

The Role of Genetic Predisposition for Chronotype in Academic Performance

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The Role of Genetic Predisposition for Chronotype in Academic Performance*

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Abstract

Does chronotype affect academic performance? Chronotype is an expression of a person's circadian rhythm. The combination of its biological variation across individuals with rigid social constraints inevitably results in different degrees of alignment between the biological and the social clock, potentially altering efficiency when performing tasks. Using data from Add Health, which combines official high school transcripts with DNA-based information, this paper examines whether the genetic predisposition for a morning-oriented chronotype affects high school GPA. Exploiting the natural experiment of random genetic inheritance among full siblings, I estimate causal effects. Results indicate that, holding the genetic predisposition for educational attainment fixed, a higher propensity for morningness has a positive and statistically significant impact on high school GPA. Findings suggest that this enhancing effect derives from a closer synchronisation between their biological and the social clocks.

JEL: I10, I21, J24, J13, I14.

Keywords: chronotype; genetics; polygenic index; academic performance; GPA.

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1 Introduction

Chronotype reflects an individual's biological clock and the underlying circadian rhythm. The term was originally coined in the 1970s to define "the temporal phenotype of an organism" [Ehret, 1974], as already at that time research had documented substantial variation in the *timing* of human sleep-wake cycles [Horne and Ostberg, 1976]. The well-known labels "early birds" and "night owls", for instance, respectively denote individuals who naturally wake up and fall asleep early, but also peak in mental and physical functioning in the morning, versus individuals with a delayed circadian phase, who naturally wake up, are mostly alert and fall asleep later in the day. More generally, chronotype has been conceptualised as a continuous trait spanning from morningness to eveningness, with early birds and night owls at the extremes and most individuals falling in between - sometimes categorised as intermediate types [Roenneberg et al., 2003].

This natural variation in biological clocks, in a society with rather rigid schedules, implies that some individuals are inevitably required to work outside their optimal timing, therefore experiencing reduced efficiency when completing tasks compared to individuals with similar abilities but a chronotype that better aligns with the regular social constraints. For example, in academic settings, the typical morning timetables are likely to advantage students with a natural predisposition for morningness, while creating an obstacle to the performance of more evening-oriented ones.

The purpose of this study is to bring evidence on this. Specifically, I aim to answer the question: does chronotype affect school performance? To do so, I use data from Add Health, a longitudinal study of a representative sample of US adolescents that uniquely integrates official high school transcripts with DNA-based information. I characterise chronotype through a polygenic index (PGI) for morningness, a summary measure of the genetic propensity for this trait that combines the explanatory power of all the measured genetic variants [Becker et al., 2021], and assess its effect on high school GPA.

Extensive research has shown that chronotype has a considerable genetic basis [Jones et al., 2019a,b, Jansen et al., 2019, Merikanto et al., 2018], which supports the use of a PGI as a meaningful measure of this trait. Identification relies, in the first place, on the fact that genes are fixed at birth, making the effect of any polygenic index immune to reverse causality. Nonetheless, naively estimated PGI coefficients would capture not only genetic endowment, but also the environment they may be correlated with - for instance through the vertical genetic inheritance from parents, who shape the rearing environment, or due to assortative mating and population stratification. Hence, to address residual confounding potentially arising from these forms of gene-environment correlations, I exploit a natural experiment occurring

within families: among full siblings, who share the same biological parents, these sources of confounding are cancelled out and genetic inheritance is a random lottery, thereby allowing identification of causal effects.

Moreover, extensive research demonstrated that not only "biological", but also "social" trait are heritable. First and foremost, educational attainment has been proved to be determined by a non-negligible genetic component, and its corresponding polygenic index to be a strong predictor of several academic outcomes [Lee et al., 2018, Okbay et al., 2016, 2022, Becker et al., 2021, Sanz-de Galdeano and Terskaya, 2023, Muslimova et al., 2024]. Thus, to better isolate the role of the genetic predisposition for chronotype, all estimations control for the educational attainment (EA) PGI. This allows to compare individuals with the same genetic predisposition for educational attainment, but with a genetic predisposition for different chronotypes.

Results indicate that, all else equal, a higher genetic propensity for morningness has a positive and significant effect on high school GPA. As a quantification, within-family estimates reveal that a 1 SD increase in the morningness PGI raises GPA by 0.201 SD, or 0.168 points, which is equivalent to a 6% gain relative to this sample's mean. A comparison of early birds and night owls (defined as individuals with extreme morning and night predisposition, respectively) with the intermediate chronotype reveals that the effect is primarily driven by an advantage of early birds, whose circadian rhythm better aligns with school schedules, whereas the performance of night owls is not statistically different from that of intermediate types. This result is not confined to specific settings, but it is persistent across high school years and extends across academic subjects, indicating that the advantage of an early chronotype is widely spread. Moreover, heterogeneity analysis reveals that it is more pronounced at the lower end of the GPA distribution, with little to no impact at the top.

To better understand this main result, I explore potential mechanisms. Early chronotypes may sleep longer; or, thanks to reduced tiredness, exert more effort, behave better in school, engage in a different use of time; be driven by higher learning or achievement goals; or they could evoke either reinforcing or compensatory parental responses. Yet, I find no evidence that genetically-predicted chronotype is associated with differences in effort, academic ambition and expectations, time use, or parental investment, suggesting these are unlikely to be the key channels mediating the main effect. Instead, students with a higher genetic propensity for morningness are more likely to perceive their sleep as sufficient, despite having sleep durations similar to those of their night-orientated peers. This distinction isolates the effect of chronotype from sleep quantity, and suggests that morningness itself - rather than longer rest - enhances performance. Indeed, while night owls can compensate for early school starts by anticipating bed time, and thus sleeping as much as early birds, their alertness peak still remains misaligned from school schedules, impairing

their efficiency. This provides suggestive evidence in support to the main hypothesis, namely, that the performance-improving effect of morningness propensity stems from better alignment of biological and school clocks. Also, students with a higher genetic predisposition for morningness report fewer difficulties in their interactions with teachers. Although the direction of causality between relationships with teachers and GPA is unclear, this association might suggest that differences in alertness levels depending on chronotype can be noticed by teachers, who react with different responses, with beneficial influences on grades.

High school is a crucial period of life, as it marks the starting point of future trajectories. Therefore, such results on grades may have important long-term implications. Nonetheless, despite its robust effect on GPA, the genetic predisposition for a morning-oriented chronotype does not seem to influence completed years of education later on. This finding may indicate that, differently from EA, which has a wide influence on various academic-related outcomes, the predisposition for chronotype affects performance through some channels, such as short-term efficiency, but not others that are plausibly relevant for long-term results, such as learning and achievement goals - as found in the mechanisms analysis. Alternatively, this result may be explained by psychological theories arguing that later chronotype individuals develop coping strategies and skills to deal with daily challenges stemming from unfavourable social constraints [Preckel et al., 2011]. Yet, without available data, this hypothesis remains within the realm of speculation.

Motivated by the mismatch between early school schedules and adolescents' circadian rhythms, previous economic research explored how adjustments to school start times can affect human capital and academic outcomes, demonstrating that later start times, by better aligning with students' biological needs, can boost performance at different educational stages, but especially among older teenagers [Carrell et al., 2011, Heissel and Norris, 2018, Groen and Pablonia, 2019, Diette and Raghav, 2017, Albrecht et al., 2026]. Nonetheless, given the substantial genetic variation in biological rhythms, the optimal schooling time is likely to vary across individuals. This study speaks to this body of research and adds to it by taking a different perspective. Rather than leveraging variation in school schedules, I exploit the individual genetic variation in circadian timing and assess its effect on performance within a given school setting. Although chronotype is new in economic research,¹ its importance, also for education, has been underscored in various disciplines, including medicine, biology, psychology, and neuroscience [Enright and Refinetti, 2017, Beşoluk et al., 2011, Bettencourt et al., 2024, Escribano and Díaz-Morales, 2016, Preckel et al., 2013, Zerbini and Merrow, 2017]. Overall, studies provide evidence of a positive association between

¹Two exceptions are Bonke [2012] and Conlin et al. [2023]. However, they focus on labour market outcomes and rely on sleep patterns or questionnaire-based measures of chronotype, which are arguably endogenous.

an early chronotype and academic performance, which is attributed to a "synchrony effect" - in line with what I find.

Thus, I contribute to research by bringing up chronotype as a novel determinant of economic outcomes and combining causal inference techniques with genetic data to identify its causal effect on GPA.

Indeed, another contribution of this study is to the growing field of research in geno-economics, or social-science genomics. Leveraging the increasing availability of DNA-based information in large datasets, enabled by recent advances in molecular genetics, this stream of research integrates genetic data into traditional economic analyses, in order to better understand the links between genetic differences and socio-economic factors. However, for what concerns academic outcomes, most studies have focused on the genetic propensity for educational attainment [Biroli et al., 2026, Muslimova et al., 2024, Arold et al., 2025], and less frequently for cognitive and non-cognitive skills [Buser et al., 2024]. This study extends the literature by bringing in another trait that has thus far been overlooked and, on top of attainment predisposition, plays a role in academic performance.

The rest of this paper is structured as follows. First, [Section 2](#) provides a general definition of PGIs, which in this work underlies the measurement of chronotype, together with key information for the identification and interpretation of PGIs' effect. Next, [Section 3](#) describes the data and [Section 4](#) the empirical strategy adopted in the analysis. [Section 5](#) contains the main results, tests their robustness and explores heterogeneity, potential mechanisms and other performance outcomes. Finally, [Section 6](#) concludes.

2 Genetics fundamentals and Polygenic Indexes

2.1 Key insights of genetic structure

The human genome comprises roughly 21,000 genes grouped into 23 pairs of chromosomes, with one chromosome in each pair randomly inherited from the two of the mother and the other from the two of the father. In turn, each chromosome is made up of two bounded DNA strands, composed of sequences of nucleotides - adenine (A), thymine (T), cytosine (C) and guanine (G) - always coupling in a complementary manner (A with T, C with G) to form a total of about 3 billions base pairs [Burt, 2024].

Over 99% of the human genome is shared by all individuals. Yet, two unrelated persons picked at random are estimated to differ in 3.5 to 4.5 million genetic variants - called polymorphisms [Burt, 2024]. The simplest and most common form of genetic variation is called single nucleotide polymorphism (SNP): a substitution, in a given location, of a reference nucleotide for another (e.g., C instead of A).² SNPs represent approximately 87% of the variants [Burt, 2024]. An individual's genotype, at any location of the genome is conventionally described through the two bases inherited from the parents on the reference DNA strand (e.g., AA or CG). Alternative nucleotide variants, at each position, are named alleles, and the less frequent version in a population is known as the minor allele. Therefore, practically, individual genotype can be expressed as a vector of minor allele counts (0, 1 or 2) for each location of the genome.

Recent expansions in large datasets containing comprehensively genotyped individuals have accelerated understanding of the relationships between specific genetic variants and phenotypes. Research has increasingly shown that trait heritability is not limited to anthropometric characteristics and diseases, but also extends to behavioural and socioeconomic outcomes [Baselmans et al., 2019, Lee et al., 2018, Karlsson Linnér et al., 2019, Liu et al., 2019, Okbay et al., 2022], and that nearly all measured traits are polygenic [Visscher et al., 2017].³

2.2 Genome-Wide Association Studies and Polygenic Indexes

To uncover the genetic basis of phenotypes, modern research largely employs Genome-Wide Association Studies (GWAS), which systematically scan the genome to identify associations between SNPs and a trait of interest, producing estimates. Despite a substantial growth in both the number and the sample size of available datasets, the max-

²Figure B1 provides an illustration.

³A phenotype, or trait, is any measurable characteristic of an organism. A phenotype is defined as monogenic if it depends on variation of a single gene; while it is defined as polygenic, or complex, if it is affected by multiple genetic variants, rather than by a single gene.

imum number of genotyped individuals - up to few million - is still far smaller than the total number of measured SNPs, making it infeasible to simultaneously estimate the effect of all SNPs on a phenotype y through multivariate regressions. Therefore, GWASs rely on separate univariate regressions for each j SNP $\forall j = 1, \dots, J$, typically conditioning on a vector C of controls to adjust for basic demographic differences between population subgroups.

$$y_i = \hat{\beta}_j^{GWAS} SNP_{ji} + \alpha_j C_i + \epsilon_{ji} \quad (1)$$

The coefficients in the vector $\hat{\beta}^{GWAS} = (\hat{\beta}_1^{GWAS}, \hat{\beta}_2^{GWAS}, \dots, \hat{\beta}_J^{GWAS})$, called GWAS summary statistics, represent the estimated associations of each variant j with the phenotype y in the discovery sample, and can be used as weights to construct DNA-based predictors of that phenotype in an independent prediction sample, as follows

$$\widehat{PGI}_i = \sum_{j=1}^J \hat{\beta}_j^{GWAS} X_{ij} \quad (2)$$

In [Equation \(2\)](#), X_{ij} is the minor allele count (0, 1 or 2) of SNP j for individual i , and $\hat{\beta}_j$ is the association between the SNP j and the trait y estimated in [Equation \(1\)](#).⁴

A polygenic index (PGI) is thus a measure of individual genetic predisposition for the phenotype of interest, calculated as a weighted sum of allele counts for each SNP, with weights derived from GWAS summary statistics.⁵ In sum, the PGI is an empirical proxy for the "additive SNP factor" and captures the combined explanatory power of all measured SNPs.

2.3 Identification and Interpretation of PGIs effects

The random transmission of one of the two alleles by each parent at each location of the genome through Mendelian segregation represents a source of exogenous variation that can be exploited in empirical analyses. Moreover, the predetermined nature of genes with respect to any life event completely rules out reverse causality. However, causal interpretation of PGIs is complicated by correlations between genetic endowment and the environment, which can confound estimated effects [[Biroli et al., 2026](#), [Benjamin et al., 2024](#)]. In particular, passive gene-environment correlations consist in associations between an individual genotype and the external environment, despite no influence of genetic markup on the other. For example, the vertical genetic transmission from parents to offsprings creates a correlation between the inherited genetic endowment and the rearing environment chosen and shaped by parents (in-

⁴[Figure B2](#) offers an illustrative example of a PGI construction.

⁵I acknowledge that "genetic predisposition" or "genetic propensity" would be more correct. Yet, I would henceforth not use quotation marks to ease readability.

cluding parents themselves) - a phenomenon known as "genetic nurture".⁶ Other forms of confounding are assortative mating and population stratification. The former is a type of non-random mating (mostly phenotypic selection in this setting), which results in a non-random inheritance of genetic variants that correlates with the environment. The latter refers to the environmental separation of population or ancestry subgroups, which may result in associations between genetic and phenotypic differences due to cultural or environmental reasons, even without genetic effects. Standard strategies to address confounding include controls for a set of principal components (PCs) of the population structure with other demographic and socioeconomic variables, but are still imperfect methods that do not fully remove these biases in simple analyses of the relationship between a PGI and an outcome.

Nevertheless, a natural experiment occurring within family overcomes these obstacles: among full siblings, born from the same pair of biological parents, genetic inheritance is a perfect random lottery and the transmission of a genetic variant versus another is as good as random. With family-fixed effect absorbing the whole common environment that siblings share, this strategy allows to eliminate those forms of confounding and to cleanly identify the causal PGI effect.

A final point on the interpretation of PGI effects deserves mention. Beyond the passive, two other types of gene-environment correlations exist: active, where individuals self-select into environments that align with their genotypes, and evocative, where certain genotypes triggers specific environmental responses. Both forms would persist even after accounting for family-fixed effects, and may be captured in the estimated PGI coefficients. However, since in either case the environment is influenced by an individual's genotype, this is part of the causal effect aimed to be captured, and to some extent they will be examined in the mechanism analysis (in [Section 5.5](#)).

