

Supplementary Appendix to:

The genetics of political participation: Leveraging poly-genic indices to advance political behavior research

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A.1 Data

Table A.1 displays the question wordings for all items used to construct the political participation measures in each of the four samples or cohorts used in this study. Table A.2 lists all polygenic indices included in the PGI repository described in Becker et al. (2021) and used in this study. The 10 PGIs included in the study are in **boldface**. The criteria for being included in the study are as follows: i) a health or psychological target trait and ii) an expected incremental $R^2 \geq 2\%$. The four samples included in the study are in **boldface**. The criteria for being included in the study are as follows: i) relevant political outcomes should be included; and ii) the sample should be family-based/include sibling pairs. It should be noted here that several of the remaining repository cohorts include phenotypic data of relevance to political behavior researchers, for instance the English Longitudinal Study of Ageing (ELSA) and Dunedin Multidisciplinary Health and Development Study.

We use restricted individual level information obtained from each of the four cohorts. As part of our contractual agreement with each cohort, we agreed not to disseminate the data to other individuals. However, researchers can access the restricted data directly from each cohort.

Genome-wide DNA information is available from the subset of respondents in the four cohorts who contributed saliva samples (Add Health and WLS) or both saliva and blood samples (MTFS and STR).¹ This was done during the fourth wave of data collection in 2008 for Add Health, the 2011 survey round for WLS, between 2005 and 2008 for MTFS, and from 2004 onwards for STR. Genotyping – that is, the process of determining the DNA sequence (the genotype) at different positions within the genome of an individual – was performed using a range of commercially available genotyping platforms: Illumina Omni 1.1 and 2.5 platforms for Add Health; Illumina 660W-Quad for MTFS; Illumina Human Omni Express BeadChip for WLS; and Illumina Human Omni Express BeadChip, Illumina Human Core Exome 550K array, and Illumina Global Screening 650K for STR. Genotyping on these platforms results in the identification of several hundred thousand SNPs.

Importantly, these genotyping platforms do not directly genotype all SNPs in the genome. Therefore, it is common practice to impute the genotypes to a reference panel that has been genotyped for a greater number of variants. This greatly enhances the coverage of genomic variation beyond the original genotypes. This means it is possible to perform genome-wide association analyses on many more SNPs than those present on the original genotyping platform. Moreover, imputation increases the overlap of variants available for analysis between different genotyping platforms, facilitating meta-analyses of datasets genotyped on different arrays (as is the case for the cohorts included in the PGI repository). The genotype information in the four PGI repository cohorts used in this study has been imputed based on either the Haplotype Reference Consortium (Add Health, MTFS, and STR) or the 1000 Genomes (WLS) reference panels. The GWAS underlying the PGIs are based on these imputed genotypes.

¹However, the specific tissue that is tested is of no consequence for the resulting data since all somatic cells contain the same genetic information.

Add Health: Access to the polygenic indexes and full phenotype data in Add Health is publicly available via a restricted data use contract with the University of North Carolina at Chapel Hill. Obtain access by visiting the CPC Data Portal at data.cpc.unc.edu/projects/2/view or see the Add Health data page at <https://www.cpc.unc.edu/projects/addhealth/documentation/restricteduse>. Add Health genotype data can be accessed via the database of Genotypes and Phenotypes (dbGaP, www.ncbi.nlm.nih.gov/gap, accession number phs001367.v1.p1).

MTFS: Access to the MTFS PGIs is available by contacting Matt McGue (mcgue001@umn.edu), who will provide access authorization. Access to MTFS phenotypic data will require a research proposal the structure of which can be provided by Matt McGue. Use of phenotypic data requires an approved proposal that is approved by the MTFS Principal Investigator Committee; access to the MTFS PGIs does not require an approved proposal.

WLS: Both the WLS polygenic index and phenotypic data is publicly available. As of November 2022, researchers who wish to use these polygenic indexes should email a brief research proposal and a copy or link to their CV to Carol.Roan@wisc.edu. Researchers will additionally need to receive IRB approval from their home institution and enter into a Data Use Agreement between the researcher’s home institution and the University of Wisconsin-Madison. For the most up-do-date instructions, see www.ssc.wisc.edu/wlsresearch/documentation/GWAS/.

STR: Researchers interested in using STR data must obtain approval from the Swedish Ethical Review Authority and from the Steering Committee of the Swedish Twin Registry. Researchers using STR data are required to follow the terms of a number of clauses designed to ensure protection of privacy and compliance with relevant laws. For further information please visit <https://ki.se/en/research/swedish-twin-registry-for-researchers>.

It should be noted that the PGI repository will be significantly expanded in its second release in the near future, and will then contain both more cohorts, more PGIs, and imputed parental PGIs.

Table A.1: Measures of the political outcomes per cohort

Minnesota Center for Twin and Family Research (MTFS)	
Political participation	N/A
Turnout - self-stated	I vote in national or state elections is “not true at all”, “not very true”, “pretty true”, or “very true”.
Turnout - high stake	1996, 2000, 2004, 2008, 2012, 2016 and 2020 elections
Turnout - low stake	1994, 1998, 2002, 2006, 2010, 2014 and 2018 elections.
National Longitudinal Study of Adolescent to Adult Health (Add Health)	
Continued on next page	

Table A.1: Measures of the political outcomes per cohort

Political participation	Question wording: “Which of the following things have you done during the last 12 months?” Response alternatives: i) Contributed money to a political party or candidate; ii) Contacted a government official regarding political or community issues; iii) Run for a public office; iv) Run for a non-public office; v) Attended a political rally or march. Political participation is an additive index including the 3 subitems that are also present in the other datasets (contacted gov’t official, contributed, attended rally/march/demonstration), rescaled to the 0-1 range.
Turnout - self-stated	Question wording: “How often do you usually vote in local or statewide elections?” Response alternatives: i) Never; ii) Sometimes; iii) Often; iv) Always. The turnout scale will be rescaled to the 0-1 range.
Turnout - high stake	N/A
Turnout - low stake	N/A
Swedish Twin Registry (STR)	
Political participation	Question wording: “During the last five years, have you done any of the following to express your political opinions?” Response alternatives: i) Contacted a politician personally, or in writing, or some other way; ii) Contacted a public sector official; iii) Made financial contributions; iv) Participated in a protest action or demonstration; v) Boycott, for example certain goods; vi) Signed a petition. Political participation is an additive index including the 3 subitems that are also present in the other datasets (contacted gov’t official, contributed, attended rally/march/demonstration), rescaled to the 0-1 range.
Turnout - self-stated	N/A
Turnout - high stake	Validated turnout data from the national elections in 1970, 1994, 2010, and 2018. Turnout is measured as the per-individual average turnout across all 4 elections.
Turnout - low stake	Validated turnout data from the European Parliament elections in 2009 and 2019. Turnout is measured as the per-individual average turnout across the 2 elections.
Wisconsin Longitudinal Study (WLS)	
Political participation	NA
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Table A.1: Measures of the political outcomes per cohort

Turnout - self-stated	Which statement best describes your decision to vote in the November 2002 election? i) I did not vote in the election in November 2002 ii) I thought about voting in November 2002 but did not iii) I usually vote but did not vote in November 2002 iv) I am sure I voted at the polls in the election in November 2002 v) I am sure I voted by absentee ballot in November 2002. Which statement best describes your decision to vote in the November 2008 election? i) I did not vote in the election in November 2008 ii) I thought about voting in November 2008 but did not iii) I usually vote but did not vote in November 2008 iv) I am sure I voted at the polls in the election in November 2008 v) I am sure I voted by absentee ballot in November 2008.
Turnout - high stake	2000, 2004, 2008, and 2012 elections
Turnout - low stake	2002, 2006, and 2010 elections.

