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Genetic predictors of educational attainment and intelligence test performance predict voter turnout

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Although the genetic influence on voter turnout is substantial (typically 40–50%), the underlying mechanisms remain unclear. Across the social sciences, research suggests that 'resources for politics' (as indexed notably by educational attainment and intelligence test performance) constitute a central cluster of factors that predict electoral participation. Educational attainment and intelligence test performance are heritable. This suggests that the genotypes that enhance these phenotypes could positively predict turnout. To test this, we conduct a genome-wide complex trait analysis of individual-level turnout. We use two samples from the Danish iPSYCH case–cohort study, including a nationally representative sample as well as a sample of individuals who are particularly vulnerable to political alienation due to psychiatric conditions ($n = 13{,}884$ and $n = 33{,}062$, respectively). Using validated individual-level turnout data from the administrative records at the polling station, genetic correlations and Mendelian randomization, we show that there is a substantial genetic overlap between voter turnout and both educational attainment and intelligence test performance.

Representative democracies are premised on the electoral participation of their citizens. Without politically engaged citizens, democracy lacks its central impetus, and if some groups are systematically excluded from the political process or decide to opt out, key societal problems will most likely remain unaddressed, because the parliaments do not represent the views and priorities of their citizens. However, although the act of voting in this sense can be considered to be a fundamental public good, a large body of literature documents strong disparities among citizens in their level of electoral participation; some do not vote in elections at all[1–4]. These disparities exist both in the general population and in segments that are particularly vulnerable to political exclusion and alienation (such as those with psychiatric conditions). Why do some people devotedly vote in elections while others consistently remain unengaged?

A large body of twin-based studies has shown that individual differences in voter turnout are strongly associated with genetic variation, with heritability estimates that are typically between 40–50% but up to 72% (refs. [5–9]). Importantly, these findings raise the fundamental question of how genes and voter turnout are linked[5,10]. As emphasized by social scientists, "[a]t this early point in the research, there remains a black box"[11]. Together with a call that better theorizing 'must occur'[12], critiques have also been raised against existing twin and candidate gene studies owing to methodological limitations[13,14].

To advance our understanding of how genes and voter turnout are linked, we followed recent studies[5,10] and integrated the genetic and the social science paradigms of the microlevel foundations of voter turnout. Classic social science models of electoral participation emphasize that voting imposes opportunity costs on voters in the form of time, money, effort and cognitive investment[15–17]. In the face of these costs, individual differences in educational attainment and intelligence test performance constitute key predictors of political participation because they index 'resources for politics' that reduce the costs of voting. The correlations predicted by the resource model between political participation and educational attainment and cognitive performance, respectively, have found wide empirical support[17,18], and can also be observed in Denmark, the site of this study[19] (Supplementary Appendix 2.3 and Supplementary Table 5). However, from a genetics perspective, it is important to note that educational attainment and performance on intelligence tests are themselves genetically influenced and correlated traits[20–24]. Thus, the genetic variation that predicts individual differences in educational attainment and intelligence test performance may also

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predict variation in voter turnout. By integrating the genetic and social science paradigms regarding the roots of voter turnout, we therefore investigated the following question: to what extent is the genetic variation that underlies educational attainment and intelligence test performance associated with individual differences in voter turnout? We argue and demonstrate that the genotypes that predict individual differences in educational attainment and intelligence test performance also predict individual differences in voter turnout.

For decades, scholars have especially considered education to be the 'universal solvent' that predicts high political participation and, therefore, the functioning of democracy (p. 324 in[25]; see also refs. [4,17,26,27]). Education "increases civic skills and political knowledge", which function as mechanisms underlying participation[28], and these mechanisms are associated with participation through "knowledge about where, when and for whom to vote"[6], the ability to communicate effectively, cognitive skills to understand policies, a sense of civic duty and responsibility[4,17], and the socioeconomic status that provides access to decision makers and politically relevant information[26,29]. Educational attainment is also associated with personality traits, such as openness to experience, that predict individual awareness and interest in politics[10,12,30], and previous research has argued that a higher educational achievement could also reflect an individual's motivation and self-control[31,32]. Importantly, these cognitive and non-cognitive mechanisms all reduce the individual's voting costs[17]. The causal interpretation of these relationships remains an ongoing discussion. In the genetics literature, research has found that the single-nucleotide polymorphism (SNP) associations that predict variation in educational attainment and cognitive performance also, to some extent, predict personality traits[33], socioeconomic status[34,35] and wealth[36]. In line with these results, there is ongoing debate in the political science literature regarding the causal interpretation of the relationship between phenotypic educational attainment and voter turnout and the underlying mechanisms (reviewed previously[28,37]). Persson[28] concluded that education might capture an individual's cognitive skills, motivation and social network. The question of the exact causal pathways notwithstanding, "[t]he relationship between education and voter turnout ranks among the most extensively documented correlations"[37], and educational attainment is considered to be one of the most important predictors of political participation[4,17,27,28].

In addition to educational attainment, cognitive performance indicators are also studied to predict voter turnout[38,39]. Although intelligence test performance should be interpreted with care, intelligence test performance has been viewed as an indicator of cognitive skills (discussed in ref. [40]) and a reliable predictor of political efficacy, which is associated with higher political participation[41]. Studies in western countries have found that those who perform well on intelligence tests during childhood are more interested in politics in adulthood and are more likely to participate in elections and engage in other forms of political participation[38]. Importantly, similar to education, it has also been found that intelligence test performance indicators are correlated with socioeconomic status[42,43], which is also associated with higher political participation[17].

Intelligence test performance and educational attainment are highly heritable and correlated polygenic traits ($r_g = 0.73$)[44,45]. The trait-enhancing markers are spread throughout the human genome[46–48] (reviewed previously[49]), with estimates of 51% narrow-sense heritability for intelligence[50] and 21% for educational attainment[51] obtained from the genomic-relatedness-based restricted maximum-likelihood approach implemented in genome-wide complex trait analysis (GCTA-GREML). Thus, integrating the social science resource model of political participation with the genetic literature on the heritability of educational attainment and intelligence test performance gives rise to the key correlational hypothesis that the genetic variance that predicts educational attainment and intelligence test performance, respectively, predicts individual differences in electoral participation.

In testing this hypothesis, we also sought to overcome methodological shortcomings in the existing literature. Previous studies investigating the heritability of voter turnout have relatively low external validity owing to an almost exclusive reliance on samples of twins ($n = 396$–$3{,}616$)[5–10]. One molecular genetics study relied on a candidate-gene approach conducted on a random sample of participants in the United States[52], but concerns have been raised about its replicability[14]. A recent study performed the first genome-wide association study (GWAS) for voting behaviour, but used regional data on voting behaviour and did not analyse individual-level turnout[53]. All but three studies have used data from individuals from the United States, which further limits the external validity. Interestingly, one of these studies with Swedish participants produced mixed findings regarding the heritability of voting[5]. Furthermore, it is important to note that all research efforts have been directed towards investigating the heritability of voting[5–9,52] and the underlying mechanisms[5] in the general population. Investigation into the genetics of individual-level turnout in vulnerable subpopulations in which political non-participation is a more wide-spread phenomenon than in the general population is lacking[54,55] (Supplementary Appendix, Supplementary Table 1 and Supplementary Fig. 1).

