



Genetic predisposition for morningness-eveningness and economic disadvantage: Evidence from Finland over 25 years

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ABSTRACT

Objectives: Individual chronotype may shape economic outcomes through alignment or misalignment with work and societal schedules. Genome-wide association studies suggest that morningness–eveningness has a partially genetic basis. This study examines how genetic predisposition to chronotype relates to economic disadvantage, using polygenic indices for morningness–eveningness both as predictors and as instruments for phenotypic chronotype.

Methods: Employing various regression and extended regression models, we studied data from 20,121 working-aged adults representative of Finnish regions, combining genetic, registry, and survey data from 1992 to 2017.

Results: Genetic markers for morningness were monotonically negatively associated with educational attainment ($p = 0.002$)—a key determinant of economic success—particularly in males. Conversely, the same genetic markers were also monotonically negatively associated with the likelihood of belonging to the lowest income quintile in males ($p = 0.012$), suggesting differential valuation of chronotype traits in education versus the labour market. This pattern emerged in post-2000. Furthermore, among males with higher education, genetic predisposition to eveningness was linked to a higher likelihood of falling into the lowest income quintile ($p < 0.001$), indicating reduced economic returns to their education. No significant associations between chronotype-related genetic markers and income were observed in females across education levels.

Conclusions: This study reveals emerging, gender-specific inequalities in how genetically influenced chronotype traits relate to economic outcomes. Genetic predisposition to eveningness favoured education but hindered income—especially in highly educated males—via phenotypic chronotype pathways. Though modest, these effects highlight the need for workplace inclusion through recognition of chronotype diversity, public sleep health initiatives, and flexible work structures.

1. Introduction

Individual diurnal preferences significantly influence functioning in modern society. Variations in chronotype are associated with educational attainment [1,2], work performance [3,4], health status [5,6], social interactions [7,8], and overall well-being [6–9]. These

associations likely arise because individuals with different chronotypes experience varying levels of alertness and productivity throughout the day, affecting their educational outcomes, labour market performance, and returns on skills. Aligning with societal schedules at the expense of one's preferred circadian rhythm can disrupt sleep patterns, adversely affecting health [10–12] and potentially impairing economic

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performance. Conversely, aligning daily activities with one's chronotype may benefit health and productivity but may also lead to socioeconomic disadvantages when societal norms favour alternative schedules.

Twin studies estimate chronotype heritability at around 50 %, while SNP-based estimates suggest lower heritability (12–14 %) [13–16]. Recent genome-wide association studies (GWAS) have identified genetic variants underlying morningness and eveningness (M-E), enabling the construction of the Morningness Polygenic Index (MPGI) [13], which typically explains 2–3 % of the variance in phenotypic M-E [17,18]. Although chronotype arises from a complex interplay of genetic, individual, and environmental factors [10], and MPGI captures only a fraction of this variation and does not account for environmental influences, it offers valuable insights into unmodifiable determinants. Nevertheless, MPGI may be subject to pleiotropy, where genetic variants associated with chronotype also influence other traits such as mental health [13–19].

This study examines the relationship between economic disadvantage and chronotype-associated genetic factors identified by MPGI [13]. While environmental factors primarily shape income disparities [20], understanding genetic associations can help inform of labour market inequalities. Inclusive labour markets that accommodate diverse chronotypes may enhance both individual well-being and broader economic development [21].

Previous research suggests a link between eveningness and lower income [22,23] but prior studies relied on self-reported or time-use-based chronotype measures that may suffer from reverse causality and unobserved confounding. Recent GWAS findings reported weak negative genetic correlations between morningness and both income ($r_g = -0.10$) and educational attainment ($r_g = -0.13$) [20]. Extending these earlier approaches, we use MPGI as a fixed genetic proxy for chronotype to examine associations with actual educational attainment—an essential driver of economic outcomes—and income, thereby mitigating concerns of reverse causality and confounding [24–27]. Given that males have been more likely than females to exhibit an evening chronotype across diverse populations and age groups [9], we also assess gender-specific associations, an area requiring further research [28,29]. Furthermore, widening income inequalities may amplify chronotype-related economic disparities over time.

To investigate potential causal pathways linking MPGI to economic outcomes via phenotypic M-E, we apply extended regression models. In these models, survey-assessed M-E is treated as an endogenous variable—potentially influenced by unmeasured factors—and instrumented with MPGI. Using a genetic instrument fixed at conception allows us to isolate variation in habitual chronotype attributable to genetic predisposition. This strategy helps reduce bias from environmental confounding and reverse causality, allowing us to assess whether MPGI predicts habitual M-E and whether the instrumented M-E is significantly associated with economic outcomes.

