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# The relationship between chronotype and intelligence: the importance of work timing

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Sleep-wake patterns show substantial biological determination, but they are also subject to individual choice and societal pressure. Some evidence suggests that high IQ is associated with later sleep patterns. However, it is unclear whether the relationship between IQ and later sleep is due to biological or social effects, such as the timing of working hours. We investigated the association between habitual sleep timing during work days and work-free days, working time and membership in Mensa, an organization of highly intelligent individuals ( $IQ \geq 130$ ) using a sample of 1,172 adults split between Mensa members and age- and sex-matched volunteers from a large web-based database. We found no difference in chronotype, and the later sleep timing of Mensa members on work days was fully accounted for by later work start times. Our results indicate that later sleep timing in those with higher IQs is not due to physiological differences, but rather due to later work schedules. Later working times and the resulting lower social jetlag may be one of the reasons why higher IQ is associated with lower prospective morbidity and mortality.

Sleep-wake patterns depend on genetic effects, social pressure and previously accumulated sleep debt<sup>1</sup>. Previous research has established a potential link between sleep-wake timing or chronotype (phase of entrainment to the day-night cycle) and IQ, but it is unclear whether this effect is biological or social—that is, whether more intelligent people are biologically (most likely genetically) predisposed to sleep at a different time, or whether this is a consequence of environmental factors. A large study<sup>2</sup> of 15,197 respondents from the Add Health longitudinal study showed a small but significant linear relationship between childhood IQ and self-reported bedtimes and wake-up times between the ages of 18–28 years ( $r = 0.013–0.053$ ). A meta-analysis of the literature up to 2011<sup>3</sup> excluding the Kanazawa *et al.* study found a similar association between diurnal preference and cognitive ability (morningness:  $N = 2,177$ ,  $r = -0.042$ ; eveningness  $N = 1519$ ,  $r = 0.075$ ), with no evidence for publication bias. Some recent work, however, found weak positive associations between an earlier chronotype and intelligence in school-age children and adolescents<sup>4–6</sup>. Additionally, very large genome-wide association studies<sup>7,8</sup> have also revealed that cognitive ability and morningness are in a negative genetic correlation—that is, genetic variants associated with an earlier chronotype are also associated with lower cognitive ability ( $r_g = -0.15–0.17$ ). Paradoxically, academic performance, itself moderately correlated with IQ<sup>9</sup>, is positively correlated with morningness<sup>3,10</sup>.

Almost all previous research was conducted with adolescents or young adults, and most studies did not systematically measure work day and free day sleep-wake preferences and lifestyle factors which may influence this relationship. Specifically, it is currently unknown whether the relationship between IQ and sleep-wake timing reflects individual differences in the biological chronotype or rather in social influences (such as different work and school schedules or differences in the use of stimulants or hypnotics). Even genetic studies are not exempt from this, due to the not always causative nature of genetic correlations<sup>11</sup>.

The goal of our study was to specify to what extent sleep time differences as a function of IQ are mediated by the different social environments of high-IQ individuals. We assessed the IQ–sleep timing relationship using a sample of working age, high-IQ adults and age- and sex-matched controls with data about both work day and free day sleep timings and work schedules. We compared the self-reported sleep-wake patterns of adult German Mensa ( $IQ \geq 130$ ) members to an age- and sex-matched random sample of the MCTQ database<sup>12</sup> ( $N_{total} = 1172$ ,