Previous studies addressing the heritability of voter turnout also face issues regarding internal validity. Most previous studies used the classic reared-together twin design, which typically confounds the potential influence of genes and shared environment (discussed previously[56]). Given these limitations to the internal validity, scholars have warned that twin studies "can neither prove nor refute the argument for a genetic basis of political traits such as […] voting turnout"[57]. Finally, most studies rely on self-reported voter turnout, which is known to be associated with misreporting and over-reporting and, therefore, limits measurement validity[58].

To address these limitations to external, internal and measurement validity, we conducted a comprehensive investigation of the link between genetics and electoral participation. To increase internal and external validity, we moved beyond the classic twin design and relied on a dataset of comprehensively genotyped individuals from the Danish-population-based iPSYCH case–cohort sample[59]. It includes a representative sample of the entire population born between 1981–2005 in Denmark and a psychiatric sample of people diagnosed with anorexia, schizophrenia, affective disorder (including depression), bipolar affective disorder, autism and attention-deficit/hyperactivity disorder (ADHD) born in Denmark during the same period[59] (Supplementary Appendix 1).

As the overall setting, the Danish electoral context is substantially more inclusive than the United States, where most previous studies of the heritability of turnout have been conducted and where, for example, comprehensive voter identification laws shape turnout[60]. In Denmark, all eligible citizens are automatically registered to vote, and much is done to enable disabled people and others on the margins of society to vote in elections. The barriers to electoral participation are therefore relatively low in Denmark.

Individuals with a psychiatric condition are an important subpopulation to study. According to the WHO, one in four people in the world become affected by a psychiatric or neurological condition during their lives[61]. Individuals with a psychiatric condition are potentially vulnerable to social and political exclusion[54,62–67]. First, we might expect that psychiatric condition, on average, will lower turnout. Using the logic of the resource model, previous research therefore argues that poor health may "hinder the acquisition of other resources such as civic skills, time and money"[66]. Second, we might expect that individuals with certain psychiatric conditions are particularly vulnerable to exclusion. For example, previous studies have argued that impeded cognitive functioning—which is central in psychiatric diagnoses such as ADHD—"negatively

influence[s] individuals' ability to process information related to elections"[55]. Having a psychiatric condition may also lead people to actively deprioritize voting as too costly in terms of time, effort and attention and therefore voluntarily opt out of the political process. Finally, other studies argue that some types of mental conditions such as depression can reduce "the somatic capacity of an individual and therefore reduce the resources an individual has for political participation"[54]. If individuals with psychiatric conditions do not vote in elections, parliaments may not represent the views and priorities of this group of citizens, and this augments the risk that key problems and needs relevant to them will remain underprioritized.

In our psychiatric sample, individuals with a psychiatric diagnosis were on average 15–32% less likely to vote in the three elections under study than the general Danish population (Supplementary Appendix 2, Supplementary Table 1 and Supplementary Fig. 1). Individuals who were diagnosed with anorexia have the highest turnout frequency and are descriptively indistinguishable from the representative sample. Individuals who were diagnosed with ADHD have the lowest turnout and are 28–54% less likely to vote than the average Danish population (Supplementary Appendix 2, Supplementary Table 1 and Supplementary Fig. 1). Studying the link between genes and voter turnout in both a nationally representative sample and a subpopulation of individuals with a psychiatric diagnosis who are vulnerable to political exclusion increases the external validity of the results.

To maximize measurement validity, we moved beyond self-reports of turnout and integrated the genetic data with population-based turnout data obtained directly from the administrative records at the polling place of whether Danish citizens voted in the 2015 national, 2014 European and 2013 municipal elections (Supplementary Appendix 1.2). The saliency of the election differs substantially across these types of elections, most simply apparent from the general turnout in the elections. The national election constitutes a high-saliency election with a turnout of 86% in 2015. The municipal election in 2013 represents a medium-high-saliency election with a turnout of 72%. Finally, European elections generally have low saliency in Denmark, and turnout in 2014 was only 56%. Investigating our prediction across three types of elections further increases external validity.

With 46,946 unrelated individuals, this provides a dataset that excels in terms of (1) sample size; (2) measurement validity, as electoral participation is objective rather than self-reported; (3) internal validity, as the research design moves beyond the limitations of the classic reared-together twin design; and (4) external validity, as the dataset includes a random, nationally representative sample of more than 13,000 individuals and one of the world's largest samples of a subpopulation of individuals with a psychiatric diagnosis, and covers three types of elections.

Results

Using GCTA, we first estimated the proportion of phenotypic variation in electoral participation in each of the three elections that can be accounted for by SNPs (hereafter refered to as h^2_{SNP}), which are the most common type of genetic variations between individuals. Previous estimates from twin and family studies typically conflate heritability because they are "biased by factors shared by close relatives, such as non-additive genetic and common environmental effects"[68,69]. The h^2_{SNP} is not inflated by these confounds (h^2_{SNP} captures only variance explained by common SNP variants). It can therefore be seen as an estimate of the lower bound of the total (or broad sense) heritability (H^2). The h^2_{SNP} estimates are reported in Fig. 1 with 95% confidence intervals (CIs), enabling us to compare the effect of heritability between samples (national and psychiatric) and across the types of election (varied by the election saliency).

In the psychiatric sample, we found a statistically significant h^2_{SNP} for electoral participation phenotypes with the point estimates, suggesting that the common SNPs explain approximately 8–10% (s.e. = 0.0155–0.021) of the phenotypic variation in electoral participation (all $P \leq 2.61 \times 10^{-7}$). Previous American twin studies of the heritability of electoral turnout have estimated that up to 53–72% of individual differences in electoral turnout can be accounted for by genetic variability. Previous studies focusing on height[70], intelligence[50], personality traits[71], and political and economic preferences[72] found that h^2_{SNP} estimates are approximately 1/2 to 1/4 the size of twin-study estimates. On this basis, the observed SNP-based heritability estimates in the psychiatric sample are proportionally consistent with previous American twin-study estimates.

In the nationally representative sample, the h^2_{SNP} estimate for the 2014 European election appears to be qualitatively similar to the estimates from the psychiatric sample ($h^2_{SNP} = 0.1096$, s.e. = 0.0044, $P = 6.43 \times 10^{-3}$; Fig. 1a). The h^2_{SNP} estimates for voting in the in the high- to medium-high-saliency elections in the 2015 and 2013 elections are statistically non-significant, and the point estimates are lower compared to the psychiatric sample. Importantly, the substantial overlap between the CIs for the heritability estimates in the two samples indicates that the h^2_{SNP} estimates in the nationally representative sample are not statistically significantly different compared to the psychiatric sample, and that the overall trends are the same in the two samples. Part of the explanation for the non-significant h^2_{SNP} estimates in the nationally representative sample is the lower statistical power in this sample (a power analysis is provided in Supplementary Appendix 3 and Supplementary Fig. 2) and that the h^2_{SNP} estimate represents a lower bound for narrow sense heritability. To further explore this interpretation, we repeated the analysis in Fig. 1 using pedigree-defined familial relationships. These analyses provide highly statistically significant ($P < 1.24 \times 10^{-40}$) upper-bound heritability estimates of voter turno\ut that range between 0.39 (s.e. = 0.02) and 0.49 (s.e. = 0.02) across the three elections (Supplementary Appendix 4.2 and Supplementary Tables 6.1 and 6.2). Although the family estimates may be biased upwards due to the expected common environment effect in a trait such as voting, the results overall support that the statistically non-significant h^2_{SNP} estimates in the nationally representative sample may be due to the lower statistical power in this sample and that the h^2_{SNP} estimate represents a lower bound for the narrow sense heritability.

