BMJ Public Health

Sleep duration, chronotype, health and lifestyle factors affect cognition: a UK Biobank cross-sectional study

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To cite: West R, Wong RTC, Park J-E, *et al*. Sleep duration, chronotype, health and lifestyle factors affect cognition: a UK Biobank cross-sectional study. *BMJ Public Health* 2024;**2**:e001000. doi:10.1136/bmjph-2024-001000

RW and DM are joint senior authors.

Received 31 January 2024
Accepted 24 April 2024

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ABSTRACT

Objective To explore the nuanced relationship between sleep patterns, chronotype, quality and the influence of health and lifestyle factors on cognitive performance.

Design, setting, participants This cross-sectional analysis used ordinary least squares regression within the UK Biobank database, assessing 26 820 participants aged 53–86 years, categorised into two cohorts: Cohort 1 (10 067 participants, 56% female; completed all four cognitive tests of Fluid Intelligence/reasoning, Pairs Matching, Reaction Time and Prospective Memory) and Cohort 2 (16 753 participants, 56% female; completed only two cognitive assessments of Pairs Matching and Reaction Time).

Exposures Participant's self-reported sleep duration, chronotype and quality. Cognitive function was assessed through standardised computerised tests. The analysis was adjusted for demographic and comorbidity covariates.

Main outcomes and measures Cognitive performance scores were evaluated against sleep parameters and health and lifestyle factors including sex, age, vascular and cardiac conditions, diabetes, alcohol intake, smoking habits and body mass index.

Results The regression highlighted a positive association between normal sleep duration (7–9 hours) and cognitive scores in Cohort 1 (β=0.0567, 95% CI 0.0284 to 0.0851), while extended sleep duration negatively impacted scores across both cohorts (Cohort 1: β=−0.188, 95% CI −0.2938 to −0.0822; Cohort 2: β=−0.2619, 95% CI −0.3755 to −0.1482). Chronotype distinctions, particularly intermediate and evening types, were linked to superior cognitive function. Gender, age, angina, high blood pressure, diabetes, alcohol intake and smoking emerged as significant cognitive influencers.

Conclusions and relevance The study delineates a multifaceted and nuanced relationship between sleep variables, health and lifestyle factors in determining cognitive outcomes. These findings highlight the vital role of sleep quality on cognitive health.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Sleep quality affects cognitive abilities with both short and long sleep durations linked to cognitive impairments.
⇒ Sleep quality is correlated with cognitive performance, but less is known about the impact of circadian rhythms (chronotypes) on cognition.

WHAT THIS STUDY ADDS

⇒ The significant influence of sleep chronotypes on cognitive function adds new depth to our understanding of the role of sleep in cognitive health.
⇒ Our findings highlight the complex interplay between sleep duration, chronotypes and various health and lifestyle factors on cognitive performance.

HOW MIGHT THIS AFFECT RESEARCH, PRACTICE AND POLICY

⇒ This study sheds light on the role and importance of sleep chronotypes on cognitive function.
⇒ Further research and practices should focus on promoting interventions to improve sleep patterns in the general population.

INTRODUCTION

Sleep is a fundamental biological behaviour that is universally conserved throughout evolution. However, despite its significance, the function of such a process remains a long-debated subject of interest. Emerging studies suggest that sleep plays a crucial role in optimising cognitive function by contributing to bodily restoration,[1] memory consolidation,[2] learning[3] and emotional regulation.[4] Sleep impairment, particularly common among elderly people, has been consistently linked to an increased risk of cognitive decline and dementia.[5–8] Homeostatic immune function is also profoundly influenced by sleep, and sleep impairment has been linked to immune-related neurodegenerative, metabolic, autoimmune and vascular diseases.[9] Animal studies have further shown that, mechanistically, sleep disturbance induces neuroinflammation, complement activation, impaired learning and memory, and affects hippocampus-dependent learning.[10 11]

Population-based research has highlighted the relationship between sleep duration and

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cognitive abilities, indicating potential cognitive impairments associated with both short (≤4 hours) and long (≥10 hours) sleep durations.[12] Severe sleep deprivation has been shown to induce alterations in synaptic plasticity and impairments in learning and memory, thus affecting cognition.[13] Furthermore, the quality of sleep is also a determinant of cognitive performance, where poor sleep quality is correlated with lower cognitive functioning.[14] Sleep provides a restorative and protective function on cognition by the removal of toxic metabolites from the central nervous system.[15] A positive feedback relationship has been suggested between sleep and Alzheimer's disease, whereby poor sleep quality and duration induces amyloid-β peptide cumulation which, in turn, also causes poor sleep quality and sleep deprivation.[16] Beyond sleep duration and quality, the role of circadian preferences or chronotypes and their impact on cognitive abilities are less clear. Despite valuable insights from existing UK Biobank studies on sleep and cognition,[17–19] there is a knowledge gap regarding the influence of chronotypes. Sleep pattern, or chronotypes, reflects an individual's inclination to sleep at a particular time of the day as a manifestation of one's circadian rhythm.[20] Although studies have shown that disruption in circadian rhythms, such as those from shift work or jet lag, negatively impacted cognitive performance,[21–24] little is known about how different circadian rhythms or chronotypes (eg, morningness or eveningness) are associated with cognitive function.

