# A polygenic score for educational attainment partially predicts voter turnout

Christopher T. Dawes[a,1], Aysu Okbay[b], Sven Oskarsson[c], and Aldo Rustichini[d]

[a]Wilf Family Department of Politics, New York University, New York, NY 10012; [b]Department of Economics, School of Business and Economics, Vrije Universiteit Amsterdam, 1081HV Amsterdam, The Netherlands; [c]Department of Government, Uppsala Universitet, 751 20 Uppsala, Sweden; and [d]Department of Economics, University of Minnesota, Minneapolis, MN 55455-0462

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**Twin and adoption studies have shown that individual differences in political participation can be explained, in part, by genetic variation. However, these research designs cannot identify which genes are related to voting or the pathways through which they exert influence, and their conclusions rely on possibly restrictive assumptions. In this study, we use three different US samples and a Swedish sample to test whether genes that have been identified as associated with educational attainment, one of the strongest correlates of political participation, predict self-reported and validated voter turnout. We find that a polygenic score capturing individuals' genetic propensity to acquire education is significantly related to turnout. The strongest associations we observe are in second-order midterm elections in the United States and European Parliament elections in Sweden, 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 within-family analysis suggests that individuals' education-linked genes directly affect their voting behavior, but, for second-order elections, it also reveals evidence of genetic nurture. Finally, a mediation analysis suggests that educational attainment and cognitive ability combine to account for between 41% and 63% of the relationship between the genetic propensity to acquire education and voter turnout.**

education | voting | polygenic score | turnout | cognitive ability

Why do some people vote in elections whereas others abstain? This question has profound implications for democratic accountability and representation (1) and has, accordingly, generated a vast empirical literature (2). A consistent finding in this literature is that educational attainment is among the strongest correlates of voter turnout (2). The conventional explanation is that education causally influences political engagement, the logic being that education provides citizens skills (3) and cognitive resources (4) that lower the cost of participating in politics as well as foster a sense of civic duty and political efficacy (3, 5). The implication of this explanation is that exogenously increasing an individual's education attainment will increase their likelihood of voting. However, other scholars have argued that the correlation between education and turnout may be spurious: preadult characteristics such as cognitive ability, personality traits, and genetic factors as well as parental socialization cause both the acquisition of education and adult political participation (6, 7). Thus, more education may not necessarily translate into a higher likelihood of voting.

To better understand the relationship between education and voter turnout, we focus on the role of genes related to education and cognitive ability. A genetic basis of voter turnout has been established by studies of twins (8–10) and adoptees (11, 12), and a recent study has reported an association between education-linked genes and voter turnout in a Danish sample of unrelated individuals (13). Since estimated genetic effects based on unrelated individuals may be confounded by assortative mating, population stratification, and environmentally mediated parental genetic effects (14), we conduct a within-family analysis based on more than 10,000 sibling pairs in order to evaluate the direct effects of an individual's education-linked genes on their voting behavior. In addition, comparing estimates from between- and within-family models allows us to evaluate the degree to which parents' education-linked genes influence their offspring's political participation via the family environment they create for them (14), a possibility hinted at by past research showing parental education is strongly correlated with their offspring's voting behavior (15, 16). Finally, in an effort to trace the pathway through which genes influence political participation, we test whether genes confound the relationship between educational attainment and voter turnout (because they separately influence both education and turnout) as well as assess how much of the relationship between the education-linked genes and turnout is mediated by education, cognitive ability, and personality traits.

Using three genetically informative samples from the United States and one from Sweden which combined include more than 50,000 individuals, we test whether an individual-level index aggregating the effect of genes associated with educational attainment, referred to as a polygenic score (17), predicts voter turnout. We find that the educational attainment polygenic score (EA PGS) is significantly related to self-reported and validated measures of voting. This relationship is significantly stronger in second-order elections (midterm elections in the United States and European Parliament [EP] elections in Sweden) in which information is likely to be harder to obtain or less desirable (18, 19) compared to first-order elections (presidential elections in the United States and national parliament elections in Sweden).