By investigating these relationships in Finland—a country with universal education, healthcare, and social support systems that seek to reduce individual disadvantages—we offer novel insights into the role of genetic chronotype predispositions in shaping economic disadvantage. The study also underscores the relevance of sleep health as a modifiable determinant of socioeconomic inequalities, with implications for population health and social equity.

2. Methods

2.1. Study cohorts

We used pooled genetic and repeated cross-sectional survey data from the Finnish National FINRISK Study (FR; 1992, 1997, 2002, 2007, 2012) [30] and the FinHealth (FH; 2017) [31] study cohorts, merged with national registry data on education. These cohorts are representative of the adult population across different Finnish regions.

The sample included adults aged 25–64 years, representing prime and pre-retirement working ages. Participants were excluded if they were outside this age range, lacked genetic or registry data, were from Lapland (due to limited coverage), or had inconsistent household size data. Participants with multiple observations were excluded. To improve income measurement precision from household income reports, we restricted the sample to households with a maximum of 10 members, including children, and no more than one additional adult besides the participant, following evidence of assortative mating patterns in Nordic countries [32]. The final analytic sample included 20,121 participants: 7811 from 1992 to 1997 and 12,311 from 2002 to 2017. The data and methodological approach are consistent with our research group's other studies on genetic proxies for brain health traits and their links to economic outcomes, with the participant flowchart detailed in Hazak et al. (2025) [33].

2.2. Ethical approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. Approval for this study was granted by the Finnish Institute for Health and Welfare, and the FR and FH study protocols were approved by the Finnish Institute for Health and Welfare and/or the Coordinating Ethical Committee of the Helsinki and Uusimaa Hospital District (approval numbers 38/96, 558/E3/2001, 229/E0/06, 162/13/03/00/2011 and 37/13/03/00/2016). All participants provided written informed consent, and the study adhered to the Declaration of Helsinki.

2.3. Genetic data and methodology: polygenic proxies for morningness-eveningness

DNA was extracted from blood samples collected during FR and FH study visits. MPGI was derived from a large-scale GWAS of 697,828 UK Biobank and 23andMe participants aged 40–69 years, where M-E was measured by self-reports, and accelerometer-derived sleep timing in a subsample [13]. Using the UK Biobank subsample ($N = 449,734$), we constructed the MPGI via PRS-CS Bayesian regression with continuous shrinkage priors, referencing European ancestry samples from the 1000 Genomes Project. Each MPGI was calculated as a weighted sum of risk allele counts across 1,102,758 variants and standardised (mean 0, SD 1) using R 3.6.0. FR and FH cohorts were genotyped separately with minimal participant overlap. Further genotyping details are in Hazak et al. (2025) [33].

2.4. Phenotypic measures of morningness-eveningness and validation of polygenic proxies

Phenotypic chronotype was assessed using two survey-based measures. First, perceived M-E type was obtained from FR 2007, FR 2012, and FH 2017 ($N = 8268$) using a Likert scale question categorising participants as morning or evening types (Table 1). Wilcoxon rank-sum tests showed highly significant differences ($p < 0.001$) in MPGI distributions between these groups.

Second, a shortened six-item version of the Horne-Östberg Morningness-Eveningness Questionnaire (sMEQ) [34,35] was used in FR 2007 and 2012 ($N = 5109$). Participants were classified into Morning (19–27 points), Intermediate (13–18), and Evening types (5–12). The sMEQ showed acceptable internal consistency (Cronbach's $\alpha = 0.81$). Wilcoxon rank-sum tests showed that MPGI distribution differences across all the chronotype pairs were statistically significant ($p < 0.001$). In our sample, MPGI explained 6.0 % of the variance between Morning and Evening types based on pseudo- R^2 from probit models adjusting for the first three genetic principal components (PC1–PC3) ($p(\chi^2) < 0.001$). Controlling for population stratification using PC1–PC3 follows established best practices in Finnish genetic studies and reflects the high genetic homogeneity of the Finnish population.

Extended regression models further validated MPGI as a statistically

Table 1
Descriptive statistics of the pooled FR 1992–2007 and FH 2017 sample.