Using the comprehensive dataset from the UK Biobank, this study investigates the intricate relationships between sleep duration, quality and chronotype and their collective impact on cognitive performance. We consider a range of demographic, lifestyle and comorbidity factors as potential covariates to unravel their influence on the interplay between sleep patterns and cognitive function.

METHODS

Study design, data source and study population

The study data were derived from the UK Biobank, a population-based prospective study established by the UK Medical Research Council and Wellcome Trust. The study recruited approximately 501 718 men and women aged ≥40 years across the UK registered with the UK National Health Service. Details of the UK Biobank with Ethics Committee approval have been previously described.[25] As this study involved secondary analysis of anonymised data, no additional ethical approval was needed. This report follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline for cross-sectional studies. Our analysis capitalised on the comprehensive data available from the UK Biobank, encompassing participants with complete information on key variables such as sex, birth year, body mass index (BMI), smoking status, frequency of alcohol intake and medical histories including diabetes, vascular/heart problems and cancer diagnosis. To optimise the analysis and enhance the representativeness of our findings, participants were divided into two cohorts based on the different combinations of cognitive assessments completed. Cohort 1, consisting of 10 067 participants (mean age 71 years, 56% female), included those who completed all four cognitive tests (Fluid Intelligence/reasoning, Pairs Matching, Reaction Time and Prospective Memory). In contrast, Cohort 2, comprising a larger group of 16 753 participants (mean age 72 years, 56% female), included individuals who completed only two of these cognitive assessments (Pairs Matching and Reaction Time). This division allowed us to incorporate a larger number of participants, thus making effective use of the extensive UK Biobank dataset. The two distinct cohorts were analysed separately. In selecting participants, we prioritised the inclusion of a comprehensive set of sleep parameters and relevant confounding variables. However, we did not adjust for educational attainment due to incomplete data in this area. Additionally, responses indicating unclear chronotypes such as "I don't know" were excluded from our analysis.

In the selection of our study participants from the UK Biobank, we prioritised the inclusion of a comprehensive set of sleep parameters and relevant confounding variables to rigorously assess their relationship with cognitive performance. As a result, the final sample size was determined based on the availability of complete data across these chosen variables. This approach, while resulting in a relatively smaller sample size compared with some previous UK Biobank sleep studies, ensured that our analysis was focused and specific to our research questions. It represents a trade-off between sample size and the depth and relevance of data for the specific variables under investigation. Furthermore, the cognitive assessments employed in the UK Biobank data are well validated and were administered through a novel brief computerised platform. The cognitive assessments—Fluid intelligence, Pairs Matching, Reaction Time and Prospective Memory—are used to evaluate different parameters of cognition including logic and reasoning, visual memory, processing speed and prospective memory, respectively.[26] Therefore, given its large sample size and extensive cognitive assessments, the UK Biobank data has been used across numerous studies.[26–28]

Assessments were conducted between 2006 and 2010 across 22 centres in England, Scotland and Wales. Health information and sleep-related variables were obtained using self-report questionnaires, and cognitive assessments were conducted digitally.

Cognitive variables

Cognitive performance was assessed through four (Cohort 1) or two (Cohort 2) cognitive tests designed for the UK Biobank. The cognitive tests examined the performance of cognitive function and the stability of such ability over time has been well established previously.[27]

The assessment procedures of the four cognitive tests were as follows:

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Fluid Intelligence/reasoning (Data Field ID: 20016): participants were given 13 verbal and numerical fluid intelligence questions designed by the UK Biobank. Each question was given a 2 min answering time to select five possible response answers. The dependent variable measured is the unweighted sum (0–13) of the number of correct answers from the 13 questions.

Pairs Matching (Data Field ID: 399): participants were presented with 12 cards consisting of six pairs of symbols. The cards then were turned face-down on the computer touchscreen and participants were tasked to identify and match as many pairs of cards as possible. The dependent variable was the number of errors made during the test.

Reaction Time (Data Field ID: 20023): Reaction time was assessed through the card game Snap. Participants were requested to press a snap button in response to the appearance of matched cards displayed on the computer touchscreen. The dependent variable was measured by the mean duration of time taken in milliseconds to react to the 12 matching trials. Values were rounded to the nearest whole number.

Prospective Memory (Data Field ID: 20018): participants were given the following instructions on the computer touchscreen: "At the end of the games, we will show you four coloured symbols and ask you to touch the blue square. However, to test your memory, we want you to actually touch the orange circle instead". The participants were then given up to two attempts to recall the above instruction correctly after a filled interval. The dependent variable was measured by whether the participants had successfully recalled the instruction.

Sleep variables

The study focused on three sleep-related variables: sleep duration, sleep pattern (ie, chronotype) and sleep quality.