## Significance

**The strong correlation between education and voting is among the most robust findings in social science. We show that genes associated with the propensity to acquire education are also associated with higher voter turnout. A within-family analysis suggests education-linked genes exert direct effects on voter turnout but also reveals evidence of genetic nurture in second-order elections. Our findings have important implications for the study of political inequality. Scholars have argued that parental education is the main driver of the reproduction of political inequality across generations. By separating the effect of genes from parental nurturing, our findings suggest that the roots of individual-level political inequality run deeper than family background.**

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[1]To whom correspondence may be addressed. Email: cdawes@nyu.edu.

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In addition, we find that the effect of the EA PGS and voting in second-order elections is stronger among younger voters. Taken together, this suggests that the political environment may moderate the genetic influence on voting. Our within-family analysis suggests that education-linked genes exert a significant direct effect in both first- and second-order elections, but we also find evidence of genetic nurture in second-order elections. Finally, after controlling for the EA PGS, the effect of education shrinks by between 8% and 17%, signaling that genes associated with education partially confound the relationship between education and turnout. Our mediation analysis suggests that educational attainment and cognitive ability combine to account for between 41% and 63% of the relationship between the EA PGS and voter turnout.

## Results

To conduct our analysis, we use individual-level information from the National Longitudinal Study of Adolescent to Adult Health (Add Health), the Minnesota Twin and Family Study (MTFS), the Wisconsin Longitudinal Study (WLS), and the Swedish Twin Registry (STR). All four studies collected measures of self-reported and/or validated voter turnout as well as made available to researchers polygenic scores for educational attainment. Relevant summary statistics for each sample are reported in *SI Appendix*, Table S1.

Table 1 presents our baseline estimates of the association between the EA PGS and the three different measures of voter turnout—self-reported voting and validated measures of turnout in first-order (presidential elections in the US samples and national parliament elections in the Swedish sample) and second-order (midterm elections in the US samples and EP elections in the Swedish sample) elections. Self-reported turnout has been standardized to have a mean equal to zero and a standard deviation (SD) equal to one. First-order and second-order election turnout are measured on the unit interval scale.

Looking first at the self-reported voting column in Table 1, a 1-SD increase in the EA PGS is associated with about a sixth of an SD increase in self-reported voting in the Add Health and MTFS samples. Turning to the results for validated turnout in the first-order voting and second-order voting columns, the effect of the EA PGS is larger in second-order elections than in first-order elections; the effect is twice as large in the MTFS parent and WLS samples and more than 4 times as large in the MTFS twin and STR samples. In *SI Appendix*, Table S4, we show that the relative difference in polygenic score effect size decreases when using a generalized linear model instead of a linear probability model. However, with the exception of the MTFS parents, the pattern of statistically significant larger EA PGS effects in second-order elections is discernible irrespective of modeling choice.

In order to put the magnitude of these effects in perspective, in *SI Appendix*, Table S3, we estimate the effect of years of schooling, one of the strongest documented predictors of voter turnout (2), in each sample. The effect of the EA PGS on voter turnout reported in Table 1 is between 55% and 70% of the corresponding effect of years of schooling.

In addition to the estimated effects, we also report the incremental $R^2$ ($\Delta R^2$), or the increase in the coefficient of determination accounted for by the EA PGS. To put the amount of variation explained by the EA PGS in perspective, Fig. 1 compares the incremental $R^2$ for the EA PGS to years of education as well as other factors identified by political behavior scholars to be strong predictors of voter turnout: parental education and income (15), cognitive ability (20, 21), and personal income (22). Fig. 1 illustrates that the polygenic score's explanatory power is on par with that of personal income, parental income, and parental education and accounts for about half as much variation as years of education.

