6.65; 12.78]
$\text{Covariance}_{\text{Preterminal decline/Terminal decline}}$	0 [0; 0]	0 [−0.01; 0]	0.03 [0.03; 0.04]	−0.02 [−0.02; −0.01]

Note: The numbers represent regression coefficients (for fixed effects, *B*) or variance (for random effects, $\omega 2$) with the corresponding 95% confidence intervals (in brackets) and *p* values, adjusted for multiple testing (in superscript), for all model terms (rows) in each of the four statistical models (columns). Statistically significant effects are highlighted in bold.

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TABLE 5 | Changes in cognitive functioning throughout aging depending on problems with night awakenings as estimated by piecewise mixed models with a random changepoint.

	Immediate recall	Delayed recall	Information processing speed	Global cognition
Subjects	2094	2078	2085	2303
Observations	11,393	11,265	11,351	13,110
Fixed effects				
Score at 100 years	4.62 [4; 5.24]	1.38 [0.68; 2.09]	12.27 [10.73; 13.8]	25.36 [24.54; 26.19]
Night awakenings (Score at 100 years)	−0.06 [−0.35; 0.22] $^{0.77}$	0.18 [−0.16; 0.51]$^{0.30}$	0 [−0.69; 0.69] $^{1.00}$	**−0.83 [−1.21; −0.45]$^{<0.01}$**
Preterminal decline	−0.02 [−0.05; 0]	0.02 [−0.01; 0.04]	−0.23 [−0.26; −0.2]	−0.05 [−0.07; −0.04]
Night awakenings (Preterminal decline)	0 [−0.01; 0.01] $^{0.77}$	−0.01 [−0.02; 0] $^{0.20}$	0.01 [0; 0.03]$^{0.13}$	0 [−0.01; 0.01] $^{0.67}$
Terminal decline	−0.18 [−0.21; −0.15]	−0.22 [−0.25; −0.18]	−0.74 [−0.83; −0.65]	−0.1 [−0.15; −0.04]
Night awakenings (Terminal decline)	0 [−0.02; 0.01]$^{0.80}$	0.01 [−0.01; 0.02]$^{0.30}$	0.04 [0; 0.07]$^{0.09}$	**−0.04 [−0.07; −0.02]$^{<0.01}$**
Change point	−26.07 [−27.71; −24.44]	−28.22 [−29.72; −26.73]	−19.61 [−20.99; −18.22]	−18.66 [−19.64; −17.67]
Night awakenings (change point)	−0.17 [−0.93; 0.59]$^{0.77}$	0.67 [−0.07; 1.42]$^{0.15}$	**−0.81 [−1.46; −0.16]$^{0.02}$**	0.45 [0.01; 0.89]$^{0.05}$
Error	1.64	1.88	2.50	1.61
Random effects				
$\omega 2_{\text{Score at 100 years}}$	2.32 [1.85; 2.79]	3.17 [2.52; 3.82]	24.7 [20.99; 28.4]	1.24 [0.66; 1.83]
$\omega 2_{\text{Preterminal decline}}$	0 [0; 0.01]	0.01 [0; 0.01]	0.03 [0.02; 0.03]	0.01 [0.01; 0.01]
$\omega 2_{\text{Terminal decline}}$	0 [0; 0]	0 [0; 0]	0.04 [0.03; 0.05]	0.02 [0.01; 0.02]
$\omega 2_{\text{Change point}}$	13.07 [7.23; 18.9]	12.35 [7.43; 17.27]	32.02 [26.75; 37.29]	10.37 [7.16; 13.57]
$\text{Covariance}_{\text{Preterminal decline/Terminal decline}}$	0 [0; 0]	0 [−0.01; 0]	−0.03 [−0.03; −0.02]	1.24 [0.66; 1.83]

Note: The numbers represent regression coefficients (for fixed effects, *B*) or variance (for random effects, $\omega 2$) with the corresponding 95% confidence intervals (in brackets) and *p* values, adjusted for multiple testing (in superscript), for all model terms (rows) in each of the four statistical models (columns). Statistically significant effects are highlighted in bold.