Sleep duration (Data Field ID: 1160) was collected from the touchscreen question "How many hours of sleep do you get in every 24 hours (please include naps)?". The inputted responses were then systematically filtered by the following criteria: if the answer was <1 hour or >23 hours it was rejected; if the answer was <3 hours or >12 hours then the participants were asked to confirm. For this study, sleep duration was categorised into short (<7 hours), normal (7–9 hours) and long (>9 hours) in accordance with the guidelines of the American Academy of Sleep Medicine and Sleep Research Society.[29]

Sleep pattern (Data Field ID: 1180) was determined by an individual's chronotype (ie, a morningness person is active and alert predominantly in the morning while dormant at night while an eveningness person is active and alert predominantly at night while dormant in the morning). This was assessed through the touchscreen question: "Do you consider yourself to be?" Participants were then given six answer options to select: 'definitely a morning person', 'more a morning than an evening person', 'more an evening than a morning person', 'definitely an evening person', 'do not know' and 'prefer not to answer'. For this study the data were re-categorised into three groups: 'Morningness', which consisted of participants who answered 'definitely a morning person'; 'Intermediate', which consisted of participants who answered 'more a morning than evening person' and 'more an evening than morning person'; and 'Eveningness', which consisted of participants who replied 'definitely an evening person'.

Sleep quality (Data Field ID: 1200) was assessed through the degree of sleeplessness/insomnia a participant experienced. Participants were asked the question "Do you have trouble falling asleep at night or do you wake up in the middle of the night?" and given the four answer choices of 'never/rarely', 'sometimes', 'usually' and 'prefer not to answer'. For this study, the data were re-categorised into two categories of sleeplessness/insomnia: 'never/rarely' or 'sometimes/usually'.

Covariates

The study included covariates to account for potential factors that might confound the association between the three sleep parameters and cognition in the analyses. These covariates encompassed sex (male/female) (Data Field ID: 31), year of birth (Data Field ID: 34), BMI (Data Field ID: 21001), smoking status (Data Field ID: 20116), alcohol intake frequency (Data Field ID: 1558), diabetes diagnosis (Data Field ID: 2443), vascular/heart diagnosis (heart attack, angina, stroke and high BP) (Data Field ID:6150) and cancer diagnosis (Data Field ID: 2453).

Statistical analysis

Descriptive statistics were used for data characterisation. To normalise the cognitive test scores and ensure comparability, we transformed the raw scores into z-scores using the formula: z-score = (raw score – mean baseline)/baseline standard deviation (SD). This transformation standardised scores from different tests into a unified scale.

Our primary analytical approach was the ordinary least squares (OLS) regression. This model was chosen for its ability to handle multiple predictors and to assess their independent effect on cognitive scores. We included sleep parameters and a range of other covariates such as demographic, health and lifestyle factors to control for potential confounding effects.

To address heteroskedasticity, which can lead to biased standard error (SE) estimates and affect the reliability of test statistics, robust SE were integrated into the regression model. This approach strengthens the validity of our inferences under potential heteroskedastic conditions.

Multicollinearity among independent variables was rigorously evaluated using the Variance Inflation Factor (VIF), with all values confirming below the threshold of 10, indicating that our model did not suffer from multicollinearity issues.

Residual diagnostics were thoroughly conducted. We performed skewness and kurtosis tests to assess the normality of the residuals. Additionally, we visually inspected residual plots against fitted values to confirm the assumptions of linear regression were met. Cook's

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distance was used to identify and evaluate the impact of potentially influential observations on the regression model, thereby ensuring its robustness and reliability.

All statistical analyses were performed using Stata/BE 18 software.

Data availability

Full study data cannot be openly shared under the material transfer agreement with UK Biobank. Prospective researchers can apply for access to the UK Biobank data from www.ukbiobank.ac.uk.

RESULTS

Demographics and sample sizes of the UK Biobank data

The present study included 26820 participants who completed all baseline surveys, including all demographic, lifestyle and comorbidity covariates. Of the 26820 participants, 10067 who completed all four cognitive assessments were grouped as Cohort 1 (mean (SD) age 71 (8) years; 56% female (5650/10 067)) while 16753 who completed only two cognitive assessments were grouped as Cohort 2 (mean (SD) age 72 (8) years; 56% female (9440/16 753)). The baseline demographic background covariates and sleep-related variables of the two independent cohorts from the UK Biobank data are shown in table 1.

Regression diagnostics

Our regression analysis for both cohorts involved several diagnostic checks to confirm the appropriateness of the OLS regression model. For Cohort 1, the Breusch–Pagan test suggested the presence of heteroskedasticity ($\chi^2(1) = 242.37$, $p<0.0001$), prompting the use of robust SE. Cohort 2 showed similar signs of heteroskedasticity with a χ^2 value of 709.60 ($p<0.0001$) from the Breusch–Pagan test, indicating the need for robust SE in this group as well.

The VIF was computed for each predictor in both cohorts, with all VIF values remaining below the threshold of 10, suggesting that multicollinearity was not a concern for our regression models. Skewness and kurtosis tests for normality indicated a departure from normality in the residuals for both cohorts ($p<0.0001$); however, the central limit theorem was anticipated to mitigate the impact of this non-normality due to the large sample sizes.