⁶For example, if parents establish structured routines that align with their own chronotype (i.e., morning-oriented parents organising early schedules, and night-oriented parents promoting evening routines to effectively manage daily responsibilities), genetic nurture would result into a positive gene-environment correlation, upward biasing the estimated effect of the offspring PGI. Conversely, if parents implement compensatory routines (e.g., encouraging night-oriented children to adapt to standard daytime schedules to help them develop those habits, or if morning-oriented parents are available only in the evening), genetic nurture would bias the PGI effect downward due to a negative gene-environment correlation.

3 Data

3.1 The National Longitudinal Study of Adolescent to Adult Health (Add Health)

Data for the analysis come from the National Longitudinal Study of Adolescent Health (Add Health), a longitudinal study designed to follow a representative sample of US adolescents along their transition to adulthood [Harris et al., 2019]. The core sample, composed of 20,745 adolescents enrolled in grades 7-12 (ages 12-20) in 1994-95, was formed from a stratified group of 80 high schools and their feeder schools, through school rosters and an in-school survey. These participants were interviewed at home in Wave I and in follow-up surveys in Wave II (1996), Wave III (2000-2001), Wave IV (2008) and Wave V (2016-2018). In-home interviews encompassed a wide range of topics, including education, lifestyle, family relationships, social networks, and personal attitudes. Additionally, several supplemental datasets were added throughout the waves. In this analysis I leverage three of them.

First, the Adolescent Health and Academic Achievement (AHAA) data: official school transcripts obtained during Wave III fieldwork directly from the high schools attended by respondents, regardless of graduation.⁷ These records contain detailed information on, amongst all, course-taking histories, curricular content, failure trajectories, and grades (GPA). This is the source of information for the outcome variable.

Second, biological data. The biological data collection operated in Wave IV included DNA-based information for consenting participants. From this, the Social Science Genetics Association Consortium (SSGAC) created a set of Polygenic Indexes (PGIs) for numerous traits with high prediction accuracy [Becker et al., 2021], including the PGI for morningness that will be used to capture the genetic predisposition for a more morning- or evening-oriented chronotype.

Third, sibling files. Provided during Wave III, they allow for the identification of full siblings (i.e., siblings sharing the same biological parents), who will form the sample for within-family analyses aimed at establishing causal effects.

The combination of rich academic, genetic, and family-level information makes Add Health an ideal resource for understanding the relationships between individual chronotype and academic performance.

⁷Around 91% of Wave III respondents (N = 12,000) signed the Transcript Release Form (TRF).

3.2 Variables

3.2.1 The outcome variable: overall high school GPA

The main outcome in this analysis is high school Grade Point Average (GPA), coded in AHAA data by converting letters into numerical values (F=0, D=1, C=2, B=3, and A=4). The data contains GPA for each subject studied in each year of course taking, as well as cumulative indicators that average performance across all subjects and/or school years. To have a measure of the general performance in high school, I focus on the overall high school GPA, across all subjects taken and all years, standardised to have mean 0 and SD of 1 in the initial sample. Single measures will be objects of secondary analyses, as well as other dimensions of academic performance, such as course failure and completed years of education.

3.2.2 Chronotype in Add Health

The SSGAC morningness PGI used to capture the genetic propensity for chronotype was constructed through a meta-analysis of the summary statistics of large-scale GWASs conducted in UK Biobank and 23andMe, thereby maximising predictive accuracy through large discovery sample sizes [Becker et al., 2021, Okbay et al., 2018]. Importantly, as all SSGAC PGIs, it is only available for individuals from European descent, the same as discovery samples [Becker et al., 2021].⁸

In the GWAS on the UK Biobank the phenotype was assessed through the question: *"Do you consider yourself to be..?"*, with response options: *"Definitely a morning person"* (recoded as 4), *"More a morning than evening person"* (3), *"More an evening than a morning person"* (2), and *"Definitely an evening person"* (1), excluding responses *"Do not know"* and *"Prefer not to answer"*. In 23andMe, the phenotype was derived from two separate questions: *"Are you naturally a night person or a morning person?"* with possible answers *"Night owl"*, *"Early bird"*, or *"Neither"*, and a second version with *"Night person"*, *"Morning person"*, *"Neither"*, *"It depends"* and *"Im not sure"* as response options, excluding individuals providing inconsistent answers across the two questions. The resulting PGI measures the genetic predisposition toward a morning chronotype on a continuous, standardised scale [Becker et al., 2021] ranging from eveningness to morningness. The proportion of the variance in the phenotype ex-

⁸However, limiting the estimation sample to individuals from European ancestry is currently standard practice [Biroli et al., 2026, Sanz-de Galdeano and Terskaya, 2023, Muslimova et al., 2024, Buser et al., 2024]. Indeed, as to date large-scale GWAS are conducted on discovery samples from European descent, derived PGIs would be less accurate (with lower predictive ability of the phenotype and higher chance of measurement error) in prediction samples of different ancestries - an issue known as "portability problem" [Becker et al., 2021, Benjamin et al., 2024]. Other PGIs constructed by the SSGAC have been used in recent economic research. See for instance Sanz-de Galdeano and Terskaya [2023] who focus on the PGI for EA, or Buser et al. [2024] for a study on cognitive and non-cognitive skills.

plained by the measured genetic factor, the "SNP heritability" (h_{SNP}^2), is estimated to be 15.9% [Becker et al., 2021].⁹

Beyond the continuous PGI, I also define chronotype categories: early birds, as individuals in the top PGI quintile, night owls, as those in the bottom quintile, and intermediate types, as those in between.

3.2.3 Phenotype proxies

Another set of variables will be used to ensure that the PGI does actually reflect chronotype. Indeed, any PGI estimates provide evidence of the relationship between a phenotype and an outcome to the extent that the genetic variants associated with the PGI actually predict the related trait. Add Health data do not contain a direct measure of phenotype as targeted in the GWASs (see Section 3.2.2), thus, I use bed times and wake-up times measured over Add Health waves [Roenneberg et al., 2003, Preckel et al., 2013].¹⁰ Specifically, all Waves from I to IV report self-assessed usual bed time on weekdays, Wave II additionally includes usual bed time on weekdays during summer, while Waves III and IV contain usual bed times on both work-week and free days. Usual wake-up times are only collected in Wave III and IV, separately for week and free-days. A more detailed description of these variables is provided in Appendix A.1.

3.2.4 Controls

Finally, other variables will serve as covariates and include demographic and socioeconomic characteristics (sex, age in months in September 1994, an indicator of birth in the US, number of siblings, birth order and an index of socioeconomic status (SES) in the first wave¹¹), school-related factors (school and academic year of entry) and the first ten principal components (PCs) of the genetic relatedness matrix to address residual within-ancestry population stratification [Okbay et al., 2018, Biroli et al., 2026]. Furthermore, since recent GWAS evidence has shown that 12-16% of the variation in educational attainment can be explained by a polygenic index for EA [Okbay

⁹Recent evidence shows that sleep-related PGIs constructed with summary statistics from GWAS in adult samples are already predictive of the corresponding traits in childhood and adolescence [Kocevska et al., 2024].

¹⁰In a large GWAS on sleep related-traits, Jones et al. [2019b] find a strong correlation between accelerometer-derived measurement of sleep timing and self-reported chronotype, leading to the conclusion that a self-reported question as used in the GWAS is a valid and practical proxy for the information that can be obtained by repeated sleep tracker data. Nonetheless, it is worthy to clarify that, beyond sleep timing, studies have shown that variation in chronotype also correlates with differences in the timing of other physiological processes regulated by the circadian system, such as alertness, body temperature or melatonin and cortisol secretion, hormones regulating sleep and wakefulness [Adan et al., 2012, Enright and Refinetti, 2017].

¹¹The index is constructed as in Sanz-de Galdeano and Terskaya [2023] and it is based on parental education, parental occupation, household income, public assistance receipt, and housing quality.

et al., 2022], which has been proven to be a strong predictor of several academic and related outcomes [Arold et al., 2025, Becker et al., 2021, Biroli et al., 2026, Buser et al., 2024, Muslimova et al., 2024, Sanz-de Galdeano and Terskaya, 2023], the EA PGI will also be used as a control variable in all regressions.¹²

3.3 Sample Selection and Descriptive Statistics

The final estimation sample is obtained after several selection steps. First, the exclusion of all respondents without the morningness PGI, leaves with 5,599 observations out of the initial 20,772. Second, a further 282 are discarded due to missing sampling weights. Third, intersecting these observations with those who authorised the high school transcript release reduces the sample size to 3,839; and excluding those not enrolled in high school (i.e., in grade 7 or 8) in Wave I lowers it to 2,721.¹³ Fourth and finally, dropping observations with missing controls results in a final sample of 2,662 observations suitable for between-family estimations; including 300 full sibling, in pairs or trios, for within-family analysis. Despite a modest size, this sample offers the advantage of allowing to exploit random genetic inheritance, thereby enabling the identification of causal effects.

Table 1 reports the summary statistics of the main variables of interest, comparing the initial sample (composed of all the observations with non-missing information on each single variable and sampling weights) with the two estimation samples used in either the between- or within-family analysis.

As a result of the selection criteria, which exclude students enrolled in middle schools in Wave I, observations in the final sample are slightly older. Moreover, perhaps due to the restriction to individuals of European descent (the only group with available genetic data), selected observations are characterised by largely better socioeconomic conditions, measured with the SES index, and are slightly more likely

¹²Specifically, I use the "multi-trait" PGI. The rationale of multi-trait PGIs lies in leveraging the phenomenon of pleiotropy, for which the same SNPs that predict the "target" trait may also partially predict other "supplementary traits". Thus, if some traits are highly associated, it is possible to exploit strong genetic correlations and integrate the SNPs related to the "supplementary traits", bolstering the predictive power of the "target" [Turley et al., 2018, Becker et al., 2021, Buser et al., 2024]. Although the interpretation of "multi-trait" PGIs is less clear than for their single-trait correspondents - since they may also capture other traits - they exhibit higher predictive power. For the purpose of this analysis, the use of the multi-trait EA PGI as control conveniently helps to mitigate more confounding related to EA, as it captures the genetic propensity to attainment in a more comprehensive manner. Specifically, the "multi-trait" EA PGI is constructed accounting for the genetic overlap among educational attainment, cognitive performance, math attainment, delay discounting, age at first birth and religious attendance [Becker et al., 2021].

¹³In spite of the well-defined correspondence between selected high schools and feeder schools designed by Add Health sampling process, I adopt a conservative approach to address potential mobility of middle school students, for whom it is not possible to determine with certainty their final high school choice. Nevertheless, I will also re-estimate the main results including them in the sample as a robustness check.

Table 1: Descriptive Statistics

	Initial sample			Final sample			Sibling sample		
	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs
<i>Panel A: Chronotype</i>									
Morningness PGI	0.00	1.00	5317	0.04	0.99	2662	-0.12	1.01	300
Night Owl	0.20	0.40	5317	0.19	0.39	2662	0.24	0.42	300
Intermediate	0.60	0.49	5317	0.60	0.49	2662	0.58	0.49	300
Early Bird	0.20	0.40	5317	0.21	0.41	2662	0.18	0.39	300
<i>Panel B: Main outcomes</i>									
Overall GPA	2.58	0.84	9990	2.66	0.80	2662	2.78	0.77	300
Failed a course	0.50	0.50	7364	0.47	0.50	2662	0.40	0.49	300
Failure rate	0.08	0.15	7364	0.07	0.14	2662	0.06	0.12	300
Years of education	14.19	2.20	10861	14.48	2.12	2662	14.54	1.92	300
<i>Panel C: Controls</i>									
Female	0.49	0.50	14788	0.51	0.50	2662	0.46	0.50	300
Age in September '94	181.39	21.50	14783	192.44	14.90	2662	192.36	15.34	300
Born in the US	0.95	0.22	14786	0.99	0.09	2662	0.99	0.11	300
SES index WI	0.02	0.99	14788	0.27	0.90	2662	0.33	0.85	300
Num. of siblings	1.55	1.39	14576	1.47	1.16	2662	2.04	1.16	300
Birth order	1.80	1.13	14576	1.75	1.00	2662	2.12	1.10	300

Notes: Summary statistics for the initial and the estimation samples used in the between- and within-family analyses. Variable means are weighted using Add Health sample weights.

to be born in the US. Individuals in the final samples also have a higher average high school GPA, especially in the sibling subgroup, which also presents a lower likelihood of failure and a lower failure rate with respect to the other two samples. Instead, completed years of education later in life are rather similar across the three samples. Finally, observations in the sample for within-family analysis have more siblings. Summary statistics of the phenotype proxies used to assess the predictive ability of the PGI are reported in [Table B1](#).

Focusing on genetics, [Figure B3](#) plots the empirical pairwise correlations among a set of PGIs spanning across ability, personality traits, health or satisfaction in several life domains.¹⁴ The first column illustrates the correlations with the PGI for morningness, capturing chronotype. The long series of values close to zero shows that it is almost unrelated to other characteristics. This aligns with the very low genetic correlations with other traits found by [Becker et al. \[2021\]](#), as well as with results from recent GWASs, finding very little phenotypic correlation of chronotype with numerous traits, including anthropometric measures, psychiatric and metabolic conditions, educational attainment [[Jansen et al., 2019](#)], but also both sleep quality and quantity, either using self-reported or accelerometer-derived data [Jones et al. \[2019a,b\]](#). This reassures that the morningness PGI represents chronotype, rather than or in addition to other traits, supporting the credibility of results. Yet, the correlation with the EA

¹⁴See the Supplementary Materials from [Becker et al. \[2021\]](#) for a detailed description of multi-traits PGIs' construction.

PGI, although small, is not exactly zero. Thus, given the well-recognised importance of this polygenic index for academic outcomes, including GPA [Sanz-de Galdeano and Terskaya, 2023], it will be controlled for in the empirical analysis. This allows to better isolate the role of chronotype, by comparing individuals with the same genetic predisposition for educational attainment. In addition, Table B3 presents results of a balance test, showing that the morningness PGI is not correlated with any of the characteristics that will later be used as controls in the empirical part.

Finally, since conducting within-family estimations requires sufficient variation in the PGI among siblings, I provide two pieces of validating evidence. First, Table B4 presents the mean and standard deviation of the morningness PGI in the full and sibling samples (as reported in Table 1), also after residualising it with respect to family fixed-effects. While the variation among siblings is about half that observed between families, because siblings share on average 50% of their DNA, the third row indicates that even within families there is variation in the genetic predisposition for chronotype. To complement this evidence, Figure B4 shows the CDF of the absolute difference between the PGIs of sibling pairs. The vertical line depicts the median difference, equal to 0.67 SD, very close to the values found by Buser et al. [2024] for PGIs related to other traits. The figure also shows that more than 30 percent of sibling pairs differ by more than 1 SD of their genetic predisposition for morningness, again in line with Buser et al. [2024]. Hence, even within families there is variability to exploit.

