# The genetics of political participation: Leveraging polygenic indices to advance political behavior research

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**Abstract**

Previous research has found that political traits have some degree of genetic basis, but researchers have had less success unpacking the relationship between genes and political behavior. We propose an approach for examining the relationship between genetic predispositions and political variables that can overcome many of the limitations of previous research on the genetic underpinnings of political behavior: polygenic indices (PGIs). PGIs are DNA-based individual-level variables that capture the genetic propensity to exhibit a given trait. We begin by outlining how PGIs are derived, how they can be utilized in conventional regression-based research, and how results should be interpreted. We then provide proof of concept illustrating the fruitfulness of the PGI approach by examining the relationship between PGIs for psychological and health-related traits and various measures of political participation. Using data on over 50,000 individuals in four samples from the U.S. and Sweden, we find that PGIs for 10 different health and psychological traits significantly predict four measures of political participation. We also conduct within-family analyses, which suggest that a fair amount of the relationship between the molecular genetic markers and political participation is causal in origin. We conclude by outlining several ideas and providing empirical examples for researchers who may be interested in building on the PGI approach used in this paper.

## Introduction

"Why do people think and act politically in the manner they do?" With this fundamental question in political science, Alford, Funk and Hibbing opened their widely debated 2005 article in the *American Political Science Review*. The question is of course deceptively simple, but the answer provided by the authors was, at least at the time, surprising to large parts of the discipline. Alford, Funk and Hibbing (2005) showed that genetics play an important role in shaping political attitudes and ideologies among individuals. The attention the study garnered both inside and outside academia was instant and significant, and the article quickly became the most downloaded in *APSR* history and heralded as among "the most important articles the *APSR* has ever published" (Sigelman, 2006, 172).

In the wake of the Alford et al. study, a new subfield combining the lessons from behavioral genetics, psychology, and political science emerged. Up until that point, the idea that political predispositions were linked to biological factors had been more or less absent in studies on the determinants of political attitudes and behavior.

During the last decade and a half, researchers active within this subfield have produced a growing number of studies demonstrating that political traits are, to some degree, genetically heritable. The bulk of these studies are based on the classical twin design, in which differences in concordance between monozygotic (identical) and dizygotic (fraternal) twins are used to trace out the share of different traits that can be accounted for by genetic and environmental factors, respectively. Such studies have shown sizeable heritability estimates for political orientation and ideology (Alford, Funk and Hibbing, 2005; Bell, Schermer and Vernon, 2009; Hatemi et al., 2014; Oskarsson et al., 2015), strength of party identification (Hatemi et al., 2009; Settle, Dawes and Fowler, 2009), and political participation (Fowler, Baker and Dawes, 2008; Klemmensen et al., 2012; Dawes et al., 2014, 2015).[1]

All of the above-mentioned studies have been criticized due to the assumptions that are necessary to estimate the heritability component in the classical twin design (see, e.g., Beckwith and Morris 2008; Charney 2008). Above all, the equal environments assumption, stating that identical and fraternal twin pairs experience equivalent trait-relevant environments, has been questioned. In response to this, a few studies have utilized alternative research designs, such as using extended families (Hatemi et al., 2010; Kornadt et al., 2018; Kandler, Bleidorn and Reimann, 2012), adoptees (Cesarini, Johannesson and Oskarsson, 2014; Oskarsson, Dawes and Lindgren, 2018; Oskarsson et al., 2022), and molecular genetic data (Benjamin et al., 2012) to study the genetic heritability of both political orientations and political participation and their findings largely corroborate those from twin studies. Thus, there is substantial evidence that political attitudes and behavior are partly genetically transmitted.

Despite providing a substantial challenge to conventional models of the formation of political attitudes and behavior, the findings from this subfield have yet to be fully integrated into mainstream political science research. One reason for this may be that while studies using different samples and measures consistently find that a moderate to large share of the variance in political traits can be accounted for by genetic factors, researchers have had less success in explaining *how* genetic factors are related to political attitudes and behavior. For instance, some studies have tested whether specific genes, so called "candidate genes," are associated with political traits (Fowler and Dawes, 2008; Dawes and Fowler, 2009; Settle et al., 2010; Fowler and Dawes, 2013; Deppe et al., 2013) in the hope that their functions can help reveal how they may influence political traits. However, the growing realization that most, if not all, complex human traits are influenced by a very large set of genetic variants, each with very small effect sizes (Chabris et al., 2015), has largely put a halt to such efforts (Charney and English, 2012; Fowler and

[1] Reviews of recent research on the genetic basis of political behaviors and attitudes have been conducted by Hatemi and McDermott (2016), Ksiazkiewicz and Friesen (2017), and Dawes and Weinschenk (2020).