Cook's distance was used to detect influential cases, identifying 5.93% of the data points in Cohort 1 and 4.83% in Cohort 2 as potential outliers. These points were, however, retained for analysis as they did not present with data entry errors or anomalies that would warrant exclusion. Visual inspection of residual plots against fitted values did not reveal any discernible patterns, suggesting that the assumptions of linearity were met for the regression models applied to both cohorts.

The study used a multivariable linear regression model with robust SE to evaluate predictors and their relationship with the global cognitive z-score, ensuring a robust inference in the presence of heteroskedasticity as indicated by our diagnostics checks for both cohorts. A comprehensive summary of the regression model results for Cohorts 1 and 2 is presented in table 2.

Association between sleep factors and cognitive performance

In Cohort 1, normal sleep duration was associated with a slightly higher global cognitive score (β coefficient of 0.0567, $p<0.001$) than short sleepers. In contrast, long sleepers had significantly lower scores (β coefficient of −0.1880, $p<0.001$). The pattern was similar for long sleepers in Cohort 2 (β coefficient of −0.2619, $p<0.001$), although normal sleep duration had no significant effect.

Chronotype had a considerable impact on cognitive performance in both cohorts. Intermediate sleepers had higher z-scores (β coefficients of 0.1061 and 0.0632 for Cohorts 1 and 2, $p<0.001$) compared with morningness, as did the eveningness types (β coefficients of 0.1351 and 0.0750 for Cohort 1, $p<0.001$ and Cohort 2, $p=0.002$, respectively). Sleeplessness/insomnia, however, showed no significant association in either cohort.

Association between health and lifestyle factors and cognitive performance

Health and lifestyle factors showed variable correlations with cognitive performance. Gender, age, diabetes and alcohol consumption influenced cognitive performance in both cohorts. Women had lower scores than men (β coefficients of −0.0649 and −0.0761 for Cohorts 1 and 2, $p<0.001$). Age was inversely correlated with cognitive performance (β coefficients of −0.0177 and −0.0264 for Cohorts 1 and 2, $p<0.001$). Participants with diabetes had lower cognitive scores in both cohorts (β coefficients of −0.1204, $p<0.001$ and −0.0847, $p=0.011$ for Cohorts 1 and 2, respectively). Never or occasional alcohol consumers had significantly lower cognitive scores (β coefficients of −0.2971 and −0.1644 for Cohorts 1 and 2, $p<0.001$), compared with daily or almost daily consumers. For those who consumed alcohol up to four times a week (weekly), β coefficients of −0.0607 ($p<0.001$) and −0.0396 ($p=0.006$) for Cohorts 1 and 2 or up to three times a month (monthly), β coefficients of −0.0802 ($p<0.001$) and −0.0433 ($p=0.034$) for Cohorts 1 and 2, respectively, also scored lower in the cognitive tests.

Participants with angina and high BP did significantly worse in the cognitive test only in Cohort 1 (β coefficients −0.1513 and −0.0551, respectively, $p<0.001$). Current smokers in Cohort 1 had lower scores (β coefficient of −0.1182, $p<0.001$), while former smokers in Cohort 2 had higher scores (β coefficients of 0.0297, $p=0.019$) than never smoking. A diagnosis of cancer, heart attack, stroke and BMI did not significantly correlate with the cognitive scores.

Our analysis using predictive margins delineates the relationship between sleep duration and cognitive function as measured by the global z-score (figure 1). For Cohort 1, the data show an inverted U-shaped curve, signifying optimal cognitive function among individuals

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Table 1 Demographic characteristics of participants in two independent cohorts at baseline (total of 26 820 for both cohorts)

			Cohort 1		Cohort 2	
			Male (n=4417) Female (n=5650)		Male (n=7313) Female (n=9440)	
Participant background	Age	50–59 years	573	739	726	857
		60–69 years	1116	1651	1962	2759
		70–79 years	1984	2515	3057	4085
		≥80 years	744	745	1568	1739
	BMI (kg/m^2)*	<18.5 (underweight)	12	35	19	66
		18.5–24.9 (healthy)	908	1983	1506	3289
		25–29.9 (overweight)	2239	2192	3793	3644
		>29.9 (obese)	1258	1440	1995	2441
	Smoking status	Never	2161	3334	3498	5631
		Previous smoker	1773	1813	2910	2956
		Current smoker	483	503	905	853
	Alcohol intake frequency	Daily	1198	917	1885	1476
		Weekly	2236	2560	3864	4611
		Monthly	422	743	621	1243
		Never	561	1430	943	2110
Comorbidity	Diabetes	Yes	309	228	502	328
		No	4108	5422	6811	9112
	Vascular heart diseases	Angina	130	87	239	141
		Heart attack	161	48	294	68
		High blood pressure	1193	1180	1846	2106
		Stroke	54	57	108	85
		No	2879	4278	4826	7040
	Cancer	Yes	292	546	427	855
		No	4125	5104	6886	8585
Sleep parameters	Chronotype	Morningness	1123	1509	1958	2549
		Intermediate	2870	3636	4651	6069
		Eveningness	424	505	704	822
	Sleep quality	Good	1358	1126	2197	1771
		Intermediate	2002	2704	3371	4652
		Poor	1057	1820	1745	3017
		<5	41	56	78	103
		5	169	274	296	442
		6	931	1067	1381	1684
		7	1742	2141	2953	3630
		8	1226	1688	2031	2846
		>8	308	424	574	735