Table A.2: Polygenic indices in the PGI repository

Target trait	Expected ΔR^2	h_{SNP}^2
<i>Anthropometric</i>		
Body mass index (BMI)	17.03%	24.26%
Height	36.20%	46.60%
<i>Cognition and education</i>		
Alzheimer's	0.22%	4.62%
Childhood reading	1.14%	6.90%
Cognitive performance	10.73%	23.18%
Educational attainment	7.27%	11.04%
Highest math	7.85%	15.10%
Self-reported math ability	8.47%	14.62%
<i>Fertility and sexual development</i>		
Age at first birth	6.98%	19.58%
Age first menses (women)	9.88%	19.65%
Age voice first deepened (men)	0.79%	9.63%
Number ever born (men)	0.58%	4.40%
Number ever born (women)	1.72%	7.96%
<i>Health and health behaviors</i>		
Alcohol - misuse	1.62%	9.83%
Allergy - cat	0.67%	9.61%
Allergy dust	0.49%	8.17%
Continued on next page		

Table A.2: Polygenic indices in the PGI repository

Allergy - pollen	0.87%	11.03%
Asthma	1.72%	5.91%
Asthma/eczema/rhinitis	3.37%	8.18%
Attention deficit hyperactivity disorder	4.09%	22.84%
Cannabis use	1.46%	8.25%
Cigarettes per day	3.49%	11.07%
COPD	0.36%	2.64%
Depressive symptoms	3.08%	7.22%
Drinks per week	2.17%	5.46%
Eczema	0.07%	1.03%
Ever smoker	5.43%	8.73%
Hayfever	2.59%	7.63%
Migraine	1.76%	5.97%
Nearsightedness	7.53%	16.57%
Physical activity	3.95%	15.13%
Self-rated health	5.48%	9.34%
<i>Personality and well-being</i>		
Adventurousness	3.51%	8.14%
Agreeableness	0.67%	8.58%
Cognitive empathy	0.77%	10.33%
Conscientiousness	0.87%	9.81%
Delay discounting	0.21%	7.48%
Extraversion	3.88%	19.78%
Left out of social activity	1.90%	5.79%
Life satisfaction - family	1.04%	7.17%
Life satisfaction - finance	0.99%	7.16%
Life satisfaction - friends	1.14%	7.61%
Life satisfaction - work	0.58%	7.55%
Loneliness	0.86%	3.99%
Morning person	7.76%	15.86%
Narcissism	1.23%	4.69%
Neuroticism	5.67%	12.61%
Openness	1.44%	11.17%
Recharge by socializing	0.73%	3.43%
Religious attendance	1.28%	5.17%
Religious belief	0.64%	7.01%
Risk tolerance	2.45%	5.13%
Subjective well-being	3.01%	7.68%

A.2 Auxiliary results and robustness checks

In this section we provide some details on the auxiliary results and robustness checks briefly discussed in the main text.

A.2.1 Summary statistics

Table A.3 displays summary statistics for the four samples included in this study.²

Table A.3: Summary Statistics

	MTFS	Add Health	WLS	STR
Self-Reported Turnout	0.742 (0.323)	0.436 (0.386)		
Presidential Vote	0.915 (0.194)		0.862 (0.300)	
Midterm Vote	0.715 (0.335)		0.810 (0.344)	
National Vote				0.934 (0.203)
European Parliament Vote				0.625 (0.430)
Participation index		0.029 (0.122)		0.090 (0.195)
Birth Year	1969.1 (15.95)	1979.0 (1.771)	1939.5 (4.310)	1970.9 (23.66)
Male	0.470 (0.499)	0.389 (0.488)	0.480 (0.500)	0.457 (0.498)
<i>N</i>	2,333–7,525	4,791–5,652	8,534–8,937	9,598–43,669

Notes: Means and standard deviations (in parentheses) for some key variables. Presidential, midterm, national, and European Parliament election turnout are measured as average turnout across all the elections for which we have information for the individuals.

A.2.2 Full regression results

In the main text we present the results using graphs. The corresponding regression tables are presented below. That is, Figure 1 in the main text is based on the estimates displayed in Table A.4 whereas the estimates displayed in Figure 2 are found in Table A.5.

A.2.3 Baseline results in separate samples

In the main text we present results based on a pooled sample. To check whether the estimates are unduly driven by any of the subsamples we have re-estimated the baseline models from Table A.4 in each of STR, WLS, MTFS and Add Health samples. The results are reported in Tables A.6 through A.9. For

²We do not include summary statistics for the PGIs. As discussed above, various genotyping arrays, which tag different but overlapping sets of SNPs, have been used to genotype the individuals in the different cohorts. Imputation leads to better, but not perfect, overlap between the cohorts. Consequently, slightly different sets of SNPs and weights are used to construct the PGIs in each sample, implying that the average values and measures of dispersion for the unstandardized PGIs are not comparable across the cohorts. In all analyses, the PGIs are standardized within each cohort to have a mean of zero and a standard deviation of one.

Table A.4: Baseline results

	1st order	2nd order	Self-reported	Participation
EA	0.016*** [0.001]	0.067*** [0.002]	0.050*** [0.004]	0.016*** [0.002]
ΔR^2	0.514	2.588	1.578	0.760
CP	0.010*** [0.001]	0.042*** [0.002]	0.023*** [0.004]	0.009*** [0.001]
ΔR^2	0.195	1.019	0.343	0.265
EX	-0.001 [0.001]	0.003 [0.002]	0.012*** [0.004]	0.005*** [0.002]
ΔR^2	0.001	0.006	0.092	0.087
NE	-0.004*** [0.001]	-0.022*** [0.002]	-0.020*** [0.004]	-0.000 [0.001]
ΔR^2	0.025	0.274	0.263	0.000
RI	-0.002** [0.001]	0.006*** [0.002]	0.011** [0.004]	0.009*** [0.001]
ΔR^2	0.011	0.023	0.075	0.244
AD	-0.001 [0.001]	0.007*** [0.002]	0.012*** [0.004]	0.006*** [0.001]
ΔR^2	0.003	0.027	0.095	0.103
MO	0.002** [0.001]	-0.001 [0.002]	0.002 [0.004]	-0.003 [0.002]
ΔR^2	0.009	0.001	0.004	0.028
SRH	0.013*** [0.001]	0.038*** [0.002]	0.033*** [0.004]	0.001 [0.002]
ΔR^2	0.332	0.853	0.695	0.004
DEP	-0.009*** [0.001]	-0.024*** [0.002]	-0.022*** [0.004]	0.002 [0.002]
ΔR^2	0.148	0.339	0.315	0.008
SWB	0.008*** [0.001]	0.024*** [0.002]	0.020*** [0.004]	-0.001 [0.001]
ΔR^2	0.119	0.348	0.249	0.003
ACT	0.005*** [0.001]	0.019*** [0.002]	0.017*** [0.004]	0.003 [0.001]
ΔR^2	0.057	0.206	0.193	0.022
$\bar{Y}$	0.920	0.664	0.525	0.070
N	54,031	54,351	7,966	14,389