Dawes, 2013; Deppe et al., 2013). Some researchers have employed multivariate twin models to examine the extent to which psychological traits can be traced back to the same genetic sources as political traits (Dawes et al., 2014, 2015; Weinschenk and Dawes, 2017; Weinschenk et al., 2019). Here again, though, researchers have encountered important limitations. For instance, it has been difficult to demonstrate that psychological traits are causal mediators linking genes and political attitudes and behavior (see Verhulst, Eaves and Hatemi 2012; Dawes et al. 2014; Rasmussen, Ludeke and Hjelmborg 2019). Thus, despite the initial promise of work on the genetic underpinnings of political traits, the recent output of empirical work has slowed considerably.

Another reason for the lack of integration is likely due to structural limitations related to research on the genetic basis of political traits. For example, the type of data used in these studies, i.e., samples of twins, adoptees, or genotyped individuals including information on relevant political traits, has been somewhat limited. Moreover, it is often necessary to collaborate across disciplinary lines in order to access relevant data. Further, to analyze such data, learning and mastering new and oftentimes unfamiliar methodological skills is often required.

Finally, it is also important to note that researchers in the subfield, including the authors of this paper, have largely failed, thus far, in clarifying the relevance of their findings for the larger political science community in a convincing way. A common reaction when presenting research on the genetic basis of political traits is one of polite skepticism. On the one hand, most political behavior researchers admit that the evidence that genetics influence political attitudes and behaviors seems quite strong. On the other hand, fewer are convinced that this research adds to our understanding of politics or that the potential scientific payoff is worth the effort. Put differently, as social scientists we should study social causes and since genetic differences per definition are not social causes, genes are somebody else's problem.

We argue that this pragmatic partitioning between social and genetic causes is at best untenable and at worst harmful for our understanding of social phenomena. Our point of departure is instead that any human behavior is the product of the presumably very complex interplay between genetic and past and current environmental factors and that failing to account for either runs the risk of rendering our conclusions about the factors we *do* consider flat out wrong. To reach a better understanding of the causes (be those social or genetic) of individual differences in political behavior, we must take this interplay into account. Against this background, our study has two main aims. First, we introduce a new approach for examining the relationship between genetic predispositions and political traits that can overcome many of the limitations of the previous lines of research discussed above. In short, we argue that the integration of polygenic indices (PGIs) into conventional empirical models provides a way forward. As a quick overview, a PGI for a given trait is a DNA-based predictor of the trait, calculated as a weighted sum of an individual's relevant genetic variants (Becker et al., 2021). Thus, rather than focusing on the relationship between political participation and assumed genetic similarity (as in traditional twin or

adoption studies), or a handful of specific genes (as in the candidate gene paradigm), the approach we advocate for makes use of an individual-level measure of genetic propensity, summarizing the effect of a large number of variants, that can be used in empirical frameworks already part of the existing political science toolkit. Furthermore, the dramatically decreasing costs for genotyping individuals has led to a rapid expansion in the number of datasets that contain the necessary type of data.

Our second aim is to provide a proof of concept illustrating the fruitfulness of the PGI approach. More concretely, this study serves as an example of how PGIs can be used to improve our understanding of a set of well-known and strong empirical relationships between, on the one hand, psychological and health-related traits, and, on the other, different measures of political participation. Political scientists have been increasingly interested in the link between health and psychological traits and political participation. However, determining whether there is an underlying causal effect of these traits on political outcomes is challenging. Above all, any psychological or health trait might be correlated with observable and unobservable characteristics, including genetic and environmental factors, that independently affect outcomes.[2] Moreover, we can not rule out the risk of reverse causality such that the psychological and health-related traits are partially determined by the act of political engagement. Instead of using contemporary trait measures, we suggest a different approach. We use PGIs for the traits of interest - that is, indices of genetic variants linked to different psychological and health traits - and study how these predispositions affect political participation. There are two distinct advantages with this approach. First, genes are fixed over the life cycle, precluding reverse causality issues. Second, we make use of the fact that our data includes a large share of full siblings. Since genetic differences (and therefore differences in PGIs) between full siblings are random in accordance with Mendel's first law, a within-family PGI effect on an outcome can be given a causal interpretation.