4 Empirical Strategy

The relationship between the genetic predisposition for chronotype and academic performance is estimated with the following econometric specification

$$GPA_{is} = \beta PGI_{is}^{Morn} + \gamma X_{is} + \delta PGI_{is}^{EA} + \zeta_s + \psi_y + \varepsilon_{is} \quad (3)$$

where GPA_{is} is the overall GPA of individual i in high school s and PGI_{is}^{Morn} captures the genetic propensity to morningness on a continuous scale. X is a vector of covariates including demographic and socioeconomic factors, school-related factors, family-related factors and ancestry subgroup (the first 10 PCs of the genetic relatedness matrix), described in [Section 3.2.4](#). PGI_{is}^{EA} is also controlled for, in order to net out the genetic predisposition to educational attainment. ζ_s and ψ_y absorb school and year-of-entry fixed-effects, respectively. Finally, the idiosyncratic error term ε_{is} is clustered at the school level. [Equation \(3\)](#) allows me to compare students within the same high school and academic years, with identical observable characteristics and the same genetic predisposition for educational attainment, but a different genetic predisposition for chronotype. Still, β should not be interpreted in causal terms due to unobserved environmental confounding factors (i.e., passive gene-environment correlations). Therefore, I exploit the random genetic variation among full siblings to identify the causal effect.

$$GPA_{if} = \beta PGI_{if}^{Morn} + \gamma Z_{if} + \delta PGI_{if}^{EA} + \varphi_f + \psi_y + \varepsilon_{if} \quad (4)$$

The f subscript in [Equation \(4\)](#) denotes a family, φ_f are fixed-effects absorbing all time-invariant characteristics of the family, including parental genotypes and their manifestation in the rearing environment. Z is a subset of the vector X , only containing factors that vary between siblings, namely gender, age and birth order.¹⁵ ε_{if} is allowed to be correlated among siblings by clustering at the family level.

In order to shed light on eventual non-linearities, I also estimate parallel specifications, [Equation \(5\)](#) and [Equation \(6\)](#), which replace the continuous PGI^{Morn} with two dummy variables, *EarlyBird* and *NighOwl*, that capture extreme predispositions for morningness and eveningness, respectively defined as the top and bottom quintile of the PGI^{Morn} distribution.

$$GPA_{is} = \eta EarlyBird_{is} + \nu NighOwl_{is} + \gamma X_{is} + \delta PGI_{is}^{EA} + \zeta_s + \psi_y + \varepsilon_{is} \quad (5)$$

$$GPA_{if} = \eta EarlyBird_{if} + \nu NighOwl_{if} + \gamma Z_{if} + \delta PGI_{if}^{EA} + \varphi_f + \psi_y + \varepsilon_{if} \quad (6)$$

¹⁵Since all siblings in the sample attend the same high school, family fixed-effects also remove the need to control for school fixed-effects. Moreover, while controlling for the PCs is unnecessary when using family fixed-effects, their inclusion as covariates yields almost identical results (see for instance [Table B18](#)).

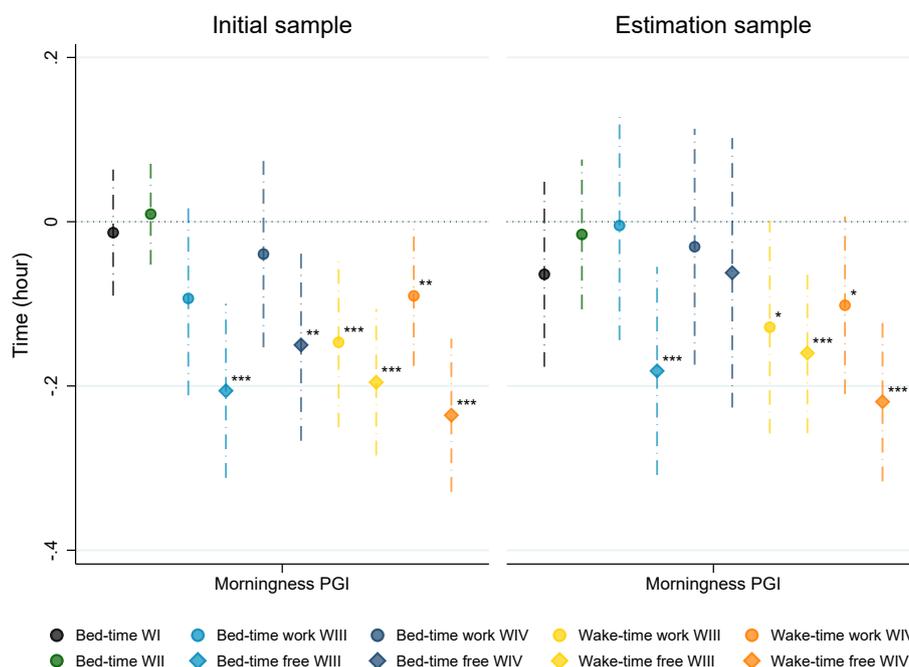
The coefficients of interest are β , η and ν . Since the PGI is standardised, β captures the effect of a standard deviation increase (from the mean) in the genetic propensity for a morning-oriented chronotype on GPA, whereas η and ν the effect of being an Early Bird or a Night Owl, relative to the intermediate type.

Before the main analysis, to validate the use of the morningness PGI as a measure of chronotype - and thus support the interpretation of results - I assess the ability of the genotype to predict the respective phenotype. This is done by regressing the bed or wake-up times introduced in Section 3.2.3, which proxy the chronotype phenotype, on the morningness PGI, controlling for the same vector of covariates X as in the main specifications.

5 Results

5.1 Predictive ability of the PGI

Figure 1: Predictive ability of the PGI



Notes: The graphs show coefficients associated with the morningness PGI from separate regressions of each outcome, conditional on controls. Cold colours represent bed times, hot colours wake up times. For each outcome, circles indicate work-week days, diamonds free days. The vertical axis denotes timing, measured in hours: downward (upward) movements from the horizontal line represent earlier (later) bed or wake-up time. The left panel reports results in the total sample with data on both the PGI and phenotypes (to maximise power), the right panel in the estimation sample used in the main analysis. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 1 presents the coefficients from regressions of bed and wake-up times on the morningness PGI throughout Add Health waves. For the purpose of assessing the predictive ability of the genotype, I report results using all observations with data on both the PGI and phenotypes, as well as in the estimation sample. Despite a slight reduction in statistical significance due to the sample size loss in the latter, at a first glance, both panels clearly show that individuals with a higher predisposition for morningness have an earlier timing of the sleep-wake cycle. A couple of further details are worthy of notice. First, the chronological ordering of the outcomes on the horizontal axis from the first to the last wave reveals a downward trend, indicating that the predictive ability of the PGI increases as individuals age - and plausibly gain more decision-making autonomy. This is in line with Merikanto et al. [2018], who find that the genetic variants associated with chronotype are predictive of an earlier sleep midpoint already during childhood, and that the relation intensifies in adolescence. Second, a closer look at Waves III and IV uncovers substantial differences between work-week and free days (respectively illustrated with circles and diamonds): the systematic gap between the two, with stronger effects on the latter, reveals that the genetic predisposition for chronotype is more evident when social constraints are relaxed. This is in line with Roenneberg et al. [2003], who document an evident gap in sleep onset between early and late chronotypes on workdays, which widens to more than two hours on free days. Together, these results confirm that the PGI is a valid and reliable measure of chronotype.

5.2 Main results

Estimates of the relationship between the genetic propensity for chronotype and GPA are presented in Table 2. Columns (1) to (3) report the coefficient associated with the morningness PGI. The first column presents results using the between-family specification in the full sample, the second replicates this in the sibling sample, and the third introduces family fixed-effects. Results reveal that a higher genetic predisposition for morningness has a positive and statistically significant effect on high school GPA. Notably, this holds keeping several observables, including the EA PGI, fixed. Specifically, a 1 SD increase in the morningness PGI is associated with a 0.044 SD increase in high school GPA, equivalent to 0.037 (0.044×0.84) GPA points, in the full sample, and with a 0.110 SD increase in GPA, or 0.092 points, in the sibling sample. However, the difference between the two coefficients is not statistically different at conventional levels, and it is most likely driven by diverse sample compositions. Finally, moving to within-family estimates reveals a positive effect of the morningness PGI on GPA equal to 0.201 SD, or 0.168 (0.201×0.84) GPA points, corresponding to a 6% increase relative to this sample's average GPA.

To shed more light on this relationship, columns (4) to (6) report estimates of the effect of extreme chronotypes' predispositions, with respect to the intermediate one. Although the direction of coefficients is in line with the linear model, it emerges that the effect is driven by an advantage of the early birds, whose circadian rhythm is more closely aligned with the school schedule. In contrast, night owl students do not exhibit significant differences in performance relative to those with an intermediate chronotype.

Table 2: Main results

	(1)	(2)	(3)	(4)	(5)	(6)
	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA
	Full sample	Sibling sample		Full sample	Sibling sample	
Morningness PGI	0.044** (0.018)	0.110** (0.041)	0.201* (0.108)			
Early Bird chronotype				0.114** (0.045)	0.422*** (0.109)	0.500** (0.234)
Night Owl chronotype				-0.011 (0.052)	-0.038 (0.085)	-0.150 (0.212)
EA PGI	0.250*** (0.018)	0.322*** (0.050)	0.319** (0.124)	0.249*** (0.018)	0.322*** (0.047)	0.298*** (0.113)
Observations	2,662	300	300	2,662	300	300
R-squared	0.370	0.501	0.820	0.371	0.515	0.829
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	No	Yes	Yes	No
Family FE	No	No	Yes	No	No	Yes
School FE	Yes	Yes	No	Yes	Yes	No
Academic Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.111	0.247	0.247	0.111	0.247	0.247
N of clusters	79	45	148	79	45	148
Early Birds VS Nigh Owls				0.125* (0.0710)	0.460*** (0.145)	0.650** (0.263)

Notes: The tables reports estimates of the genetic predisposition for chronotype on overall high school GPA, conditional on controls. Columns (1) and (2) are obtained from estimation of Equation (3), column (3) of Equation (4), columns (4) and (5) of Equation (5) and column (6) of equation Equation (6). For each specification, the first column reports results from estimate in the full sample, the second and the third columns in the sibling sample. Estimates are weighted using Add Health sample weights. Standard errors in parentheses are clustered at the school level in the between-family specification and at the family-level in the within-family specification. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Overall high school GPA is a more informative and representative measure of performance than single test scores, but it is important to understand whether results are driven by a particular context. Table B5 presents estimates of the effect on GPA in each school year (averaging across subjects), while Table B6 disaggregates performance by subject (averaging over years). Despite a slight reduction in statistical significance in within-family analysis, all coefficient estimates remain fairly stable, indicating that students with a stronger predisposition for an earlier circadian timing

are not better off in one particular setting only, but experience widespread advantages lasting throughout their high school path.

These results provide evidence that a higher predisposition for a morning-oriented chronotype has a positive and significant effect on high school performance. In the next section I will further address potential reasons underlying the differences in the estimates between the two samples, potentially attributable to compositional differences (as discussed in [Section 3.3](#)) and/or imperfect control of unobservables in the between-family specifications. After having proved robustness of the main result, I will dive into its heterogeneous effects and possible mechanisms.

5.3 Robustness Tests

5.3.1 Sibling spillovers

Within-family analyses have become a benchmark for identifying the causal effects of a PGI thanks to the ability to fully remove confounding from genetic nurture with the family fixed effect. However, a potential concern with this approach is the possibility of spillovers between siblings [[Bertoni et al., 2020](#), [Black et al., 2021](#), [Altmejd et al., 2021](#)]. The within-family estimator effectively captures the difference between an individual’s PGI effect and the average of their siblings ($\beta - \beta_{sib}$); hence, if an individual outcome is affected by his/her sibling’s genotype, the resulting coefficient would be a biased estimate of the direct effect (in the opposite direction of the sibling’s effect). I address potential spillover concerns in the sibling sample in two ways. First, I re-estimate the between-family specification but augmented with the sibling’s morningness PGI. As shown in Column (2) of [Table B7](#), the self PGI effect remains robust, while the sibling’s PGI coefficient is smaller and statistically indistinguishable from zero, in line with [Young et al. \[2022\]](#) who find evidence of the absence of sibling spillovers in many phenotypes. Second, I replace the individual’s PGI with the sibling’s PGI in [Equation \(3\)](#), obtaining an insignificant estimate even without accounting for the self PGI (Column (3)). Although controlling for parental genotype would further improve identification [[Young et al., 2022](#)], this exercise points to minimal spillover effects, mitigating bias concerns and reinforcing their validity of within-family estimates.

5.3.2 Role of unobservables

Between and within-family estimates from [Table 2](#) present some dissimilarities, which the intermediate columns suggest to stem from sample composition. To prove it, I employ entropy balancing [[Hainmueller, 2012](#)] to reweight the full sample so that its covariate distribution precisely matches the first and second moments of the distribution in the sibling sample.¹⁶ [Table B8](#) presents the results of [Equation \(3\)](#) and [Equation \(5\)](#), comparing estimates on the reweighted full sample with the baseline. In every specification, coefficient magnitudes increase but remain below within-family estimates. This pattern confirms the role of sample composition, but also supports the importance of within-family analysis for establishing causal effects.¹⁷

Next, to gauge an idea of the degree of omitted variable bias in the between-family analysis relies, which relies on selection on observables assumptions, I use the methods developed by [Oster \[2019\]](#). I calculate the "bias-adjusted treatment effects", known as β , under the conservative assumptions of equal selection on observables and observables ($\delta = 1$) and of a maximum $R_{max}^2 = 1.3R$, as recommended. [Table B9](#) displays "bias-adjusted treatment effects" very close to those in [Table 2](#), implying that omitted variable bias is unlikely to be a major concern. Assuming $R_{max}^2 = 1.5R$ confirms the results.¹⁸

To further address concerns about potential confounders, I perform a falsification exercise by reshuffling the morningness PGIs among observations in the estimation sample and re-estimating [Equation \(3\)](#) and [Equation \(4\)](#) 10,000 times. Since the permutation breaks the relationship between chronotype and GPA, no effect should be observed. The resulting empirical distributions of the placebo PGI effect, shown in Panels A and B of [Figure B5](#), are centred around zero, far from estimated effects. The same holds with the categorical classification of chronotype used for re-estimating [Equation \(5\)](#) and [Equation \(6\)](#), as shown in Panels C and D. Results are virtually identical when reshuffling the morningness PGI among students in the same school, preserving the original sampling design. These findings reassure that the estimated effects are unlikely to be observed only due to chance, or driven by spurious correlation between individual chronotype and potential confounding factors.

¹⁶The sibling sample is chosen as the target group since it is the one allowing to obtain causal insights. This choice also allows to exploit the larger sample size of the other, as well as to provide complementary evidence to the intermediate columns of [Table 2](#). Reweighting the sibling sample to match the covariate distributions of the full sample yields to qualitatively coherent conclusions.

¹⁷Although results from an Hausman test comparing the within- and the between-family specification in the sibling sample do reject the null hypothesis, theoretical reasons motivate the preference for the within-family specification to obtain causal estimates.

¹⁸Coherently, the estimates for δ - the ratio of selection on unobservables relative to observables required to explain the estimated effect away - either assuming a maximum $R_{max}^2 = 1.3R$ or $1.5R$, are well above 1. Assuming a value of $R_{max}^2 = 1$ would be hardly plausible given the estimated $R^2 = 0.37$.