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TABLE 6 | Changes in cognitive functioning throughout aging depending on problems with early morning awakenings as estimated by piecewise mixed models with a random changepoint.

	Immediate recall	Delayed recall	Information processing speed	Global cognition
Subjects	2120	2103	2111	2330
Observations	11,508	11,376	11,466	13,226
Fixed effects				
Score at 100 years	4.75 [4.12; 5.39]	2 [1.3; 2.7]	16.75 [15.31; 18.19]	25.37 [24.56; 26.18]
Early morning awakenings (Score at 100 years)	−0.09 [−0.38; 0.2]$^{0.84}$	−0.17 [−0.5; 0.16]$^{0.49}$	**−2.05 [−2.72; −1.39]$^{<0.01}$**	**−0.55 [−0.91; −0.19]$^{<0.01}$**
Preterminal decline	−0.03 [−0.05; −0.01]	0 [−0.02; 0.02]	−0.23 [−0.26; −0.2]	−0.05 [−0.07; −0.03]
Early morning awakenings (Preterminal decline)	0 [−0.01; 0.01]$^{0.84}$	−0.01 [−0.02; 0.01]$^{0.49}$	**0.02 [0; 0.03]$^{0.03}$**	−0.01 [−0.01; 0]$^{0.10}$
Terminal decline	−0.19 [−0.22; −0.16]	−0.2 [−0.24; −0.17]	−0.51 [−0.59; −0.43]	−0.12 [−0.18; −0.07]
Early morning awakenings (Terminal decline)	0 [−0.01; 0.02]$^{0.84}$	0 [−0.02; 0.01]$^{0.93}$	**−0.07 [−0.1; −0.03]$^{<0.01}$**	−0.01 [−0.04; 0.01]$^{0.30}$
Change point	−25.34 [−27.07; −23.6]	−27.06 [−28.56; −25.56]	−21.33 [−22.72; −19.95]	−16.27 [−17.19; −15.35]
Early morning awakenings (Change point)	−0.08 [−0.89; 0.72]$^{0.84}$	0.53 [−0.18; 1.25]$^{0.29}$	−0.27 [−0.86; 0.33]$^{0.38}$	−0.36 [−0.78; 0.06]$^{0.11}$
Error	1.63	1.87	2.48	1.59
Random effects				
$\omega 2_{\text{Score at 100 years}}$	2.27 [1.79; 2.76]	2.83 [2.17; 3.49]	25.03 [21.48; 28.59]	0.94 [0.39; 1.48]
$\omega 2_{\text{Preterminal decline}}$	0 [0; 0.01]	0.01 [0; 0.01]	0.03 [0.02; 0.03]	0.01 [0.01; 0.02]
$\omega 2_{\text{Terminal decline}}$	0 [0; 0]	0 [0; 0.01]	0.03 [0.02; 0.04]	0.02 [0.02; 0.02]
$\omega 2_{\text{Change point}}$	13.55 [7.18; 19.92]	16.09 [11; 21.17]	30.38 [25.22; 35.54]	13.52 [10.13; 16.91]
$\text{Covariance}_{\text{Preterminal decline/Terminal decline}}$	0 [0; 0]	0 [−0.01; 0]	−0.03 [−0.03; −0.02]	−0.02 [−0.02; −0.02]

Note: The numbers represent regression coefficients (for fixed effects, *B*) or variance (for random effects, $\omega 2$) with the corresponding 95% confidence intervals (in brackets) and *p* values, adjusted for multiple testing (in superscript), for all model terms (rows) in each of the four statistical models (columns). Statistically significant effects are highlighted in bold.

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linearly across the whole trajectory (e.g., in the association between early morning awakenings and global cognition). This enforced change point might have been the reason why problems with falling asleep were associated with a *slower* decline in immediate recall later in life, which was not detected by the quadratic model despite the visually similar trajectories. Overall, we conclude that the piecewise model was more flexible and could differentiate the effects of sleep problems specific to the earlier and later phases of cognitive decline.