To study these relationships, we make use of data from four samples across two national contexts (the U.S. and Sweden). In addition to different measures of political participation such as self-reported and validated voter turnout and engagement in various other political acts, these samples include ten PGIs that capture the genetic propensity for traits related to health (e.g., depression and physical activity) and various psychological attributes (e.g., cognitive ability and risk tolerance). To preview our findings, we demonstrate that the ten PGIs constructed to capture genetic variation in different health-related and psychological traits significantly predict our four measures of political participation. Moreover, we report within-family analysis estimates suggesting that the psychological and health trait-linked genes captured by the ten PGIs exert significant causal effects on political participation.

The rest of this paper proceeds in the following manner. In the next section, we provide an overview of some of the important technicalities and interpretational issues around polygenic indices, which are

[2]Many studies in these areas have highlighted the genetic underpinnings of health and psychological traits. For example, Mondak et al. (2010) note that "because personality is substantially rooted in biology, any effects of personality on political behavior likely signal the mediated influence of biology" (106). Similarly, Pacheco and Fletcher (2015) point out that "health may also be a potential mechanism by which genetic factors influence political behavior as health is inherited" (113).

likely to be new to many social and political scientists. We then provide an overview of how the 10 traits of interest in this paper have previously been related to political engagement. To be clear, previous research on the relationship between health and psychological traits and political participation has not focused on measuring genes related to these traits. Next, we describe our empirical framework, data, and measures. We note that a pre-analysis plan for this paper was registered prior to us getting access to the data or conducting any analyses.[3] After we present the results of these analyses and discuss the findings, we conclude by providing a host of suggestions and empirical examples for researchers who may be interested in using and building on the framework discussed in this paper. The approach employed here can be used to study a wide range of relationships of interest to political behavior scholars and may be used in a variety of other interesting ways. For example, we discuss and provide some simple illustrative examples on how PGIs can be used to further political socialization research and to study how the effects of certain interventions vary by individual genetic endowments, or so-called gene-environment interactions.

## Polygenic Indices

As a first step, we need to establish a basic understanding of PGIs, how they are constructed, and how results from analyses where they are used should be interpreted. In order to accomplish this, it is also necessary to delve very briefly into human genetics.

The human genome, which is packed into the nucleus of each of our cells, consists of several long strings (of deoxyribonucleic acid, or *DNA*), with a double helix structure, consisting of pairs (*base pairs*) of *nucleotides*. Each string is called a chromosome, and every cell holds 23 pairs of these chromosomes, such that there are two versions of each - one inherited from the mother and one from the father. At each position (*locus*) on a chromosome, the nucleotide base pair will be one of two types (*alleles*): adenine paired with thymine (i.e. AT), or cytosine paired with guanine (CG). For the vast majority of the loci across the genome (approximately 99.9%), humans are identical. The remaining loci, therefore, make up all of our genetic variation. Base pairs that differ between individuals are called single nucleotide polymorphisms (henceforth SNPs, pronounced 'snips').[4] The least/most common allele at a certain locus (that is, AT or CG) in a population is referred to as the minor/major allele. In effect, each position on the genome can have three different values: it will have the minor allele on neither of the two chromosomes in a pair (0), on one of the chromosomes (1), or on both (2), meaning that a locus can come in three different versions. Longer strings of base pairs (from a few hundred to a few million in a row) are what

[3]This paper is the composite of two separate pre-analysis plans. These were posted on the OSF website on April 14, 2021 and are available at the following links: `https://osf.io/gnv9t/`, and `https://osf.io/vrd75/`. Any deviations from, or additions to, the pre-analysis plan that were deemed necessary and/or helpful are mentioned throughout.

[4]While the focus here is on SNPs, there are other types of variation as well – such as insertions or deletions of particular segments of the genome, or copy-number repeats, where specific segments of the genome are repeated varying numbers of times.

forms a *gene*, i.e. the instructions for the cell to produce a particular protein. When a gene contains SNPs, i.e. base pairs that differ between people, there are, in effect, different versions of the gene, that may result in slightly different versions of the protein.