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

Table A.5: Within-family results

	1st order		2nd order		Self-reported		Participation	
	BF	WF	BF	WF	BF	WF	BF	WF
CP	0.011*** [0.002]	0.012*** [0.003]	0.041*** [0.003]	0.023*** [0.005]	0.017* [0.009]	0.029 [0.014]	0.007** [0.003]	0.008 [0.005]
EX	-0.002 [0.002]	-0.001 [0.003]	-0.001 [0.003]	0.005 [0.005]	0.017 [0.009]	0.037** [0.014]	0.010*** [0.003]	0.004 [0.006]
NE	-0.005** [0.002]	-0.008** [0.003]	-0.024*** [0.003]	-0.012** [0.005]	-0.016 [0.009]	-0.009 [0.014]	-0.003 [0.003]	-0.005 [0.005]
RI	-0.002 [0.002]	-0.002 [0.003]	0.014*** [0.003]	0.011** [0.005]	0.020* [0.010]	0.008 [0.015]	0.013*** [0.003]	0.006 [0.005]
AD	-0.002 [0.002]	-0.005 [0.003]	0.006** [0.003]	-0.000 [0.005]	0.020 [0.010]	0.014 [0.016]	0.005 [0.003]	0.001 [0.005]
MO	0.003 [0.002]	-0.001 [0.003]	-0.000 [0.003]	-0.011** [0.005]	-0.000 [0.010]	0.009 [0.015]	-0.001 [0.003]	-0.015 [0.006]
SRH	0.016*** [0.002]	0.015*** [0.003]	0.042*** [0.003]	0.019*** [0.005]	0.040*** [0.010]	0.043** [0.015]	0.002 [0.003]	0.001 [0.006]
DEP	-0.010*** [0.002]	-0.011*** [0.003]	-0.024*** [0.003]	-0.012** [0.005]	-0.017 [0.010]	-0.024 [0.015]	-0.002 [0.003]	0.002 [0.006]
SWB	0.009*** [0.002]	0.009*** [0.003]	0.031*** [0.003]	0.025*** [0.005]	0.028** [0.010]	0.020 [0.015]	0.001 [0.003]	-0.005 [0.005]
ACT	0.007*** [0.002]	0.008** [0.003]	0.020*** [0.003]	0.010* [0.005]	0.021* [0.009]	0.031 [0.016]	0.004 [0.003]	0.001 [0.006]
$\bar{Y}$	0.919	0.919	0.667	0.667	0.560	0.560	0.080	0.080
N	20,488	20,488	20,402	20,402	1,660	1,660	4,206	4,206

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. The within-family models (WF) also include fixed effects for twin pairs. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

convenience we also display the estimates from Table A.4 in the first column of each table. Finally, the last column in each table reports whether or not (using F-tests) the coefficient estimates vary significantly across the subsamples.³

The estimated PGI effects on first-order voting, self-reported voting and political participation do not seem to vary systematically across the different samples. There are some exceptions to this rule – for instance, the positive effects of the PGIs for risk attitudes and adventurousness on the participation index are driven by the STR sample. Moreover, the associations between voting in first-order elections and the PGIs for cognitive performance (strongest in STR), risk attitudes (strongest in MTFS) and self-rated health (strongest in WLS) differ significantly across the three samples. However, the overall pattern is one of consistency. Looking instead at Table A.7, the results for second-order voting stick out and the pattern is rather clear: The PGI effects are, in most cases weaker in the WLS sample compared to the STR and MTFS samples. The most obvious difference between these samples is the age composition of the study participants. The individuals in the WLS sample are significantly older than the individuals in the other two samples (see Table 1 in the main text). We speculate that experience and habit are more important for the decision to vote than genetic predispositions among older citizens. Moreover, this may be especially so in second-order elections, which tend to be viewed as less important by voters, parties, and the media and thus present a more information-poor electoral environment for citizens to navigate.

A.2.4 Baseline results for separate participation items

In Table A.10 we present the baseline estimates for models using as outcomes the three separate items included in the political participation index – whether the respondents contacted a government official, contributed money to a political cause, and attended a political rally or march during the last 12 months (Add Health)/5 years (STR).⁴ For convenience we also display the estimates for the index from Table A.4 in the first column of the table. The last column reports whether or not (using Wald tests) the coefficient estimates vary significantly across the three outcomes.⁵

The overall pattern of estimates across the three different participation measures is one of consistency. In all five cases in which the PGI effects on the participation index are statistically significant, the corresponding effects on the constituent parts have the same sign and are, with few exceptions, also significant. Conversely, in all cases but two (the effects of the PGIs for morningness and activity on attending a political rally) in which the PGI effects on the participation index are non-significant, the corresponding effects on the constituent parts are also non-significant. Still, it is interesting to note that in two cases – the PGIs for risk attitudes and adventurousness – the effect seems to be driven by the association with the propensity to contact government officials and to a lesser degree by contributing

³To test for significant variation in coefficient estimates we have included interaction terms between each PGI and indicator variables for the subsamples in pooled models. We then test (using an F-test) if all interaction coefficients are equal to 0.

⁴The separate analyses for each participation item were not pre-registered.

⁵That is, we combine the parameter estimates and associated (co)variance matrices in each of the three models into one parameter vector and simultaneous (co)variance matrix and then test for cross-model equality in the PGI coefficients.