4 | Discussion

4.1 | Summary of the Findings and Their Relation to Earlier Studies

This study used two complementary types of statistical models to demonstrate how sleep problems affect nonlinear trajectories of changes in four indicators of cognitive functioning in the age period between 55 and 100 years in a large population sample of older adults from the Netherlands. We used a quadratic and piecewise statistical models, each with pros and cons, that showed highly similar general patterns of cognitive decline but diverged in their estimation of the effects of sleep problems on these trajectories: Although the quadratic model estimated significantly different decline trajectories in participants with more severe sleep problems, its parabolic curve could not incorporate two phases and, unlike the piecewise models, could not offer interpretable estimates to describe these associations or detect the effects of some sleep problems that concerned only specific stages of cognitive decline. According to the piecewise models, sleep problems were predominantly associated with the later (acceleration) phase of cognitive decline, supporting the role of healthy sleep in healthy cognitive aging. Furthermore, some sleep problems were more specific in predicting performance on certain cognitive domains, complementing the evidence of the differing mechanisms behind these associations.

Decline in memory function accelerated from the age of 74 years, and a faster rate of decline in immediate recall was associated with short/long sleep duration and severe problems falling asleep, while a faster decline in delayed recall was associated with long sleep duration. Despite a number of earlier genetic (Henry et al. 2019), within-subject (Zuidersma et al. 2022) and between-subject (Beydoun et al. 2021; Lai et al. 2022; Zaheed et al. 2023; Cox et al. 2019; Gildner et al. 2019; Overton et al. 2024; Schneider et al. 2022; Li, Sahakian, et al. 2022) longitudinal studies that showed the associations between poor sleep and memory decline, there were only two that separated effects on immediate and delayed recall (Klaming et al. 2017; Lai et al. 2022). The first study, in line with ours, found that worse sleep quality predicted worse functioning on both immediate and delayed recall years later (Lai et al. 2022), but it did not distinguish the effects of separate sleep problems. Another study of memory decline across five measurement waves of LASA (Klaming et al. 2017) used a similar quadratic model that, in agreement with our analyses, detected the effects of long sleep on immediate and delayed recall, but it did not catch the significant association with problems with falling asleep on immediate recall that could only be found by the piecewise model. Earlier studies in older adults similarly found that abnormal sleep duration and severe sleep problems can also be associated with other measures of short-term memory, but they could not distinguish the effects on immediate and delayed recall, which may explain occasional inconsistencies in results, such as no association with problems falling asleep (Owusu et al. 2023; Overton et al. 2024) or sleep duration (Owusu et al. 2023; Ma et al. 2020; Lücke et al. 2022). Similar to our results, several studies that separated the effects of individual sleep problems found no association between memory and night/early morning awakenings (Klaming et al. 2017; Zaheed et al. 2023; Owusu et al. 2023). Furthermore, our findings based on quadratic models were similar for immediate and delayed recall, but piecewise models that separated two phases revealed substantial differences in the acceleration of memory decline at the end of life.

Information processing speed started to decline already before the age of 55 years, with a steadily accelerating rate toward the end of life that accelerated earlier in participants with short sleep duration, and at a faster rate in those with more severe early morning awakenings. Few other studies investigated the long-term effects of sleep on information processing speed. In agreement with our findings, two studies showed that information processing speed during aging declined faster in those with more early morning and night awakenings, and, contradicting our results, problems with falling asleep (Zaheed et al. 2023; Overton et al. 2024). Another study that included sleep duration with other health-related predictors in one model reported that the decline in information processing speed was associated with long sleep duration, and only in the Doetinchem cohort study and not LASA (Jaarsma et al. 2024): This finding agreed with the result of our quadratic model but not the piecewise model which linked processing speed to short sleep instead, and specifically, with the earlier time of acceleration rather than the rate of decline in processing speed. Furthermore, we showed that early morning awakenings have opposing effects during the early and later stages of aging, which can be missed when estimating a single linear trajectory of decline during this wide age period.