A PGI is an individual level measure that is intended to summarize at least part of an individual's genetic endowment that may predispose them to have a particular trait (which we will call the *target* trait). In order to be able to construct a PGI for a target trait, we first need to know which SNPs should be included, and how strongly each of these SNPs are associated with our target trait. This information comes from a so-called genome-wide association study (henceforth GWAS) in which researchers separately regress an outcome (or phenotype) of interest on the number of alleles (0, 1 or 2) subjects possess for each genotyped SNP (typically in the hundreds of thousands to millions) using a very large sample. A SNP is declared to be "genome-wide significant" if the p-value associated with its effect on the outcome is below a stringent threshold, adjusted for multiple testing, which is typically $p < 5 \times 10^{-8}$. As an example, a recent GWAS of educational attainment based on more than 3 million individuals found 3,952 SNPs that were significantly associated with years of education (Okbay et al., 2022).

The summary information from the GWAS – the estimated effect of each genotyped SNP on the outcome – is then used to construct a PGI in a new sample. The PGI is simply the coefficient-weighted sum of the number of alleles an individual has for each SNP analyzed in the GWAS. More formally, the PGI for individual $i$ is defined as

$$\text{PGI}_i = \sum_{k=1}^{K} \hat{\beta}_k X_{ki} \tag{1}$$

where $\hat{\beta}_k$ is the coefficient for SNP $k$, as estimated in the discovery sample in the GWAS, and $X_{ki}$ is the number of alleles (0, 1 or 2) that individual $i$ has for the same SNP. Typically, the resulting index will then be standardized to have mean zero and standard deviation one.

The final PGI can now be used, much like any individual level quantitative variable, in a regression framework as a measure of the individual's genetic propensity for the trait in question. However, the results of this regression must be carefully interpreted and it is important to be aware of what PGIs do and do not capture, as well as under what circumstances the resulting estimates can be interpreted as causal genetic effects.

When used in a cross-sectional design, the PGI may capture not only the causal genetic effects, but also remaining population structure and genetic nurture. By population structure we mean the confounding that stems from certain alleles simply being more common in segments of a population that, for entirely unrelated reasons, differ on average on the outcome of interest. That is, if a population is stratified into groups that are partially "separated" (i.e. not mating randomly across groups) due to social, cultural or natural barriers, and the outcome in question is more prevalent in one of these strata for non-genetic reasons, a GWAS would suggest that SNPs that happen to be more common in that group

are correlated with the outcome. The problem of population stratification in cross-sectional designs can be somewhat ameliorated by the standard practice of restricting the analyses to people who have similar genetic ancestry and including the top principal components of the genetic-relatedness matrix that capture broad patterns of genetic similarity produced by human demographic history (Price et al., 2006). However, converging evidence has shown that some amount of population structure confounding likely still remains even after controlling for these principal components (e.g. Selzam et al., 2019).

Genetic nurture, on the other hand, refers to the phenomenon where a genetic factor shared between children and their parents, siblings, or other relatives due to inheritance, is actually related to a parenting behavior. Thus, we could observe a correlation between a genetic marker and an outcome simply because that marker was also present in the parents, and caused the parents to behave in a certain way. For example, Kong et al. (2018) demonstrate that a PGI for educational attainment based on parental SNPs that were *not* transmitted to the children was significantly associated with offspring education even after controlling for the offspring's own educational attainment PGI. Such an association could well be environmental in nature (i.e. non-transmitted parental SNPs may influence parental behaviors that have downstream effects on offspring traits).

Both of these issues (population stratification and genetic nurture effects), however, will be substantially reduced, if not completely removed, if one has access to data for more than one sibling. Including family fixed effects in a regression analysis based on a sample of biologically related siblings is tantamount to holding constant the parental genotypes and only using the random genetic variation between the siblings. Thus, within-family estimates of the PGI effects are not confounded by genetic nurture effects and will eliminate population stratification since individuals sharing the same parents are by definition in the same genetic population strata. Thus, since differences between siblings in a PGI are random, downstream effects are credibly causal. Therefore, we present PGI coefficient estimates based on both between- and within-family models.

Several remaining caveats must be taken into account, however. The first thing to keep in mind is that genetic effects are causal in the distal counterfactual sense: changing the genotype will *ultimately* lead to a change in a trait. However, they do not at all imply a proximal biologically deterministic interpretation of the etiology of the trait. In fact, it is completely possible that most or all of the genetic effect is transmitted through entirely environmental pathways. Consider, for example, if a PGI for height was also found to be associated with political participation. This could conceivably be partially explained by a social context characterized by wage discrimination based on height, which then has downstream effects on political participation. Thus, the chain of causation from genetic variation to a social outcome will be long and complex.