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		N (Control)	N (Mensa)	M (Control)	M (Mensa)	SD (Control)	SD (Mensa)	Cohen's <i>d</i>	<i>p</i>
Work day	Age (years)	586	569	38.17	38.22	10.66	10.74	<0.01	0.94
	Bedtime (h <sub>midnight</sub> )	577	518	-0.69	-0.65	1.39	1.28	-0.03	0.67
	Sleep preparation (h <sub>midnight</sub> )	577	518	-0.56	-0.31	1.21	1.28	-0.20	<0.001
	Sleep latency (min)	576	517	16.99	13.36	16.92	15.32	0.22	<0.001
	Sleep onset (h <sub>midnight</sub> )	577	517	-0.27	-0.09	1.25	1.32	-0.14	0.02
	Wake time (h <sub>midnight</sub> )	578	516	6.69	6.91	1.21	1.31	-0.17	<0.001
	Waking inertia (min)	574	517	15.98	15.41	16.54	17.08	0.03	0.57
	Sleep duration (h)	577	518	6.98	6.99	1.07	1.07	-0.01	0.89
	Midsleep (h <sub>midnight</sub> )	576	517	3.21	3.40	1.10	1.22	-0.17	0.01
Free day	Bedtime (h <sub>midnight</sub> )	585	518	0.44	0.28	1.74	1.44	0.10	0.11
	Sleep preparation (h <sub>midnight</sub> )	585	518	0.54	0.58	1.66	1.44	-0.02	0.69
	Sleep latency (min)	580	517	13.92	11.61	14.53	13.17	0.17	0.01
	Sleep onset (h <sub>midnight</sub> )	574	517	0.71	0.76	1.51	1.48	-0.04	0.56
	Wake time (h <sub>midnight</sub> )	570	515	8.67	8.75	1.71	1.72	-0.04	0.48
	Waking inertia (min)	578	512	19.40	24.19	18.87	22.58	-0.23	<0.001
	Sleep duration (h)	571	518	8.03	7.98	1.48	1.32	0.03	0.59
Midsleep (h <sub>midnight</sub> )	573	518	4.77	4.77	1.63	1.51	0.02	0.94	
Chronotype (corrected midsleep, h <sub>midnight</sub> )	Chronotype (corrected midsleep, h <sub>midnight</sub> )	551	485	4.30	4.32	1.39	1.37	-0.02	0.75
	Light exposure (work day, h)	571	516	2.54	1.12	1.93	0.97	0.92	<0.001
	Light exposure (free day, h)	574	517	3.37	2.06	1.86	1.31	0.81	<0.001
	Social jetlag (h)	551	485	1.58	1.39	1.06	0.97	0.19	<0.001
	Social jetlag (h), corrected for oversleeping	552	485	1.23	1.14	0.87	0.79	0.11	0.08
	Sleep deprivation/week (h)	552	484	1.97	1.61	1.85	1.52	0.21	<0.001
	Workdays/week	564	485	4.92	5.16	0.77	0.75	-0.31	<0.001

**Table 1.** A comparison of Mensa and control samples in terms of chronotype-related and miscellaneous variables. Statistical significance is derived from independent sample *t*-tests. Bold *p*-values indicate differences which were significant after correcting for multiple comparisons. Effect sizes are reported in Cohen's *d*, and positive signs correspond to higher values in controls. h<sub>midnight</sub> indicates that the value is a time point expressed in fractions of hours before (negative) or after (positive) midnight. Chronotype refers to free day midsleep corrected for oversleeping.

mean age: 38.2 years). In doing so, we investigated differences in sleep timing on work days and weekends separately in order to separate the preferred chronotype from the effects of socially pressure on sleep-wake timing<sup>1</sup>, and specifically investigated the role of work schedules and additional lifestyle variables.

## Results

We compared the self-reported sleep-wake timing of adult German Mensa (IQ > 130) members to an age- and sex-matched random sample of the MCTQ database<sup>12</sup> ( $N_{\text{total}} = 1172$ ).

Mensa members were characterized by later lights-off, sleep onset midsleep and wake-up times as well as shorter sleep latency, but only during work days. During free days, Mensa members still experienced less sleep latency and longer waking inertia (time in bed after waking up), but there was no difference in the main measures in sleep timing. Mensa members suffered from less social jetlag (but not after correcting for oversleeping) and less weekly sleep deprivation, despite having slightly more work days on average. Importantly, Mensa members had nearly a standard deviation less light exposure, both on free days and working days. Group averages, sample sizes, standard deviations as well as *t*-values and significance levels are reported in Table 1.

So far, our results were in line with some of the literature indicating generally later sleep timing among higher IQ adult participants; however, this difference was limited to work days and not present in the corrected chronotype. Next, we used linear regression models to test whether later work day sleep patterns were accounted for by non-endogenous variables, including workplace characteristics (work start and commute duration) and substance use (consumption of alcohol, cigarettes, caffeine, and hypnotics) (Table 2).