**Table 1. EA PGS and voting, baseline results**

| | Self-reported voting | First-order voting | Second-order voting |
|---|---|---|---|
| Add Health | | | |
| EA PGS | 0.153*** | | |
| | (0.013) | | |
| $\Delta$ $R^2$ | 0.023 | | |
| Average birth year | 1979 | | |
| Observations | 5,633 | | |
| MTFS twins | | | |
| EA PGS | 0.178*** | 0.011* | 0.052*** |
| | (0.025) | (0.005) | (0.008) |
| $\Delta R^2$ | 0.028 | 0.002 | 0.018 |
| Average birth year | 1979 | 1983 | 1983 |
| Observations | 2,264 | 3,013 | 3,035 |
| MTFS parents | | | |
| EA PGS | | 0.011*** | 0.028*** |
| | | (0.002) | (0.004) |
| $\Delta R^2$ | | 0.009 | 0.017 |
| Average birth year | | 1953 | 1953 |
| Observations | | 3,448 | 3,452 |
| WLS | | | |
| EA PGS | | 0.014*** | 0.027*** |
| | | (0.004) | (0.004) |
| $\Delta R^2$ | | 0.002 | 0.006 |
| Average birth year | | 1939 | 1939 |
| Observations | | 7,042 | 7,042 |
| STR | | | |
| EA PGS | | 0.018*** | 0.080*** |
| | | (0.001) | (0.004) |
| $\Delta R^2$ | | 0.007 | 0.034 |
| Average birth year | | 1967 | 1968 |
| Observations | | 39,333 | 39,633 |

Self-reported voting and the EA PGS are standardized (mean = 0, SD = 1) within each sample. First-order (presidential in the United States and national in Sweden) and second-order (midterm in the United States and EP in Sweden) election turnouts are measured as average turnout across all the elections for which we have information for the individuals. All models include controls for gender, birth year, and the first 10 principal components of the genetic relatedness matrix. Standard errors, shown in parentheses, allow for clustering at the family level. ***/**/*, indicate significance at the 0.1/1/5% level.

It should be noted that, in Fig. 1, the EA PGS, as well as other predictors of voter turnout, explains less of the variation in validated turnout in the WLS sample than the MTFS and STR samples. This may be due to the fact that the older WLS subjects are at a point in their lives where voting has become an ingrained habit (23), meaning that individual- and contextual-level factors become less influential in determining turnout (24). Further, among older citizens, factors such as health (25) and social isolation (26, 27) become more prominent in determining whether or not to vote. In *SI Appendix*, Table S5 and Fig. S2, we make use of the wide and approximately uniform age distribution in the STR sample and show that the estimated effects and predictive power of both the EA PGS and years of education on validated voter turnout are significantly stronger among younger individuals.

To check the representativeness of our samples we show, in *SI Appendix*, Table S6, that the amount of variation in validated turnout accounted for by education in the MTFS and WLS samples are similar to what we find based on the Cooperative