Interestingly, in the nationally representative sample, the SNP heritability is significantly higher in the low-saliency European parliamentary election compared with in the national election ($P = 0.044$, $z = 1.7052$). Although the difference in point estimates is similarly wide in the municipal elections, it is not statistically significant owing to a larger s.e. of the estimate ($P = 0.0534$, $z = 1.6126$); details of these analyses are provided in the 'Analyses of differences in heritability across elections' section in the Methods). The statistically significant difference in the SNP heritability in the low-saliency European parliamentary election compared with the high-saliency national election is consistent with Tingsten's[73] law of dispersion, which predicts that the participatory gap between high- and low-propensity voters is higher in low-saliency elections because low saliency increases the costs of voting[74]. In such circumstances, the effect of individual differences including genetic dispositions should take precedent, which is the pattern that we observed. This perspective could also potentially explain the general low heritability estimates in the Danish nationally representative sample compared with previous studies, given the general inclusiveness of the Danish electoral context. Regarding the psychiatric sample, note that we found no statistically significant difference between SNP heritability of turnout in the European election of 2014 and in the national election of 2015 ($P = 0.4099$, $z = 0.2277$) or the municipal election of 2013 ($P = 0.2561$, $z = 0.6551$); details of these analyses are provided in the 'Analyses of differences in heritability across elections' section in the Methods). Thus, the heritability of turnout is possibly less affected by election saliency in this population.

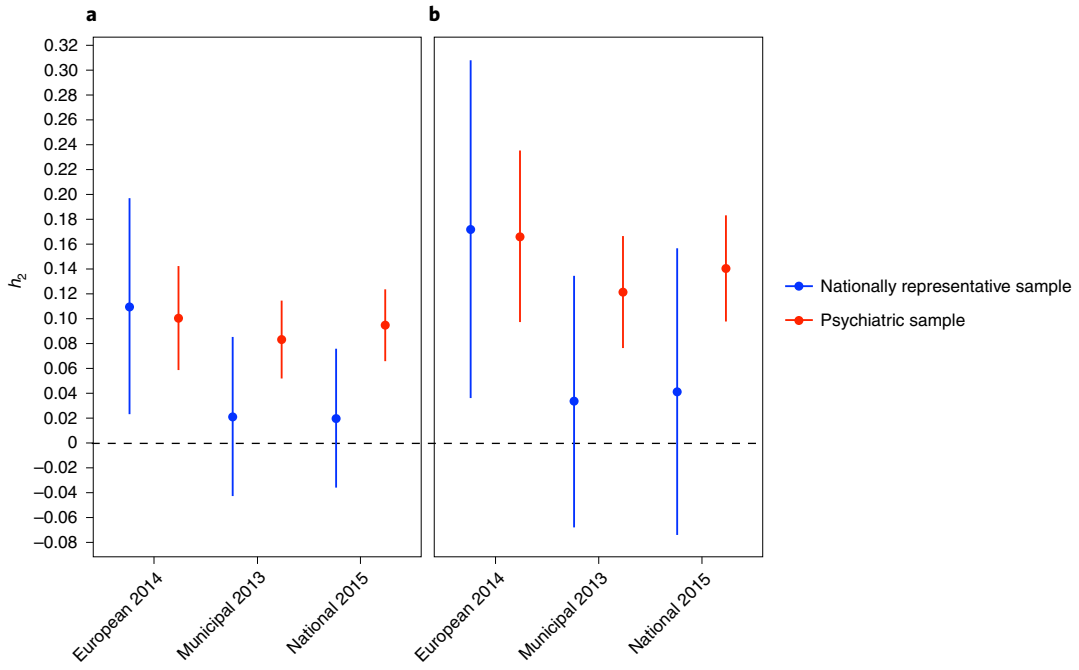

Fig. 1 | Heritability estimates for voting in municipal, European and national elections. **a**,**b**, Observed scale (**a**) and a transformed liability scale (**b**). Data points refer to point estimates for narrow-sense SNP heritability. Error bars incidcate 95% CIs.

Predictive use of polygenic scores. Next, we investigated whether the genotypes that predict individual differences in educational attainment and performance on intelligence tests also predict individual differences in voter turnout. We used polygenic scores (PSs) to measure genetic correlates of educational attainment and performance on intelligence tests (compare with refs. [44,75,76]). A PS is an index that aggregates the effects of all of the DNA variants associated with a given trait to predict an outcome[24]. Specifically, a PS is the sum of the products of additive counts of SNP alleles (for example, 0, 1 or 2 copies of the rarer of two alleles at a given variant) and the corresponding per-allele effect sizes estimated on the basis of a GWAS for a given trait. PSs have been shown to reliably predict educational attainment[75] and intelligence test performance[44]. However, the predictive ability of PSs should be interpreted knowing that the proportion of explained variation is typically low[77]. About 11–13% of the variance in education and 7–10% of the variance in cognitive performance can be predicted from the PS for educational attainment, and 5.2% of the variation in intelligence test performance can be predicted from the PS for intelligence test performance[44,75].

Figure 2 shows voter turnout within each percentile of the distribution for the PS for educational attainment and performance on intelligence tests across the three elections in the psychiatric and nationally representative samples. The results show that higher PSs for educational attainment and intelligence test performance correspond with increased voter turnout across all three elections in both samples. Details of the variance in voter turnout explained by PSs for educational attainment and intelligence test performance for each election across both the nationally representative and psychiatric samples and the associated P values are provided in Supplementary Appendix 5.2 and Supplementary Table 7. These results indicate that the relationships between voter turnout and PS for educational attainment and intelligence test performance, respectively, are highly statistically significant ($P < 1.61 \times 10^{-7}$ or lower) and that the gain in Nagelkerke pseudo r^2 ranges from 0.0041 (95% CI = 0.0016–0.0078) to 0.0317 (95% CI = 0.0235–0.0413) when one of the PSs was added to a baseline logistic model that had the first ten principal components of genetic ancestry as explanatory variables (Supplementary Appendix 5.2 and Supplementary Table 7). Although voter turnout is overall higher in the nationally representative sample, the predictive value of the PSs retains statistical significance (Fig. 2 and Supplementary Table 7). The consistency in predictive performance of the PSs in the two samples suggests that the overlap among SNPs associated with voter turnout and educational attainment or intelligence test performance is comparable in the general population and in the subpopulation of individuals with a psychiatric condition.

When interpreting the phenotypic processes producing these findings, it is relevant to consider previous studies that showed that SNPs associated with educational attainment may in part exert their influence through personality traits[78]. Social science studies have also found evidence of a relationship between the big five personality traits and political participation[12,30,79–81] (reviewed previously[82]). In particular, some studies suggest that personality factors can account for part of the correlation between genetics and political participation[5,10] and highlight extraversion as a factor that "may possibly mediate the relationship between genes and political predispositions that are known to be strongly related to political participation"[5].