Table A.6: Baseline results - first-order voting

	All	STR	WLS	MTFS	F
EA	0.016*** [0.001]	0.017*** [0.001]	0.013*** [0.003]	0.012*** [0.003]	3.24**
ΔR^2	0.514	0.730	0.178	0.368	
CP	0.010*** [0.001]	0.011*** [0.001]	0.007* [0.003]	0.005 [0.003]	4.05**
ΔR^2	0.195	0.314	0.048	0.054	
EX	-0.001 [0.001]	-0.001 [0.001]	0.003 [0.003]	-0.002 [0.003]	0.72
ΔR^2	0.001	0.004	0.009	0.014	
NE	-0.004*** [0.001]	-0.004*** [0.001]	-0.004 [0.003]	-0.001 [0.003]	0.40
ΔR^2	0.027	0.032	0.021	0.005	
RI	-0.002** [0.001]	-0.001 [0.001]	-0.006 [0.003]	-0.007** [0.002]	3.47***
ΔR^2	0.011	0.002	0.034	0.134	
AD	-0.001 [0.001]	-0.001 [0.001]	-0.003 [0.003]	-0.001 [0.003]	0.42
ΔR^2	0.003	0.001	0.012	0.002	
MO	0.002** [0.001]	0.002** [0.001]	0.003 [0.003]	0.000 [0.002]	0.30
ΔR^2	0.009	0.014	0.010	0.000	
SRH	0.013*** [0.001]	0.013*** [0.001]	0.016*** [0.003]	0.006* [0.003]	4.01***
ΔR^2	0.332	0.420	0.283	0.105	
DEP	-0.009*** [0.001]	-0.008*** [0.001]	-0.013*** [0.003]	-0.005 [0.003]	1.50
ΔR^2	0.148	0.162	0.177	0.070	
SWB	0.008*** [0.001]	0.008*** [0.001]	0.007* [0.003]	0.004 [0.003]	1.42
ΔR^2	0.119	0.171	0.051	0.041	
ACT	0.005*** [0.001]	0.005*** [0.001]	0.009** [0.003]	0.002 [0.003]	1.30
ΔR^2	0.057	0.062	0.092	0.009	
$\bar{Y}$	0.920	0.934	0.862	0.915	
N	54,031	39,166	8,534	6,331	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

Table A.7: Baseline results - second-order voting

	All	STR	WLS	MTFS	F
EA	0.067*** [0.002]	0.080*** [0.002]	0.028*** [0.004]	0.039*** [0.004]	89.05***
ΔR^2	2.588	3.415	0.628	1.293	
CP	0.042*** [0.002]	0.051*** [0.002]	0.014*** [0.004]	0.026*** [0.004]	40.60***
ΔR^2	1.019	1.378	0.171	0.570	
EX	0.003 [0.002]	0.003 [0.002]	0.005 [0.004]	0.003 [0.004]	0.03
ΔR^2	0.006	0.005	0.018	0.006	
NE	-0.022*** [0.002]	-0.025*** [0.002]	-0.009** [0.004]	-0.018*** [0.004]	6.18***
ΔR^2	0.274	0.332	0.066	0.295	
RI	0.006*** [0.002]	0.010*** [0.002]	-0.002 [0.004]	-0.005 [0.004]	6.43***
ΔR^2	0.023	0.051	0.005	0.021	
AD	0.007*** [0.002]	0.008*** [0.002]	0.001 [0.004]	0.006 [0.004]	1.55
ΔR^2	0.027	0.036	0.000	0.033	
MO	-0.001 [0.002]	-0.003 [0.002]	0.004 [0.004]	0.007 [0.004]	2.66*
ΔR^2	0.001	0.006	0.014	0.038	
SRH	0.038*** [0.002]	0.043*** [0.002]	0.022*** [0.004]	0.031*** [0.004]	13.25***
ΔR^2	0.853	1.005	0.397	0.805	
DEP	-0.024*** [0.002]	-0.026*** [0.002]	-0.016*** [0.004]	-0.023*** [0.004]	2.46*
ΔR^2	0.339	0.361	0.215	0.451	
SWB	0.024*** [0.002]	0.029*** [0.002]	0.010** [0.004]	0.019*** [0.004]	9.73***
ΔR^2	0.348	0.437	0.077	0.316	
ACT	0.019*** [0.002]	0.021*** [0.002]	0.015*** [0.004]	0.008 [0.004]	4.06**
ΔR^2	0.206	0.245	0.178	0.050	
$\bar{Y}$	0.664	0.625	0.810	0.715	
N	54,351	39,459	8,534	6,358	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

Table A.8: Baseline results - self-reported voting

	All	MTFS	AddHealth	F
EA	0.050*** [0.004]	0.039*** [0.008]	0.056*** [0.005]	3.87**
ΔR^2	1.578	1.436	2.018	
CP	0.023*** [0.004]	0.022*** [0.008]	0.025*** [0.005]	0.17
ΔR^2	0.343	0.466	0.403	
EX	0.012*** [0.004]	0.022*** [0.007]	0.008 [0.005]	2.62
ΔR^2	0.092	0.459	0.042	
NE	-0.020*** [0.004]	-0.010 [0.007]	-0.024*** [0.005]	2.58
ΔR^2	0.263	0.092	0.396	
RI	0.011** [0.004]	0.009 [0.007]	0.012** [0.005]	0.11
ΔR^2	0.075	0.069	0.089	
AD	0.012*** [0.004]	0.011 [0.007]	0.012** [0.005]	0.00
ΔR^2	0.095	0.119	0.093	
MO	0.002 [0.004]	-0.003 [0.008]	0.005 [0.005]	0.76
ΔR^2	0.004	0.010	0.014	
SRH	0.033*** [0.004]	0.034*** [0.008]	0.033*** [0.005]	0.00
ΔR^2	0.695	1.091	0.720	
DEP	-0.022*** [0.004]	-0.021** [0.008]	-0.023*** [0.005]	0.03
ΔR^2	0.315	0.423	0.347	
SWB	0.020*** [0.004]	0.029*** [0.008]	0.017*** [0.005]	1.67
ΔR^2	0.249	0.790	0.184	
ACT	0.017*** [0.004]	0.020** [0.007]	0.016*** [0.005]	0.26
ΔR^2	0.193	0.400	0.169	
$\bar{Y}$	0.642	0.742	0.436	
N	3,407	2,333	5,633	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

Table A.9: Baseline results - participation index

	All	STR	AddHealth	F
EA	0.016*** [0.002]	0.018*** [0.002]	0.011*** [0.002]	5.15**
ΔR^2	0.760	0.796	0.751	
CP	0.009*** [0.001]	0.010*** [0.002]	0.006*** [0.002]	2.74*
ΔR^2	0.265	0.282	0.268	
EX	0.005*** [0.002]	0.006*** [0.002]	0.003 [0.002]	1.27
ΔR^2	0.087	0.099	0.076	
NE	-0.000 [0.001]	0.000 [0.002]	-0.001 [0.002]	0.02
ΔR^2	0.000	0.000	0.005	
RI	0.009*** [0.001]	0.012*** [0.002]	0.003 [0.002]	10.35***
ΔR^2	0.244	0.348	0.074	
AD	0.006*** [0.001]	0.008*** [0.002]	0.001 [0.002]	6.88***
ΔR^2	0.103	0.165	0.003	
MO	-0.003 [0.002]	-0.004 [0.002]	-0.002 [0.002]	0.030
ΔR^2	0.028	0.035	0.018	
SRH	0.001 [0.002]	0.001 [0.002]	0.000 [0.002]	0.02
ΔR^2	0.004	0.003	0.004	
DEP	0.002 [0.002]	0.002 [0.002]	0.001 [0.002]	0.12
ΔR^2	0.008	0.011	0.004	
SWB	-0.001 [0.001]	-0.000 [0.002]	-0.003 [0.002]	1.16
ΔR^2	0.003	0.000	0.053	
ACT	0.003 [0.001]	0.004 [0.002]	0.000 [0.002]	1.11
ΔR^2	0.022	0.034	0.000	
$\bar{Y}$	0.070	0.090	0.029	
N	14,389	9,598	4,791	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

money or attending rallies.