The table shows the demographic background of participants in two independent cohorts from the UK Biobank data. Cohort 1 contained 10 067 participants who undertook four cognitive tests (Fluid Intelligence, Prospective Memory, Paired Associated Learning and Reaction Time). Cohort 2 contained 16 753 participants who undertook two cognitive tests only (Paired Associated Learning and Reaction Time).
Sleep parameters were collected from self-report questionnaires and were characterised into three groups: chronotypes, sleep quality and sleep duration. Sleep chronotypes indicate the activity level of an individual throughout the day, whereby a morningness person is active in the morning and dormant at night. In contrast, an eveningness person is active at night and dormant in the morning. Sleep quality is defined by the extent of sleeplessness/insomnia an individual experiences at night.
*BMI categories were determined in accordance with the suggested BMI ranges from the National Health Service United Kingdom.

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Table 2 Results of combined table of multivariable linear regression to model for Cohorts 1 and Cohort 2

Variable	Coefficient (β)	Coefficient (β)	SE	SE	95% CI	95% CI
	Cohort 1	Cohort 2	Cohort 1	Cohort 2	Cohort 1	Cohort 2
Sleep duration (normal)	0.0567***	0.0052^{ns}	0.0140	0.0136	0.0292 to 0.0842	−0.0214 to 0.0318
Sleep duration (long)	−0.1880***	−0.2619***	0.0468	0.0432	−0.2797 to −0.0963	−0.3465 to −0.1773
Sleep pattern (intermediate)	0.1061***	0.0632***	0.0140	0.0131	0.0787 to 0.1335	0.0375 to 0.0889
Sleep pattern (evening person)	0.1351***	0.0750***	0.0231	0.0219	0.0898 to 0.1804	0.0320 to 0.1180
Sleeplessness/insomnia (sometimes/usually)	0.0021^{ns}	−0.0160^{ns}	0.0142	0.0137	−0.0258 to 0.0300	−0.0428 to 0.0108
Sex (female)	−0.0649***	−0.0761***	0.0127	0.0120	−0.0897 to −0.0401	−0.0995 to −0.0526
Age	−0.0177***	−0.0264***	0.0008	0.0008	−0.0193 to −0.0162	−0.0279 to −0.0249
Cancer (yes)	0.0119^{ns}	0.0344^{ns}	0.0219	0.0215	−0.0310 to 0.0547	−0.0078 to 0.0766
Heart attack (yes)	−0.0657^{ns}	0.0271^{ns}	0.0431	0.0402	−0.1501 to 0.0187	−0.0516 to 0.1059
Angina (yes)	−0.1513***	−0.0095^{ns}	0.0422	0.0391	−0.2340 to −0.0686	−0.0861 to 0.0671
Stroke (yes)	−0.1149*	−0.1405**	0.0577	0.0537	−0.2279 to −0.0019	−0.2457 to −0.0353
High BP (yes)	−0.0551***	−0.0078^{ns}	0.0151	0.0143	−0.0847 to −0.0256	−0.0357 to 0.0202
Diabetes (yes)	−0.1204***	−0.0847**	0.0276	0.0272	−0.1745 to −0.0663	−0.1380 to −0.0313
Alcohol intake (weekly)	−0.0607***	−0.0396**	0.0159	0.0152	−0.0919 to −0.0296	−0.0694 to −0.0098
Alcohol intake (monthly)	−0.0802***	−0.0433*	0.0224	0.0217	−0.1241 to −0.0363	−0.0858 to −0.0008
Alcohol intake (never)	−0.2971***	−0.1644***	0.0195	0.0190	−0.3354 to −0.2587	−0.2017 to −0.1271
Smoking (previous)	0.0245^{ns}	0.0297*	0.0132	0.0127	−0.0014 to 0.0505	0.0048 to 0.0545
Smoking (current)	−0.1182***	−0.0239^{ns}	0.0210	0.0194	−0.1593 to −0.0772	−0.0619 to 0.0141
BMI	0.0007^{ns}	0.0021^{ns}	0.0013	0.0013	−0.0019 to 0.0033	−0.0004 to 0.0046

In Cohort 1 (n=10 067) all participants answered four types of cognitive test: Fluid Intelligence/reasoning, Pairs Matching, Reaction Time, and Prospective Memory and in Cohort 2 (n=16 753) all participants only answered two types of cognitive test: Pairs Matching and Reaction Time. The coefficient references for the variables were: short sleep for the sleep duration, morningness for the sleep pattern, never/rarely for the sleeplessness/insomnia, being male for sex, not having a diagnosis of cancer or heart attack or angina or high BP or diabetes, and never smoking. Daily or almost daily was the reference for alcohol, where weekly alcohol was those who consumed alcohol 1–4 times a week, monthly was 1–3 times a month and never were those who never consumed alcohol or on special occasions only.
*p≤0.05; **p≤0.01; ***p≤0.001; ns, p>0.05.
BMI, body mass index; BP, blood pressure; SE, standard error.