Although we believe that it is probable that phenotypic personality constitutes part of the causal pathway from genetics to turnout, we cannot test whether the correlation between PSs for educational attainment and turnout is mediated by the phenotypic big five personality traits or phenotypic education, as these variables are not available in our data. However, further analyses reported in Supplementary Appendix 5.2 and Supplementary Tables 7 and 8 show that the PSs for educational attainment or intelligence test performance predict individual differences in turnout more consistently than the PSs for the big five personality traits. The PSs for the big five personality traits display mixed predictive utility for electoral turnout in which the gain in Nagelkerke pseudo r^2 ranges between 2×10^{-10} and 0.0014 when the PSs were added to a baseline logistic regression model that had the first ten principal components of genetic ancestry as explanatory variables, and P values for the statistical significance of the PSs for the big five personality traits range

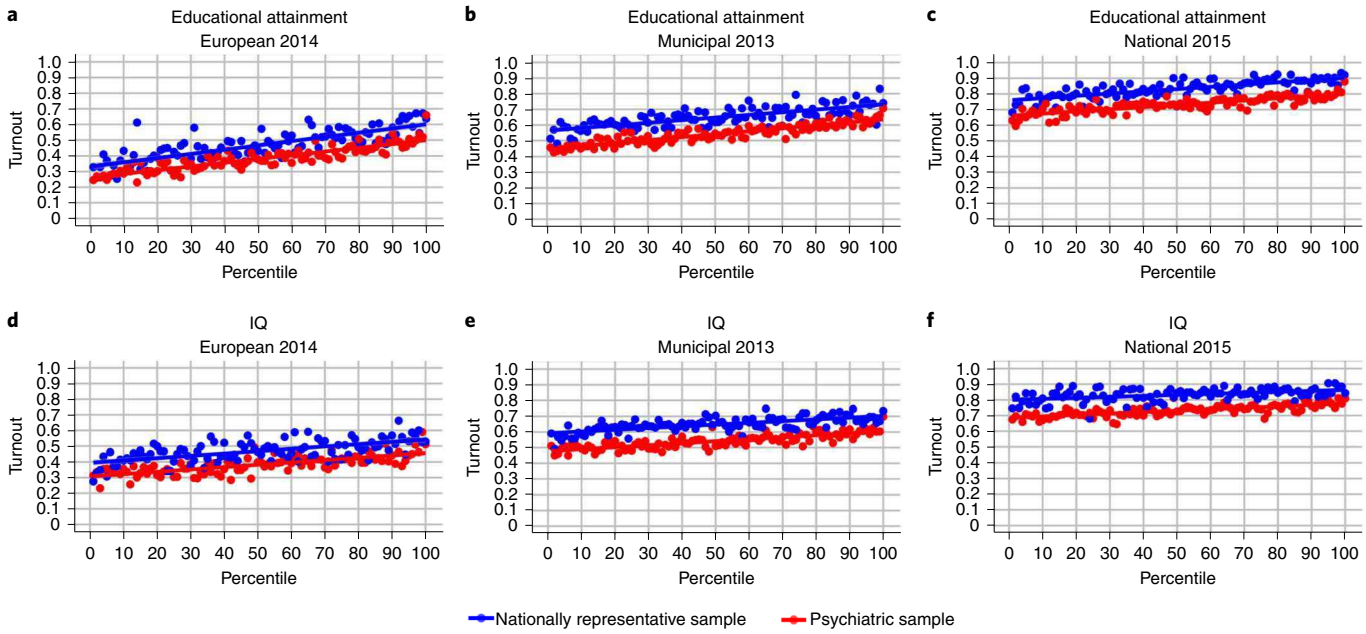

Fig. 2 | Prevalence of voter turnout by polygenic score percentiles. a–f, Voter turnout within each percentile of the distribution for the PSs for educational attainment (**a**–**c**) and intelligence test performance (**d**–**f**) for the European (**a**,**d**), municipal (**b**,**e**) and national elections (**c**,**f**).

between 0.9754 and 4.32×10^{-5} (Supplementary Appendix 5.2 and Supplementary Table 8). For example, across the three elections and our two samples, only in the 2014 European election in the psychiatric sample did we find that the PS for extraversion significantly accounts for variation in turnout. PS for openness to experience also has mixed predictive use and no significant predictive utility of conscientiousness were observed. Only the PS for agreeableness is consistently significantly associated with turnout across the three elections in the two samples. Note that these results could reflect the lower sample size of the dataset that was used to generate the genome-wide summary statistics for calculating the PS for the big five personality traits. Still, these findings support the more consistent predictive use of PS for educational attainment and intelligence test performance, respectively, compared to the PSs for the big five personality traits.

Genetic correlation analysis. Next, we investigated the genetic correlation between voter turnout and traits that are related to educational attainment and intelligence test performance. Furthermore, we analysed genetic correlations between voter turnout and socioeconomic factors as well as health indicators using linkage disequilibrium score regression (LDSC)[83] as implemented in LDHub, which makes it possible to assess genetic correlation with many traits using online tools[45]. Specifically, we used LDHub to test the correlation between 72 traits and voter turnout. Separating the correlations by election and sample, Fig. 3 shows the genetic correlations between voter turnout and non-voting traits for all traits with at least one significant correlation across elections, and sample after false-discovery rate (FDR) adjustment (the full list of genetic correlations and all estimates is provided in Supplementary Appendix 11).

In the psychiatric sample, we found positive genetic correlations for electoral turnout across all three elections with indicators of educational attainment (that is, college completion and years of schooling) and intelligence test performance (that is, childhood IQ). As expected, the correlations in the nationally representative sample follow the same pattern, albeit with higher P values and larger error bars, most likely due to the smaller sample size (an analyses of robustness is provided in Supplementary Appendix 7.1). None of the genetic correlations in Fig. 3 show any discernible difference between the two samples for any of the elections (Supplementary Appendix 7.2 and Supplementary Fig. 7).

Interestingly, negative socioeconomic factors—such as number of children, younger age at first childbirth and younger parental age at death—also show genetic correlations with voter turnout (Fig. 3). This suggests that, in addition to the PSs for education and intelligence test performance, a broad set of socioeconomic resources could connect genetics to electoral participation. Furthermore, Fig. 3 shows a negative correlation between voter turnout and adverse physical and mental health outcomes (for example, depressive symptoms, neuroticism and ADHD). The genetic correlations with depressive symptoms and ADHD symptoms underline the importance of these psychiatric conditions as predisposing factors for non-voting. Even among individuals for whom the additive sum of conferred risk does not cross the threshold to manifest as a psychiatric diagnosis, instances of significant correlation with voter turnout are identified for each of these traits.

Summary-statistics-based Mendelian randomization analysis. As the final test of our prediction, we performed a Mendelian-randomization-based analysis[84] of whether the genetic variance that predicts educational attainment and intelligence test performance, respectively, predicts voter turnout. Specifically, we used markers surpassing the threshold for genome-wide significance ($P \leq 5 \times 10^{-8}$) in association studies of educational attainment and intelligence test performance as instrumental variables. These genetic markers for educational attainment and intelligence test performance, respectively, are treated as exposures and voter turnout in each election as an outcome.