A.2.5 Mediation Analysis

In the main text we show that the ten psychological and health trait PGIs are significantly related to voter turnout and political participation in both between- and within-family models. A natural next step is to test whether and, if so, the degree to which the different PGIs influence political participation via their respective target traits. For instance, cognitive performance is likely to partly mediate the effect of the cognitive performance PGI on political participation. The same expectation, of course, also holds for the other PGI-target trait combinations. It is important to keep in mind that there are multiple reasons for not expecting the target traits to *fully* mediate the PGI-participation links. Above all, as discussed in the main text, pleiotropy - the fact that one gene can influence multiple traits - makes it highly likely that any PGI effect will be mediated via several different pathways in addition to the target trait in question. For example, Selzam et al. (2019) recently showed that PGIs for cognitive performance, self-rated health, and neuroticism all predict educational attainment which may have downstream effects on a multitude of human traits, among others political participation. Second, due to measurement error and imprecise estimation of beta weights, the PGIs will not perfectly capture the true genetic propensity for the target traits. Third, differences in how the target traits are measured between the discovery sample used to estimate the weights for the PGIs and the replication samples (Add Health, WLS, MTF5 and STR in our case) may further reduce the degree to which a PGI effect is mediated via its target trait.

With these caveats in mind, Table A.11 displays results for the effect of each PGI on its corresponding target trait. The analysis is restricted to target traits for which we have information from at least two samples.⁶ The target traits are standardized within each sample (mean=0, s.d.=1) and the target traits are measured such that we should expect positive relationships with the corresponding PGIs. Column 6 reproduces the expected explanatory power of the PGIs displayed in Table A.11.

From the results, we can draw several conclusions. Firstly, and as expected, the effects of the PGIs on their respective target traits are generally quite strong. Secondly, the average explanatory power of the PGIs across the four samples (column 5) follows the calculated predictive ability for the PGIs in the repository (see column 6) fairly well.⁷ For example, the explanatory power of the PGIs for educational attainment and cognitive performance is significantly higher than that of the PGI for risk attitudes. Thirdly, it is also evident that the actual explanatory power of the PGIs (columns 1-5) varies in how closely it matches the expected explanatory power (column 6). There are several reasons for this discrepancy, such as demographic differences (age, gender, etc.) and differences in institutional context (samples from different countries) between the GWAS sample used to construct the PGI and the sample

⁶Details about how the target traits are measured in the four samples can be found in the pre-analysis plans: <https://www.dropbox.com/sc1/fi/7dv8en9uh5hj6ib2yv3/PAP-Cognitive-and-personality-traits.pdf?rlkey=8aqhwsf8d4n4xrkgqanwdh0s4&dl=0> and <https://www.dropbox.com/sc1/fi/9hs7ao3rkr6njjr6xmaj/PAP-Health-traits.pdf?rlkey=cmgf3a0l3pep6bqysqj8gm7x5&dl=0>.

⁷These calculated predictive abilities (taken from Becker et al. (2021)) are a function of the degree of heritability in the outcomes in question, as well as the size of the samples used in the GWAS analyses that underpin the PGIs.

Table A.10: Baseline results - separate participation items

	Index	Contact	Contribute	Rally	χ^2
EA	0.016*** [0.002]	0.022*** [0.003]	0.010*** [0.002]	0.015*** [0.002]	18.01***
ΔR^2	0.760	0.476	0.226	0.430	
CP	0.009*** [0.001]	0.012*** [0.003]	0.007*** [0.002]	0.008*** [0.002]	2.54
ΔR^2	0.265	0.134	0.123	0.145	
EX	0.005*** [0.002]	0.007** [0.003]	0.005** [0.002]	0.004 [0.002]	1.69
ΔR^2	0.087	0.050	0.060	0.025	
NE	-0.000 [0.001]	-0.002 [0.003]	0.002 [0.002]	-0.001 [0.002]	3.28
ΔR^2	0.000	0.002	0.010	0.003	
RI	0.009*** [0.001]	0.016*** [0.003]	0.004** [0.002]	0.006*** [0.002]	18.15***
ΔR^2	0.244	0.253	0.047	0.069	
AD	0.006*** [0.001]	0.012*** [0.003]	0.002 [0.002]	0.003 [0.002]	12.79***
ΔR^2	0.103	0.135	0.015	0.017	
MO	-0.003 [0.002]	-0.002 [0.003]	-0.003 [0.002]	-0.004* [0.002]	0.67
ΔR^2	0.028	0.003	0.025	0.032	
SRH	0.001 [0.002]	0.004 [0.003]	-0.001 [0.002]	-0.001 [0.002]	3.49
ΔR^2	0.004	0.019	0.001	0.000	
DEP	0.002 [0.002]	0.000 [0.003]	0.003 [0.002]	0.001 [0.002]	1.35
ΔR^2	0.008	0.000	0.023	0.004	
SWB	-0.001 [0.001]	-0.000 [0.003]	-0.003 [0.002]	0.000 [0.002]	2.30
ΔR^2	0.003	0.000	0.018	0.000	
ACT	0.003 [0.001]	0.003 [0.003]	-0.000 [0.002]	0.005** [0.002]	5.72*
ΔR^2	0.022	0.010	0.000	0.049	
$\bar{Y}$	0.070	0.113	0.044	0.052	
N	14,389	14,389	14,387	14,388	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. ***/**/*, indicates significance at the 1/5/10% level.

in which the PGI is used. However, the most important reason for a PGI’s actual explanatory power being lower than expected is differences in how outcomes are measured. In some cases, there is a high conceptual and operational alignment in how an outcome was defined in the GWAS sample and the samples we use in this study. A clear example here is educational attainment, which was measured as years of education in both the original GWAS study that underpins the educational attainment PGI and in all four of our samples. The same applies to cognitive performance, where the alignment between the GWAS study and our four samples is strong. In both of these cases – the PGIs for educational attainment and cognitive performance – the actual explanatory power comes quite close to the expected explanatory power. This can be compared to the PGIs for physical activity and neuroticism (especially for MTF5 and STR samples), where the actual explanatory power is significantly lower than expected. Unsurprisingly, the alignment between how the target outcomes for these two PGIs were measured in the GWAS studies and our four samples is weaker. In general, this shows something quite obvious: measures that are conceptually and operationally clearer perform better. Ultimately, this highlights the need for better harmonization in terms of both concepts and measures.