5.3.3 Other PGIs and placebo phenotypes

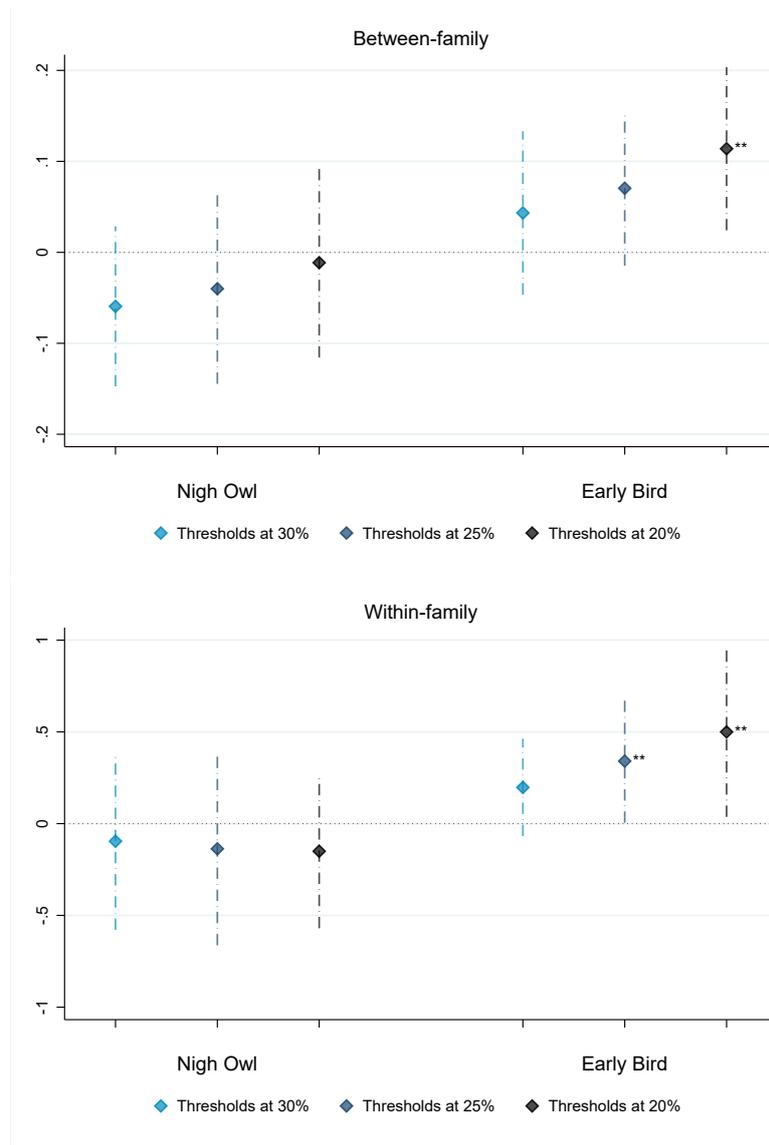
Although [Figure B3](#) shows no evidence of considerable correlation among polygenic indexes, and [Section 5.3.2](#) mitigates concerns about potential omitted variable bias, I explicitly check the stability of results to the addition of PGIs for economic preferences, physical and mental health, or personality traits as controls. [Table B10](#) shows that introducing these PGIs one at a time yields almost identical estimates to the main ones. [Table B11](#) compares the baseline specifications against alternative versions that either remove the EA PGI from the set of controls, or add all the PGIs simultaneously. Results underscore the importance of controlling for the genetic predisposition for educational attainment, without which the morningness PGI coefficient is still positive, but imprecisely estimated (except for the causal effects of the *EarlyBird* dummy in the sibling sample); beyond that, incorporating extra PGIs has only a minimal influence on the estimates. This implies that, conditional on the genetic predisposition for educational attainment, omitted variable bias from these other PGIs is negligible.

Finally, a placebo test reported in [Table B12](#) shows that the morningness PGI does not affect other traits that in principle should be unrelated with chronotype, namely chronic physical limitations, allergies and height.

5.3.4 Functional form

Although the early bird and night owl dummies - set at the top and bottom 20 percent of the chronotype distribution following chronobiological literature [[Adan et al., 2012](#), [Bettencourt et al., 2024](#)] - already offer intuitive insight into non-linear relationships between chronotype and academic performance, I further assess functional form misspecification. First, I estimate a quadratic specification, but results reported in [Table B13](#) do not point to a U-shaped relationship. Then, I assess sensitivity of the chosen thresholds to define early birds and night owls. [Figure 2](#) plots coefficients based on alternative cut-off points at 25 and 30 percent, other thresholds occasionally adopted in the literature. While the night owl coefficient remains statistically indistinguishable from zero, the magnitude of the early bird one further diverges from zero with more stringent cut-offs, corresponding to stronger morningness predisposition. Taken together, these results suggest that the relationship between the genetic predisposition for chronotype and GPA is not symmetric, but characterised by an early bird advantage, plausibly stemming from the alignment between their biological and the school timing - the so-called "synchrony effect".

Figure 2: Sensitivity of between-family estimates at different thresholds



Notes: The graph shows the coefficient estimates from Equation (5) (upper panel) and Equation (6) (bottom panel) obtained at various cut-off points for defining the early bird and night owl dummy variables.

Darker colours denote stricter thresholds.

5.3.5 Specification

The main within-family estimates rely on a linear parametrisation of controls to remove residual confounding from factors not absorbed in the family fixed effects. As a robustness check, I adopt a more flexible, less parametric approach that also mitigates extrapolation concerns. I begin by re-estimating the main specifications without all controls except for the EA PGI. Then, I also exclude the EA PGI and gradually restrict the sample to families where the difference between the EA PGIs of the siblings is below the 90th or 75th percentile of the difference distribution across families. Reassuringly, results in Table B14 are very similar to the baseline.

The baseline between-family specification accounts for unobserved factors that are common within a school and cohort (i.e., academic year of entry), as captured by the fixed effects ζ_s and ψ_y . Nevertheless, their inclusion in an additive manner may not completely absorb unobserved factors specific to a particular combination of school *and* cohort. Thus, to more accurately control for this unobserved heterogeneity, I replace the two set of school and cohort fixed effects with school-by-cohort fixed effects. [Table B15](#) shows that the two specifications yield almost equivalent results.

In the main specifications standard errors are clustered at the family or school level, in order to account for either the level of variation (the pool of parental genotypes) or the sampling design (which started from a stratified sample of schools), as also standard practice [[Arold et al., 2025](#), [Harden et al., 2020](#), [Muslimova et al., 2024](#), [Buser et al., 2024](#)]. Yet, very similar results obtained with heteroscedasticity-robust standard errors, in [Table B16](#), demonstrate that their validity does not hinge on specific assumptions on the standard error structure [[Abadie et al., 2020, 2023](#)].

5.3.6 Sample selection

Despite the Add Health design originally determined a well-defined mapping between high schools and their feeder schools, I conservatively excluded middle school students in Wave I from the estimation sample, since their final high school placement is uncertain due to potential mobility. Nevertheless, results in [Table B17](#), obtained while retaining also those students, are very similar to the baseline, meaning that sample selection does not affect the main findings.

5.4 Heterogeneity analysis

Table 3: Heterogeneous effects on different quantiles of the GPA distribution

	(1)	(2)	(3)	(4)	(5)
	10th	25th	50th	75th	90th
	GPA quantile				
Morningness PGI	0.063* (0.032)	0.077*** (0.027)	0.080*** (0.024)	0.018 (0.031)	0.027 (0.029)
EA PGI	0.188*** (0.043)	0.273*** (0.032)	0.326*** (0.027)	0.292*** (0.030)	0.223*** (0.039)
Observations	2,662	2,662	2,662	2,662	2,662
R-squared	0.214	0.257	0.274	0.218	0.148
Controls	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	Yes	Yes
Family FE	No	No	No	No	No
School FE	Yes	Yes	Yes	Yes	Yes
A.Y. FE	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4
Mean dep. var.	0.111	0.111	0.111	0.111	0.111
N of clusters	79	79	79	79	79

Notes: The table reports estimates of [Equation \(3\)](#) in the full sample at different unconditional quantiles of the GPA distribution. Standard errors in parentheses are clustered at the school level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Results in [Table 2](#) indicate a positive relationship between an earlier chronotype and academic performance, on average. Yet, the effect may be heterogeneous along several dimensions. A relevant one is the ability level, since evaluating how the effect of chronotype on academic performance varies by ability allows understanding the role of school scheduling in either reinforcing or alleviating educational disparities.

To explore this, [Table 3](#) replicates the main specification in the full sample at different quantiles of the GPA distribution, using unconditional quantile regressions [[Firpo et al., 2009](#)]. It emerges that the positive relationship between the morningness PGI and academic performance is mainly concentrated in the bottom half of the GPA distribution. In a similar spirit, I estimate [Equation \(3\)](#) and [Equation \(5\)](#) but augmented with an interaction term between the morningness PGI and the educational attainment PGI. In line with results from unconditional quantile regressions, the negative coefficient associated with the interaction term in [Table 4](#) indicates that the marginal benefit of an earlier chronotype decreases with a higher genetic predisposition for educational attainment (or, alternatively, is stronger among students with a lower genetic predisposition for educational attainment).

Table 4: Heterogeneous effects by genetic predisposition to educational attainment

	(1)	(2)
	Overall GPA	Overall GPA
	Full sample	Sibling sample
Morningness PGI	0.044** (0.018)	0.209** (0.102)
EA PGI	0.251*** (0.019)	0.320*** (0.122)
Morningness PGI × EA PGI	-0.037* (0.020)	-0.021 (0.109)
Observations	2,662	300
R-squared	0.372	0.820
Controls	Yes	Yes
Genotype PCs	Yes	No
Family FE	No	Yes
School FE	Yes	No
A.Y. FE	Yes	Yes
Weights	W4	W4
Mean dep. var.	0.111	0.247
N of clusters	79	148

Notes: The table reports estimates of Equation (3) in columns (1) and of Equation (4) in columns (2), but adding an interaction between the morningness and the EA PGIs. Standard errors in parentheses are clustered at the school level in column (1) and at the family level in column (2). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5.5 Mechanisms analysis

The main hypothesis underlying the main result is that students with a stronger propensity for morningness achieve greater efficiency when completing school tasks thanks to better alignment between their biological and social clocks - a phenomenon known in biology and psychology as the "synchrony effect" [Bettencourt et al., 2024, Preckel et al., 2013]. However, in principle, there might be several alternative potential explanations. In general, genetic endowments can influence outcomes in either a direct way, by enhancing or lowering the efficiency with which outcomes are generated, or in an indirect way, operating through behavioural or environment responses (e.g., active or evocative gene-environment correlations) [Biroli, 2015, Biroli et al., 2026]. This section explores possible mechanisms mediating the main relationship, including sleep, time use, academic ambition and effort, troubles at school and parental investment (all described in detail in Appendix A.2). While potential mediators are potentially numerous, failing to find evidence in favour of some of the arguably most important ones would at least support the main hypothesis.¹⁹ For brevity, I will present estimates based on the continuous PGI, with further explorations when needed to corroborate the results.

¹⁹In the spirit of a falsification exercise, and in the absence of significant results by independent testing, I will not correct for multiple testing.

5.5.1 Sleep

An option is that results actually reflect the influence of sleep duration, which is increasingly recognised as a relevant determinant of academic achievement [Avery et al., 2025, Giuntella et al., 2024, Jagnani, 2022] and may correlate with chronotype. To disentangle these hypotheses, I assess whether chronotype affects an objective and a subjective measure of sleep duration. The former refers to self-reported sleep duration, in hours and minutes; the latter captures the subjective perception of having slept enough or not, independently of the actual number of hours of rest. In the absence of a direct measure of sleep quality, this variable serves as a proxy for the sensation of morning fatigue. According to the findings in Table 5, students with an earlier chronotype tend to have better sleep patterns. However, the additional time spent sleeping is rather small. Column (1) shows that a 1 SD increase in the morningness PGI corresponds to 5 more minutes (0.09×60) of sleep per night - or 16 minutes (0.268×60) difference between early birds and night owls. Moreover, there is no statistical evidence of this effect among siblings, which makes it hard to point to sleep duration as the the main channel mediating the effect to GPA. In contrast, the increased likelihood of more morning-oriented students to report enough sleep is robust across both samples and specifications, and persists over time, as shown in the columns referring to Wave II.²⁰ These findings support the main hypothesis that chronotype influences academic performance *per se*, independently of sleep duration. In principle, night owls could anticipate their bed time to compensate for the earlier wake-up time, thus sleeping as much as the early birds. Yet, the mismatch between their natural alertness peak and school hours would still hinder efficiency.

5.5.2 Time use

Another plausible channel is time use. If students with an earlier circadian rhythm experience less fatigue from waking up for school, they could have more energy to study afterwards. Unfortunately, Add Health does not contain direct data on study hours or frequency. Therefore, I use a proxy based on students' participation in study, sport or any other type of school clubs. With fixed mandatory class schedules and the weak evidence of differences in sleep duration, variations in post-class activities offer insights of the use of the remaining time available for studying. Table B19 shows no evidence that participation in any kind of club is related to chronotype, ruling out the conclusion that time allocation in school-related activities is the pathway to effects on achievement.

Another possibility could be that night-oriented students more likely engage in

²⁰I report only Wave I and II values because respondents in wave III have already finished high-school, meaning that eventual results could not be interpreted as mechanisms.

Table 5: Chronotype effect on sleep duration

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Objective	Subjective	Objective	Subjective	Objective	Subjective	Objective	Subjective
	Wave I		Wave II		Wave I		Wave II	
	Full sample				Sibling sample			
Morningness PGI	0.092*** (0.032)	0.023** (0.010)	0.063* (0.037)	0.037*** (0.010)	0.083 (0.178)	0.072 (0.082)	-0.056 (0.200)	0.198** (0.084)
EA PGI	-0.041 (0.041)	-0.011 (0.014)	-0.083* (0.044)	-0.010 (0.020)	-0.267 (0.182)	-0.065 (0.081)	-0.445** (0.174)	-0.020 (0.086)
Observations	2,661	2,660	1,976	1,976	300	298	286	286
R-squared	0.089	0.070	0.093	0.101	0.720	0.633	0.657	0.549
Early Bird chronotype	0.013 (0.093)	0.057* (0.030)	0.103 (0.083)	0.085** (0.033)	0.236 (0.334)	0.348** (0.153)	-0.495 (0.389)	0.372* (0.194)
Night Owl chronotype	-0.255*** (0.069)	-0.037 (0.033)	-0.061 (0.107)	-0.027 (0.038)	0.282 (0.350)	0.161 (0.126)	-0.079 (0.407)	-0.067 (0.159)
EA PGI	-0.044 (0.041)	-0.010 (0.014)	-0.085* (0.044)	-0.010 (0.020)	-0.309* (0.169)	-0.085 (0.065)	-0.437** (0.179)	-0.057 (0.071)
Early Birds VS Nigh Owls	0.268*** (0.101)	0.0940*** (0.0304)	0.163 (0.128)	0.112*** (0.0322)	-0.0456 (0.481)	0.187 (0.210)	-0.416 (0.482)	0.440** (0.218)
Observations	2,661	2,660	1,976	1,976	300	298	286	286
R-squared	0.090	0.071	0.092	0.102	0.723	0.657	0.664	0.549
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	Yes	No	No	No	No
Family FE	No	No	No	No	Yes	Yes	Yes	Yes
School FE	Yes	Yes	Yes	Yes	No	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4	W4	W4
Mean dep. var.	7.664	0.706	7.505	0.673	7.673	0.635	7.539	0.714
N of clusters	79	79	78	78	148	147	141	141

Notes: The table reports estimates of the morningness PGI (in the upper panel) and of the two chronotype dummies (in the bottom panel) on objective and subjective measures of sleep duration as dependent variables. Columns (1) to (4) report results in the full sample and columns (5) to (8) in the sibling sample. Standard errors in parentheses are clustered at the school level in columns (1) to (4) and at the family level in columns (5) to (8). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

work activities during the evening, thereby reducing study time or increasing fatigue during school hours. However, [Table B20](#) provides no support for this channel too.