reporting 6–9 hours of sleep, with performance diminishing among those with shorter or longer sleep durations. In contrast, Cohort 2 shows a relatively stable cognitive function across individuals sleeping 5–9 hours. Notably, there is a pronounced decline in function for those reporting sleep durations beyond 9 hours, with a

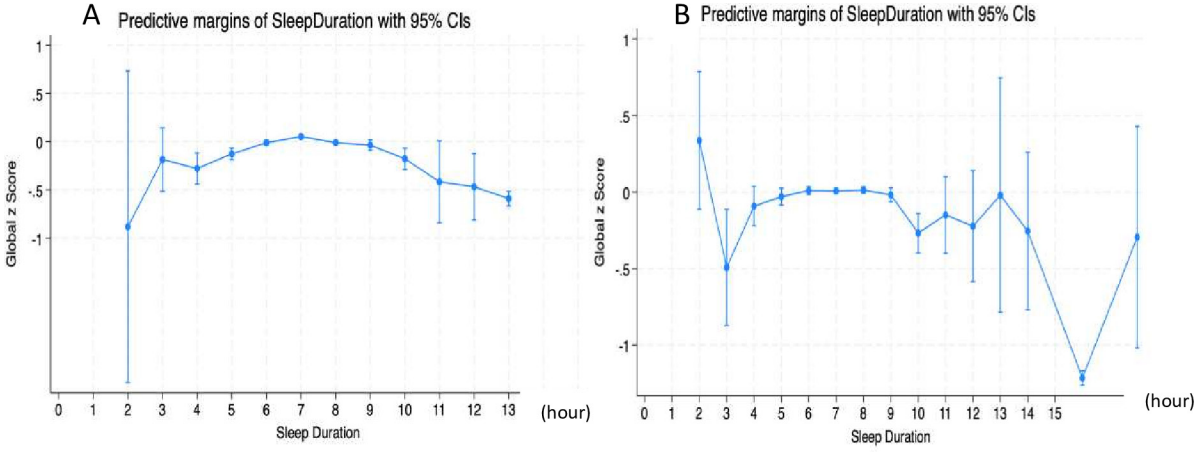

Figure 1 Predictive margins of sleep duration and global z-score across cohorts 1 (A) and 2 (B). The solid line in the graphs depicts the adjusted mean global cognitive z-score for each sleep duration category, with 95% confidence intervals, providing insights into the average cognitive function corresponding to each sleep duration after controlling for other factors.

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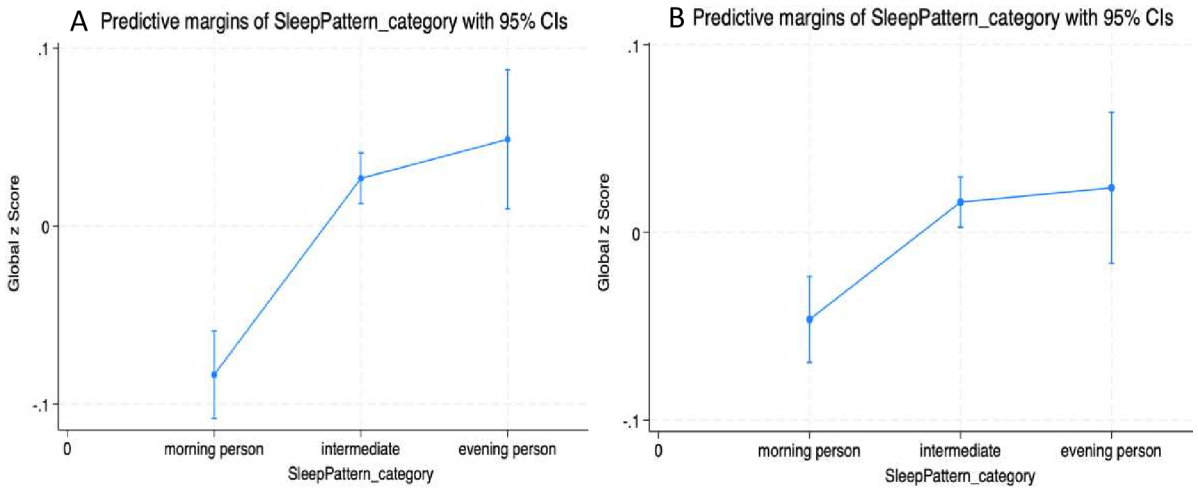

Figure 2 Predictive margins of sleep chronotype and global z-score across cohorts 1 (A) and 2 (B). The solid line in the graph depicts the adjusted mean global cognitive z-score for each sleep chronotype, with 95% confidence intervals, providing insights into the average cognitive function corresponding to each sleep chronotype after controlling for other factors.

marked decrease at 3 hours followed by a modest recovery at the 2-hour mark.