Table A.12 presents coefficient estimates from a simple mediation analysis. We restrict the mediation analysis such that we only include results for PGIs that are significantly related (at the 0.05 level) to an outcome when restricting the sample to individuals for which we have information on the corresponding target trait. In the end, this leaves us with five PGI–target trait combinations for the turnout in first-order elections, seven PGI–target trait combinations for turnout in second-order elections, eight PGI–target trait combinations for self-reported voting, and three PGI–target trait combinations for the political participation index.⁸

We use a very simple two-step procedure to test for mediation (Baron and Kenny, 1986). First, we re-estimate the effect of a specific PGI (e.g., the PGI for cognitive performance) on a specific outcome for individuals with complete data on the target trait. Second, we include the target trait as a regressor in the model. We will measure mediation as the percentage decrease in the PGI effect size between the two models.

The results in Table A.12 show that the effects of the PGIs on the four outcome measures shrink somewhat when controlling for the respective target trait. The decrease in PGI effect magnitude is modest. On average, the target traits account for around 30% of the corresponding PGI effect. However, there is a great deal of variation in the degree to which the the PGI effects decrease when controlling for the corresponding target trait. The pattern here follows the results presented in Table A.11 above. In cases where a PGI’s actual explanatory power regarding the target variable aligns with its expected explanatory power, the target variable will also capture much of the PGI effect on political outcomes.

⁸The pre-analysis plan stipulated that only within-family models should be used for the mediation analysis. However, given the decrease in sample size and the loss of variation in the within-family models this would lead to significantly fewer PGI–target trait combinations that could be examined in the mediation analysis (two PGI–target trait combinations for the turnout in first-order elections, four PGI–target trait combinations for turnout in second-order elections, four PGI–target trait combinations for self-reported voting, and no PGI–target trait combinations for the political participation index). In light of this, we decided to deviate from the original pre-analysis plan and instead present between-family results.

Clear examples here are the effects of the PGIs for education and cognitive ability. Conversely, when the alignment between how the target variable (e.g., physical activity and neuroticism) is measured in the GWAS study and our four samples is poorer, the target variable can only explain a smaller part of the relationship between the PGI and the political outcomes.

While this analysis suggests that the effects of the PGIs are partly mediated by the traits they are constructed to predict, it also suggests that there are other PGI mechanisms influencing political participation that are unrelated to the target traits as there are still significant and sizeable effects of the PGIs on the four outcomes.

A.2.6 Confounding

We next check the degree to which the PGIs confound the relationship each target trait and the four participation outcomes. Our approach here is very simple. First, we estimate the effect of a specific target trait on a specific outcome (e.g., the effect of cognitive performance on second order turnout). In the second step we control for all eleven PGIs used in this study (the ten health and psychological trait PGIs and the educational attainment PGI). We will measure confounding as the decrease in target trait effect size between the two models. We restrict these models such that we only include results for target traits that are significantly related (at the 0.05 level) to an outcome when not controlling for the PGIs. The results are presented in Table A.13. The odd-numbered columns show results of the trait effects when not controlling for the PGIs. The estimates displayed in the even-numbered columns are adjusted for the PGIs.

The estimates show that controlling for the PGIs reduces the effect of the health and psychological traits by, on average, about 11% across all models. While these results suggest only a modest amount of confounding, it is important to remember that the PGIs do not capture all of the genetic propensity to exhibit the health and psychological traits in focus. When more precise polygenic indices become available in the future, it is likely that the amount of effect size reduction from controlling for relevant PGIs will significantly increase (Becker et al., 2021).

A.2.7 Sibling models

In Table A.14 we follow the approach taken by Cawley et al. (2020) and report results from models restricted to sibling pairs including both subjects' (ego) and siblings' (alter) PGIs as predictors. We also restrict the analysis such that we only include results for PGIs that are significantly related (at the 0.05 level) to an outcome in between-family models in the sibling sample (see the estimates in the odd-numbered columns in Table A.5). This approach should not be interpreted as capturing genetic nurture exclusively, but will also pick up e.g. population stratification that remains after control for the genomic principal components. The sibling (alter) PGI controlling for the ego PGI will, however, pick up a mixture of *non-genetic* effects.

Looking first at the results for first-order and self-reported turnout, the sibling PGIs do not pick

Table A.11: Results - PGI effects on target traits

	MTFS	Add Health	WLS	STR	All	exp ΔR^2
EA PGI	0.273*** [0.015]	0.343*** [0.012]	0.223*** [0.011]	0.256*** [0.006]	0.265*** [0.004]	
ΔR^2	7.17	11.56	4.85	5.85	6.52	7.27
N	5,250	5,651	8,222	29,276	48,849	
CP PGI	0.249*** [0.017]	0.223*** [0.015]	0.257*** [0.010]	0.296*** [0.010]	0.267*** [0.006]	
ΔR^2	5.97	4.89	6.48	8.66	7.05	10.73
N	4,917	4,667	8,410	10,966	28,960	
EX PGI	0.072*** [0.016]	0.119*** [0.013]	0.124*** [0.012]	0.938*** [0.011]	0.104*** [0.006]	
ΔR^2	0.51	1.41	1.49	0.86	1.07	3.88
N	5,664	5,648	7,774	9,484	28,570	
NE PGI	0.078*** [0.014]	0.143*** [0.013]	0.152*** [0.011]	0.058*** [0.008]	0.096*** [0.005]	
ΔR^2	0.59	2.03	2.25	0.34	0.92	5.67
N	5,664	5,647	7,762	16,025	35,098	
RI PGI	0.064*** [0.013]	0.082*** [0.013]	0.061*** [0.012]	0.132*** [0.010]	0.090*** [0.006]	
ΔR^2	0.38	0.68	0.36	1.72	0.80	2.45
N	5,748	5,645	7,040	9,431	27,864	
SRH PGI		0.197*** [0.013]	0.155*** [0.011]	0.131*** [0.007]	0.148*** [0.005]	
ΔR^2		3.77	2.34	1.72	2.17	5.48
N		5,651	8,651	22,196	36,498	
DEP PGI		0.136*** [0.013]	0.093*** [0.012]	0.126*** [0.010]	0.118*** [0.006]	
ΔR^2		1.83	0.84	1.58	1.39	3.08
N		5,651	7,757	12,645	26,053	
SWB PGI	0.100*** [0.015]	0.101*** [0.013]	0.100*** [0.013]	0.131*** [0.011]	0.111*** [0.006]	
ΔR^2	0.96	1.02	0.96	1.72	1.21	3.01
N	5,742	5,650	6,446	9,452	27,290	
ACT PGI		0.021 [0.013]	0.061*** [0.011]	0.100*** [0.011]	0.065*** [0.007]	
ΔR^2		0.04	0.36	0.98	0.42	3.95
N		5,648	8,936	8,991	23,575	

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data. Standard errors, shown in parentheses, allow for clustering at the family level. ***/**/*, indicates significance at the 1/5/10% level.