Variable	Explanation	All		Male		Female		Evening ^a		Intermediate ^a		Morning ^a	
		Mean /%	Min Max SD	Mean /%	Min Max SD	Mean /%	Min Max SD	Mean /%	Min Max SD	Mean /%	Min Max SD	Mean /%	Min Max SD
N		20,121		9248		10,873		2016		16,115		1990	
MPGI		0.000	−4.13 4.01 0.997	−0.007	−3.83 4.01 1.002	0.006	−4.13 3.80 0.993	−1.755	−4.13 −1.30 0.396	0.004	−1.30 1.30 0.666	1.743	1.30 4.01 0.385
Income quintile ^b													
1	Lowest	22 %		21 %		22 %		22 %		22 %		20 %	
2	Lower	20 %		20 %		21 %		20 %		20 %		21 %	
3	Medium	19 %		18 %		20 %		17 %		19 %		20 %	
4	Higher	20 %		20 %		19 %		20 %		20 %		20 %	
5	Highest	20 %		21 %		18 %		22 %		19 %		19 %	
Education													
1	Primary	20 %		22 %		18 %		18 %		20 %		20 %	
2	Secondary (reference)	42 %		45 %		40 %		41 %		42 %		44 %	
3	Higher	38 %		33 %		42 %		40 %		38 %		36 %	
Gender	Male = 0 (reference)	46 %		100 %		0 %		48 %		46 %		44 %	
Age		44.9	25 64 11.7	45.3	25 64 11.6	44.5	25 64 11.7	44.8	25 64 11.4	44.8	25 64 11.7	45.5	25 64 11.5
Perceived M-E type ^c													
1	Absolutely morning	19 %		18 %		19 %		10 %		18 %		31 %	
2	More morning (reference)	31 %		31 %		32 %		22 %		31 %		39 %	
3	More evening	33 %		35 %		32 %		39 %		34 %		21 %	
4	Absolutely evening	17 %		17 %		17 %		29 %		16 %		8 %	
sMEQ ^d													
1	Morning	44 %		47 %		42 %		32 %		44 %		62 %	
2	Intermediate (reference)	42 %		41 %		43 %		44 %		43 %		31 %	
3	Evening	13 %		12 %		15 %		24 %		13 %		7 %	

^a All sample members in the 1st decile (Evening), pooled 2nd–9th deciles (Intermediate) and 10th decile (Morning) of the Morningness Polygenic Index (MPGI) [13].

^b Some income quintile sizes in the full sample differ slightly from 20 % because income was measured in range categories in the FR and FH surveys, which did not allow for allocation of participants into income groups of exactly 20 %.

^c sMEQ denotes the shortened six item version [34] of the Horne–Östberg Morningness-Eveningness Questionnaire (MEQ) [35]; available for years 2007 and 2012; total N = 5109.

^d Self-reported M-E type denotes perceived morningness-eveningness type derived from Likert scale responses to the survey question “There are so-called “morning people” (early to rise, early to bed) and “evening people” (late to rise, late to bed). Which are you?”; available for years 2007–2017; total N = 8268.

significant predictor of perceived M-E type and sMEQ scores ($p < 0.001$; see Statistical Analysis and Results). Together, these complementary validation approaches confirm that MPGI—derived from a UK sample of individuals aged 40–69—effectively differentiates between survey-based phenotypic chronotypes in our Finnish sample of adults aged 25–64.

2.5. Income data

Income was based on self-reported annual household income categories. Household income was translated into a continuous variable using category midpoints, and adjusted for the number of adult household members (1 or 2) to provide a standardised measure of economic resources available to each participant. The adjusted household income variable was then divided into quintiles to allow relative comparisons across the income distribution and across years. The main outcome for economic disadvantage was defined as the probability of being in the lowest income quintile, which is a widely used indicator of relative poverty and persistent economic disadvantage in socioeconomic research (e.g., OECD, Eurostat).

2.6. Registry data on education and controls

Educational attainment was obtained from Statistics Finland registry data, measured at the beginning of each study year. Control variables obtained from the Finnish National Population Register included gender, age, and categorical birth cohort.

Since chronotype polygenic proxies are fixed throughout life and educational attainment typically stabilises by age 25, the repeated cross-sectional design does not substantially limit inference.

2.7. Descriptive statistics

Descriptive statistics have been presented in Table 1, histograms of MPGI by gender in Supplemental Fig. 1, education by study year and by gender in Supplemental Fig. 2, and pairwise linear correlation matrix in Supplemental Table 1.

2.8. Statistical analysis

We first examined the association between MPGI and educational attainment using ordered probit (oprobit) regression models. Next, we

assessed economic disadvantage—defined as belonging to the lowest income quintile—using probit models.

A two-stage approach was employed: first, testing the association between MPGI and income disadvantage; second, stratifying the sample by educational level to investigate whether MPGI associations with income persisted beyond educational attainment. Income differences across education groups are shown in Supplemental Fig. 3.