Global cognition started to decline very subtly from the age of 55 years, but after 83 years, this decline was faster in participants with short/long sleep duration and more night and early morning awakenings. Few earlier studies measured the association between sleep and the *rate* of cognitive decline, which included only the effects of sleep duration: These studies similarly concluded that global cognitive functioning declined faster with longer sleep (Cox et al. 2019; Gildner et al. 2019; Ma et al. 2020), which agrees with the effect of long sleep on the risk of cognitive disorders (Sindi et al. 2018; Cavaillès et al. 2022; Bokenberger et al. 2017; Lysen et al. 2020; Palpatzis et al. 2021; Westwood et al. 2017; Benito-León et al. 2009), with few exceptions (Sabia et al. 2021; Lutsey et al. 2018; Lysen et al. 2018). Short sleep duration similarly has been linked to an elevated risk in cognitive disorders in multiple population-based studies (Bokenberger et al. 2017; Sabia et al. 2021; Palpatzis et al. 2021; Ma et al. 2020; Robbins et al. 2021), also with few exceptions (Sindi et al. 2018; Benito-León et al. 2009; Lysen et al. 2018). Our findings agreed with these trends in that the rate of terminal decline in global cognition was

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similarly faster in both short and long sleepers. However, no earlier research explored the effects of individual sleep problems on the rate of age-related changes in global cognitive functioning. The studies that predicted the risk of dementia instead were generally in line with our results, showing that it was higher in those with severe night and early morning awakenings (Sindi et al. 2018; Selbaek-Tungevåg et al. 2023) but not problems falling asleep (Sindi et al. 2018; Lysen et al. 2020; Robbins et al. 2021; Lysen et al. 2018).

4.2 | Implications

Our study demonstrated that the choice of a statistical model of cognitive changes during aging can lead to very different results for the same research question. Random piecewise change point models allowed us to estimate more realistic, two-phase trajectories of cognitive decline and confirm that sleep characteristics are not only linked to a higher incidence of cognitive disorders shown by previous research but can also predict unusual trajectories of cognitive decline in various domains, including in individuals not yet diagnosed with dementia. The association between sleep characteristics and the age and rate of acceleration during the later phase of cognitive decline has not been investigated before, but this distinction demonstrated how the associations between sleep problems and specific phases (early and late) of cognitive decline can be missed in linear models, including the quadratic model, and even more so when the age range in the (longitudinal) study includes both phases of cognitive decline.

Domain-specificity in these associations supports potential differences in the mechanisms behind the associations between sleep and cognitive functioning, which include the intermedial effects of various factors related to genetics, mental and physical wellbeing (Li, Sahakian, et al. 2022). Due to this complexity, many inconsistencies in the findings across studies may have resulted from methodological differences related to the choice of measuring instruments, types of statistical models, and chosen covariates. For example, some research suggests that short sleep may be related to cognitive functioning only in the physically robust (Chuang et al. 2022) and cognitively intact individuals (Bokenberger et al. 2017), while effects of certain sleep problems may be considerable only in specific (clinical) subgroups, such as those with cognitive (Li, Gao, et al. 2022) and mood disorders (Pearson et al. 2023), which explains why the effects of some sleep characteristics change when the models are adjusted for comorbidities. However, most of such explanations are post hoc interpretations of what may have been chance findings.