Table A.12: Results - mediation

	1st order		2nd order		Self-reported		Participation	
EA PGI	0.013***	0.009***	0.059***	0.038***	0.050***	0.019*	0.015***	0.007***
	[0.001]	[0.001]	[0.002]	[0.002]	[0.004]	[0.004]	[0.002]	[0.002]
EA Trait		0.018***		0.080***		0.097***		0.027***
		[0.001]		[0.002]		[0.004]		[0.002]
% mediated	34.7%		34.3%		62.8%		52.4%	
N	42,163		41,830		7,966		14,388	
CP PGI	0.007***	0.002	0.031***	0.014***	0.022***	0.008*	0.009***	0.004**
	[0.001]	[0.002]	[0.002]	[0.003]	[0.005]	[0.005]	[0.002]	[0.002]
CP Trait		0.016***		0.066***		0.060***		0.020***
		[0.002]		[0.003]		[0.005]		[0.002]
% mediated	66.0%		56.9%		64.2%		55.3%	
N	23,328		23,279		6,626		8,884	
EX PGI			0.006**	0.003	0.012***	0.006	0.005***	0.003**
			[0.003]	[0.003]	[0.004]	[0.004]	[0.002]	[0.002]
EX Trait				0.028***		0.050***		0.021***
				[0.002]		[0.004]		[0.002]
% mediated			49.6%		47.7%		42.3%	
N			21,913		7,749		14,273	
NE PGI	-0.004**	-0.004***	-0.017***	-0.016***	-0.020***	-0.016***		
	[0.001]	[0.001]	[0.002]	[0.002]	[0.004]	[0.004]		
NE Trait		-0.005***		-0.005**		-0.037***		
		[0.001]		[0.002]		[0.004]		
% mediated	10.2%		2.3%		22.5%			
N	28,404		28,087		7,748			
RI PGI					0.011***	0.011***	0.009***	0.007***
					[0.004]	[0.004]	[0.001]	[0.001]
RI Trait						-0.009*		0.016***
						[0.005]		[0.002]
% mediated					-5.8%		20.5%	
N					7,777		14,218	
SRH PGI	0.011***	0.009***	0.035***	0.031***	0.033***	0.024***		
	[0.001]	[0.001]	[0.002]	[0.002]	[0.005]	[0.005]		
SRH Trait		0.016***		0.024***		0.047***		
		[0.002]		[0.003]		[0.005]		
% mediated	19.1%		9.4%		28.5%			
N	30,443		30,439		5,633			
DEP PGI	-0.007***	-0.005***	-0.019***	-0.016***	-0.023***	-0.016***		
	[0.002]	[0.002]	[0.003]	[0.003]	[0.005]	[0.005]		
DEP Trait		-0.013***		-0.022***		-0.050***		
		[0.002]		[0.003]		[0.005]		
% mediated	21.9%		13.4%		30.2%			
N	20,058		20,061		5,633			
SWB PGI	0.006***	0.005***	0.018***	0.016***	0.020***	0.015***		
	[0.002]	[0.002]	[0.003]	[0.003]	[0.004]	[0.004]		
SWB Trait		0.008***		0.020***		0.049***		
		[0.002]		[0.003]		[0.004]		
% mediated	13.8%		12.8%		24.1%			
N	20,662		20,673		7,773			
ACT PGI			0.015***	0.013***	0.016***	0.016***		
			[0.003]	[0.003]	[0.005]	[0.005]		
ACT Trait				0.018***		0.003		
				[0.003]		[0.005]		
% mediated			9.8%		0.5%			
N			17,509		5,633			

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. ***/**/*, indicates significance at the 1/5/10% level.

Table A.13: Results - confounding

	1st order		2nd order		Self-reported		Participation	
	1	2	3	4	5	6	7	8
EA Trait	0.021*** [0.001]	0.018*** [0.001]	0.090*** [0.002]	0.079*** [0.003]	0.103*** [0.004]	0.097*** [0.005]	0.029*** [0.002]	0.027*** [0.002]
% confounded	12.0%		12.6%		6.6%		6.8%	
<i>N</i>	42,163		41,830		7,966		14,388	
CP Trait	0.017*** [0.002]	0.015*** [0.002]	0.069*** [0.002]	0.061*** [0.003]	0.062*** [0.005]	0.053*** [0.005]	0.021*** [0.002]	0.018*** [0.002]
% confounded	10.8%		11.9%		14.7%		13.2%	
<i>N</i>	23,328		23,279		6,626		8,884	
EX Trait	0.008*** [0.002]	0.007*** [0.002]	0.028*** [0.002]	0.026*** [0.002]	0.050*** [0.004]	0.049*** [0.004]	0.022*** [0.002]	0.020*** [0.002]
% confounded	2.9%		6.9%		3.2%		5.5%	
<i>N</i>	21,903		21,913		7,749		14,273	
NE Trait	-0.005*** [0.001]	-0.005*** [0.001]	-0.006*** [0.002]	-0.004* [0.002]	-0.039*** [0.004]	-0.034*** [0.004]	0.006*** [0.002]	0.006*** [0.002]
% confounded	9.9%		32.1%		14.1%		4.7%	
<i>N</i>	28,404		28,087		7,748		13,880	
RI Trait			0.015*** [0.003]	0.014*** [0.003]			0.017*** [0.001]	0.016*** [0.001]
% confounded			10.8%				7.5%	
<i>N</i>			21,247				14,218	
SRH Trait	0.017*** [0.001]	0.016*** [0.001]	0.028*** [0.002]	0.022*** [0.002]	0.052*** [0.005]	0.044*** [0.005]		
% confounded	9.2%		20.9%		14.7%			
<i>N</i>	30,443		30,439		5,633			
DEP Trait	-0.014*** [0.002]	-0.013*** [0.002]	-0.024*** [0.003]	-0.020*** [0.003]	-0.052*** [0.005]	-0.047*** [0.005]		
% confounded	7.2%		15.7%		9.9%			
<i>N</i>	20,058		20,061		5,633			
SWB Trait	0.008*** [0.002]	0.008*** [0.002]	0.022*** [0.003]	0.020*** [0.003]	0.050*** [0.004]	0.047*** [0.004]		
% confounded	6.1%		5.9%		5.6%			
<i>N</i>	20,662		20,673		7,773			
ACT Trait	0.010*** [0.002]	0.008** [0.002]	0.019*** [0.003]	0.015** [0.003]				
% confounded	13.1%		21.3%					
<i>N</i>	17,504		17,509					

Note: All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. The second model for each outcome also includes controls for 11 PGIs. The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). ***/**/*, indicates significance at the 1/5/10% level.