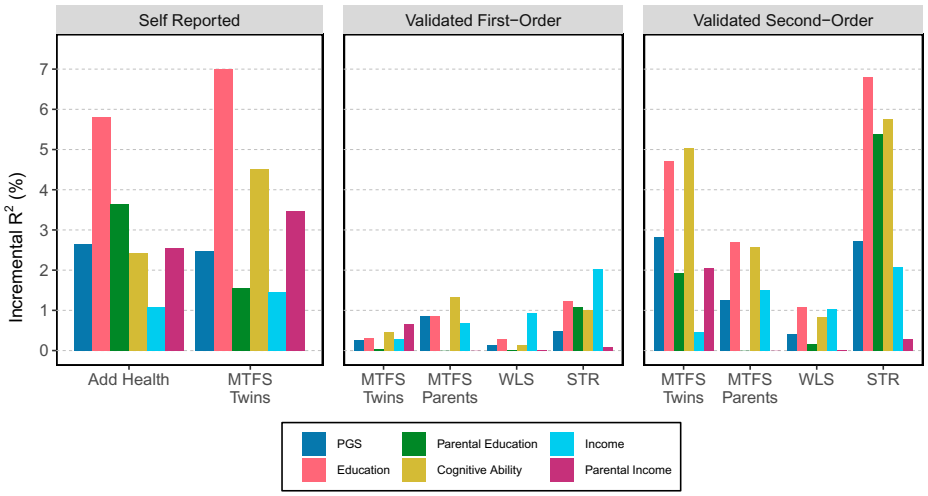


**Fig. 1.** Incremental $R^2$ for the EA PGS compared to other predictors of voter turnout. The height of each bar represents the increase in the coefficient of determination ($R^2$) when each variable indicated is added as a covariate to a regression of voter turnout on a set of baseline controls that includes gender, birth year, and 10 principal components of the genetic relatedness matrix. Parental income and education are not available for the MTFS parents. The income measure for Add Health and the MTFS twins is personal income before taxes and, for the MTFS parents, household income before taxes. For the WLS sample, the figure reports personal net worth rather than income, since it is a more appropriate measure of financial resources for older individuals in the sample, many of whom are retired. For the STR sample, we use annual register information on gross total wage income and income from business to calculate average income between ages 25 and 65 years.

Congressional Election Study, a US survey based on a very large national representative sample. We also show that the results obtained in the STR sample are comparable to corresponding estimates based on individual-level population data.

We next check the degree to which the EA PGS confounds the relationship between education and turnout. A recent discordant twin study based on a similar Minnesota sample found that approximately half of the effect of education on political participation was due to genetic factors and/or family environment (28). In *SI Appendix*, Table S12, we show that controlling for the EA PGS reduces the effect of education by, on average, about 12% across the four samples. While these results suggest only a modest amount of confounding, it is important to remember that the EA PGS does not capture all of the genetic propensity to acquire education. When more-precise polygenic scores become available in the future, it is possible that the effect of education falls by closer to 30 to 40% (29).

We also test whether genes associated with educational attainment influence voter turnout via education and cognitive ability. Cognitive ability is likely to be a mediator, given that it has been shown to be associated with the EA PGS (30) and is strongly related to voter turnout (20, 21) and both have been shown to be influenced by shared genetic factors (9, 10). As shown in Fig. 1, voter turnout is strongly related to both years of schooling and cognitive ability in all four samples. In Fig. 2, we present results from a mediation analysis (31, 32) for the two traits (the full regression results are presented in *SI Appendix*, Tables S13 and S14). We acknowledge that, since we cannot account for potential alternative mediators, this analysis should be considered descriptive rather than causal (33).

Consistent with a mediated relationship, Fig. 2 shows that the effect of the EA PGS on voter turnout shrinks considerably when controlling for educational attainment and cognitive ability. This is the case across all four samples. The amount mediated differs depending on the measure of turnout; educational attainment and cognitive ability account for ∼63% of the effect of the EA PGS on self-reported voting, 47% of the effect on voting in first-order elections, and 50% of the effect on voting in second-order elections. While this analysis suggests that the genetic propensity to acquire education is mediated by educational attainment and cognitive ability, it also suggests there are EA PGS mechanisms influencing voter turnout that are unrelated to education and cognitive ability, as there are still significant and sizeable effects of the EA PGS on the three outcomes. Another possible mediator is personality, given that the EA PGS is correlated with personality traits (34, 35), a growing literature has demonstrated personality traits to be important for turnout (36), and personality and turnout have been shown to be influenced by shared genetic factors (9, 10). In *SI Appendix*, Tables S15–S19, we present results from regression models in which we include different measures of personality traits included in the four samples as additional controls. The inclusion of these variables does not reduce the estimated effect of the EA PGS any further.