5.5.3 Ambition and effort

While the morningness predisposition does not seem to influence time in school, it might influence effort intensity or learning and performance goals [[Escribano and Díaz-Morales, 2016](#)]. As shown in [Table B21](#), there is no evidence that either ambition or expectations of college completion are affected by the genetic predisposition for chronotype. Neither the reported exerted effort is. This could possibly suggest that early birds and night owls put equal effort, but while the former succeed, the latter, who are not biologically functioning at their best during school times, are not able to get the returns. Alternatively, it might be that early birds do not need to strongly apply themselves, by naturally performing well at those times, whereas night owls lack the energies to do so. In either case, no difference would be observed.

5.5.4 Troubles at school

Another option relates to the different behaviour and atmosphere in the school environment. For instance, higher tiredness of later chronotypes may induce irritability and conduct issues, in turn affecting evaluation and grades. Yet, [Table B22](#) does not indicate differences in problems with peers. Instead, it shows that students with a higher predisposition for morningness are less likely to report troubles getting along with teachers. This may indicate that differences in alertness are evident to others, possibly leading to evocative gene-environment correlation. Finally, there is no evidence that the predisposition to morningness affects the perceived difficulty in paying attention (in line with results on effort in [Table B21](#)).

5.5.5 Parental investment

Beyond students themselves, teachers, and peers, also parents may play a role in shaping the relationship between chronotype and grades, although the nature of this influence is *a priori* uncertain. On one hand, they might invest more in children with higher alertness and responsiveness; but on the other hand they may exert greater effort to sustain children who struggle more to keep up with unnatural schedules (an argument in a similar vein to [Sanz-de Galdeano and Terskaya \[2023\]](#)). Differential parental investments determined by genetic differences would generate a negative gene-environment correlation, confounding the estimated effects. However, empirical results in [Table B23](#) finds no evidence in favour of either hypothesis.²¹

5.5.6 Summary of mechanism analysis

The list of possible mechanisms is in principle infinite, and the one presented here does not claim to be exhaustive. Yet, these findings are unable to confirm that several of the presumably most important candidates actually underlay the observed effect.²² Instead, they provide at least suggestive evidence in line with the main hypothesis, grounded in the biological and psychological literature, that earlier chronotype affects performance through a closer alignment between social and biological timings, increasing efficiency.

²¹Results are unaltered if using the index proposed by [Sanz-de Galdeano and Terskaya \[2023\]](#), which differs from that presented by capturing also parental investments not strictly related to school.

²²Non-reported results fail as well to prove hours/frequency of screen time (TV, computer, video games) or physical exercise, working during the high school years and three of the Big 5 personality traits (conscientiousness, neuroticism and extraversion, coded following [Young and Beaujean \[2011\]](#)) as channels.

5.6 Other academic performance outcomes

High school is a crucial period of life, as it marks the starting point of future life trajectories. Therefore, the observed effect on grades could have further implications in the long-run. To gauge an idea, I assess the role of the genetic propensity for chronotype on other two dimension of academic performance. [Table B24](#) replicates the main specifications on the extensive and intensive margin of course failure, i.e., on the probability of failing a course and a standardised measure of failure rate, respectively. Overall, it emerges that a higher morningness predisposition also reduces the likelihood of course failure, even between siblings of the same family. Although not always at conventional level of statistical significance, the pattern is confirmed also with the non-linear specification and on the failure rate. This indicates that the effect of chronotype extends also to more severe measures of achievement.

Next, [Table B25](#) assesses the role of the genetic predisposition for chronotype on years of education, failing to detect any significant effect. This might suggest that the genetic predispositions for educational attainment and for chronotype affect different aspects of performance. While the former influences a wide set of academic outcomes [[Muslimova et al., 2024](#), [Sanz-de Galdeano and Terskaya, 2023](#), [Buser et al., 2023](#)], the latter seems to only impact a short-term dimension, perhaps through efficiency and not through other drivers that may conduct to long-term qualifications, as those considered in [Section 5.5](#). Otherwise, it is could be that students sort into different tracks or fields, possibly different in their difficulty [[Buser et al., 2023](#)].²³ Alternatively, this result can be explained in light of some psychological research suggesting that later chronotypes, facing everyday inconveniences due to the misalignment between the social and their biological timings, develop higher problem solving capabilities to cope with these difficulties and and adjust to their best to social schedules [[Preckel et al., 2011](#)]. However, without suitable data to investigate it,

²³Following [Domingue et al. \[2015\]](#) and [Arold et al. \[2025\]](#), years of education are coded through the following transformation of the highest education level reported in Wave IV: eight grade or less (8 years); some high school (10 years); high school graduate or GED (12 years), some vocational/technical training (13 years); some college, some community college, completed vocational/technical training, associate or junior college degree (14 years); completed college (16 years); some graduate school (17 years); completed a masters degree or some post-baccalaureate professional education (18 years); some graduate training beyond a masters degree (19 years); completed post-baccalaureate professional education (19 years); completed a doctoral degree (20 years). A downside of this coding is that it is still possible that individuals with a different morningness PGI complete the same educational level, but in different numbers of years. Nonetheless, equivalent results are obtained on the likelihood of engagement in any post-secondary education (+12 years) as coded by [Trejo et al. \[2018\]](#), or on the likelihood of high school drop-out. For what concerns the study field, a replication exercise of [Buser et al. \[2023\]](#) in the estimation sample yields some suggestive evidence of statistical variation of the morningness PGI across college majors. However, due to the limited number of individuals with complete field information (around 700 observations covering 37 two-digit CIP (Classification of Instructional Programs) codes), conclusions should be drawn with caution.

this possibility remains within the realm of speculation, eventually offering potential scope for future research.

6 Conclusions

This study investigates whether the genetic predisposition for chronotype, measured with a polygenic index for the morningness trait, is a relevant predictor of academic success. Leveraging the unique combination of official high school transcript and DNA-based information contained in Add Health, I find that a higher genetic propensity for morningness has a significantly positive effect on GPA - a widespread result that persists across subjects and high school years. A within-family approach, which exploits the conditionally exogenous variation between full siblings who randomly inherit different genetic variants from the same biological parents' genotypes, yields the same conclusions and allows causal interpretation of the results. Specifically, a 1 SD increase in the morningness PGI has a positive and statistically significant effect on GPA equal to 0.201 SD, or 0.168 points, corresponding to a 6% increase relative to the mean. To benchmark these results, the estimated magnitudes are broadly within the range of effects on test scores of start-time delays, [Carrell et al., 2011, Heissel and Norris, 2018, Groen and Pabilonia, 2019], interventions to boost achievement in post-secondary education [Angrist et al., 2009, 2014, Clotfelter et al., 2018, Scott-Clayton, 2011] or sleep among college students [Giuntella et al., 2024]. The positive effect on GPA is also accompanied by a lower likelihood of course failure, while there is no influence of chronotype on completed years of education later in life.

These findings indicate that success in education partly depends on genetic luck. Notably, the positive effect of a more morning-oriented chronotype holds while keeping the genetic predisposition for educational attainment fixed. Such a conditional result implies that later chronotype students could perform as well as earlier ones if classes and exams were better matched with their own biological clock.

Indeed, the analysis of the potential mechanisms brings no evidence that the genetic predisposition for morningness is associated with different levels of learning and achievement goals, attention, exerted effort or time use. Hence, early chronotype students do not seem to outperform peers through higher inputs. Instead, they seem to benefit from higher alertness, possibly evident to teachers, during the relevant times, which enhances their efficiency - as argued by biological and psychological research.

Of course, genes cannot be modified over the life course, but there is room for intervention in the social environment. Prior research in economics has documented the achievement benefits of school start times delays, attributed to the higher alertness

deriving from a better alignment of schedules with adolescents' circadian rhythm [Carrell et al., 2011, Edwards, 2012, Heissel and Norris, 2018, Groen and Pabilonia, 2019, Diette and Raghav, 2017]. In a similar vein, but complementarily, this study demonstrates that, for a given timetable, students genetically predisposed to morningness benefit from an advantage. This implies that such interventions would help students on average, but especially those with a more evening-oriented chronotype. Alternatively, adjustments promoting flexibility or at least a more equitable balance of learning and testing times, without harming early chronotypes, would reduce the systematic disadvantage that students with a higher predisposition for eveningness are exposed to.

Results show that while during work-week days early and late chronotypes exhibit minor differences in their sleep timing, plausibly due to adaptation to social rhythms, when given more freedom they diverge in their wake and bed timing, revealing their biological preferences. Beşoluk et al. [2011] find that students entering university, when given the option to choose among a morning and an afternoon teaching period, without the possibility to change afterwards, tended to select in the one *designed for their chrono-type*. This evidence highlights that students are aware of their biological needs and implies that a reorganisation of school timetables could reduce educational inequalities.

Moreover, in line with the beneficial effects of later school start times among low-achievers found by Edwards [2012], results from this study are particularly strong at the bottom of the GPA distribution and for students with lower genetic predisposition for educational attainment. This means that such scheduling reforms could be especially helpful for students more in need.

Despite its strengths and contributions, this study is not free of limitations.

A first one is that, while the use of a PGI to capture chronotype is an improvement over traditional measures, based on either questionnaire instruments [Horne and Ostberg, 1976] or sleep-midpoints [Roenneberg et al., 2003], polygenic indexes are noisy measures of the underlying genetic factor [Becker et al., 2021, van Kipper-sluis et al., 2023]. When larger genotyped samples will become available, allowing better powered GWAS, more precise estimations of this relationship could be done. If anything, measurement error in the PGI would mean that results in this work are conservative estimates of the true, larger, effect. In addition, as in almost the whole research employing PGIs, the indexes used here rely on GWAS weights estimated in European-ancestry samples, limiting their portability to individuals of other genetic backgrounds. Future GWAS conducted in more diverse populations will be essential to assess the generalisability of these findings to other ancestry subgroups.

Moreover, although cumulative GPA is probably a better proxy of general performance than scores in single tests, and high school classes are typically scheduled in

the morning, with available data it is not possible to distinguish between morning and afternoon, learning and testing times. However, studies evaluating the impact of later start times [Carrell et al., 2011] or incentives to improve students' sleep [Giuntella et al., 2024] find that the benefits of the interventions, boosting efficiency, are not limited to the first class, but extend throughout the whole day. This is possibly due to the accumulated fatigue at the end of the day [Enright and Refinetti, 2017]. Nevertheless, with more detailed data, an analysis of the interaction between timing and chronotype would be an interesting and important future research to understand how the misalignment between the school and circadian clocks affects learning and testing in exams.

In conclusion, these findings underscore that innate genetic differences in circadian timing contribute to differences in academic performance, which are not due to different abilities, motivation or effort. Ignoring this evidence would create or preserve systematic obstacles for some individuals who, under more equal conditions, could perform as well as others. Instead, taking action and implementing changes would create a fairer environment and reduce educational inequalities.

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A Variables description

A.1 Chronotype proxies description

Bed times

1. Bed time in Wave I: *"What time do you usually go to bed on week nights?"*
2. Bed time in Wave II: *"What time do you usually go to bed on week nights?"*
3. Bed time in Wave III: *"On days when you go to work, school, or similar activities, what time do you go to sleep the night (or day) before?"*
4. Bed time in Wave III, on free days: *"On days that you don't have to get up at a certain time, what time do you go to sleep the night (or day) before?"*
5. Bed time in Wave IV: *"On days when you go to work, school, or similar activities, what time do you go to sleep the night (or day) before?"*
6. Bed time in Wave IV, on free days: *"On days that you don't have to get up at a certain time, what time do you go to sleep the night (or day) before?"*

All answers are provided in hours and minutes. Bed times after midnight are coded as the indicated hour +24 (e.g., 2 AM is coded as 26).

Wake-up times

1. Wake-up time in Wave III: *"On days when you go to work, school, or similar activities, what time do you usually wake up?"*
2. Wake-up time in Wave III, on free days: *"On days that you don't have to get up at a certain time, what time do you usually get up?"*
3. Wake-up time in Wave IV: *"On days when you go to work, school, or similar activities, what time do you usually wake up?"*
4. Wake-up time in Wave IV, on free days: *"On days that you don't have to get up at a certain time, what time do you usually get up?"*

All answers are provided in hours and minutes.

A.2 Mechanism variables description

Sleep

1. Sleep duration: *"How many hours of sleep do you usually get?"*
2. Sleep enough: *"Do you usually get enough sleep?"*

Time use

1. Study clubs. Whether the respondent reports to participate in at least one of the following clubs: *French club, German club, Latin club, Latin club, Book club, Computer club, History club, Math club, Science club*
2. Sport clubs. Whether the respondent reports to participate in at least one of the following clubs: *Cheerleading/dance team, Baseball/softball, Basketball, Field hockey, Football, Ice hockey, Soccer, Swimming, Tennis, Track, Volleyball, Wrestling, Other sport*
3. Other clubs. Whether the respondent reports to participate in at least one of the following clubs: *Debate team, Drama club, Future Farmers of America, Band, Chorus or choir, Orchestra, Other club or organization, Newspaper, Honor Society, Yearbook*

Responses were collected in the "in-school" survey, where students could mark all clubs they belonged to. However, since the vast majority has a total of 0²⁴ I create binary variables taking value 1 in case of positive total and 0 otherwise.

4. Working status. *"How many hours do you spend working for pay in a typical non-summer week?"*

The question was asked during the "in-home" survey in Wave I. I create a binary variable taking value 1 in case of a non-zero answer and 0 otherwise.

Ambition and effort

1. College ambition: *"On a scale of 1 to 5, where 1 is low and 5 is high, how much do you want to go to college?"*
2. College expectation: *"On a scale of 1 to 5, where 1 is low and 5 is high, how likely is it that you will go to college?"*
3. School effort: *"In general, how hard do you try to do your school work well?"*, with possible answering options: 1 *"I try very hard to do my best"*, 2 *"I try hard enough, but not as hard as I could"*, 3 *"I don't try very hard"* and 4 *"I never try at all"*.

²⁴Add Heath codebooks suggest 82% of students belonged to no club. Disaggregating into the selected groups, these figures correspond to 90% for study clubs, and 75% to sport and other clubs, both in the initial and in the estimation samples.

Items 1 and 2 are asked during the "in-home" survey, item 3 in the "in-school" survey. I reverted the original coding of the latter so that higher values indicate higher exerted effort. All the variables are standardised to have mean 0 and SD of 1 in the Wave I sample with the relative sampling weights.

Troubles at school

1. Troubles with students: *"Since school started this year/During the 1994-1995 school year, how often have you had trouble getting along with other students?"*
2. Troubles with teachers: *"Since school started this year/During the 1994-1995 school year, how often have you had trouble getting along with your teachers?"*
3. Troubles paying attention: *"Since school started this year/During the 1994-1995 school year, how often have you had trouble paying attention in school?"*

Each question is asked in the Wave I "in-home survey" and has possible answering options: 0 "never", 1 "just a few times", 2 "about once a week", 3 "almost everyday" and 4 "everyday". All the variables are standardised to have mean 0 and SD of 1 in the Wave I sample with the relative sampling weights.

Parental investment

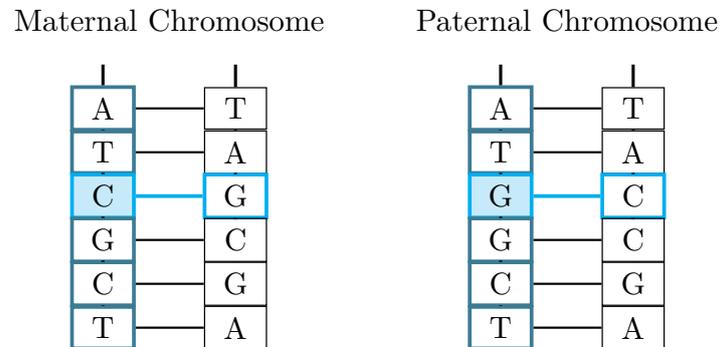
During the "in-home survey" in Wave I respondents were asked to mark from a list which things they had done with their mother and their father figures, separately, in the past 4 weeks. To measure parental investment I select the following two questions, referring to both the mother and the father.