Table A.14: Sibling models

	1st order voting	2nd order voting	Self-reported voting	Participation index
CP EGO	0.009*** [0.002]	0.031*** [0.003]		0.007** [0.003]
CP ALTER	0.000 [0.002]	0.013*** [0.003]		0.001 [0.003]
EX EGO				0.007** [0.003]
EX ALTER				0.004 [0.003]
NE EGO	-0.005*** [0.002]	-0.019*** [0.003]		
NE ALTER	0.000 [0.002]	-0.008*** [0.003]		
RI EGO		0.010*** [0.003]		0.012*** [0.003]
RI ALTER		0.002 [0.003]		0.005* [0.003]
AD EGO		0.006** [0.003]		
AD ALTER		0.004 [0.003]		
SRH EGO	0.013*** [0.002]	0.029*** [0.003]	0.035*** [0.011]	
SRH ALTER	0.002 [0.002]	0.016*** [0.003]	0.004 [0.009]	
DEP EGO	-0.008*** [0.002]	-0.019*** [0.003]		
DEP ALTER	-0.001 [0.002]	-0.007** [0.003]		
SWB EGO	0.007*** [0.002]	0.025*** [0.003]	0.011 [0.010]	
SWB ALTER	0.001 [0.002]	0.006** [0.003]	0.003 [0.009]	
ACT EGO	0.006*** [0.002]	0.015*** [0.003]		
SWB ALTER	-0.000 [0.002]	0.006** [0.003]		
$\bar{Y}$	0.920	0.682	0.662	0.081
N	26,567	26,764	2,948	4,585

Note: The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genomic data. ***/**/*, indicates significance at the 1/5/10% level.

up any clear effects. The estimates are inconsistently signed and never statistically significant. Turning instead to the results for second-order voter turnout the results are much clearer and suggest the presence of effects of some unknown bundle of environmental factors.

When one has access to both parental and sibling genotype information, these models will become a lot more interesting since they will allow population stratification to be held constant (via control for parental PGI). In this situation, the sibling PGI will therefore pick up strictly genetic nurture effects – i.e. a quantity with a much more specific, and concrete, interpretation than “a bundle of unknown environmental factors”. Fortunately, efforts are underway to produce such data. Large-scale genetically informed datasets with politically relevant outcomes that includes genotype information (or highly precisely imputed genotype information⁹) on not just both siblings, but also on the parents, are on the cusp of being available to the larger community of researchers.

This difference in results for first- and second-order voting is particularly interesting. Second-order elections are generally considered by voters, parties, and the media to be less important than first-order presidential or national elections. Thus, citizens typically have to navigate a more information-poor electoral environment, making it more challenging to participate. Our estimates suggest that such conditions will increase the relative importance of the social context - here proxied by the predispositions of one’s siblings - over individual resources in determining an individual’s decision to engage in the political sphere.

A.2.8 Gene-by-environment interaction

As our last applied example of relevant uses of PGIs, we present a simple case of a gene-by-environment interaction, the example being the interaction between a depression PGI and educational attainment (measured as years of education) for voter turnout. Previous research has documented consistent negative effects of depressive symptoms on political participation (Ojeda and Pacheco, 2019; Landwehr and Ojeda, 2021; Gerber et al., 2011; Engelman et al., 2021), a picture that is confirmed by our main PGI results. Meanwhile, the so-called diathesis-stress model of depression claims that it is the combination of vulnerability (diathesis) with an environmental stressor that triggers actual depressive episodes (Monroe and Simons, 1991; Colodro-Conde et al., 2018). Educational attainment, in this context, thus acts as a possible proxy for a variety of stressors induced by different socioeconomic environments.

We use data from the STR on the latest available elections, and basic cross-sectional specifications with a simple multiplicative interaction term between a genetic factor (the depression PGI) and a non-genetic variable (years of education), controls for sex and birth year fixed effects (and their interaction), as well as the top ten principal components (and their interactions with the PGI and the moderator).

As evidenced by the significant and positive interaction terms in columns 1 and 2 in Table A.15, the negative effect of the depression PGI on turnout is moderated by educational attainment. The marginal

⁹To a large extent, large-scale trio data has been made possible by recent breakthroughs in methods for imputing average parental genotypes from two or more siblings (Young et al., 2022).

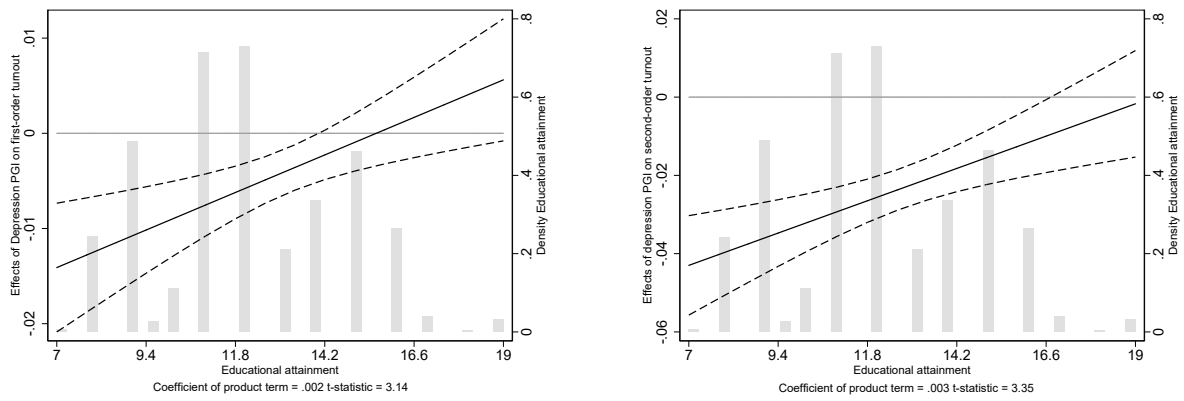
Table A.15: GxE interaction models

VARIABLES	(1) 1st Order	(2) 2nd Order	(3) 1st Order	(4) 1st Order	(5) 2nd Order	(6) 2nd Order
EA	0.008*** (0.001)	0.041*** (0.001)	0.008*** (0.001)	0.007*** (0.001)	0.042*** ^{uv} (0.001)	0.041*** (0.001)
Depression PGI	-0.026*** (0.007)	-0.067*** (0.013)				
Depression PGI $\times$ EA	0.002*** (0.001)	0.003*** (0.001)				
Neuroticism PGI			-0.017** (0.007)		-0.054*** (0.013)	
Neuroticism PGI $\times$ EA			0.001* (0.001)		0.003*** (0.001)	
SRH PGI				0.036*** (0.007)		0.081*** (0.013)
SRH PGI $\times$ EA				-0.002*** (0.001)		-0.004*** (0.001)
Constant	0.944** (0.013)	-0.491** (0.038)	0.948** (0.014)	0.953** (0.015)	-0.507** (0.038)	-0.540** (0.039)
Observations	29,461	29,067	29,461	29,461	29,067	29,067
Conotrols	YES	YES	YES	YES	YES	YES
R-squared	0.048	0.077	0.048	0.049	0.076	0.078

Note: All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, and the first ten principal components of the genomic data, as well as interactions between these and educational attainment and the PGI. ***/**/*, indicates significance at the 1/5/10% level.

effects plots (Fig. A.1) further reveal that the negative effect of the depression PGI is entirely driven by the lower end of the education distribution and disappears at the higher end. Thus, it may be that education (or variables associated with education) acts as a dampener on the negative turnout effects of having a genetic susceptibility to depression. As a robustness check, the same interaction is tested with PGIs for related traits (neuroticism and self-rated health) in columns 3–6.

Figure A.1: Marginal effects plots, depression PGI effect on 1st/2nd Order Voting



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