MPGI was modelled continuously, either as a linear term, or in both linear and squared terms to account for the possibility that both morningness and eveningness may be associated with economic disadvantages. Models adjusted for gender, age (linear and squared), birth cohort (by decades), study year dummies, and PC1–PC3. Alternative models included MPGI–gender interaction terms to examine the hypothesised greater disadvantage among males, given their overall higher propensity for eveningness (Table 1; earlier studies [9]), and MPGI–birth cohort interactions to explore whether the associations with education have intensified over time. As a sensitivity analysis, the main results are also presented from comparable models that include PC1–PC10.

Income analyses were stratified by pre- and post-2000 periods, reflecting the rise in income inequality in Finland. The Gini coefficient of income inequality, which ranged from 20.3 to 22.7 between 1986 (when data became available) and 1996 (the reference year for the FR 1997 cohort), rose to 26.9–29.1 from 2001 (the reference year for the FR 2002 cohort) to 2023, according to Statistics Finland’s Income Distribution Statistics. Wilcoxon rank-sum tests confirmed significant shifts ($p = 0.03$) in income distributions, supporting the decision to examine these periods separately.

To investigate potential causal pathways from genetic predispositions for M-E to economic outcomes via phenotypic M-E, we applied Extended Regression Models (ERM; StataCorp LLC, 2023) using maximum likelihood estimation. These recursive systems jointly estimated: (1) a first-stage model predicting a phenotypic M-E trait—either

perceived M-E type or sMEQ score (both specified as ordered probit)—instrumented by MPGI; and (2) a second-stage model predicting a socioeconomic outcome—educational attainment (ordered probit) or probability of belonging to the lowest income quintile (probit)—with the phenotypic M-E trait included as an endogenous regressor potentially influenced by unobserved confounders. Both equations included identical covariates: gender, age (linear and squared), birth cohort dummies, study year dummies, and PC1–PC3. MPGI was included exclusively in the first-stage equation, serving as an instrument to isolate variation in phenotypic chronotype attributable to genetic predisposition. This approach helps disentangle genetic effects on socioeconomic outcomes from those driven by environmental factors or reverse causality operating through phenotypic M-E.

Robust standard errors accounted for heteroscedasticity, non-normality, and data clustering. Statistical significance was defined as $p < 0.005$, and findings at $p < 0.05$ were considered suggestive. As each model tested a distinct hypothesis without multiple subgroup comparisons, no additional correction for multiple testing was applied—except in the exploratory analysis of MPGI–birth cohort interactions. Analyses were performed using STATA 18.

3. Results

3.1. Morningness Polygenic Index and education

MPGI was monotonically negatively associated with educational attainment ($p = 0.002$), with no evidence of non-linearity (Table 2). Although the MPGI–gender interaction term was positive but not statistically significant ($\beta = 0.027$, $p > 0.05$), the negative main effect of MPGI ($\beta = -0.036$, $p = 0.002$) suggests that the association is primarily driven by males (Table 2), as gender-stratified models further clarified this pattern: MPGI was significantly negatively associated with

Table 2
Coefficient estimates from ordered probit regression models of educational attainment in the pooled 1992–2017 sample.

Sample	All	All	Males	Males	Females	Females
MPGI specification	Linear	Linear and squared	Linear	Linear and squared	Linear	Linear and squared
Model	oprobit	oprobit	oprobit	oprobit	oprobit	oprobit
N	20,121	20,121	9248	9248	10,873	10,873
Dependent variable: Education						
Explanatory variables:						
MPGI	-0.036** (0.012)	-0.021* (0.008)	-0.036** (0.012)	-0.036** (0.012)	-0.009 (0.011)	-0.010 (0.011)
MPGI squared		0.005 (0.006)		0.003 (0.008)		0.008 (0.008)
Female	0.199*** (0.016)	0.200*** (0.016)				
MPGI # Female	0.027 (0.016)					
Age	0.009 (0.007)	0.009 (0.007)	0.007 (0.010)	0.007 (0.010)	0.013 (0.010)	0.013 (0.010)
Age squared	-0.000*** (0.000)	-0.000*** (0.000)	-0.000* (0.000)	-0.000* (0.000)	-0.001*** (0.000)	-0.001*** (0.000)
PC1	8.831*** (1.156)	8.809*** (1.156)	11.362*** (1.666)	11.359*** (1.666)	6.406*** (1.612)	6.391*** (1.612)
PC2	-3.683** (1.194)	-3.704** (1.194)	-2.271 (1.650)	-2.270 (1.649)	-5.214** (1.720)	-5.207** (1.719)
PC3	-0.319 (1.110)	-0.308 (1.110)	-0.762 (1.578)	-0.758 (1.578)	0.231 (1.568)	0.248 (1.568)
Year dummies	Yes	Yes	Yes	Yes	Yes	Yes
Birth cohort dummies	Yes	Yes	Yes	Yes	Yes	Yes
Cut: primary/secondary education	-1.007*** (0.174)	-1.004*** (0.174)	-0.949*** (0.250)	-0.950*** (0.250)	-1.178*** (0.244)	-1.172*** (0.244)
Cut: secondary/higher education	0.269 (0.175)	0.272 (0.175)	0.340 (0.251)	0.340 (0.251)	0.097 (0.244)	0.103 (0.244)
Pseudo-R ²	0.075	0.075	0.047	0.047	0.097	0.097
$p(\chi^2)$	***	***	***	***	***	***