1. *"Have you talked about your school work or grades?"*
2. *"Have you worked on a project for school?"*

To create the index I add the 4 items, standardised to have mean 0 and SD of 1 in the Wave I sample with the relative sampling weights.

B Additional tables and figures

Figure B1: Chromosomes and SNP



Notes: The figure illustrates a short segment of individual genome. It is composed of a pair of chromosomes, each inherited from one parent. Either chromosome is made up of a double-stranded piece of DNA (the vertical chains), consisting in a sequence of base pairs (the couples of nucleotides horizontally linked). The thick DNA strand represents the reference one. The cyan coloured boxes represent a SNP in that location of the genome. The genotype at that position is "CG". If the minor allele at that locus was G, the conventional coding would be "1".

Figure B2: PGI Construction

Loci	1	2	3	4	5
M. Chr.	A	C	G	A	T
P. Chr.	C	C	G	T	T
Minor Allele	C	G	T	A	G
β^{GWAS}	0.3	0.1	0.5	0.4	0.3

Notes: The figure illustrates two short segments of the maternal and paternal chromosomes, each composed of a sequence of nucleotides on the reference DNA strand. The minor allele in the population at each locus, with the corresponding estimated associations with the target phenotype, β^{GWAS} , are reported below. In this example the polygenic index would be equal to $PGI = 1 \times 0.3 + 0 \times 0.1 + 0 \times 0.5 + 1 \times 0.4 + 0 \times 0.3 = 0.7$

Table B1: Descriptive statistics of chronotype proxies

	All			Early birds			Intermediate			Night owls		
	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs
Bed time on work-week days WI	22.57	2.33	2654	22.48	2.57	541	22.55	2.27	1615	22.74	2.24	498
Bed time on work-week days WII	22.77	1.80	1971	22.72	1.67	389	22.78	1.76	1195	22.79	2.03	387
Bed time on work-week days WIII	23.75	2.93	2643	23.75	3.18	535	23.69	2.88	1613	23.95	2.80	495
Bed time on work-week days WIV	23.31	3.04	2653	23.38	2.96	539	23.27	3.06	1616	23.35	3.05	498
Bed time on free days WIII	24.51	2.88	2657	24.17	3.19	539	24.53	2.81	1619	24.81	2.72	499
Bed time on free days WIV	23.78	3.11	2656	23.67	3.16	541	23.86	2.91	1616	23.68	3.63	499
Wake-up time on work-week days WIII	7.38	2.58	2645	7.29	2.67	537	7.28	2.38	1613	7.79	3.03	495
Wake-up time on work-week days WIV	6.81	2.52	2653	6.66	2.44	539	6.85	2.62	1616	6.84	2.29	498
Wake-up time on free days WIII	9.52	2.42	2657	9.36	2.45	539	9.51	2.52	1619	9.72	2.01	499
Wake-up time on free days WIV	8.48	2.31	2658	8.20	1.96	541	8.47	2.42	1617	8.78	2.29	500

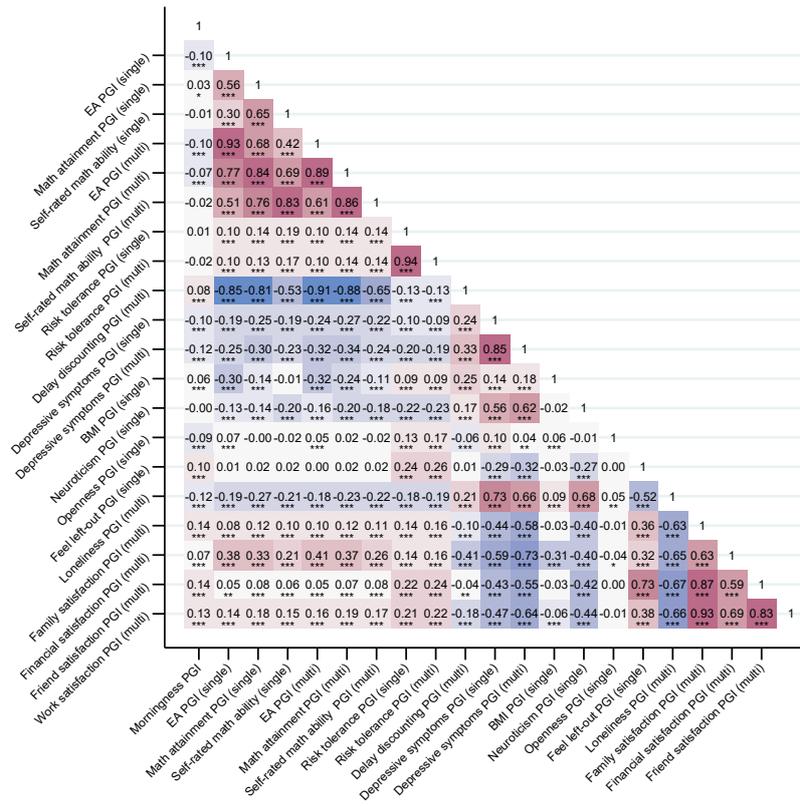
Notes: Summary statistics for the variables used as proxies of the chronotype phenotype in the whole estimation sample, and disaggregated by chronotype. Variable means are weighted using Add Health sample weights.

Table B2: Descriptive statistics of mechanisms' variables

	All			Early birds			Intermediate			Night owls		
	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs	Mean	Std.Dev.	Obs
<i>Panel A: Sleep</i>												
Objective duration (WI)	7.66	1.36	2661	7.72	1.32	541	7.71	1.38	1620	7.47	1.31	500
Subjective duration (WI)	0.71	0.46	2660	0.76	0.43	541	0.70	0.46	1621	0.66	0.47	498
Objective duration (WII)	7.51	1.35	1978	7.61	1.23	389	7.50	1.38	1201	7.43	1.39	388
Subjective duration (WII)	0.67	0.47	1978	0.75	0.43	389	0.66	0.47	1201	0.64	0.48	388
<i>Panel B: Time use</i>												
Study club	0.15	0.36	2662	0.14	0.35	541	0.14	0.35	1621	0.17	0.37	500
Sport club	0.41	0.49	2662	0.43	0.50	541	0.40	0.49	1621	0.42	0.49	500
Other club	0.37	0.48	2662	0.37	0.48	541	0.35	0.48	1621	0.41	0.49	500
Working status	0.66	0.47	2641	0.69	0.46	538	0.65	0.48	1608	0.67	0.47	495
<i>Panel C: Ambition and effort</i>												
College ambition	0.01	1.01	2659	0.05	0.96	541	-0.01	1.02	1619	0.01	1.03	499
College expectation	0.09	0.99	2659	0.11	0.98	541	0.08	1.00	1619	0.09	1.00	499
School effort	-0.19	1.00	2014	-0.11	0.99	412	-0.21	1.00	1221	-0.22	1.00	381
<i>Panel D: Troubles and difficulties</i>												
Troubles w/ students	-0.09	0.89	2612	-0.10	0.87	532	-0.08	0.90	1581	-0.12	0.88	499
Troubles w/ teachers	-0.08	0.91	2613	-0.18	0.83	532	-0.04	0.94	1582	-0.09	0.89	499
Troubles w/ attention	0.11	0.98	2613	0.05	1.00	532	0.11	0.97	1582	0.19	0.97	499
<i>Panel E: Parental investment</i>												
Parental investment	-0.04	2.33	2662	0.09	2.53	541	-0.09	2.25	1621	-0.04	2.34	500

Notes: Summary statistics for the variables studied as mechanisms in the estimation sample, then disaggregated in the three chronotypes. Variable means are weighted using Add Health sample weights.

Figure B3: Pairwise PGIs correlations



Notes: Pairwise PGIs correlation in the estimation sample. Red tones indicate a positive correlation, blue tones a negative one. Colour intensity reflects the magnitude. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B3: Balance tests

	(1) Female	(2) Age	(3) Birth order	(4) SES index	(5) N siblings	(6) US born
Morningness PGI	-0.009 (0.013)	0.207 (0.378)	-0.030 (0.025)	0.014 (0.021)	-0.008 (0.030)	0.001 (0.004)
Observations	2,662	2,662	2,662	2,662	2,662	2,662
R-squared	0.008	0.010	0.008	0.058	0.008	0.005
Weights	W4	W4	W4	W4	W4	W4
Genotype PCs	Yes	Yes	Yes	Yes	Yes	Yes
Family FE	No	No	No	No	No	No

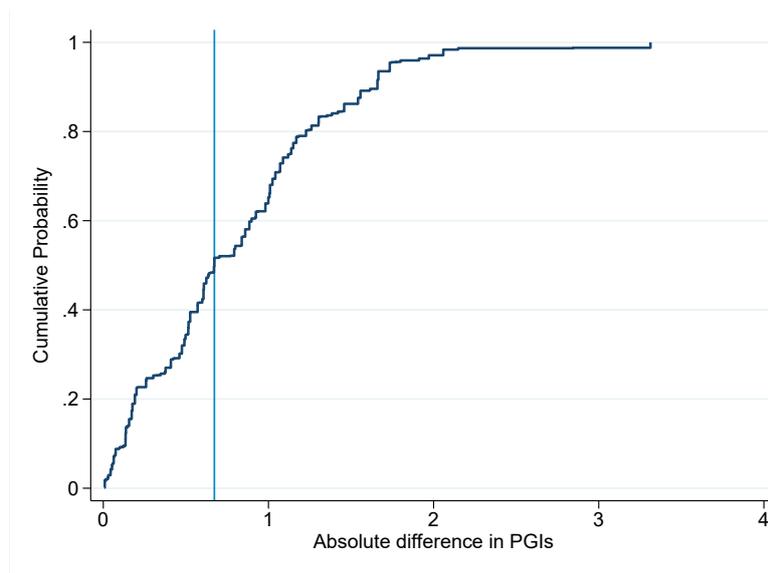
Notes: The table reports coefficients from separate regressions of each control variable on the Morningness PGI and the first 10 PCs of the genetic relatedness matrix. Estimates are weighted using Add Health sample weights. Robust standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B4: Identifying variation

	Mean	Std.Dev.	Obs
<i>Full sample</i>			
Morningness PGI	0.04	0.99	2662
<i>Sibling sample</i>			
Morningness PGI	-0.12	1.01	300
Residualised PGI	0.00	0.51	300

Notes: Mean and standard deviation of the Morningness PGI before and after residualisation to family fixed effects. Variable means are weighted using Add Health sample weights.

Figure B4: CDF of sibling differences in the morningness PGI



Notes: Cumulative distribution function of the difference in the standardised PGI within sibling pairs.

The vertical line represents the median absolute difference.

Table B5: GPA across high school years

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Overall GPA	1 year GPA	2 year GPA	3 year GPA	4 year GPA	Overall GPA	1 year GPA	2 year GPA	3 year GPA	4 year GPA
Full sample										
Morningness PGI	0.044** (0.018)	0.037* (0.019)	0.038** (0.017)	0.054** (0.022)	0.037* (0.022)					
Early Bird chronotype						0.114** (0.045)	0.081* (0.048)	0.126** (0.052)	0.128*** (0.043)	0.094** (0.038)
Night Owl chronotype						-0.011 (0.052)	-0.021 (0.054)	-0.002 (0.055)	0.007 (0.053)	-0.018 (0.060)
EA PGI	0.250*** (0.018)	0.251*** (0.017)	0.219*** (0.019)	0.227*** (0.026)	0.227*** (0.025)	0.249*** (0.018)	0.250*** (0.017)	0.219*** (0.019)	0.225*** (0.026)	0.226*** (0.025)
Observations	2,662	2,659	2,631	2,533	2,408	2,662	2,659	2,631	2,533	2,408
R-squared	0.370	0.326	0.290	0.298	0.308	0.371	0.326	0.292	0.298	0.308
Controls	Yes									
Genotype PCs	Yes									
Family FE	No									
School FE	Yes									
A.Y. FE	Yes									
Weights	W4									
Mean dep. var.	0.111	0.110	0.0953	0.0613	0.0710	0.111	0.110	0.0953	0.0613	0.0710
N of clusters	79	79	78	77	77	79	79	78	77	77
Early Birds VS Nigh Owls						0.125* 0.0710	0.103 0.0673	0.128* 0.0682	0.121 0.0738	0.111 0.0678
Sibling sample										
Morningness PGI	0.201* (0.108)	0.184* (0.103)	0.156 (0.096)	0.202 (0.151)	0.144 (0.133)					
Early Bird chronotype						0.500** (0.234)	0.215 (0.219)	0.523*** (0.180)	0.450 (0.332)	0.524** (0.236)
Night Owl chronotype						-0.150 (0.212)	-0.341 (0.213)	-0.029 (0.184)	-0.135 (0.260)	0.158 (0.256)
EA PGI	0.319** (0.124)	0.285** (0.134)	0.302*** (0.100)	0.278 (0.169)	0.282* (0.152)	0.298*** (0.113)	0.272** (0.128)	0.285*** (0.086)	0.254 (0.161)	0.263* (0.154)
Observations	300	300	300	292	270	300	300	300	292	270
R-squared	0.820	0.801	0.807	0.760	0.780	0.829	0.806	0.819	0.765	0.790
Controls	Yes									
Genotype PCs	No									
Family FE	Yes									
School FE	No									
A.Y. FE	Yes									
Weights	W4									
Mean dep. var.	0.247	0.282	0.232	0.109	0.140	0.247	0.282	0.232	0.109	0.140
N of clusters	148	148	148	145	134	148	148	148	145	134
Early Birds VS Nigh Owls						0.650** (0.263)	0.556** (0.270)	0.552** (0.222)	0.585 (0.377)	0.366 (0.285)

Notes: The upper panel report estimates of Equation (3) in columns (1) to (5) and of Equation (5) in columns (6) to (10) in the full sample, but substituting the main outcome with GPA in each high school year. The bottom panel replicates the same with estimates of Equation (4) in columns (1) to (5) and Equation (6) in columns (6) to (10) in the sibling sample. Standard errors in parentheses are clustered at the school level in the top panel and at the family level in the bottom panel.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B6: GPA across subjects