The results (Supplementary Appendix 8.2, Supplementary Table 12 and Supplementary Fig. 8) show that an individual in the nationally representative sample who has a genetic disposition to obtain education 1 s.d. higher than the population mean was predicted to be 2.66 (s.e. = 0.1064, $P = 3.76 \times 10^{-20}$) times more likely to vote in the municipal election in 2013, 3.14 (s.e. = 0.129, $P = 7.51 \times 10^{-19}$) times more likely to vote in the European Parliament election in

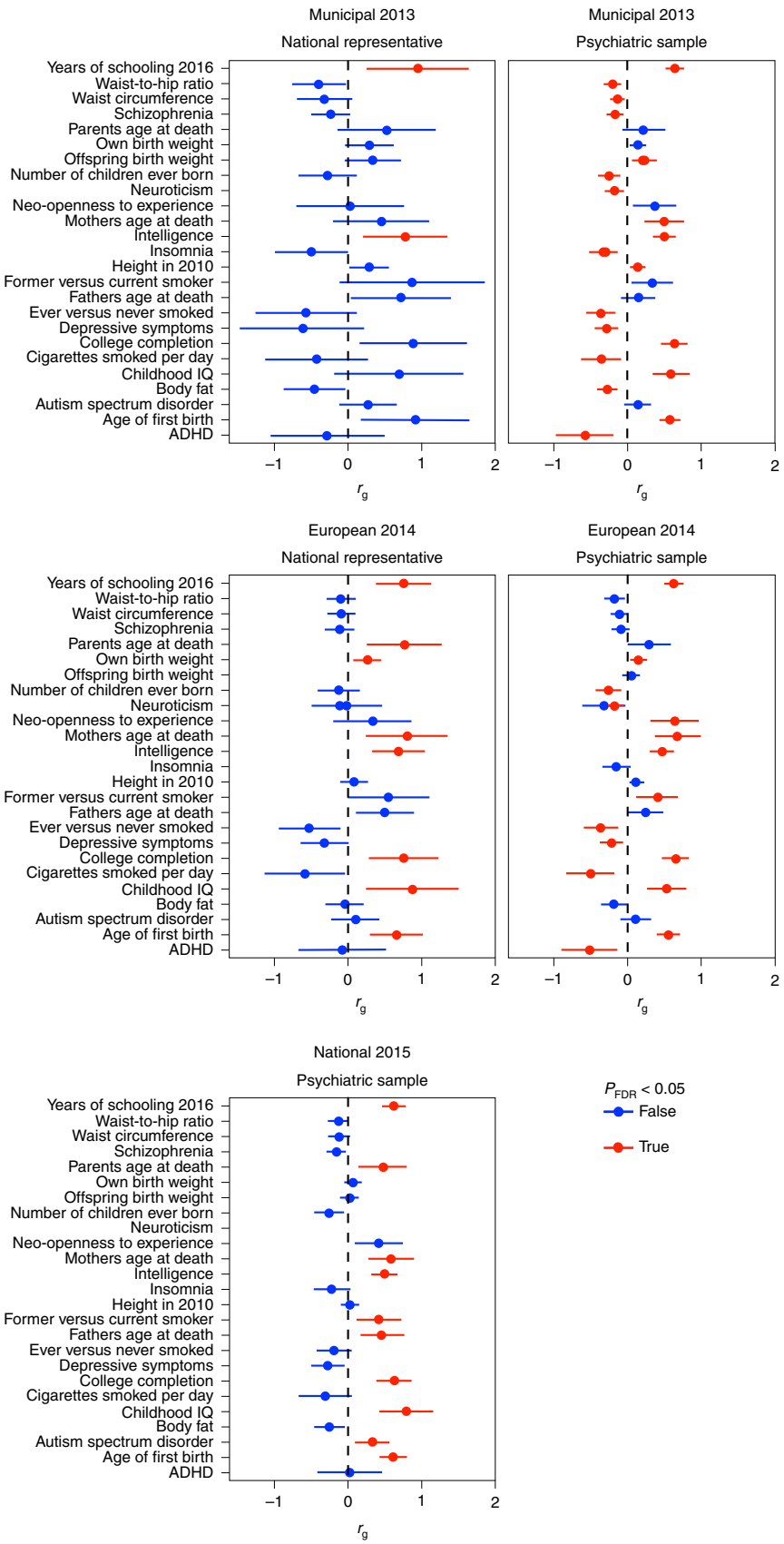

Fig. 3 | Genetic correlations between voter turnout and non-voting traits that show significant genetic correlation after FDR adjustment in at least one of the turnout phenotypes in the two samples by election. The LDSC-estimated heritability of turnout in the 2015 national election was not significantly different from zero in the nationally representative sample; therefore, no correlations were computed. Data points refer to point estimates for genetic correlations. Error bars incidcate 95% CIs.

2014 and 3.32 (s.e. = 0.1439, $P = 6.66 \times 10^{-17}$) times more likely to vote in the national election in 2015. An individual in the psychiatric sample with a genetic disposition to obtain education 1 s.d. higher than the mean in the psychiatric sample was predicted to be 2.97 (s.e. = 0.0866, $P = 3.44 \times 10^{-36}$) times more likely to vote in the 2014 European election, 2.65 (s.e. = 0.08, $P = 3.72 \times 10^{-34}$) times more likely to vote in the 2015 national election and 2.67 (s.e. = 0.0656, $P = 1.26 \times 10^{-50}$) times more likely to vote in the 2013 municipal election.

We observed a similar trend with intelligence test performance, although the magnitude of the effect size was smaller. An individual in the nationally representative sample with a genetic disposition to perform on intelligence tests 1 s.d. higher than the mean in the general population was predicted to be 1.85 (s.e. = 0.142, $P = 1.47 \times 10^{-5}$) times more likely to vote in the national election in 2015, 2.15 (s.e. = 0.1277, $P = 1.87 \times 10^{-9}$) times more likely to vote in the European Parliament election in 2014 and 1.77 times (s.e. = 0.1051, $P = 4.6 \times 10^{-8}$) more likely to vote in the municipal election in 2013. Similarly, an individual in the psychiatric sample with a genetic disposition to perform on intelligence tests 1 s.d. higher than the mean of this sample was predicted to be 1.78 (s.e. = 0.078902, $P = 2.58 \times 10^{-13}$) times more likely to vote in the national election in 2015, 2.07 (s.e. = 0.0858, $P = 1.19 \times 10^{-17}$) times more likely to vote in the European Parliament election in 2014 and 1.66 (s.e. = 0.0647, $P = 3.06 \times 10^{-15}$) times more likely to vote in the municipal election in 2013.

As a negative control, we repeated the analysis above using markers that surpass the genome-wide significance threshold for height obtained using summary statistics from the GIANT consortium as instrumental variables. The GSMR analysis of height did not suggest that there was any significant relationship between genetic dispositions towards height and turnout in any election across either of the samples (Supplementary Appendix 8.2 and Supplementary Table 12).