Model	Independents	$\beta$	$p$
1	Mensa	0.096	0.009
2	Mensa	0.036	0.204
	Work start time	0.626	<0.001
	Commute duration	-0.150	<0.001
3	Mensa	0.044	0.116
	Work start time	0.630	<0.001
	Commute duration	-0.143	<0.001
	Cigarettes/day	0.094	0.002
	Beer/day	-0.010	0.732
	Glass wine/day	-0.023	0.426
	Liquor/day	0.032	0.252
	Coffee/day	0.014	0.624
	Caffeine drink/day	0.071	0.014
	Tea/day	-0.023	0.404
Hypnotics/day	0.023	0.403	

**Table 2.** Results of linear regression models with work day midsleep timing as the dependent variable. Positive standardized regression coefficients for “Mensa” indicate later midsleep timing in Mensa members. Model 3 incorporates the self-reported number of various alcoholic beverages, coffee and other caffeinated beverages, cigarettes and hypnotics medication consumed on a typical day.

	$t$	$df$	$p$	Mean Difference	SE	95% CI	
						Lower	Upper
Work start time	-2.33	751.00	0.02	-0:13:59.63	0:06:00.957	-0:25:48.23	-0:02:11.02
Commute duration	0.97	756.00	0.33	1.41	1.46	-1.46	4.27
Cigarettes/day	3.44	806.00	0.00	1.27	0.37	0.55	2.00
Beer/day	2.57	807.00	0.01	0.17	0.07	0.04	0.31
Glass wine/day	0.69	807.00	0.49	0.02	0.03	-0.04	0.09
Liquor/day	0.05	807.00	0.96	<0.01	0.01	-0.02	0.03
Coffee/day	2.79	807.00	0.01	0.35	0.13	0.11	0.60
Caffeine drink/day	-1.96	807.00	0.05	-0.16	0.08	-0.32	0.00
Tea/day	-1.54	807.00	0.12	-0.16	0.11	-0.37	0.04
Hypnotic/day	-1.11	807.00	0.27	-0.01	0.01	-0.03	0.01

**Table 3.** Mensa vs. Control differences in covariates potentially influencing sleep timing. Negative effect sizes indicate higher values in Mensa members. Commute duration is given in minutes. Respondents could provide their consumption of psychoactive substances in monthly, weekly or daily quantities which was always converted to daily values.

Data on all variables were available for 480 Mensa members and 271 controls. Supplementary Table S1 reports descriptive statistics for all covariates by subgroup and Table 3 reports subgroup differences. Supplementary Table S2 provides the correlation matrix of covariates.

Work start timing and commute duration were the strongest predictors of work day midsleep timing, and later work start times ( $M_{\text{Mensa}} = 8:35$ ,  $SD = 1:18$ ;  $M_{\text{Control}} = 8:21$ ,  $SD = 1:21$ , values indicating hours and minutes) among Mensa members alone fully mediated the association between Mensa membership and work day midsleep timing (Table 2).

In sum, we found no statistically significant differences between the chronotype of highly intelligent individuals and matched controls. Sleep timing differences on work days were fully accounted for by the later work schedules of Mensa members.

## Discussion

We assessed sleep timing differences between highly intelligent subjects and matched controls. We did find later midsleep timing and waking time in Mensa members, but this was limited to work days, and even then, it was fully accounted for by later work start times. We found no subgroup difference in chronotype. Thus, it appears that at least in the current sample earlier sleep timing in those with lower IQs is not due to physiological differences, but rather a function of earlier work schedules. Much higher levels of both working day and free day light exposure was found in control participants. While our database does not allow the empirical investigation of the causes of this, we speculate that since IQ is associated with job prestige<sup>13</sup>, extremely intelligent individuals may be overrepresented in office jobs and underrepresented in outdoor jobs, such as agriculture or construction. Free-day differences might be explained by the well-documented<sup>14–17</sup> concentration of better educated or more intelligent individuals in urban areas, where outdoors activities are potentially less available or less attractive.

In any case, despite large differences in self-reported light-exposure, subgroup differences in sleep timing were modest and limited to work days.