It remains unclear why exactly certain sleep characteristics are stronger predictors of performance on specific cognitive domains, but the accumulating evidence from our and earlier studies offers some insight. For one, both short and long sleep may be associated with cognitive aging due to its association with other age-related health problems: some chronic diseases (e.g., cardiovascular diseases and diabetes mellitus) most prevalent in the elderly (Hoeymans et al. 2012) were repeatedly associated with both abnormal sleep duration (Li, Cao, et al. 2022) and an increased risk of dementia (Cao et al. 2024; Thomassen et al. 2020). Furthermore, insufficient (short) sleep is usually a result of sleep problems and is correlated with their severity (Figure S1), which may explain why worsening in some cognitive domains linked to short sleep (i.e., decline in immediate recall and information processing speed) is also predicted by certain sleep problems that may cause it (e.g., problems falling asleep and early morning awakenings). It is less clear why worse cognitive functioning is associated with long nighttime sleep (e.g., in delayed recall and global cognition in our study) and even *daytime* sleep (Palpatzis et al. 2021; Li, Gao, et al. 2022), even after adjusting for common comorbidities (Palpatzis et al. 2021; Gildner et al. 2019; Chuang et al. 2022; Li, Gao, et al. 2022) as well as fragmented sleep, as seen from more self-reported night awakenings in our study and its actigraphy-based proxy in earlier research (West et al. 2021). The fact that this long fragmented sleep in the elderly repeats the sleep pattern observed in developing children (Leong and Chee 2023; Kocevska et al. 2021) may suggest that in the elderly, especially those with cognitive problems, it may be a sign of developmental regression, i.e., the start of neurodegeneration. Thus, even with the lack of our understanding of the exact mechanisms (e.g., if sleep problems are causal or epiphenomena), some sleep characteristics can be more accurate predictors of abnormal trajectories of decline in specific cognitive domains (e.g., night awakenings more specific to global cognitive functioning and problems falling asleep to memory, alike to rapid eye movement sleep behaviour disorder being specific to Lewy body's dementia (Postuma et al. 2019)) and thus help identify participants at a higher risk of cognitive disorders.

Although substantial researcher degrees of freedom may explain the contradicting findings in the literature, it is difficult to pinpoint the best approach to estimate and identify (causal) effects of sleep problems on cognitive decline. However, a deeper understanding of these interrelations is crucial before we can use such data to formulate any practical implications, such as informing the existing healthcare policies. Despite the commonly reported prospective effects of sleep behaviour on cognitive functioning and the known role of sleep in brain health (Frank and Heller 2019), poor sleep does not necessarily *cause* cognitive disorders later in life and may rather be an *early sign* and consequence of neurodegeneration related to aging and/or cognitive disorders (Shen et al. 2023). In fact, long-term associations between sleep problems and cognitive functioning are likely bidirectional and complex, involving a wide variety of intermedial pathways (Musiek and Holtzman 2016), which differ from the effects observed after sleep deprivation (Ochab et al. 2021; García et al. 2021; Mota Albuquerque et al. 2023). Furthermore, even if sleep problems predict the trajectories of cognitive aging, the clinical relevance of these (statistically significant but relatively small) effects is still under question, which is unsurprising given that sleep is only one of many factors associated with cognitive functioning. Considerable differences in cognitive functioning mostly concerned processing speed and global cognition, and even then, were observed particularly after around 80 years; for example, global cognition scores of those with severe sleep problems crossed the threshold for qualifying for dementia unlike the group with minimal sleep problems. Altogether, these gaps in our understanding hinder the

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direct application of such data in healthcare, such as the decisions regarding the utility of sleep-targeted interventions in the prevention of cognitive disorders.

Although our results should be ideally replicated in future studies, more concerted efforts are also needed to formulate theories before simply repeating and expanding the empirical analyses on longitudinal data. Models of cognitive changes across time need to be carefully chosen as they greatly affect the findings and their interpretation. If some sleep problems are considered weak predictors of cognitive functioning, further studies of these factors may be redundant given the low relevance of these effects for clinical practice. Yet, more comprehensive sleep data is required to study the effects of multidimensional sleep health, such as the RU-SATED (Buysse 2014) model, as it is possible that only specific sleep profiles (combinations and/or course of development of sleep problems) may be strongly linked to cognitive dysfunction. Furthermore, the distinction of the short- and long-term effects of sleep on cognition in older adults can be investigated, such as by using diary data in experience sampling studies. Altogether, such focused approaches may deepen our understanding of the role of sleep health in cognitive aging.