However, as our data are observational and Mendelian randomization analysis requires strong identifying assumptions and does not account for any environmental influences on the exposures of interest towards the outcome, the results should be seen as correlation evidence that PSs for educational attainment and intelligence test performance predict voter turnout (further discussion of our analysis and the assumptions for Mendelian randomization analyses are provided in Supplementary Appendix 9).

Conclusion

It is essential to the functioning and legitimacy of democracy that citizens vote in elections. Nonetheless, large inequalities in voter turnout exist. Although a decade of twin-based studies has shown that variability in voter turnout is shaped by genetics[5,7–9,13], the fundamental question of the mechanisms that underlie this connection largely remains a 'black box'[12]. Traditionally, social science models of voter turnout have "typically ignored genetic or biological factors"[85].

Here we followed recent studies[5,10] and sought to advance our understanding of how genes and voter turnout are linked by integrating the genetic and the social science paradigms of the microlevel foundations of voter turnout, arguing that the genetic variances that predict educational attainment and intelligence tests performance predict individual differences in voter turnout. To test this argument, we conducted a genome-wide complex trait analysis of the heritability of individual-level electoral participation using a nationally representative sample and a population sample of individuals diagnosed with anorexia, schizophrenia, affective disorder, bipolar affective disorder, autism and ADHD. Our methodological triangulation combining PSs, genetic correlations and Mendelian randomization analyses consistently supports that the genetic variants that predict educational attainment and intelligence test performance, respectively, also predict voter turnout.

While our findings provide evidence for the predicted genetic associations, we are unable to draw a causal interpretation. First, as we work with observational data and Mendelian randomization analysis requires strong identifying assumptions, our results are correlational. We cannot determine whether the observed genetic correlations reflect causal effects of phenotypic education and phenotypic intelligence test performance on voter turnout. For example, some research suggests that education works at least in part as a proxy for pre-adult factors, such as family socioeconomic status[86] (reviewed previously[28]), and recent research has found evidence of genetic nurturing in which genes influence educational attainment indirectly through the family environment[86].

Second, the resources for politics associated with, for example, educational achievement are multifaceted and include particular personality traits as well as material resources. We therefore find that it is probable that part of the relationship between genetic dispositions toward educational achievement or intelligence test performance could be mediated by phenotypic personality-related resources. Furthermore, as the PS for educational attainment is also predictive of performance on cognitive tests[75], part of the significant relationship between the PSs for educational attainment and turnout could be explained by the broader set of factors underlying such performance. Although our results are consistent with the argument that educational attainment and intelligence test performance represent a link between genes and voter turnout, respectively, they cannot provide conclusive evidence in support of such a causal interpretation.

The above limitations notwithstanding, our study contributes by moving beyond the limitations of the reared-together twin design to analyse the heritability of individual-level voter turnout using data on SNPs. Given the strong critiques raised in the social sciences of existing twin-based studies of the heritability of turnout[13,14], this is important as methodological triangulation helps to overcome some of the limitations of twin-based studies and increases internal validity. Considering the typical differences in the size of h^2_{SNP} estimates and twin-study estimates[87], the h^2_{SNP} estimates in the psychiatric sample are proportionally consistent with previous (American) twin-study estimates. Interestingly, we did not observe statistically significant h^2_{SNP} estimates in the nationally representative sample in the high- and medium-high-saliency elections in 2013 and 2015, respectively, but only in the low-saliency election for the European election in 2014. However, (1) the overlapping CIs with the psychiatric sample, (2) the power analysis and (3) the heritability estimates from the pedigree-defined familial relationships suggest that the non-significant h^2_{SNP} estimates for the high- and medium-high-saliency elections in the nationally representative sample in part reflect lower statistical power in this sample.

Importantly, the differences in heritability between elections and samples probably also reflect substantive contextual differences. First, in the nationally representative sample, we found higher heritability of turnout in the low-saliency European election compared with the high-saliency national election. Second, our results from the nationally representative sample suggest that the heritability for national and highly salient elections possibly could be lower in the general Danish population sample compared with previous estimates from United States; these findings are consistent with Tingsten's[73] law of dispersion, which stipulates that individual differences in resources for politics have stronger influence on whether people vote in elections with greater participatory costs as reflected in, for example, lower general turnout. Note that these results are based on data from a single country, that is, Denmark. Our results support the value of additional investigations of the differences in the heritability of turnout between elections with different costs of participation in different types of electoral systems to further explore the replicability and external validity of this finding.

Another path for further research is to study whether the heritability of turnout varies across psychiatric conditions. Our analysis focuses on average patterns for individuals with a psychiatric condition. However, certain types of psychiatric conditions may generate particularly high costs of voting. Drawing on the logic of Tingsten's law of dispersion[73], this could imply that these conditions increase the role of genetic dispositions.

Overall, the results extend understanding of the patterns that underlie inequalities in voter turnout. They show that the genetic variance that predicts educational attainment and intelligence test performance, respectively, also predicts individual differences in voter turnout. This integration of the social science and genetic paradigms of voter turnout reveals new linkages between social and political inequalities in the general population and in the subpopulations that are particularly vulnerable to political exclusion due to psychiatric diagnoses.

Methods

Data. The iPSYCH Danish case–cohort sample consists of two large samples of comprehensively genotyped individuals. A nationally representative sample ($n=30{,}000$) of individuals born in Denmark during 1981–2005; and a population sample of all individuals born during the same period with one of the following psychiatric diagnoses: schizophrenia, affective disorder, bipolar affective disorder, autism, ADHD and anorexia ($n=63{,}080$)[59].

The iPSYCH samples were merged with population-based records of whether Danish citizens voted in the 2015 national, 2014 European and 2013 municipal elections in Denmark. To maximize measurement validity, turnout data were collected directly from the voting files at the polling stations (Supplementary Appendix 1). We excluded individuals who show second degree or higher relatedness, individuals who were not from the genetically homogenous population, as identified by PCA analysis, and individuals who failed standard GWAS quality control tests for association tests. Finally, we limited the analysis to individuals who were aged 18 years or older and had therefore reached the Danish voting age to be eligible to vote in a public election in 2015 (Supplementary Appendix 1).

The iPSYCH has been approved by the Danish Scientific Ethics Committee, the Danish Health Data Authority, the Danish Data Protection Agency, Statistics Denmark and the Danish Neonatal Screening Biobank Steering Committee[59]. The iPSYCH is "an example of Danish studies based on the combination of registers and biological material stored in biobanks"[88]. On 28 August 2012, in accordance with the Act on Research Ethics Review of Health Research Projects (in Danish, Komitéloven), Section 10 (1), the Central Denmark Region Committees on Health Research Ethics, Committee II granted the iPSYCH exemption from obtaining informed consent from participants (https://ipsych.dk/en/data-security/health-research-and-ethical-approval/). The committee's "processing and approval of the iPSYCH project was in accordance with applicable law and practice at the time of notification." (https://ipsych.dk/en/data-security/health-research-and-ethical-approval/). This waiver also applies to our study. Participants for the iPSYCH project were recruited according to consent by non-opt-out from a national research program[89,90]. Participants "can opt out of having any biological material used for research without specific informed consent"[88]. It is possible for participants to opt out of the Biobank storage at any time (further description of the briefing and opt-out procedures[90] and discussion of the scientific ethical standards in iPSYCH[88] were reported previously). The iPSYCH has data protection measures in place that comply with Danish and European legislation[88].