Having demonstrated a relationship between the EA PGS and voter turnout, we next assess the extent to which this relationship is due to direct genetic effects (effects of individuals' education-linked genes on their voting behavior) versus indirect effects via genetic nurture. As evidence of the latter, there are moderate (and significant) correlations between 1) the offspring EA PGS and parental education, 2) parental education and offspring education, and 3) offspring education and offspring turnout in all four samples used in this study.* Additionally, the EA PGS effects may still be confounded by population stratification despite the fact that the previous analysis restricted the samples to individuals of European descent and includes controls for a set of principal components of the genetic relatedness matrix.

In order to identify potential bias, we restrict our analysis to sibling pairs. Since this restriction dramatically reduces the size of each sample, we pool the US samples having similar outcome measures in order to increase power.† Table 2 displays the results

*The bivariate correlations ($p < 0.001$ in all cases) between 1) offspring polygenic score and parental education are equal to 0.294 (Add Health), 0.201 (MTFS twins), 0.239 (MTFS parents), 0.154 (WLS), and 0.245 (STR); 2) parental education and offspring education: 0.400 (Add Health), 0.314 (MTFS twins), 0.794 (MTFS parents), 0.325 (WLS), and 0.786 (STR); 3) offspring education and offspring self-reported voting: 0.273 (Add Health) and 0.291 (MTFS twins); 4) offspring education and offspring first-order voting: 0.113 (MTFS twins), 0.138 (MTFS parents), 0.076 (WLS), and 0.114 (STR); and 5) offspring education and offspring second-order voting: 0.201 (MTFS twins), 0.157 (MTFS parents), 0.112 (WLS), and 0.227 (STR).

†For self-reported voting, we have pooled the MTFS and the Add Health samples, and, for first-order (presidential) and second-order (midterm) voting, we pooled the MTFS and WLS samples.

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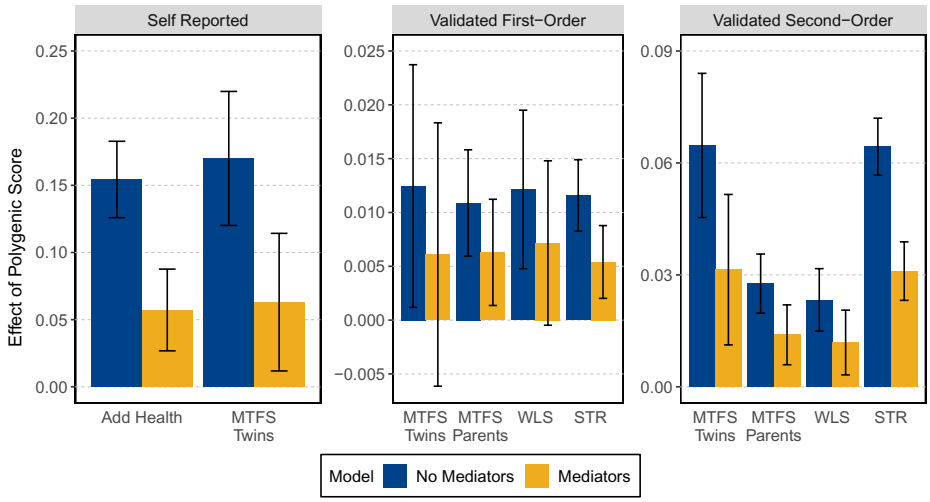


**Fig. 2.** Mediation analysis results for self-reported and validated turnout. The height of each bar represents the estimated effect of the polygenic score on turnout, and the error bars represent 95% confidence intervals. The model with mediators includes years of education and cognitive ability. Both models include controls for gender, birth year, and the first 10 principal components of the genetic relatedness matrix.

based on the pooled US samples (as well as estimates based on each of the subsamples) and the STR sample. As a point of comparison, the first column for each outcome represents the ordinary least squares (OLS) effect in these restricted samples, and the second column shows estimates from models in which we include fixed effects (FE) for each sibling pair. That is, in the latter models, we only use the within-family variation in order to account for both genetic nurture effects that are related to