Second, if a PGI for a given target trait is causally related to some outcome, this does not imply that the genetic effect on the outcome is exclusively transmitted via the given target trait. Since genes may affect multiple traits, a phenomenon known as pleiotropy, the mechanisms linking a PGI and an outcome

could involve a large number of traits that are *genetically related* to the target trait. For example, in the results section we show that a PGI for the Big Five personality trait neuroticism has a negative effect on voter turnout. As we show in the Appendix (Table A.12) it is highly likely that part of this effect is mediated via the corresponding neuroticism trait. However, extant research reports that the neuroticism PGI is linked to traits other than neuroticism, such as educational attainment, that we know are related to voter turnout (Selzam et al., 2019).

Third, a PGI is unlikely to capture all of the genetic influences on a trait. The precision of estimated genetic effects in a GWAS, which are used as weights in the construction of a PGI, depends crucially on the size of the sample being analyzed. Since social traits are highly polygenic (i.e., many variants with very small effect sizes), even with very large samples these effects are estimated with some level of noise. It is therefore not accurate say that the inclusion of a PGI can completely control for the effect of genetics in any design. However, since they do capture *some* genetic effects, they can be used to increase power and therefore precision even in studies not explicitly investigating genetic effects per se. Additionally, it is important to keep in mind that the noise in the PGI is essentially classical measurement error between families, meaning that unadjusted effect size estimates are going to be artificially small due to attenuation bias (Becker et al., 2021).

Lastly, in line with the first point above, genetic effects may depend on the environmental context in which they are expressed. As such, a PGI will capture the average treatment effect. Since we investigate main effects in this study, it is entirely possible that a null or weak finding is the result of effect sizes of different magnitudes or opposing signs in different segments of a sample.

## Political Participation

As discussed in the introductory section, our goal in this paper is to illustrate the usefulness of the PGI approach. To do so, we focus on the relationship between numerous PGIs and political participation. Understanding the underpinnings of political participation is, for good reason, a central preoccupation among political scientists. The idea behind the fundamental democratic right of "one person, one vote" is that every citizen's political preferences should carry equal weight when society makes joint decisions. At the same time, we know from earlier research that political participation, and therefore political power, is unequally distributed. Some people vote and participate in other modes of political activity more often than others and therefore increase the likelihood that their wishes are heard by political leaders (Lijphart, 1997). Given the importance of political engagement for representation (Griffin and Newman, 2005; Hajnal and Trounstine, 2005), it is critical to solve the puzzle of why some people are more participatory than others. In short, a better understanding of the causes of political participation is a precondition for creating a more equal society (Campbell, 2013).

## Health and Political Participation

Although we know a great deal about the determinants of civic engagement, in the past decade or so researchers have started to consider the relationship between a new set of factors—measures of health—and political participation. Thus far, researchers have examined the link between a number of different measures of health, some of which are more general in nature (e.g., self-rated health and subjective well-being) and some of which capture more specific dimensions of health (e.g., depression/depressive symptoms and physical ability), and political participation.

Within the literature on health and political participation, self-rated health (SRH) has been one of the most commonly used measures of health (Mattila et al., 2018), thanks to its simplicity and good measurement characteristics (e.g. Wuorela et al., 2020). Most research in this area has focused on voter turnout, showing that people who report being in poor health are less likely to vote in elections than those who are in good health (Pacheco and Fletcher, 2015; Mattila et al., 2013; Söderlund and Rapeli, 2015; Stockemer and Rapp, 2019; Rapeli, Mattila and Papageorgiou, 2020; Burden et al., 2017; Engelman et al., 2021). This finding is consistent with the resource theory of participation (Brady, Verba and Schlozman, 1995), that health is a politically-relevant resource and being in poor health creates barriers that make turning out to vote more arduous.

Researchers have also examined the relationship between SRH and non-voting measures of participation. Here, the findings are somewhat more mixed than they are for voter turnout with reports of positive (Mattila et al., 2018), negative (Söderlund and Rapeli, 2015) and null (Burden et al., 2017; Stockemer and Rapp, 2019; Adman, 2020) relationships. Interestingly, Mattila (2020) finds a more nuanced relationship between health a political participation—poor health increases the odds of non-institutional participation (e.g., boycotting, signing petitions), while good health increases the odds of traditional institutional participation (e.g., voting, contacting government).