The predictive margins analysis for chronotype revealed a consistent pattern in cognitive performance in relation to circadian preferences across both cohorts (figure 2). Morning types consistently showed the lowest cognitive scores in both cohorts, with scores improving for intermediate types and reaching higher levels for evening types. The pattern indicates that later chronotypes correlate with better cognitive performance, with this effect being more pronounced in Cohort 1 compared with Cohort 2, where the increase in scores from intermediate to evening types is less substantial.

The predictive margins analysis for sleeplessness/insomnia shows that in Cohort 1 there is a minimal decrease in cognitive performance when moving from 'never/rarely' experiencing sleeplessness/insomnia to 'sometimes/usually' experiencing it (figure 3). For Cohort 2, a similar trend is observed, with a modest decline in cognitive performance for those who 'sometimes/usually' experience sleeplessness/insomnia compared with those who 'never/rarely' do. However, in both cohorts the change in cognitive performance is minimal and the confidence intervals are broad, suggesting a lack of significant impact of sleeplessness/insomnia on the global cognitive z-score.

DISCUSSION

Our study investigated the collective impact of the sleep parameters sleep duration, chronotype and quality

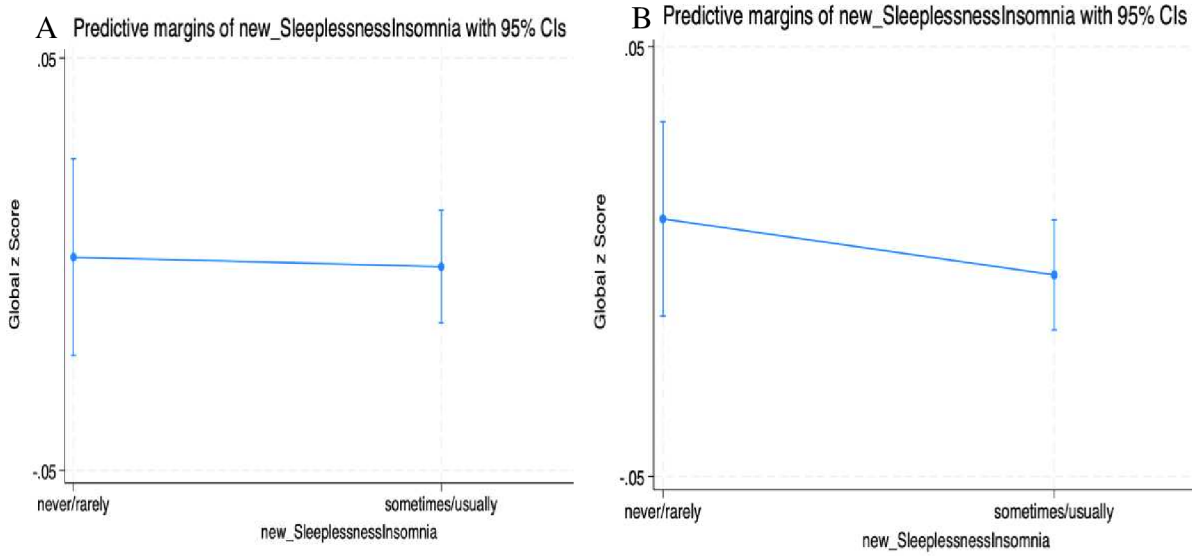

Figure 3 Predictive margins of sleeplessness/insomnia and global z-score across cohorts 1 (A) and 2 (B). The solid line in the graph depicts the adjusted mean global cognitive z-score for each sleeplessness/insomnia category, with 95% confidence intervals, providing insights into the average cognitive function corresponding to each sleeplessness/insomnia category after controlling for other factors.

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(sleeplessness/insomnia) and various health and lifestyle factors on cognitive functioning. We found significant correlations between sleep duration, chronotype and cognitive performance in both cohorts, where normal sleep duration and intermediate and evening chronotypes were generally associated with better cognitive performance than short sleep duration and morningness type.

Our study showed an inverse relationship between morningness and cognitive performance in adults, contrasting with adolescent studies where morningness correlated with better health and mental well-being.[30] This disparity suggests a nuanced age-dependent impact of chronotype on cognition. Leng *et al* explored the complex interactions between circadian rhythms and neurodegenerative diseases, implying that the effects of morningness on cognitive health may evolve over the lifespan.[13] In older adults morningness might not confer the same cognitive advantages as those seen in younger populations, possibly reflecting age-related changes in circadian mechanisms and their influence on cognitive functions.

Notably, our strongest predictors of cognitive functioning also pertained to sleep duration. This aligns with previous studies, which underscored the importance of adequate sleep duration and regular sleep patterns for optimal cognitive functioning.[12 17–19] On the other end of the sleep duration spectrum, our results indicate that long sleep is a significant negative predictor for cognitive performance, consistent with prior research suggesting a U-shaped relationship between sleep duration and cognitive impairment.[12] Interestingly, our study did not identify a significant relationship between sleep quality—that is, sleeplessness/insomnia and cognitive performance—contrary to some previous findings.[31] This may be because the specific aspects of insomnia such as severity and chronicity as well as comorbid conditions need to be considered.