Measures. *Voting.* Voting in each election was coded as 1 = did vote and 0 = did not vote. All analyses include individuals who were eligible to vote at the time of the election and were Danish citizens.

PSs for educational attainment and intelligence test performance. PSs for educational attainment and intelligence test performance were calculated using summary statistics from large recent studies of associations between genetic markers and educational attainment in 766,345 individuals (excluding the 23andme cohort)[75] and intelligence in 269,867 individuals of primarily European ethnicity[44] (details on the base datasets for the PSs and the target dataset are provided in Supplementary Appendix 5.1).

Previous research indicates that the predictive ability of educational attainment varies across samples[75]. Only limited available data exist regarding the accuracy of PSs for educational attainment in Danish samples, but previous research suggests that PSs for educational attainment predicted about 15% of the variance in educational attainment in a small sample of 1,459 Danish individuals[75]. In terms of predictive accuracy, this is comparable to the size of the general estimate of about 11–13%.

PSs for the big five personality traits. PSs for the big five personality traits were calculated using summary statistics from large recent studies of associations between genetic markers and the big five personality traits[91] using summary statistics from 175,375 adult individuals, obtained from the Genetics of Personality Consortium (http://www.tweelingenregister.org/GPC/) as described previously[91].

To construct the PSs, we used PRSice v.1.25 (https://choishingwan.github.io/PRSice/) with the default parameters (clump-r2 = 0.1, clump-kb = 250, clump-p = 1) to sum the products of the effect size of each individual marker with the associated additive genotype for every individual in the iPSYCH determined to be of European origin using principal component analysis. Summary statistics for all measures and demographic sample characteristics are provided in Supplementary Appendix 2 and Supplementary Tables 1–4.

Statistical analysis. All of the reported tests of statistical significance were performed in a two-tailed manner.

Following standard practices, all analyses focus on genetically homogenous (that is, broadly northern European) ancestry respondents (compare with ref. [76]). A detailed description of quality control, haplotype estimation and genotype imputation protocols undertaken on the iPSYCH dataset is provided in Supplementary Appendix 1.1.2. In brief, the iPSYCH cohort of 78,050 individuals were genotyped at 554,360 genomic loci in 23 waves. After initial quality control was performed on the basis of principal component analysis using common (minor allele frequency > 5%) high-quality markers common to the iPSYCH and the full 1000 Genomes phase 3 variant calls, followed by principal component analysis and outlier detection, 75,501 samples of a homogenous genetic origin were selected for SNP-level quality control. After excluding rare markers (minor allele frequency < 1%), those that failed tests for Hardy–Weinberg equilibrium within controls and markers with high missingness, a total of 246,539 SNPs were phased using SHAPEIT3 and imputed to 80,707,375 SNPs in 10 batches using IMPUTE2 and the full set of 1000 Genomes phase 3 haplotypes as the reference. Following stringent post-imputation quality control to exclude SNPs with low imputation INFO scores, rare SNPs (minor allele frequency < 0.001), SNPs with high missingness, differential missingness between cases and controls and SNPs showing significant association to a genotyping wave or imputation batch, 11,601,089 SNPs were retained. Sample-level quality control involved excluding samples with greater than second-degree relatedness, samples showing discordance from documented gender, duplicate samples, samples with abnormal heterozygosity not explained by admixture, samples with high missingness and samples that deviated from a homogenous genetic background as identified by ancestral birth records in conjunction with principal component analysis. This resulted in a total of 65,535 samples that were used for all complex trait analyses. All of the reported statistical tests were performed in a two-sided manner, except where explicitly indicated.

GCTA. We controlled for sex, age (measured in days after the election and centred to the mean) and age^2 (centred to the mean), as this is the functional form in relation to turnout in a young cohort[92] and for the first ten principal components of the SNP-based genetic relatedness matrix. The genetic relatedness matrix (GRM) for estimating the GCTA-GREML SNP heritability was computed using the standard SNP filters chosen for iPSYCH data release (MAF > 0.001, imputation INFO score > 0.2)[89] and we used a grm-cutoff of 0.05 as a threshold for relatedness between samples. We conducted an analysis of robustness in which we built a GRM from SNPs common between the HapMap3 dataset[93] and iPSYCH with a minor allele frequency of ≥0.01 and imputation INFO scores of ≥0.8 and a more stringent relatedness threshold with a grm-cutoff of 0.034. Although the point estimates of SNP heritability vary slightly, the significance of our results do not change (Supplementary Appendix 4.1)

We used the publicly available GCTA software for the analysis[70]. When reporting the h^2 estimates for voting in the 2013, 2014 and 2015 elections in Fig. 1, we reported h^2_{SNP} estimates on both the observed scale from our phenotypes and on a transformed liability scale, in which the estimates are scaled according to the percentage of voters in the nationally representative sample.

Analyses of differences in heritability across elections. To examine whether the SNP heritability of turnout in the European election of 2014 is significantly higher than in the national election of 2015 or in the municipal election of 2013 in both the nationally representative and psychiatric samples, we computed the z score of the difference $z = h^2_{\text{European election 2014}} - h^2_{\text{municipal election 2013}} \text{ OR } h^2_{\text{national election in 2015}} / \sqrt{(\text{s.e.}_{\text{European election 2014}} \times \text{s.e.}_{\text{European election 2014}} + \text{s.e.}_{\text{municipal election 2013}} \times \text{s.e.}_{\text{municipal election 2013}} \text{ OR } \text{s.e.}_{\text{national election 2015}} \times \text{s.e.}_{\text{national election 2015}})}$ where h^2 indicates the narrow sense SNP heritability of turnout in each election and s.e. indicates the standard error of the estimate obtained from GCTA-GREML. A one-sided P value was further computed as $P = \text{pnorm}(-1 \times \text{abs}(z))$ using R. This method has previously been used to test the gender-specific differences in heritability estimates[94].

Genetic correlations. To obtain summary statistics to upload to the LD Hub[45], we performed GWASs of turnout in both samples for each of the three elections. The GWASs were performed using PLINK, adjusting for age, gender and the first ten principal components of genetic ancestry. LDSC h^2 estimates are provided in the Supplementary Appendix and Supplementary Table 11, and Manhattan and Q–Q plots are provided in Supplementary Fig. 4a–f). The loci which showed

association ($P < 1 \times 10^{-6}$) with electoral turnout are reported in Supplementary Table 10 by election for each sample. We computed genetic correlations of voter turnout with 72 traits categorized as smoking behaviour, neurological diseases, personality traits, reproductive traits, sleeping, cognitive, anthropometric traits, education, psychiatric diagnoses and aging on the LDHub. Significance thresholds were adjusted to account for multiple testing using Benjamini–Hochberg FDR correction[87], implemented using R.