It is worth noting that while SRH captures physical, mental, and social factors, it has the most predictive power when it comes to physical health (Mavaddat et al., 2011). In short, measures of SRH are closely associated with the ability to perform physical functions. Recently, some research has emerged on the link between specific measures of physical functioning and/or activity and political participation. Burden et al. (2017), for example, find that walking speed, an indicator of broader physical functioning, has a limited effect on voter turnout and no effect on donating to political campaigns. In a follow-up analysis, Engelman et al. (2021) find that slow walking speed is associated with lower turnout and that this relationship is especially pronounced among the least wealthy.

Researchers have also examined the connection between subjective well-being (SWB) and political participation. Measures of SWB (also sometimes referred to as life-satisfaction or happiness) attempt to capture how well people think their lives are going. We consider this a health measure because, according to the World Health Organization, "health is a state of complete physical, mental and social *well-being* and not merely the absence of disease or infirmity."

Existing studies generally indicate that there is a positive relationship between subjective well-being and participation (Weitz-Shapiro and Winters, 2011; Flavin and Keane, 2012), with the idea being that once people reach a certain level of satisfaction with their own lives, they may then begin to look beyond themselves and try to address broader concerns by getting more involved in the political process.

Finally, some research has explored the link between more specific health measures and participation. A relatively new but growing line of research has examined the association between depressive symptoms and political behavior.[5] Similar to work on SRH, the bulk of existing research on depression and engagement has focused on voter turnout. Most studies find that turnout decreases as the severity of depressive symptoms increases (Ojeda, 2015; Ojeda and Pacheco, 2019; Landwehr and Ojeda, 2021; Ojeda and Slaughter, 2019; Engelman et al., 2021). Similar findings have emerged for non-voting measures as well (Ojeda, 2015; Landwehr and Ojeda, 2021). Overall, the negative relationship between depression and measures of political engagement fits well with the resource theory of participation—depression reduces the resources available to participate (e.g., motivation and physical energy).

## Psychological Traits and Political Participation

Over the past fifteen years, interest in the relationship between psychological traits and participation has grown. Here, we focus on both cognitive (e.g., intellectual effort such as thinking or reasoning) and noncognitive (e.g., personality traits, self-control, and socioemotional skills) traits.

Turning first to cognitive traits, scholars have devoted increasing attention to individual differences in cognitive ability. Overall, studies on cognitive ability and participation consistently find that as cognitive ability increases, the likelihood of voting and participating in other types of political activities increases (Deary, Batty and Gale, 2008; Denny and Doyle, 2008; Dawes et al., 2014; Engelman et al., 2021). Again, this pattern aligns with the resource theory of participation. In short, acts of political participation typically require people to acquire and process information. If these tasks are easier for those who have high levels of cognitive ability, then they should be more inclined to participate than their counterparts.

There has also been growing interest in the relationship between noncognitive factors and participation. Here, we focus on five noncognitive traits: extraversion, neuroticism, adventureness, risk tolerance, and chronotype. Both extraversion and neuroticism are traits within the Big Five model of personality. In most studies, extraversion is positively related to political participation (Mondak, 2010; Gerber et al., 2011; Dawes et al., 2014; Lindell and Strandberg, 2018).[6] In general, the idea is that extraverted people tend to be more inclined to participate in politics than their introverted counterparts because politics is a place where they can express many of the characteristics they exhibit (e.g., sociable, assertive). For

[5]Depression has been measured in a variety of ways. In general, it is assessed by asking respondents a series of questions related to their mood, such as "In the past 30 days, how often did you feel: So sad nothing could cheer you up?"

[6]In a recent meta-analysis based on 10 different datasets, Vitriol, Larsen and Ludeke (2020) found that the average correlation between extraversion and participation is positive and statistically significant.

neuroticism and participation, findings are a bit more mixed. Initial studies found divergent effects (e.g., Gerber et al. 2011 found a positive relationship between neuroticism and participation, while Mondak 2010 found a negative association), but a recent meta-analysis based on 10 different samples shows that the average correlation between neuroticism and participation is negative and significant (Vitriol, Larsen and Ludeke, 2020).

We are also interested in adventurousness, which refers to a preference for novel and intense experiences. Although there has not been a great deal of work on this particular trait and political engagement, adventurousness is a subfacet of the Big Five trait openness (Soto and John, 2012). Overall, research on openness has shown that this trait is positively related to measures of political participation (Gerber et al., 2010; Mondak, 2010; Vitriol, Larsen and Ludeke, 2020). People high in openness tend to like encountering new ideas and are open to different perspectives, which both have a natural connection to politics.