Health and lifestyle factors also provided considerable predictive utility. Age and diabetes consistently emerged as negative predictors of cognitive functioning across both cohorts. These findings are consistent with a vast body of research. The cognitive decline associated with increasing age is well documented.[32] Diabetes has been linked to cognitive impairment due to its potential to cause vascular and metabolic changes in the brain.[33] Despite our findings being significant only in Cohort 1, coronary heart disease and hypertension have been associated with an increased risk of cognitive decline.[34 35] Gender differences were evident, with women in both cohorts scoring lower on cognitive tests than men. This finding contradicts previous research suggesting that women might outperform men in specific cognitive tasks.[36] However, the interpretation of this finding should be cautious, considering the complexities of gender differences in cognition which may be modulated by a myriad of genetic, hormonal and societal factors.

The relationship between cognitive function and lifestyle factors such as alcohol consumption and smoking proved complex. Individuals who abstained from alcohol showed lower cognitive scores than those who consumed alcohol, conflicting with previous research that has connected moderate drinking with cognitive impairment.[37] Weekly and monthly alcohol consumption, as opposed to daily drinking, was found to somewhat correlate with lower cognitive scores. Interestingly, in a previous study in people in the age group 60–69 years, heavy alcohol drinking was significantly associated with a lower risk of cognitive impairment. Conversely, among individuals aged 70 and above, heavy alcohol drinking was associated with a higher risk of cognitive impairment.[38] This emphasises the complex relationship between alcohol consumption and cognitive function, which may vary across different age groups and consumption patterns. Similarly, current smoking in Cohort 1 is linked with lower cognitive performance, while ex-smokers in Cohort 2 outperformed never-smokers, suggesting a 'smoker's paradox' where former smokers perform better in cognitive tests.

Our study uses the extensive UK Biobank dataset to explore the complex interplay between sleep, lifestyle factors and cognitive performance. Key strengths include a large sample size and the implementation of robust statistical techniques, enhancing the reliability of our findings. Additionally, analysing two distinct cohorts allows for a broader understanding across different cognitive assessments. However, the study's cross-sectional design limits causal inferences, and residual confounding remains a possibility. Other limitations include reliance on self-reported data for sleep parameters, which may introduce biases. The study does not adjust for educational attainment, a factor potentially influential on cognitive performance and sleep patterns, due to incomplete data. Studies link depression and social isolation to an increased risk of cognitive decline[39 40] and physical activity to a reduced risk of age-related cognitive decline.[41] However, these parameters were not included to adjust the data related to the primary objective of our study which is the main limitation. This was purely due to the fact that the UK Biobank dataset does not all contain physical and social activity of all participants. Nevertheless, the covariates analysed in our study are consistent across similar studies.[12 19] Furthermore, the exclusion of unclear chronotype responses and the absence of time of day control for cognitive assessments may affect the generalisability and interpretation of our results. Finally, geographical factors and demographics of the UK Biobank's population may restrict the wider applicability of our findings.

To enhance future research it would be beneficial to include more diverse populations from different databases or geographical locations. Incorporating objective sleep measures would also provide a more accurate assessment of sleep parameters and their impact on cognitive performance.

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CONCLUSION

Our study underscores the multifaceted link between sleep parameters, health and lifestyle in cognitive performance, where sleep duration and chronotypes are strong predictors of cognitive performance in our study population. Being a woman, increasing age and having a diagnosis of angina, high blood pressure and diabetes also worsen cognitive performance, while alcohol and smoking have a more complex relationship. Our findings highlight the need for deeper exploration into these correlations, providing a foundation for tailored interventions to combat cognitive decline.

Future studies should adopt a longitudinal approach, incorporate more diverse populations, include objective sleep measures and delve into the biological mechanisms linking sleep duration with cognitive deterioration, thereby broadening the scope of cognitive functions.

Transparency

The lead author (RW) affirms that the manuscript is an honest, accurate and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned have been explained.

Acknowledgements This research has been conducted using data from UK Biobank, a major biomedical database in the UK.

Contributors JEP, SWL, RW and DM conceptualised the study. All authors were involved in the drafting, reviewing and editing and all authors have read and approved the final manuscript. The corresponding author (DM) attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.The corresponding author (DM) is also responsible for the overall content as a guarantor.

Funding This study is funded by the Korea Institute of Oriental Medicine, 1672 Yuseong Daero, Yuseong-gu, Daejeon, Korea (KSN20234113) in collaboration with Imperial College London.

Competing interests All authors have completed the ICMJE uniform disclosure form at http://www.icmje.org/disclosure-of-interest/

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval The UK Biobank study was approved by the National Health Services' National Research Ethics Service.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data may be obtained from a third party and are not publicly available. Full study data cannot be openly shared under the material transfer agreement with UK Biobank. Prospective researchers can apply for access to the UK Biobank data from www.ukbiobank.ac.uk.

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