Nagelkerke pseudo r^2. The variance explained in election turnout was calculated as the gain in pseudo r^2 after adding PS as the explanatory variable to a baseline logistic regression model with the voter turnout as the outcome and the first ten principal components of genetic ancestry, estimated using flashPCA[95], as covariates. Pseudo r^2 was calculated using the NagelkerkeR2 function in the fmsb R package (https://cran.r-project.org/package=fmsb). The bias-adjusted CIs were estimated using the R package boot[96,97] and 35,000 bootstrap replicates. The pseudo r^2 values were transformed to a liability scale using the population prevalence of voter turnout, turnout within each election and cohort using the equations in Lee et al.[98] (Supplementary Appendix 5.2; an analyses of robustness is provided in Supplementary Appendix 5.3).

Mendelian randomization analyses. We used the GCTA[70] with the summary statistics from the GWAS of voter turnout and summary statistics from the latest GWAS for educational attainment, intelligence test performance and height available in the public domain. Individuals of European origin in the 1000 Genome phase 3 dataset were utilized to supply linkage disequilibrium information between the SNPs. We conducted HEIDI outlier heterogeneity tests (further information about the Mendelian randomization analysis and robustness checks is provided in Supplementary Appendix 8 and 9).

One of the methodological assumptions with summary-statistics-based GSMR analysis is that there is no sample overlap between the GWAS samples utilized for the exposure and outcome of interest. We made sure that the iPSYCH samples used to perform GWASs of voter turnout were not used in meta-analysis of any of the exposure traits.

Another assumption of GSMR is that the effect of the exposure on the outcome is linear, which might not always be true. While we use the HEIDI test to detect and exclude pleiotropic SNPs, the power to detect pleiotropy relies on the sample size of the GWAS from which summary statistics are obtained and the effect sizes of the pleiotropic SNPs[99]. The large disparity in the sample sizes of the exposure and outcome traits restricts the elimination of all pleiotropic SNPs in our analysis.

Data analysis was not performed in a blinded manner.

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

Owing to the consent structure of the iPSYCH and Danish law, the data cannot be shared publicly owing to its sensitive nature. The data can be accessed from secure servers[59]. For further information, please contact P.B.M. (pbm@econ.au.dk). For access to the data in Supplementary Table 5, please contact K.M.H. (kmh@ifs.ku.dk) as these register data also cannot be shared publicly.

Code availability

Code and scripts available from the corresponding author on request.

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Author contributions

L.A., E.A. and M.B.P. conceived the study. L.A., V.A., A.J.S., A.B., E.A. and M.B.P. designed the study. L.A., V.A. and A.J.S. drafted the manuscript. L.A., V.A., A.J.S., K.M.H., E.A., A.B. and M.B.P. discussed the results and revised the manuscript. V.A. and A.J.S. conducted all of the analyses except that K.M.H. conducted the analyses for Supplementary Table 5. K.M.H. collected the turnout data. P.B.M., M.N., A.D.B., D.M.H., T.W., O.M. and E.A. designed, implemented, and/or oversaw the collection and generation of the IPSYCH data. All of the authors (L.A., V.A., K.M.H., A.J.S., T.W., O.M., A.D.B., D.M.H., M.N., P.B.M., W.K.T., A.B., E.A. and M.B.P.) approved the final version of the manuscript.

Competing interests
The authors declare no competing interests.

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Software and code

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Data collection	No software was used
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Because of the consent structure of the iPSYCH and Danish law, the data cannot be shared publicly due to its sensitive nature. The data can be accessed via secure servers (ref. 40: 9). For further information, please contact Professor Preben Bo Mortensen, Scientific Director of iPSYCH, (pbm@econ.au.dk).

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Behavioural & social sciences study design

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Study description	Investigation of the genetic overlap between voter turnout and resources for politics, as indexed by educational attainment and intelligence test performance.
Research sample	We rely on the Danish iPSYCH case-cohort sample which consists of two large samples of comprehensively genotyped individuals: a nationally representative sample of individuals born in Denmark 1981–2005, and a population sample of all individuals born in the same period with one of the following psychiatric diagnoses: schizophrenia, mood disorders, bipolar affective disorder, autism, ADHD, and anorexia (see ref. 40 for details). The iPSYCH samples were merged with population-based records of whether Danish citizens voted in the 2015 national, 2014 European, and 2013 municipal elections in Denmark.
Sampling strategy	The nationally representative sample was collected using random sampling from the Danish CPR - where all people alive and living in Denmark are registered - of individuals born in Denmark 1981-2005. Using the CPR and the Danish Psychiatric Central Research Register a population sample of all individuals born 1981-2005 and diagnosed with schizophrenia, mood disorders, bipolar affective disorder, autism and attention-deficit/hyperactivity disorder was identified. These samples are state-of-the-field for single cohort GWAS studies, and constiitute the largest of its kind. The iPSYCH samples were merged with population-based records of whether Danish citizens voted in the 2015 national, 2014 European, and 2013 municipal elections in Denmark. We limit the analyze sample to individuals who were 18 years old or older - and hence had reach the Danish voting age to become eligible to vote in a public election in 2015.
Data collection	DNA was extracted from dried neonatal blood spots and amplified before genotyping on the Infinium PsychChip v1.0. Blood was collected between 4-7 days after birth and retrieved from the Danish Neonatal Screening Biobank within the Danish National Biobank. Psychiatric conditions were identified from national registers. Demographic and social variables were aggregated from national civil registers. Further details can be found in Ref. 40. Since 1997, the Danish Turnout Project has collected actual and validated turnout data from the 1,387 polling stations in Denmark. The turnout data covers about 70% of the population in each of the three elections. The municipality in which the individual polling station is location decides if voting files are made available to the Turnout Project, meaning that there is no individual selection. After the voting files were collected and verified, they were merged with individual social security number (CPR). See individual reports for each election, which describe this process in detail (refs. 2–4). In this case, the voting file with individual turnout data for the 2013 local, 2014 European, and 2015 national elections were merged into the iPSYCH data using the social security number (CPR).
Timing	All iPSYCH data was initially collected in 2012 and psychiatric diagnoses were later updated, complete through 2014. Turnout data were collected in 2103, 2014, and 2015 respectively (see above).
Data exclusions	Removed individuals who were not from a genetically homogeneous as population as identified by PCA analysis for association tests, heritability estimates. Removed individuals for whom voter turnout data was not available. We limit the analyze sample to individuals who were 18 years old or older - and hence had reach the Danish voting age to become eligible to vote in a public election in 2015.
Non-participation	N/A
Randomization	N/A

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Population characteristics	See above
Recruitment	Participants were recruited according to consent by non-opt out from a national research program (see also the methods section in the main text and Schork et al 2019 in Nature Neuroscience 22, https://doi.org/10.1038/s41593-018-0320-0, and Mortensen 2018 in Molecular Genetics, https://doi.org/10.1038/s41380-018-0296-x, and Nørgaard-Pedersen & Hougaard 2007 in Journal of Inherited Metabolic Disease: Official Journal of the Society for the Study of Inborn Errors of Metabolism.
Ethics oversight	The iPSYCH has been approved by the Danish Scientific Ethics Committee, the Danish Health Data Authority, the Danish Data Protection Agency, Statistics Denmark, and the Danish Neonatal Screening Biobank Steering Committee (see also the Methods section in the main text and Mortensen 2018 in Molecular Genetics, https://doi.org/10.1038/s41380-018-0296-x)

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