* $p < 0.05$, ** $p < 0.005$, *** $p < 0.001$; Robust standard errors in parentheses; MPGI denotes Morningness Polygenic Index [13].

education in males ($\beta = -0.036$, $p = 0.002$), whereas the association was small and non-significant in females ($\beta = -0.009$, $p > 0.05$; Table 2; Fig. 1). These findings indicate that the MPGI–education association is largely specific to males, with no meaningful effect observed in females, likely due to greater heterogeneity in the latter group. For example, a male with a strong genetic predisposition toward eveningness—reflected by an MPGI value of -1.755 (the mean in the lowest decile; Table 1)—had a predicted probability of 35.0 % of attaining higher education ($p < 0.001$; 99.5 % CI: 32.6–37.4 %; 95 % CI: 33.3–36.6 %). In contrast, a genetically extreme morning-type male (MPGI = 1.743, top decile mean) had a predicted probability of 30.7 % ($p < 0.001$; 99.5 % CI: 28.3–33.0 %; 95 % CI: 29.0–32.3 %).

No significant MPGI–birth cohort interaction was found after Bonferroni correction for multiple testing (Supplemental Table 2), indicating no robust temporal trend in the MPGI–education association.

Extended regression models further supported these findings. MPGI was a significant predictor of both perceived M-E type and sMEQ score ($p < 0.001$; Supplemental Table 3). When these phenotypic chronotypes were instrumented with MPGI, perceived “absolutely morning” type was negatively associated with educational attainment ($p = 0.005$), “more evening” type was positively associated ($p = 0.005$) primarily in males, and the morning type derived from sMEQ showed a suggestive negative association ($p = 0.036$; Supplemental Table 3).

3.2. Morningness Polygenic Index and income

Probit models examining the likelihood of belonging to the lowest income quintile indicated a significant negative association between MPGI and income disadvantage during the post-2000 period ($p = 0.012$; Table 3), but not during the pre-2000 period (Supplemental Table 4). This aligns with the marked rise in income inequality in Finland and increased higher education attainment after 2000 (Supplemental Fig. 2). No evidence of non-linear associations between MPGI and income was observed (Supplemental Table 5).

Although the MPGI–gender interaction term was positive but not statistically significant ($\beta = 0.040$, $p > 0.05$) in the full 2002–2017 sample, the negative main effect of MPGI on income ($\beta = -0.049$, $p = 0.012$) suggests the association is primarily driven by males (Table 3). Stratified models confirmed this: in males, MPGI was significantly and negatively associated with income ($\beta = -0.052$, $p = 0.008$), whereas in females, the association was near zero and non-significant ($\beta = -0.008$, $p > 0.05$) (Table 3). This indicates that the MPGI–income relationship is evident in males but not among females, likely due to greater

heterogeneity in the latter group. For example, a male with an MPGI of -1.755 (lowest decile mean) had a 23.1 % predicted probability of being in the lowest income quintile ($p < 0.001$; 99.5 % CI: 19.9–26.3 %; 95 % CI: 20.9–25.3 %), compared to 18.2 % for a male in the highest MPGI decile (MPGI = 1.743; $p < 0.001$; 99.5 % CI: 15.3–21.0 %; 95 % CI: 16.2–20.1 %).

To assess whether MPGI–income associations extended beyond educational pathways, we examined models within educational subgroups. Among males with higher education (37 % of the male 2002–2017 sample; $N = 2059$), MPGI was significantly negatively associated with the likelihood of being in the lowest income quintile ($p < 0.001$; Supplemental Table 6). No significant association was observed in females (Supplemental Table 6). The MPGI–female interaction was positive (rightmost column of Table 3), offsetting the main negative MPGI effect seen in males (Fig. 2).

For instance, among highly educated males, an extreme evening-type individual (MPGI = -1.755) had a predicted 13.6 % probability of belonging to the lowest income quintile ($p < 0.001$; 99.5 % CI: 9.3–18.0 %; 95 % CI: 10.6–16.7 %), while an extreme morning-type male (MPGI = 1.743) had only a 5.2 % probability ($p < 0.001$; 99.5 % CI: 2.7–7.8 %; 95 % CI: 3.4–7.0 %) (Fig. 2).