Beyond the Big Five and related personality traits, some research has examined the link between risk preferences and participation. Kam (2012) finds that risk accepting people are more likely than their counterparts to engage in a wide range of political acts. In part, the relationship is due to the fact that risk-accepting individuals are highly motivated by novelty and excitement, which many political activities provide. Oosterhoff and Wray-Lake (2020) find similar results using data on over 100,000 high school students. Those with higher risk preferences were more likely than their counterparts to vote, donate, write to government officials, boycott, and protest. The basic idea is that political participation represents a novel behavior that entails potential rewards but also many potential risks (e.g., failure to influence government, negative appraisals by peers).

The final trait we consider is chronotype, that is, whether an individual is a morning person (those who prefer going to bed and waking earlier) or evening person (those who prefer a later bedtime and later rising time). While there has been limited research on the consequences of chronotype for political behavior, research in biology and psychology has found some evidence that morningness is associated with prosocial behavior and empathy (Lange and Randler, 2011; Zoe, Depow and Inzlicht, 2021). Given the idea that many political activities are prosocial in nature (Panagopoulos, 2010), the positive relationship that has been observed between morningness and pro-sociality may extend to political engagement. Interestingly, research by Vollmer and Randler (2012) has shown that morning types are more accepting of social values (versus individual values) than evening types, in part because they have fewer problems synchronizing with the daily social schedule. The notion that morning types are more oriented towards social values may increase their propensity to participate in social and political activities. Only one study of which we are aware has examined the association between morningness and participation. Ksiazkiewicz and Erol (2022) recently reported that morning chronotype is positively associated with voter turnout.

# Data and Samples

We use data from four different samples to examine the relationship between the ten psychological and health trait PGIs and four measures of political participation: the Minnesota Twin Family Study, the National Longitudinal Study of Adolescent to Adult Health, the Wisconsin Longitudinal Study, and the Swedish Twin Registry.

The Minnesota Twin and Family Study (MTFS) is a population-based multi-wave longitudinal study of same-sex twins and their parents from the Upper Midwest and is collected by the Minnesota Center for Twin and Family Research (MCTFR) (Iacono, McGue and Krueger, 2006). The MTFS twin sample is comprised of two age cohorts, one in which subjects were 11 years old at the time of their initial assessment and the other in which subjects were 17 years old. The younger cohort was born between 1977-1994 and the older cohort was born between 1972-1979.

The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a multi-wave longitudinal study of a nationally representative sample of adolescents in grades 7-12 in the United States, during the 1994-95 school year (Harris et al., 2013). In Wave I of the Add Health study, researchers created a sample of sibling pairs based on a screening of a sample of 90,118 adolescents. These pairs include all adolescents that were identified as twin pairs, full-siblings, half-siblings, or unrelated siblings raised together.

The Wisconsin Longitudinal Study (WLS) is a long-term multi-wave longitudinal study of a random sample of 10,317 men and women who graduated from Wisconsin high schools in 1957 (Herd, Carr and Roan, 2014). Survey data was collected from the original respondents in several waves between between 1957 and 2011 and from a selected sibling between 1977 and 2011.

The Swedish Twin Registry (STR) began in the 1950s and contains nearly all twins born in Sweden since 1886. The total sample contains more than 170,000 twins (Lichtenstein et al., 2007).

## Political Participation Measures

We focus on four different measures of political participation: self-rated voter turnout (only available in Add Health and MCTFR), validated voter turnout in high-stakes elections (presidential elections and elections to the national parliament; not available in Add Health), validated voter turnout in low-stakes elections (midterm elections and elections to the European Parliament; not available in Add Health), and a political participation index (only available in Add Health and STR). Table A.1 in the Appendix lists the question wordings for all items used to construct the outcomes in each of the four samples.

In some of the samples, we have access to information on the outcomes (e.g., self-rated voter turnout, political participation) from multiple survey waves. In those cases, we use the latest available observation for each individual. Likewise, in some of the samples we have information on validated voter turnout from multiple elections. In those cases, the outcome for each individual will be measured as average

turnout across all elections for which we have data. All outcome measures are recoded to the 0–1 range.

### Polygenic Indices

We obtain the PGIs used in the analyses from a recently established repository of polygenic indices (Becker et al., 2021). The repository is intended as a resource for researchers who wish to include genetic data in their studies. The repository contains PGIs for 47 different traits in 11 samples constructed using a consistent methodology. In the Appendix, we provide a description on how to get access to the repository samples of greatest interest to the political science community.