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Overall GPA	English GPA	Math GPA	Science GPA	Overall GPA	English GPA	Math GPA	Science GPA
Full sample								
Morningness PGI	0.044** (0.018)	0.054*** (0.018)	0.039** (0.020)	0.057*** (0.020)				
Early Bird chronotype					0.114** (0.045)	0.122*** (0.043)	0.074 (0.047)	0.126** (0.049)
Night Owl chronotype					-0.011 (0.052)	-0.045 (0.058)	-0.016 (0.054)	-0.044 (0.056)
EA PGI	0.250*** (0.018)	0.230*** (0.019)	0.235*** (0.020)	0.247*** (0.023)	0.249*** (0.018)	0.230*** (0.018)	0.234*** (0.020)	0.246*** (0.023)
Observations	2,662	2,656	2,656	2,645	2,662	2,656	2,656	2,645
R-squared	0.370	0.344	0.271	0.303	0.371	0.345	0.270	0.304
Controls	Yes							
Genotype PCs	Yes							
Family FE	No							
School FE	Yes							
A.Y. FE	Yes							
Weights	W4							
Mean dep. var.	0.111	0.0688	0.110	0.0841	0.111	0.0688	0.110	0.0841
N of clusters	79	79	79	79	79	79	79	79
Early Birds VS Nigh Owls					0.125* 0.0710	0.167** 0.0725	0.0898 0.0730	0.169** 0.0769
Sibling sample								
Morningness PGI	0.201* (0.108)	0.184 (0.125)	0.129 (0.115)	0.162 (0.102)				
Early Bird chronotype					0.500** (0.234)	0.455 (0.277)	0.302 (0.219)	0.361** (0.179)
Night Owl chronotype					-0.150 (0.212)	-0.178 (0.205)	-0.164 (0.218)	-0.170 (0.235)
EA PGI	0.319** (0.124)	0.260* (0.134)	0.488*** (0.122)	0.399*** (0.123)	0.298*** (0.113)	0.246** (0.121)	0.481*** (0.115)	0.384*** (0.121)
Observations	300	298	300	298	300	298	300	298
R-squared	0.820	0.796	0.801	0.801	0.829	0.804	0.806	0.806
Controls	Yes							
Genotype PCs	No							
Family FE	Yes							
School FE	No							
A.Y. FE	Yes							
Weights	W4							
Mean dep. var.	0.247	0.0618	0.176	0.167	0.247	0.0618	0.176	0.167
N of clusters	148	147	148	147	148	147	148	147
Early Birds VS Nigh Owls					0.650** 0.263	0.634** 0.300	0.466 0.281	0.531** 0.240

Notes: The upper panel report estimates of Equation (3) in columns (1) to (4) and of Equation (5) in columns (5) to (8), but substituting the main outcome with GPA in different high school subjects in the full sample. The bottom panel replicates the same with estimates of Equation (4) in columns (1) to (4) and Equation (6) in columns (5) to (8) in the sibling sample. Standard errors in parentheses are clustered at the school level in the top panel and at the family level in the bottom panel. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B7: Robustness to sibling spillovers

	(1)	(2)	(3)
	Overall GPA	Overall GPA	Overall GPA
Morningness PGI	0.110** (0.041)	0.126*** (0.039)	
Sibling's morn. PGI		-0.064 (0.038)	-0.032 (0.042)
EA PGI	0.322*** (0.050)	0.336*** (0.049)	0.324*** (0.047)
Observations	300	300	300
R-squared	0.501	0.504	0.492
Controls	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes
Family FE	No	No	No
School FE	Yes	Yes	Yes
A.Y. FE	Yes	Yes	Yes
Weights	W4	W4	W4
Mean dep. var.	0.247	0.247	0.247
N of clusters	45	45	45

Notes: The table reports estimates of Equation (3) in sibling sample in three versions: column (1) is the base-line, replicating the between-family specification (as in the second column of Table 2); column (2) augments this specification with the sibling's morningness PGI; column (3) replaces an individual PGI with the PGI the sibling. Standard errors in parentheses are clustered at the school level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B8: Entropy balancing

	(1)	(2)	(3)	(4)
	Overall GPA	Overall GPA	Overall GPA	Overall GPA
	Survey weights	Balancing weights	Survey weights	Balancing weights
Morningness PGI	0.044** (0.018)	0.067*** (0.025)		
Early Bird chronotype			0.114** (0.045)	0.197** (0.077)
Night Owl chronotype			-0.011 (0.052)	-0.058 (0.047)
EA PGI	0.250*** (0.018)	0.296*** (0.017)	0.249*** (0.018)	0.295*** (0.017)
Observations	2,662	2,662	2,662	2,662
R-squared	0.370	0.365	0.371	0.369
Controls	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	Yes
Family FE	No	No	No	No
School FE	Yes	Yes	Yes	Yes
A.Y. FE	Yes	Yes	Yes	Yes
Weights	W4	EB	W4	EB
N of clusters	79	79	79	79
Early Birds VS Nigh Owls			0.125* (0.0710)	0.255*** (0.0946)

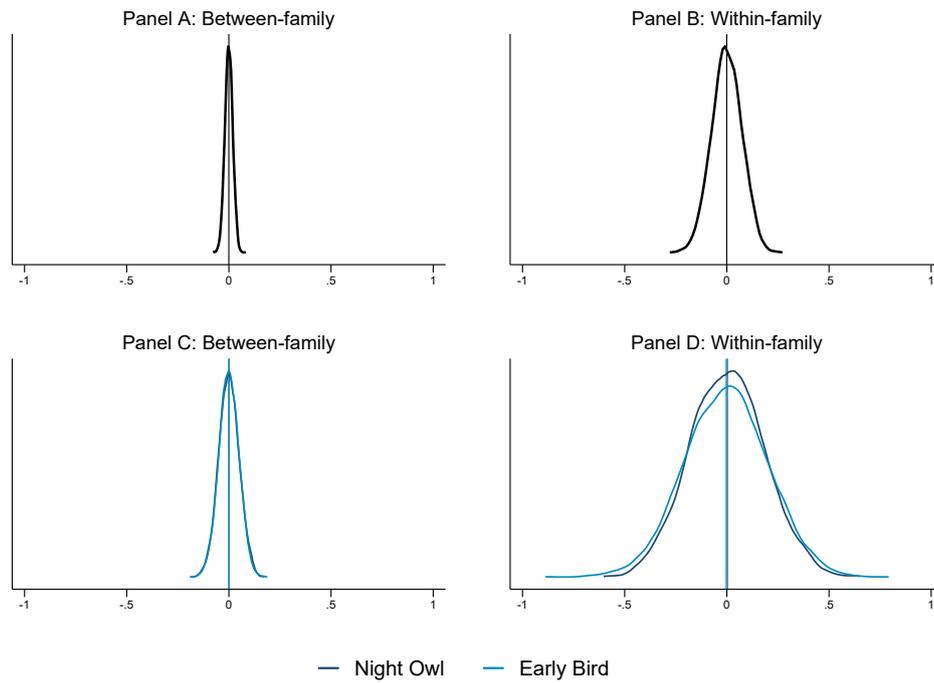
Notes: The table reports results of Equation (3) in columns (1) and (2) and Equation (5) in columns (3) and (4), comparing baseline results using sampling weights with results using balancing weights obtained reweighting the full sample to match the first and second moments of the covariate distribution in the sibling sample. Standard errors in parentheses are clustered at the school level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B9: Oster's bias-adjusted treatment effects

	(1)	(2)
	Oster β	Oster β
	$R_{max} = 1.3R$	$R_{max} = 1.5R$
Morningness PGI	0.052	0.059
Early Bird chronotype	0.120	0.125

Notes: "Bias-adjusted treatment effects" for Equation (3), in the first row, and Equation (5), in the second row, calculated with the model proposed by Oster [2019], under the assumption of equal selection ($\delta = 1$) and alternative values of R_{max}^2 as noted in column headings.

Figure B5: Falsification exercise: placebo morningness PGI effects



Notes: The figure plots the distribution of placebo effects based on 10,000 replications. The top row pertains to the placebo morningness PGI, the bottom row to the placebo Early Bird and Night Owl dummies.

Vertical lines represent the average placebo effect.

Table B10: Robustness to the inclusion of additional PGIs as controls

	Sibiling sample													
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
	Overall GPA Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA									
Morningness PGI	0.042** (0.018)	0.044** (0.018)	0.037** (0.019)	0.045** (0.018)	0.044** (0.018)	0.044** (0.018)	0.041** (0.018)	0.232** (0.109)	0.197* (0.109)	0.215* (0.111)	0.199* (0.106)	0.202* (0.108)	0.196* (0.106)	0.207* (0.114)
EA PGI	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Risk aversion PGI	✓													
Delay discounting														
Dep. symptoms PGI			✓											
BMI PGI														
Extraversion PGI				✓										
Neuroticism PGI						✓								
Openness PGI														
Observations	2,662	2,662	2,662	2,662	2,662	2,662	2,662	300	300	300	300	300	300	300
R-squared	0.374	0.371	0.372	0.372	0.371	0.371	0.371	0.831	0.822	0.822	0.822	0.822	0.822	0.820
Controls	Yes Yes	Yes	Yes	Yes	Yes									
School FE	Yes	No	No	No	No	No	No	No						
A.Y. FE	Yes Yes	Yes	Yes	Yes	Yes									
Weights	W4 W4	W4	W4	W4	W4									
Genotype PCs	Yes	No	No	No	No	No	No	No						
Family FE	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes						
Mean dep. var.	0.111	0.111	0.111	0.111	0.111	0.111	0.111	0.247	0.247	0.247	0.247	0.247	0.247	0.247
N of clusters	79	79	79	79	79	79	79	148	148	148	148	148	148	148

Notes: The table reports coefficients from separate regressions of Equation (3) in the full sample, in columns (1) to (7), and of Equation (4) in the sibling sample, in columns (8) to (14), augmented the inclusion of the PGI indicated by the check-mark as additional control. Estimates are weighted using Add Health sample weights. Standard errors in parentheses are clustered at the school level in columns (1) to (7) and at the family level in columns (8) to (14). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B11: Robustness to the inclusion of different sets of PGIs as controls

	(1)		(2)		(3)		(4)		(5)		(6)		(7)		(8)		(9)		(10)		(11)		(12)			
	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	
	Full sample		Full sample		Sibling sample		Sibling sample		Sibling sample		Sibling sample		Full sample		Full sample		Full sample		Full sample		Sibling sample		Sibling sample			
Morningness PGI	0.021 (0.017)	0.044** (0.018)	0.035* (0.019)	0.125 (0.103)	0.201* (0.108)	0.249** (0.113)	0.076 (0.046)	0.114** (0.045)	0.097** (0.047)	0.459* (0.263)	0.500** (0.234)	0.556** (0.227)														
Early Bird chronotype							0.011 (0.054)	-0.011 (0.052)	-0.003 (0.050)	-0.075 (0.203)	-0.150 (0.212)	-0.311 (0.234)														
Night Owl chronotype																										
EA PGI		0.250*** (0.018)	0.233*** (0.019)		0.319** (0.124)	0.361*** (0.122)		0.249*** (0.018)	0.233*** (0.018)																	
Other PGIs			✓			✓																			✓	
Observations	2,662	2,662	2,662	300	300	300	2,662	2,662	2,662	300	300	300	2,662	2,662	2,662	300	300	2,662	2,662	300	300	300	300	300	300	
R-squared	0.314	0.370	0.378	0.801	0.820	0.837	0.315	0.371	0.378	0.811	0.829	0.848	0.378	0.371	0.378	0.811	0.829	0.378	0.371	0.811	0.829	0.848	0.829	0.829		
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes		
Genotype PCs	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	No	No	No	No	No	No	No	No	No		
Family FE	No	No	No	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes		
School FE	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	No	No	No	Yes	Yes	Yes	No	No	No	No	No	No	No	No	No		
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes		
Weights	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4	W4		
Mean dep. var.	0.111	0.111	0.111	0.247	0.247	0.247	0.111	0.111	0.111	0.247	0.247	0.247	0.111	0.111	0.111	0.247	0.247	0.111	0.111	0.247	0.247	0.247	0.247	0.247		
N of clusters	79	79	79	148	148	148	79	79	79	148	148	148	79	79	79	148	148	79	79	148	148	148	148	148		
Early Birds VS Nigh Owls							0.0653 (0.0680)	0.125* (0.0710)	0.0998 (0.0718)	0.535** (0.268)	0.650** (0.263)	0.867*** (0.288)														
SE																										

Notes: The table reports estimates of the morningness PGI, in columns (1) to (6), and the two chronotype dummies in columns (7) to (12), with different sets of PGIs as covariates. For each specification and sample, the first column presents results obtained without controlling for any other PGI, the second column when introducing the EA PGI (as in baseline specifications), and the third column when jointly including the set of other PGIs separately controlled for in Table B10. Estimates are weighted using Add Health sample weights. Standard errors in parentheses are clustered at the school level in the full sample, columns (1) to (3) and (7) to (9), and at the family level in the sibling sample, columns (4) to (6) and (10) to (12). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B12: Placebo outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	Ph. limit	Allergies	Height	Ph. limit	Allergies	Height
	Full sample			Sibling sample		
Morningness PGI	-0.000 (0.003)	-0.003 (0.012)	-0.014 (0.011)	-0.003 (0.004)	0.100 (0.064)	0.008 (0.069)
Observations	2,662	2,370	2,661	300	275	300
R-squared	0.055	0.088	0.167	0.394	0.663	0.576
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	No	No	No
Family FE	No	No	No	Yes	Yes	Yes
School FE	Yes	Yes	Yes	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.0241	0.464	5.558	0.00427	0.349	5.578
N of clusters	79	78	79	148	136	148

Notes: The table reports estimates of [Equation \(3\)](#) in columns (1) to (3) and of [Equation \(4\)](#) in columns (4) to (6), but using as dependent variables placebo outcomes: physical limitations, allergies and height. Standard errors in parentheses are clustered at the school level in columns (1) to (3) and at the family level in columns (4) to (6).

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B13: Quadratic functional form

	(1)	(2)
	Overall GPA	Overall GPA
	Full sample	Sibling sample
Morningness PGI	0.044** (0.018)	0.207* (0.106)
Morningness PGI ²	-0.001 (0.013)	0.048 (0.078)
EA PGI	0.250*** (0.018)	0.316*** (0.120)
Observations	2,662	300
R-squared	0.370	0.821
Controls	Yes	Yes
Genotype PCs	Yes	No
Family FE	No	Yes
School FE	Yes	No
A.Y. FE	Yes	Yes
Weights	W4	W4
Mean dep. var.	0.111	0.247
N of clusters	79	148

Notes: The table reports estimates of [Equation \(3\)](#) in columns (1) and of [Equation \(4\)](#) in columns (2), but adopting a quadratic functional form specification. Standard errors in parentheses are clustered at the school level in column (1) and at the family level in column (2). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B14: Role of controls within-family

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA	Overall GPA
	Baseline		Without controls		EA PGI diff < 90 th quant		EA PGI diff < 75 th quant	
Morningness PGI	0.201*		0.236**		0.234**		0.223*	
	(0.108)		(0.115)		(0.108)		(0.116)	
Early Bird chronotype		0.500**		0.533**		0.503**		0.666***
		(0.234)		(0.226)		(0.243)		(0.244)
Night Owl chronotype		-0.150		-0.133		-0.114		-0.035
		(0.212)		(0.248)		(0.249)		(0.248)
EA PGI	0.319**	0.298***	0.268*	0.229*				
	(0.124)	(0.113)	(0.139)	(0.127)				
Observations	300	300	300	300	270	270	236	236
R-squared	0.820	0.829	0.790	0.799	0.778	0.785	0.783	0.803
Controls	Yes	Yes	No	No	No	No	No	No
Genotype PCs	No	No	No	No	No	No	No	No
Family FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
School FE	No	No	No	No	No	No	No	No
A.Y. FE	Yes	Yes	No	No	No	No	No	No
Weights	W4	W4	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.247	0.247	0.247	0.247	0.288	0.288	0.333	0.333
N of clusters	148	148	148	148	134	134	117	117
Early Birds VS Nigh Owls		0.650**		0.666**		0.617**		0.701**
		(0.263)		(0.277)		(0.278)		(0.278)

Notes: The table reports coefficient estimates from alternative specifications of Equation (4) and Equation (6) in the sibling sample. Columns (1) and (2) report the results of the baseline specifications, as in Table 2; columns (3) and (4) results without controlling for other covariates but the EA PGI; columns (5)-(6) and (7)-(8) additionally remove the EA PGI from the set of controls, while dropping families where the absolute difference between the EA PGIs of the siblings is higher than either the 90th or 75th percentile of the difference distribution across families. Standard errors are clustered at the family level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B15: School by cohort fixed effects