Extended regression models corroborated these findings. MPGI significantly predicted phenotypic chronotypes ($p < 0.001$; Supplemental Table 7). In males, both perceived “absolutely evening” chronotype and the evening type from sMEQ—when instrumented with MPGI—were suggestively associated with higher likelihood of low income ($p < 0.05$). No significant associations were observed for females. In the higher education male subsample ($N = 1438$; Supplemental Table 8), perceived “absolutely morning” type was suggestively associated with lower likelihood of low income ($p = 0.037$), while “more evening” ($p = 0.004$), and “absolutely evening” ($p < 0.001$) were associated with higher likelihood of low income. Among males with higher education ($N = 869$), the sMEQ-derived evening chronotype was positively associated with the likelihood of belonging to the lowest income quintile ($p < 0.001$), while the morning chronotype showed a negative association ($p = 0.004$; Supplemental Table 8).

3.3. Sensitivity analysis with extended population stratification

Including ten genetic principal components (PC1–PC10) underscored the robustness of the main findings (Supplemental Table 9), supporting the common practice of using PC1–PC3 in studies based on the genetically homogeneous Finnish population, as the additional

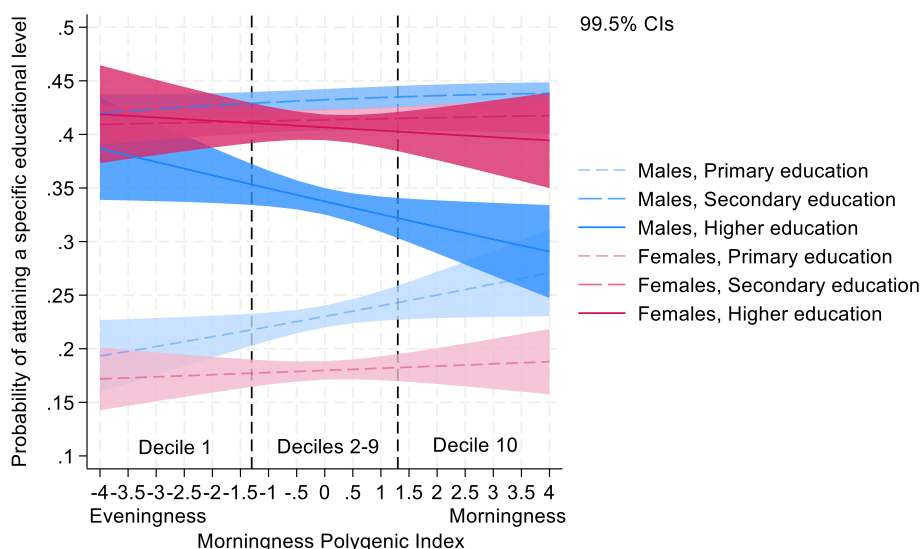


Fig. 1. Morningness Polygenic Index and predicted probabilities of attaining each educational level in the pooled 1992–2017 sample ($N = 20,121$) with 99.5 % CI.

Table 3

Coefficient estimates from the probit regression models of the probability of belonging to the lowest income quintile in the pooled 2002–2017 sample.

Sample	All	Males	Females	All with primary education	All with secondary education	All with higher education
MPGI specification	Linear	Linear	Linear	Linear	Linear	Linear
Model	probit	probit	probit	probit	probit	probit
N	12,026	5563	6463	1557	5286	5183
Dependent variable: Lowest income quintile						
Explanatory variables:						
MPGI	-0.049* (0.019)	-0.052* (0.020)	-0.008 (0.018)	-0.025 (0.045)	-0.046 (0.027)	-0.152*** (0.040)
Female	0.009 (0.026)			0.023 (0.068)	0.087* (0.038)	0.125* (0.051)
MPGI # Female	0.040 (0.027)			-0.011 (0.069)	0.019 (0.038)	0.173*** (0.051)
Age	-0.162*** (0.013)	-0.156*** (0.020)	-0.165*** (0.018)	-0.092 (0.047)	-0.174*** (0.019)	-0.132*** (0.025)
Age squared	0.002*** (0.000)	0.002*** (0.000)	0.002*** (0.000)	0.001* (0.000)	0.002*** (0.000)	0.001*** (0.000)
PC1	-3.906* (1.764)	-6.463* (2.602)	-1.562 (2.396)	-3.437 (4.328)	-4.499 (2.564)	3.468 (3.191)
PC2	7.814*** (1.751)	7.627** (2.488)	7.910** (2.491)	6.391 (4.140)	7.164* (2.581)	6.703* (3.020)
PC3	1.880 (1.735)	2.445 (2.572)	1.380 (2.351)	4.738 (4.284)	1.275 (2.429)	2.306 (3.139)
Year dummies	Yes	Yes	Yes	Yes	Yes	Yes
Birth cohort dummies	Yes	Yes	Yes	Yes	Yes	Yes
Constant	2.552*** (0.344)	2.449*** (0.520)	2.569*** (0.460)	0.829 (1.279)	3.060*** (0.477)	1.502* (0.608)
Pseudo-R ²	0.033	0.040	0.029	0.024	0.035	0.043
p(χ ²)	***	***	***	***	***	***

*p < 0.05, **p < 0.005, ***p < 0.001; Robust standard errors in parentheses; MPGI denotes Morningness Polygenic Index [13].