4.3 | Strengths and Limitations

The findings of this study were based on the data from a large sample ($N = 5132$) of older adults (52% female; followed from the age of 55–86 years) from three culturally distinct areas of the Netherlands. This large and diverse sample representative of the general population (Huisman et al. 2011) allowed us to study how different severity levels of various sleep problems are associated with individual domains of cognitive functioning. Each participant's sleep and cognitive characteristics were measured up to 10 times (mean = 3–4) over the span of 30 years, which allowed us to estimate general trajectories of changes in cognitive functioning depending on the time-varying severity of sleep problems during a wide age period from 55 to 100 years, as opposed to predicting cognitive functioning at the end of life with only baseline sleep parameters that were done in most previous studies. A final asset of our study was that two statistical models were used that could accommodate nonlinearity in the association between cognitive functioning and age, investigate the role of sleep problems in the changing rates of cognitive decline, and account for individual variation in these associations. The quadratic model has been commonly used to model non-linear change, and we showed its limitations in capturing the data. The random change point model has never been used to investigate associations between sleep and cognitive functioning before our study, and we showed that it yielded more precise and interpretable information.

Nevertheless, the interpretation of these findings is limited by several factors related to the study design and statistical methods. First, piecewise mixed models required all participants to have at least four measurement points, which may exacerbate the survivor bias characteristic of aging cohorts, that is, selectively exclude less physically and cognitively healthy participants who die earlier and have fewer datapoints (Banack et al. 2019). Thus, the findings based on this smaller subsample of younger and healthier participants were potentially less generalizable and likely to underestimate the effects of sleep. Second, we studied only four of many possible indicators of cognitive functioning, while our findings emphasised that these associations may vary per cognitive domain and sleep characteristic. Furthermore, the use of self-reported sleep data meant that sleep problems could have been reported inaccurately due to insomnia-related sleep misperception (Valko et al. 2021), recall bias due to severe cognitive problems (Stankeviciute et al. 2024), or general underestimation of sleep problem severity by older adults (Westergren et al. 2015), potentially leading to over- or underestimation of the association between cognition and short sleep duration. However, most sleep problems are subjective experiences that need to be assessed in relation to individual circumstances (e.g., sleep opportunity, burden assessment) leaving few alternatives. Although some sleep characteristics can be measured by devices, often these data are equally prone to bias (e.g., actigraphic sleep duration overestimated in individuals with insomnia (Acker et al. 2021)) or not feasible for large-scale longitudinal studies (e.g., polysomnography). Lastly, we already mentioned that our study was not designed to study whether sleep problems are epiphenomena of general neurodegeneration (Musiek and Holtzman 2016). Regarding causality, earlier studies suggested a bidirectional association, for example, progressing Alzheimer's disease predicts more daytime napping (Li, Gao, et al. 2022) and faster cognitive decline predicts worse actigraphy-based sleep fragmentation (West et al. 2021). Associations between sleep problems and cognitive functioning can also arise due to confounding: for example, different demographic factors, health conditions, and genetic predisposition may explain both poor sleep and cognitive decline. The current study did not adjust for such variables as they may also be part of the causal pathway, such as when genetic predisposition of sleep problems leads to sleep problems that, in turn, lead to cognitive decline, or when sleep problems lead to depression that, in turn, leads to cognitive decline. Adjusting for such mechanistic variables as if they were confounders would underestimate the role of sleep. Put differently, our goal was to establish the overall association between sleep and cognitive decline. Hence, it is possible that currently found associations may be explained by other variables.

5 | Conclusions

To conclude, our study was unique in comparing the effects of individual sleep problems on the rate of biphasic changes in various cognitive functions throughout multiple points during aging, which complemented the current evidence base of the associations between sleep health and cognitive functioning and added insights regarding the mechanisms behind them.

Author Contributions

Dina Sarsembayeva: conceptualization, methodology, visualization, writing – review and editing, writing – original draft, formal analysis, project administration. **Marieke J. Schreuder:** supervision, formal analysis, methodology, conceptualization, writing – review and editing. **Martijn Huisman:** data curation, writing – review and editing. **Almar Kok:** data curation, writing – review and editing. **Maude Wagner:** software, writing – review and editing. **Ana W. Capuano:** software, writing – review and editing. **Catharina A. Hartman:**

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project administration, supervision, writing – review and editing, conceptualization, methodology, formal analysis.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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