Our findings are partially at odds with some of the previous literature, which generally reported negative associations between an early chronotype and intelligence in young adults<sup>2,3</sup>, including pleiotropic genetic effects working in opposite directions<sup>18</sup>. This discrepancy may be accounted for by two study design features which were uniform in most of the previous literature but differed in ours. First, all previous studies used adolescent or very young adult ( $\leq 25$  years old) samples, while in ours, participants (38 years on average) were closer to middle age. Second, all studies referenced in the Preckel *et al.* meta-analysis used questionnaires (typically the MEQ, the LOCI [Lark-Owl Chronotype Indicator], or the CSM) to obtain a single measure of morningness and/or eveningness. These questionnaires—while containing questions about both preferred and usual activity patterns and being correlated with other measures such as the MCTQ<sup>19</sup>—do not strictly separate work day and weekend habits, in contrast to the MCTQ used in our study. Combining work day and weekend sleep timing into a general chronotype measure offers a limited opportunity to distinguish individual differences due to biological preferences (most prominent on weekends) and those due to societal pressure (most prominent on work days). Notably, the study of Kanazawa *et al.*<sup>2</sup>, which enquired about work day and free day bedtimes separately, is an exception to this. This contradiction might arise from several factors, including self-report inaccuracy (as wake times were quite late, on average 10:00 AM or later on weekends and later than 7:00 AM on work days, with some inaccuracies such as frequent 12:00 PM [noon] bedtimes manually corrected by the authors) and age-specific effects as all respondents were very young ( $< 30$  years old). Further possibilities are that IQ–sleep timing associations are culture-specific, and while brighter individuals might indeed prefer sleeping later in the United States, they do not do so in Germany, supporting the role of non-endogenous environmental factors such as differences in societal pressure in creating a later chronotype in brighter individuals. A similar caveat applies to the evidence for a negative genetic correlation ( $r_g = 0.15\text{--}0.17$ ) between morningness and IQ<sup>18</sup>. The genetic correlation between morningness and IQ is based on summary genetic association data from a chronotype GWAS<sup>7</sup>, in which morningness was assessed by respondents' self-report of being a "morning/evening person" on a subjective Likert-type scale. Genetic correlations do not necessarily implicate a common biological mechanism. They may arise from biological pleiotropy if the same genes are implicated in the actual biological processes regulating two phenotypes, but they may also arise from mediated pleiotropy, in which one trait is caused by an environmental influence which in turn is caused by the other, heritable trait<sup>20</sup>. Our results suggest that the negative genetic correlation between morningness and IQ may be a case of mediated rather than biological pleiotropy: genetic effects resulting in higher IQs do not cause lower morningness because these traits share their biological underpinnings, but because heritable high intelligence exposes individuals to environments – such as later or more permissive work schedules—in which an early sleep-wake pattern is less preferred. Future studies using Mendelian Randomization (MR) on genetic data may test this hypothesis, which would be supported by a unidirectional causal path from IQ-related genetic variants to sleep timing. We are aware of one such study<sup>21</sup>, which found minimal causal influence of education on chronotype using an education polygenic score which is also associated with intelligence<sup>22</sup>.

Our study suffers from a number of limitations. First, due to its cross-sectional design we had a limited ability to assess causation. Because IQ is stable throughout the lifetime<sup>23</sup> and highly heritable<sup>24</sup>, and employees are usually not free to choose when their work day starts, we believe that it is more likely that the correlation between IQ and sleep timing as well as work start and sleep timing reflects the effect of the former on the latter than vice versa, but only longitudinal designs could test this reliably. Second, our sample may have been underpowered to detect very small associations between IQ and free day sleep timing or chronotype. Third, we did not have IQ data available from our control sample, therefore, we are unable to perform fine-grained analyses within the normal IQ range or quantitatively assess the effects of intelligence range extension resulting from comparing a highly selected sample to random volunteers. Because participation in the control group required being interested in and filling out an internet-based questionnaire, it is likely that the average IQ of our control group was above the population average as volunteer databases usually are<sup>25,26</sup>. Fourth, it must be emphasized that the relationship between sleep timing and intelligence was – in line with previous results<sup>3</sup>—modest, amounting to a weekday mid-sleep timing about 14 minutes later and approximately 19 minutes less social jetlag per an approximately 1–2 SD IQ difference, assuming a Mensa mean higher than 130 and a control group mean above 100 but not exceeding 115. Fifth, Mensa members may not be fully representative for all individuals with an exceptionally high IQ. However, because selection into this organization is based purely on IQ, we believe that any associations between intelligence and chronotype should show up in the comparison of this sample with controls barring the very specific case when Mensa applicants are also systematically different on some other variable which has exactly the opposite relationship with chronotype as intelligence.