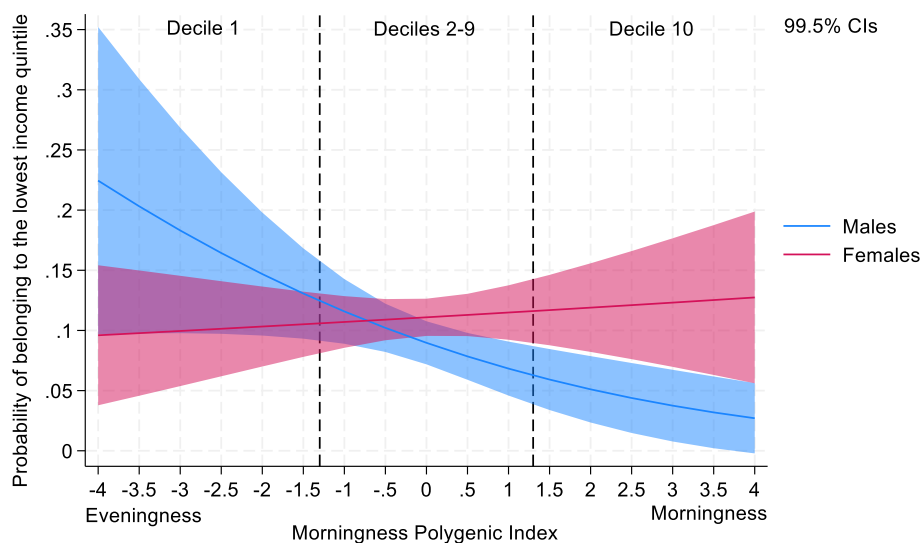


Fig. 2. Morningness Polygenic Index and predicted probabilities of belonging to the lowest income quintile in the pooled 2002–2017 higher education subsample (N = 5183) with 99.5 % CI.

inclusion of PC4–PC10 did not materially affect the estimates and only marginally increased the models’ explanatory power (pseudo-R²).

4. Discussion

This study offers novel insights into how genetic predisposition to M–E relates to educational and income outcomes. Using Finnish population-based cohorts across 25 years, we found that genetically proxied eveningness was positively associated with educational attainment in males but also with a higher likelihood of low income in this group—especially among those with higher education. Extended regression models supported these findings by isolating the component of phenotypic M–E attributable to genetic predisposition. Instrumented M–E measures confirmed that genetically influenced eveningness is

linked to both higher education and economic disadvantage in men.

Previous studies have yielded mixed results on the link between M–E and education. Although phenotypic eveningness has been associated with poorer academic performance due to misaligned school schedules [1,2], genome-wide studies have reported weak positive genetic correlations between eveningness and educational attainment [20,36]. This divergence may reflect structural features of higher education—such as schedule flexibility or the self-directed nature of advanced learning—which may favour evening types. Negative genetic correlations between morningness and both intelligence and mental disorders [13,36,37] may also contribute to these patterns. As habitual chronotype changes with age, our findings suggest that both the MPGI—derived from GWAS data based on middle- and older-aged adults—and the survey-based M–E measures in our adult sample (aged 25–64) capture stable aspects of

chronotype that are meaningfully associated with educational trajectories formed earlier in life.

At the same time, our results reinforce prior findings that phenotypic eveningness is linked to economic disadvantage [22,23], adding a genetic dimension to this association. Among males with higher education, eveningness was associated with a greater likelihood of low income, implying lower economic returns to education for this group. This contrast—where eveningness is associated with higher education but lower income—suggests that M–E traits may be differentially valued in academic and labour market contexts. A recent GWAS also reported a weak positive genetic correlation between morningness and income beyond educational attainment [20]. The convergence of evidence across studies using varied chronotype measures, analytical techniques, study samples and designs strengthens the credibility of these findings [20,22,23].