**Table 2. EA PGS and voting—OLS and fixed effects models**

| | Self-reported voting | | First-order voting | | Second-order voting | |
|---|---|---|---|---|---|---|
| | OLS | FE | OLS | FE | OLS | FE |
| Pooled US results | | | | | | |
| EA PGS | 0.172*** | 0.107* | 0.016*** | 0.022** | 0.028*** | 0.017 |
| | (0.029) | (0.048) | (0.004) | (0.009) | (0.005) | (0.011) |
| $\Delta R^2$ | 0.026 | 0.007 | 0.003 | 0.003 | 0.006 | 0.001 |
| Observations | 1,492 | 1,492 | 3,934 | 3,934 | 3,943 | 3,943 |
| Add Health | | | | | | |
| EA PGS | 0.151*** | 0.068 | | | | |
| | (0.040) | (0.066) | | | | |
| $\Delta R^2$ | 0.020 | 0.003 | | | | |
| Observations | 788 | 788 | | | | |
| MTFS twins | | | | | | |
| EA PGS | 0.202*** | 0.174* | 0.018* | 0.023 | 0.023 | −0.036 |
| | (0.043) | (0.073) | (0.008) | (0.015) | (0.012) | (0.022) |
| $\Delta R^2$ | 0.035 | 0.019 | 0.006 | 0.000 | 0.004 | 0.006 |
| Observations | 704 | 704 | 1,005 | 1,005 | 1,014 | 1,014 |
| WLS | | | | | | |
| EA PGS | | | 0.015*** | 0.021 | 0.029*** | 0.033** |
| | | | (0.005) | (0.011) | (0.006) | (0.012) |
| $\Delta R^2$ | | | 0.003 | 0.002 | 0.008 | 0.005 |
| Observations | | | 2,929 | 2,929 | 2,929 | 2,929 |
| STR | | | | | | |
| EA PGS | | | 0.017*** | 0.015*** | 0.074*** | 0.034*** |
| | | | (0.002) | (0.003) | (0.004) | (0.006) |
| $\Delta R^2$ | | | 0.006 | 0.003 | 0.028 | 0.004 |
| Observations | | | 16,386 | 16,386 | 16,520 | 16,520 |

Self-reported voting and the EA PGS are standardized (mean = 0, SD = 1) within each sample. First-order (presidential in the United States and national in Sweden) and second-order (midterm in the United States and EP in Sweden) election turnouts are measured as average turnout across all the elections for which we have information for the individuals. All models include controls for gender, birth year, and the first 10 principal components of the genetic relatedness matrix. Standard errors, shown in parentheses, allow for clustering at the family level. ***/**/*, indicate significance at the 0.1/1/5% level.

common family environment and any remaining confounding due to population stratification and assortative mating.

Comparing the OLS and FE estimates, there is no indication of any confounding due to genetic nurturing, population stratification, or assortative mating for voting in first-order elections. The effect of the EA PGS on self-reported turnout is smaller based on the fixed effects model compared to the OLS analysis. However, the estimates are not precise enough to draw any definitive conclusions regarding the existence of confounding. Looking, instead, at the results for voting in second-order elections, there is evidence of bias due to confounding, especially in the STR sample. The FE estimate for the STR sample is less than half the magnitude of the OLS estimate, and (given the sample of more than 8,000 siblings) both are precisely estimated. While less precisely estimated, the OLS and FE results based on the MTFS twins display the same pattern; the nearly significant ($p = 0.055$) OLS estimate becomes negative and insignificant in the fixed effects model. In *SI Appendix*, Tables S7 and S8, we show, by comparing results from an OLS regression controlling for parental education with those from the fixed effects model, that genetic nurturing is likely the source of most of the bias. In *SI Appendix*, Tables S9–S11, we present further evidence on genetic nurturing effects by showing that parental and sibling polygenic scores are related to second-order election turnout, also after controlling for one's own EA PGS.

## Conclusions

Based on the political participation of more than 50,000 US and Swedish citizens, we find a significant association between education-linked genes and both self-reported voting and validated turnout across a total of 23 different elections. Further, the polygenic score for education has predictive power comparable to well-studied correlates of voting such as parental education and personal income.