	(1)	(2)	(3)	(4)
	Overall GPA	Overall GPA	Overall GPA	Overall GPA
Morningness PGI	0.044** (0.018)	0.043** (0.020)		
Early Bird chronotype			0.114** (0.045)	0.129*** (0.045)
Night Owl chronotype			-0.011 (0.052)	0.003 (0.056)
EA PGI	0.250*** (0.018)	0.254*** (0.021)	0.249*** (0.018)	0.254*** (0.020)
Observations	2,662	2,614	2,662	2,614
R-squared	0.370	0.417	0.371	0.418
Controls	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	Yes
Family FE	No	No	No	No
School FE	Yes	No	Yes	No
A.Y. FE	Yes	No	Yes	No
School-by-A.Y. FE	No	Yes	No	Yes
Weights	W4	W4	W4	W4
Mean dep. var.	0.111	0.120	0.111	0.120
N of clusters	79	75	79	75
Early Birds VS Nigh Owls			0.125* (0.0710)	0.126* (0.0752)

Notes: The table reports results from alternative specification of the baseline between-family specifications, with alternative fixed-effects. The even columns report results from Equation (3) and Equation (5), while the even columns report results obtained with school-by-academic year of entry fixed effects. Standard errors are clustered at the school level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B16: Heteroscedasticity robust standard errors

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Overall GPA Overall GPA	Overall GPA	Overall GPA					
	cl. SE	hr SE	cl. SE	hr SE	cl. SE	hr SE	cl. SE	hr SE
	Full sample				Sibling sample			
Morningness PGI	0.044** (0.018)	0.044** (0.019)			0.201* (0.108)	0.201* (0.109)		
Early Bird chronotype			0.114** (0.045)	0.114** (0.049)			0.500** (0.234)	0.500** (0.236)
Night Owl chronotype			-0.011 (0.052)	-0.011 (0.049)			-0.150 (0.212)	-0.150 (0.214)
EA PGI	0.250*** (0.018)	0.250*** (0.020)	0.249*** (0.018)	0.249*** (0.020)	0.319** (0.124)	0.319** (0.129)	0.298*** (0.113)	0.298** (0.117)
Observations	2,662	2,662	2,662	2,662	300	300	300	300
R-squared	0.370	0.370	0.371	0.371	0.820	0.820	0.829	0.829
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	Yes	No	No	No	No
Family FE	No	No	No	No	Yes	Yes	Yes	Yes
School FE	Yes	Yes	Yes	Yes	No	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.111	0.111	0.111	0.111	0.247	0.247	0.247	0.247
N of clusters	79		79		148		148	
Early Birds VS Nigh Owls			0.125* (0.071)	0.125** (0.061)			0.650** (0.263)	0.650** (0.266)

Notes: The table reports results from alternative specification of the baseline estimating equations, under different standard errors assumptions. Standard errors are clustered in the odd columns (at the school level in the full sample and at the family level in the sibling sample) and robust to heteroscedasticity in the even columns. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B17: Robustness to sample selection

	(1)	(2)	(3)	(4)
	Overall GPA	Overall GPA	Overall GPA	Overall GPA
	Full sample	Sibling sample	Full sample	Sibling sample
Morningness PGI	0.041** (0.017)	0.143** (0.070)		
Early Bird chronotype			0.079** (0.037)	0.501** (0.211)
Night Owl chronotype			-0.063 (0.048)	-0.044 (0.165)
EA PGI	0.271*** (0.016)	0.192* (0.104)	0.270*** (0.016)	0.195** (0.099)
Observations	3,772	492	3,772	492
R-squared	0.384	0.798	0.384	0.808
Controls	Yes	Yes	Yes	Yes
Genotype PCs	Yes	No	Yes	No
Family FE	No	Yes	No	Yes
School FE	Yes	No	Yes	No
A.Y. FE	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4
Mean dep. var.	0.120	0.188	0.120	0.188
N of clusters	118	242	118	242
Early Birds VS Nigh Owls			0.142** (0.0598)	0.545*** (0.204)

Notes: The table replicates the estimates of [Table 2](#), but expanding the sample with students who were in grade 7 and 8 in Wave I. Standard errors in parentheses are clustered at the school level in the odd columns, reporting results in the full sample, and at the family level in the even columns, reporting results in the sibling sample.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B18: Role of PCs

	(1)	(2)	(3)	(4)	(5)	(6)
	Overall GPA					
	Full sample		Sibling sample			
Morningness PGI	0.044** (0.018)	0.040** (0.018)	0.110** (0.041)	0.072** (0.028)	0.214* (0.123)	0.201* (0.108)
EA PGI	0.250*** (0.018)	0.252*** (0.019)	0.322*** (0.050)	0.346*** (0.030)	0.317*** (0.118)	0.319** (0.124)
Observations	2,662	2,662	300	300	300	300
R-squared	0.370	0.366	0.501	0.432	0.856	0.820
Early Bird chronotype	0.114** (0.045)	0.104** (0.046)	0.422*** (0.109)	0.372*** (0.102)	0.492** (0.240)	0.500** (0.234)
Night Owl chronotype	-0.011 (0.052)	-0.011 (0.054)	-0.038 (0.085)	-0.002 (0.066)	-0.062 (0.204)	-0.150 (0.212)
EA PGI	0.249*** (0.018)	0.251*** (0.019)	0.322*** (0.047)	0.347*** (0.029)	0.275** (0.106)	0.298*** (0.113)
Early Birds VS Nigh Owls	0.125* (0.071)	0.116 (0.071)	0.460*** (0.145)	0.374*** (0.096)	0.555** (0.293)	0.650** (0.263)
Observations	2,662	2,662	300	300	300	300
R-squared	0.371	0.366	0.515	0.445	0.861	0.829
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	No	Yes	No	Yes	No
Family FE	No	No	No	No	Yes	Yes
School FE	Yes	Yes	Yes	Yes	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.111	0.111	0.247	0.247	0.247	0.247
N of clusters	79	79	45	45	148	148

Notes: The table reports results from alternative specification of the baseline estimating equations, with or without the first ten PCs of the genetic relatedness matrix for cleaner statistical comparison. Standard errors are clustered at the school level in the between-family specification and at the family level in the within-family specification. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B19: Chronotype effect on school clubs participation

	(1)	(2)	(3)	(4)	(5)	(6)
	Study club	Sport club	Other club	Study club	Sport club	Other club
	Full sample			Sibling sample		
Morningness PGI	-0.006 (0.009)	0.009 (0.011)	-0.013 (0.010)	0.069 (0.049)	0.070 (0.069)	-0.070 (0.084)
EA PGI	0.032*** (0.010)	0.008 (0.012)	0.066*** (0.012)	-0.111** (0.054)	-0.077 (0.082)	-0.038 (0.077)
Observations	2,662	2,662	2,662	300	300	300
R-squared	0.133	0.222	0.219	0.604	0.684	0.632
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	No	No	No
Family FE	No	No	No	Yes	Yes	Yes
School FE	Yes	Yes	Yes	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.148	0.411	0.366	0.168	0.547	0.447
N of clusters	79	79	79	148	148	148

Notes: The table reports estimates of Equation (3) in columns (1) to (3) and of Equation (4) in columns (4) to (6), but using participation in school clubs as dependent variables. Standard errors in parentheses are clustered at the school level in columns (1) to (3) and at the family level in columns (4) to (6). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B20: Chronotype effect on working status during the school week

	(1)	(2)
	Working status	Working status
	Full sample	Sibling sample
Morningness PGI	0.003 (0.013)	0.014 (0.090)
EA PGI	0.003 (0.012)	-0.071 (0.086)
Observations	2,641	294
R-squared	0.141	0.647
Controls	Yes	Yes
Genotype PCs	Yes	No
Family FE	No	Yes
School FE	Yes	No
A.Y. FE	Yes	Yes
Weights	W4	W4
Mean dep. var.	0.659	0.645
N of clusters	79	145

Notes: The table reports estimates of [Equation \(3\)](#) in column (1) and of [Equation \(4\)](#) in column (2), but using working status in a typical non-summer week as the dependent variable. Standard errors in parentheses are clustered at the school level in column (1) and at the family level in columns (2). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B21: Chronotype effect on ambition and effort

	(1) College ambition	(2) College expectation	(3) School effort	(4) College ambition	(5) College expectation	(6) School effort
	Full sample			Sibling sample		
Morningness PGI	0.031 (0.023)	0.022 (0.018)	0.052 (0.037)	0.201 (0.146)	0.123 (0.128)	0.318 (0.202)
EA PGI	0.119*** (0.024)	0.111*** (0.019)	0.078*** (0.029)	0.324** (0.148)	0.089 (0.150)	0.165 (0.251)
Observations	2,659	2,659	2,011	300	300	212
R-squared	0.170	0.264	0.119	0.745	0.800	0.579
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	No	No	No
Family FE	No	No	No	Yes	Yes	Yes
School FE	Yes	Yes	Yes	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.00537	0.0898	-0.189	-0.0993	0.0171	-0.102
N of clusters	79	79	72	148	148	106

Notes: The table reports estimates of Equation (3) in columns (1) to (3) and of Equation (4) in columns (4) to (6), but using self-reported measures of academic ambition, expectation and effort as dependent variables. Standard errors in parentheses are clustered at the school level in columns (1) to (3) and at the family level in columns (4) to (6). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B22: Chronotype effect on troubles at school

	(1)	(2)	(3)	(4)	(5)	(6)
	Troubles students	Troubles teachers	Troubles attention	Troubles students	Troubles teachers	Troubles attention
	Full sample			Sibling sample		
Morningness PGI	0.003 (0.017)	-0.043** (0.020)	-0.051 (0.030)	0.009 (0.150)	-0.279 (0.196)	-0.079 (0.171)
EA PGI	-0.008 (0.029)	-0.073*** (0.028)	-0.051* (0.029)	0.126 (0.197)	-0.132 (0.163)	-0.069 (0.204)
Observations	2,611	2,612	2,612	292	292	292
R-squared	0.078	0.112	0.100	0.510	0.641	0.600
Early Bird chronotype	-0.024 (0.050)	-0.148*** (0.056)	-0.078 (0.072)	-0.110 (0.394)	-0.717** (0.327)	-0.185 (0.315)
Night Owl chronotype	-0.034 (0.049)	-0.046 (0.065)	0.096 (0.067)	-0.277 (0.372)	0.045 (0.354)	0.049 (0.355)
EA PGI	-0.009 (0.029)	-0.073*** (0.028)	-0.051* (0.029)	0.144 (0.196)	-0.089 (0.141)	-0.059 (0.202)
Early birds VS Nigh Owls	0.0109 (0.0560)	-0.102* (0.0715)	-0.174 (0.0882)	0.166 (0.449)	-0.762 (0.532)	-0.233 (0.454)
Observations	2,611	2,612	2,612	292	292	292
R-squared	0.078	0.114	0.100	0.515	0.654	0.601
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	Yes	Yes	No	No	No
Family FE	No	No	No	Yes	Yes	Yes
School FE	Yes	Yes	Yes	No	No	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4
Mean dep. var.	-0.0886	-0.0808	0.112	0.0504	-0.0271	0.260
N of clusters	79	79	79	144	144	144

Notes: The table reports estimates of the morningness PGI (in the upper panel) and of the two chronotype dummies (in the bottom panel) on self-reported measures of troubles in the school environment as dependent variables. Columns (1) to (3) report results in the full sample and columns (4) to (6) in the sibling sample. Standard errors in parentheses are clustered at the school level in columns (1) to (3) and at the family level in columns (4) to (6). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B23: Chronotype effect on parental investment

	(1) Parent investment Full sample	(2) Parent investment Sibling sample
Morningness PGI	0.001 (0.054)	0.343 (0.314)
EA PGI	0.129** (0.063)	0.833** (0.399)
Observations	2,662	300
R-squared	0.085	0.693
Controls	Yes	Yes
Genotype PCs	Yes	No
Family FE	No	Yes
School FE	Yes	No
A.Y. FE	Yes	Yes
Weights	W4	W4
Mean dep. var.	-0.0411	-0.0998
N of clusters	79	148

Notes: The table reports estimates of [Equation \(3\)](#) in column (1) and of [Equation \(4\)](#) in column (2), but using parental investment as the dependent variable. Standard errors in parentheses are clustered at the school level in column (1) and at the family level in column (2). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B24: Extensive and intensive margin of failure

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Failed a course				Failure rate			
	Full sample	Sib. sample	Full sample	Sib. sample	Full sample	Sib. sample	Full sample	Sib. sample
Morningness PGI	-0.025*** (0.009)	-0.106* (0.057)			-0.037*** (0.014)	-0.176 (0.134)		
Early Bird chronotype			-0.062*** (0.021)	-0.140 (0.117)			-0.050 (0.045)	-0.385 (0.395)
Night Owl chronotype			-0.006 (0.027)	0.081 (0.129)			0.016 (0.048)	0.182 (0.201)
EA PGI	-0.103*** (0.010)	-0.142** (0.068)	-0.102*** (0.010)	-0.124* (0.066)	-0.108*** (0.018)	-0.187 (0.162)	-0.106*** (0.017)	-0.171 (0.146)
Observations	2,662	300	2,662	300	2,662	300	2,662	300
R-squared	0.262	0.759	0.262	0.756	0.311	0.686	0.310	0.696
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Genotype PCs	Yes	No	Yes	No	Yes	No	Yes	No
Family FE	No	Yes	No	Yes	No	Yes	No	Yes
School FE	Yes	No	Yes	No	Yes	No	Yes	No
A.Y. FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4	W4	W4	W4	W4
Mean dep. var.	0.469	0.403	0.469	0.403	-0.106	-0.183	-0.106	-0.183
N of clusters	79	148	79	148	79	148	79	148
Early Birds VS Nigh Owls			-0.0564 (0.0344)	-0.222 (0.136)			-0.0662 (0.0568)	-0.567 (0.386)

Notes: The table reports estimates of the morningness PGI in columns (1), (2), (5) and (6) and of the two chronotype dummies in columns (3), (4), (7) and (8) on the extensive (columns (1) to (4)) and intensive (columns (5) to (8)) margin of course failure. Standard errors in parentheses are clustered at the school level in the odd columns, reporting results in the full sample, and at the family level in the even columns, reporting results in the sibling sample. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table B25: Years of education

	(1) Years educ Full sample	(2) Years educ Sibling sample	(3) Years educ Full sample	(4) Years educ Sibling sample
Morningness PGI	0.006 (0.041)	0.156 (0.213)		
Early Bird chronotype			-0.052 (0.122)	0.350 (0.521)
Night Owl chronotype			-0.049 (0.116)	0.026 (0.423)
EA PGI	0.457*** (0.043)	0.335 (0.279)	0.456*** (0.043)	0.302 (0.272)
Observations	2,662	300	2,662	300
R-squared	0.392	0.816	0.392	0.816
Controls	Yes	Yes	Yes	Yes
Genotype PCs	Yes	No	Yes	No
Family FE	No	Yes	No	Yes
School FE	Yes	No	Yes	No
A.Y. FE	Yes	Yes	Yes	Yes
Weights	W4	W4	W4	W4
Mean dep. var.	14.48	14.54	14.48	14.54
N of clusters	79	148	79	148
Early Birds VS Nigh Owls			-0.00281 (0.138)	0.324 (0.592)

Notes: The table replicates the estimates of Table 2, but using years of education as the dependent variable. Standard errors in parentheses are clustered at the school level in the odd columns, reporting results in the full sample, and at the family level in the even columns, reporting results in the sibling sample. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.