Gender differences appear to shape these associations. Males typically exhibit stronger eveningness [9], which may amplify genetic expression. Hormonal influences, such as testosterone, may also play a role [38,39]. Social roles and expectations—such as caregiving responsibilities—may suppress chronotype expression in females, potentially explaining the weaker associations in women [6]. These dynamics point to important gene–environment interactions and biological factors warranting further research. Chronotype is a dynamic and multifaceted construct shaped by both biological and environmental factors. Physical influences such as light exposure, and social conditions such as work schedules and family responsibilities, can substantially shift sleep timing regardless of genetic predisposition [10,40]. This plasticity implies that associations between genetic markers and economic outcomes—including observed gender disparities—may be moderated or obscured by life-course circumstances.

The Finnish context introduces additional considerations, as seasonal variation in daylight is particularly pronounced at high latitudes. Circadian phase and chronotype are sensitive to these seasonal fluctuations in light exposure [40,41], and the magnitude of such effects may vary according to genetic background [42]. While the main models of economic outcomes relied exclusively on genetic measures of chronotype, which are unaffected by the timing of data collection, seasonal influences could confound associations between genetic markers and phenotypic chronotype in the extended regression models that incorporated survey-based M–E measures. However, these extended models were based on smaller samples ($N \leq 8268$), and survey dates were distributed across the calendar year, which limited the statistical power for stratification by season. In addition, eveningness has been shown to correlate with latitude and to vary with age, with more pronounced age-related changes observed in men than in women [43]. These factors may partly account for the male-specific eveningness–income relationship observed in this study, reflecting both the distribution of chronotype within the Finnish population and age-related shifts across the 25–64 age range. The repeated cross-sectional design further implies that chronotype and annual income were measured contemporaneously, making it difficult to determine whether phenotypic eveningness preceded or resulted from earlier life-course choices that shaped economic outcomes. Finally, migration or residential mobility related to chronotype—for example, morning types relocating southwards in pursuit of greater income opportunities—could also play a role, although this could not be assessed here and remains an interesting avenue for future research.

Our findings also reveal important temporal shifts. The association between eveningness and income disadvantage emerged only in the 2000s, coinciding with rising income inequality and a transition to a more knowledge-based economy in Finland. These changes likely intensified the labour market relevance of cognitive and self-regulatory traits, exacerbating chronotype-related disadvantages. As higher education becomes more widespread, understanding how M–E predispositions interact with occupational expectations gains importance.

The wide age range (25–64 years) in this study covering 25 years

(1992–2017) also implies that different generations faced distinct educational and labour market conditions. Although the models control for age, study year, and birth cohort dummies, which mitigate confounding by generational context, it remains possible that the observed associations reflect interactions between genetic predispositions, age-related chronotype shifts, and changing societal conditions. This generational dimension should be considered when interpreting the results.

The biological mechanisms linking chronotype and socioeconomic outcomes remain unclear, but circadian rhythm regulation and glutamatergic signalling—both implicated in learning and psychiatric vulnerability—are likely involved [13,44]. Pleiotropic genetic effects may influence both chronotype and mental or cognitive traits, shaping labour market performance through shared pathways. Strengths of this study include its large, population-based survey, registry and genetic data, validated chronotype measures, and their structural modelling using genetic instruments to address confounding and reverse causality. Limitations include the use of polygenic indices derived from UK samples, potential misclassification from self-reported income, and potential selection bias due to voluntary DNA sampling in the population-based study cohorts.

5. Conclusion

This study demonstrates that genetic predisposition to eveningness is modestly but consistently linked to reduced income outcomes—particularly among highly educated males—despite a positive association with education. Recognising chronotype as a partially biologically rooted trait relevant for economic inequality can inform inclusive policies and practices, and sleep health initiatives. Rather than expecting individuals to align with societal and work schedules, accommodating chronotype diversity through more flexible educational and workplace structures may help mitigate these disadvantages and promote greater inclusion, sleep health, well-being, and productivity in increasingly knowledge-based economies.

CRediT authorship contribution statement

Aaro Hazak: Writing – original draft, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Katri Kantojärvi:** Writing – review & editing, Resources. **Johanna Liuhanen:** Writing – review & editing. **Sonja Sulkava:** Writing – review & editing. **Tuija Jääskeläinen:** Writing – review & editing, Resources. **Veikko Salomaa:** Writing – review & editing, Resources. **Seppo Koskinen:** Writing – review & editing, Resources. **Markus Perola:** Writing – review & editing, Resources. **Tiina Paunio:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization.

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Data availability statement

The FR and FH data that support the findings of this study are available from the Finnish Institute for Health and Welfare subject to permission.

Declaration of competing interest

TP: Idorsia Pharmaceuticals and Biogen (unrelated to the present work). The authors declare no other competing interests.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.sleep.2025.106811>.

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