In both the United States and Sweden, we find that genes associated with educational attainment play a more influential role in explaining voting in second-order compared to first-order elections. Second-order elections are generally considered by voters, parties, and the media to be less important than first-order presidential or national elections (18). Thus, citizens typically have to navigate a more information-poor electoral environment, making it more challenging to participate. Citizens with higher education are relatively more likely to vote in these elections (19), since they are better equipped to overcome the costs of acquiring information. We also observe a stronger effect of the EA PGS among the younger Swedish twins. Perhaps, among older citizens, experience and habit reduce the importance of the genetic propensity for traits that reduce the cost of participation. More research is necessary, but these results suggest genes and the political environment both combine to influence political participation.

Our findings corroborate a recent Danish study of voter turnout (13); however, our within-family analyses based on a large number of sibling pairs and mediation analysis provide a more nuanced picture of this relationship. Since models based on unrelated individuals are vulnerable to environmental confounding (14), the significant within-family estimates we provide offer solid evidence of a direct relationship between education-linked genes and voter turnout in both first- and second-order elections.[‡] For second-order elections, the within-family estimates also reveal evidence of confounding. For the Swedish sample, the effect of the EA PGS in the fixed effects model is half the size of the estimate from a naive OLS model, and our follow-up analysis suggests that much of this confounding is due to genetic nurture. Political behavior scholars have long held that well-educated parents influence their children's civic development by providing the resources necessary to enable them to be well educated, thus facilitating their political engagement, as well as to create family environments that foster political interest and impart skills necessary to be politically active (16). However, recent work has shown that education-linked genes influence the type of environments parents choose for their children, which, in turn, impacts their children's educational attainment (39–44). Our findings suggest that these genes are also influencing voter behavior via family environment.

The availability of detailed information on subjects' educational attainment, cognitive ability, and personality traits also allowed us to test potential pathways through which education-linked genes influence turnout. While our mediation analysis suggests that these genes likely influence turnout via educational attainment and cognitive ability, surprisingly, a large fraction of this genetic influence was not mediated by these two factors. This underlines the importance of testing potential mechanisms rather than making assumptions based solely on theory or past empirical evidence; genes associated with educational attainment may influence complex traits like voting through a number of different pathways. A wealth of studies show the EA PGS is predictive of traits related to voter turnout, and thus suggest potential mediators to be explored in future research. They include socioeconomic status (35, 41, 45–47), wealth (48), labor market outcomes (47, 49, 50), geographic mobility and migration (35, 51, 52), and mate choice (53, 54). Health outcomes may be another possible mediator; recent work found that cognitive and physical well-being predict voter turnout (25), and ref. 34 showed that educational attainment is genetically linked with psychiatric and physical traits.

Our findings also contribute to the study of political inequality (1, 15, 55, 56). Ref. 15 argues that parental education is the main driver of the reproduction of political inequality across generations. However, most studies of the intergenerational transmission of political participation fail to separate the effect of genes from parental nurturing. We show that genes are nearly as predictive as parental education for self-reported and validated voting. Thus, inheriting genes beneficial for educational attainment makes individuals more likely to vote. Taken together, our findings suggest that the roots of individual-level political inequality run deeper than family background. It is important to note, however, that, since the genome-wide association study (GWAS) used to construct the EA PGS consisted of individuals of European ancestry, the EA PGS cannot shed any light on group differences in political participation (15).[§]

Finally, it is important to point out that the EA PGS does not fully capture the genetic propensity to acquire education; it accounts for between 11% and 13% of the variation in educational attainment (30), while heritability based on all genotyped single nucleotide polymorphisms (SNPs) has been shown to be ∼20% (57), and a metaanalysis of twin studies reported that about 40% of the variation in educational attainment can be attributed to genetic factors (58). Thus, our results likely represent a lower bound on the effect of genes associated with educational attainment on voter turnout. Ongoing larger GWA studies of educational attainment will provide us with more-accurate polygenic scores in the near future.