Overall, our analysis of an adult sample suggests that highly intelligent individuals do not have a biological preference for a later chronotype: while they do go to bed, sleep, and wake up later on work days, this effect is fully mediated by later working times and not present on free days or in the corrected chronotype. Highly intelligent individuals also suffer lower social jetlag, although this effect did not reach statistical significance once oversleeping was controlled for. Thus, the effect of intelligence on chronotype seems to have less to do with biological differences than with different work-related environmental pressures on sleep schedules. Eveningness and specifically social jetlag has negative effects on overall morbidity and mortality, including leading causes of death<sup>27,28</sup>, high IQ, however, is associated with a lower chance of all-cause mortality<sup>29</sup> and morbidity<sup>30,31</sup>. Our results suggest that—although the size of the effects is modest—reduced social jetlag in those with late chronotypes due to later or more permissive work schedules might be one of the factors mediating the protective role of high IQ against morbidity and mortality. Because our results indicate that these risk differences are the result of lifestyle factors rather than biological differences, policies enabling a better alignment of desired and actual work schedules could be beneficial to individuals not possessing high intelligence.

## Methods

**Sample.** We recruited 586 members of the German Mensa Society, an organization where a standardized IQ test score at least two standard deviations above the mean ( $IQ \geq 130$ ) assessed by trained testers and evaluated by a professional psychologist is a prerequisite for membership. As control, we selected a random sample of the same number ( $N = 586$ ) of age- and sex-matched controls from the main MCTQ database<sup>12,32</sup>. There were 232 females and 354 males in each subgroup with a mean age of 38.2 years (see Tables 2 and 3 for more details on subgroups and subgroup differences). The study was approved by the ethics committee of the University of Munich and all participants gave informed consent accordingly. All research was performed in accordance with the relevant guidelines and regulations.

**Materials.** All participants filled out the MCTQ questionnaire<sup>32</sup>, which contains questions about basic socio-demographic data (age, sex, place of residence, height and weight), other occupational and behavioral data (number of work days per week, working hours, light exposure on work and free days) as well as about sleep habits (bedtime, time of lights-off, time to falling asleep, wake-up time and the habitual time of getting out of bed) for both work days and free days separately. From the responses, sleep duration and midsleep (the mid-point between sleep onset and sleep end) on work and work-free days, social jetlag (absolute midsleep difference between work days and free days), weekly sleep deprivation (the difference between the weekly average sleep duration and work day sleep duration, multiplied by the number of work days) and chronotype-proxy MSFsc (free-day midsleep corrected for sleep debt accumulated during the work week) were computed. We also collected information on work start times, commute time, and the consumption of legal substances acting on the central nervous system (i.e., cigarettes, alcohol, coffee, other caffeinated beverages, and hypnotics).

**Analyses.** We calculated the differences between Mensa members and matched controls using independent-sample *t*-tests. Multiple comparisons were controlled for using the Benjamini-Hochberg method of false detection rate (FDR) correction<sup>33</sup>. IBM SPSS Statistics Version 20 was used to test the confounding effects of extraneous variables on Mensa-control differences in sleep-wake timing with hierarchical linear regression models. The absence of missing data in the MCTQ was not a prerequisite for inclusion in all analyses. For this reason, missing data were found in some subjects, and the final sample sizes for each variable are reported in the Results and Table 1.

## Data availability

The datasets generated and/or analysed during the current study are available from the corresponding author on request.

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## Author contributions

M.D. and T.G.B. designed the study and performed data collection. P.U. and R.B. performed statistical analyses. All authors wrote and reviewed the manuscript.

## Competing interests

The authors declare no competing interests.

## Additional information

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