[‡] Since any genetic differences between full siblings are random, within-family estimates are typically given a causal interpretation (14). However, recent theoretical work (37, 38) has argued that estimates from within-family models may still be biased due to environmental confounding.

[§] For example, ref. 30 shows that the EA PGS did a poor job of predicting educational attainment in a sample of African Americans.

## Methods

**Samples and Measures.** We use data from four different samples to examine the relationship between the EA PGS and voter turnout: the MTFS, Add Health, the WLS, and the STR. Please see *SI Appendix* for detailed information about each of the samples and the measures we use as well as how to access the data.

**Polygenic Score Prediction.** To test our hypothesis that genes associated with educational attainment influence voter turnout, we use an index, referred to as a polygenic score, of an individual's genetic predisposition for educational attainment. A PGS maximizes the predictive power for a trait by using information from a well-powered GWAS (17). The standard approach of a GWAS is to run $K$ separate regressions of a trait $y$ on the $K$ genotyped SNPs,

$$y_i = \mu_k + \beta_k x_{ik} + \epsilon_{ik} \quad [1]$$

for $k = 1, \ldots, K$, where $x_{ik}$ $(x \in \{0, 1, 2\})$ is the number of minor alleles individual $i$ has for SNP $k$.

To create a PGS, a GWAS is conducted in a discovery sample, and the estimated effects of the SNPs ($\hat{\beta}_k$) are subsequently used as a weights in the aggregation of SNPs in a target sample of interest,

$$PGS_i = \sum_{k=1}^{K} \hat{\beta}_k x_{ik}. \quad [2]$$

The polygenic scores for educational attainment we use were made available to researchers by the four studies (described in greater detail in the *SI Appendix*) we analyze as part of our study. All of the scores were constructed in the same manner by the Social Science Genetic Association Consortium (https://www.thessgac.org/) as part of the Polygenic Index Repository (29).[¶] For a more detailed discussion of the polygenic score framework, please refer to *SI Appendix*.

**Empirical Framework.** As a starting point for our analysis, we analyze the EA PGS in the following simple regression framework:

$$y_i = \beta_0 + \beta_1 PGS_i + C_i \beta_C + \epsilon_i, \quad [3]$$

where $y_i$ is a measure of voter turnout for individual $i$; $PGS_i$ is the polygenic score of educational attainment; $C_i$ is a vector of control variables; and $\epsilon_i$ is an independent and identically distributed error term. The control variables include fixed effects for each birth cohort (i.e., birth year), a dummy indicator for being male, and the top 10 principal components of the covariance matrix of the individuals' genotypic data to mitigate the risk of population stratification.

In Eq. **3**, $\beta_1$ provides us with an estimate of the effect of the EA PGS on voter turnout. However, this estimate may be biased due to population stratification, assortative mating, or genetic nurturing. Since our samples comprise families including biological siblings, we are able to estimate sibling fixed effect models that eliminate the first two sources of potential bias. For a more detailed discussion of the empirical framework, please refer to *SI Appendix*.

**Data Availability.** We use restricted individual level information obtained from each of the studies. As part of our contractual agreement with each study, we agreed not to disseminate the data to other individuals. However, researchers can access the restricted data directly from each study. Details on how to access restricted data for each sample can be found at: MTFS (https://mctfr.psych.umn.edu/); Add Health (https://www.cpc.unc.edu/projects/addhealth/documentation/restricteduse); WLS (https://www.ssc.wisc.edu/wlsresearch/data/); and STR (https://ki.se/en/research/swedish-twin-registry-for-researchers and https://www.scb.se/en/services/ordering-data-and-statistics/ordering-microdata/).

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[¶]The EA PGS is constructed using the results of a published GWAS of educational attainment based on over 1,100,000 subjects (30). Information about the construction of the EA PGS can be found in ref. 29.

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