We restrict the repository data in several ways. First, we only use data from samples that include a large number of sibling pairs, information on relevant political outcomes, and measures of the PGIs of interest. This leaves us with the four samples included in the this study: Add Health, WLS, MCTFR and STR. Second, we only include PGIs with an estimated capacity to predict the target trait of 2% or more (see Table A.2 in the Appendix). In addition to the 10 health and psychological PGIs, we use the PGI for educational attainment in each sample as a benchmark against which the effects of the other 10 PGIs of interest can be compared. PGIs for educational attainment have a moderate level of predictive power (Becker et al., 2021) and two studies have recently established that PGIs for education are related to voter turnout (Dawes et al., 2021; Aarøe et al., 2021).

In total, we have four PGIs for health traits (depressive symptoms, physical activity, self-rated health, and subjective well-being) and six PGIs for psychological traits (cognitive performance, adventurousness, extraversion, chronotype/morning person, neuroticism, and risk tolerance). We standardize all PGIs (within each sample and birth-year) to have a mean equal to 0 and a standard deviation equal to 1.

Table 1 below displays summary statistics for the four samples and Table A.4 in the Appendix presents bivariate correlations between all PGIs used in this study.

## Results

### Baseline Estimates

Figure 1 presents our baseline estimates and 95% confidence intervals for the associations between the ten PGIs and the four different measures of political participation. We report results from the pooled sample and with all available observations included; the full regression results (Table A.5) and separate baseline results for the Swedish and US subsamples (Tables A.7 through A.10) are reported in the Appendix.[7]

We regress each political participation measure on each of the ten PGIs separately, controlling for the first 10 genetic principal components of the genetic-relatedness matrix as well as birth-year dummies,

[7]The analysis plan stipulated that only complete sibling pairs be used, to facilitate comparisons with within-family results. These results are therefore found in Figure 2 (circles). In order to instead utilize the full samples, Figure 1 also contains singletons.

Table 1: Summary Statistics

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

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

gender, interaction terms between the birth-year dummies and the gender indicator, and fixed effects for subsample. We also restrict the samples to individuals of European descent (as an additional way of addressing population stratification). We use OLS for all models and all standard errors are clustered at the family-level. To account for multiple comparisons and adjust for the false discovery rate, we also use the Benjamini-Hochberg procedure per outcome (Benjamini and Hochberg, 1995). Filled circles indicate significance at the .05-level.

In addition to the estimated effects displayed in Figure 1, we report the incremental $R^2$ ($\Delta R^2$), or the increase in the coefficient of determination accounted for by the PGIs in Table A.5 in the Appendix. The incremental $R^2$ for the PGI is obtained by subtracting the $R^2$ from a model only including the control variables as predictors from the $R^2$ from a model that also includes the PGI as a regressor.

To put the effects of the ten psychological and health trait PGIs on the participatory outcomes in

perspective, the first row in each panel displays corresponding estimates for the educational attainment (EA) PGI. As recently established by Dawes et al. (2021) and Aarøe et al. (2021), education linked genes are significantly related to validated and self-reported voter turnout. We extend these results by showing that the EA PGI is also positively associated with an index measuring non-voting participatory acts.

For two reasons, we expect the EA PGI to be particularly strongly related to the four outcomes. First, previous research has shown that EA is among, if not the most important predictor of participation (Verba, Schlozman and Brady, 1995; Smets and van Ham, 2013; Persson, 2015). Second, the predictive capacity of a PGI is dependent on the precision of the weights used to construct the PGI. Since the separate effects of individual genetic markers (SNPs) on complex human traits are bound to be very small, large discovery samples are needed to obtain reasonably precise estimates of the weights. The GWAS estimates used to construct the EA PGI are derived from a very large discovery sample ($> 1,000,000$) and the predictive capacity of the resulting PGI is consequently substantial.[8] In line with this, Dawes et al. (2021) report that the effect of the EA PGI on voter turnout is on par with that of other well-known and strong predictors of political participation such as personal and parental income and parental education.

Turning now to the estimated effects of the psychological and health trait PGIs in rows 2 through 11 and looking first at the results for validated first- and second-order turnout in the two leftmost panels, a couple of things stand out. First, a majority of the PGIs are significantly related to voter turnout. Moreover, the directions of these associations are consistent with previous research on the effects of psychological and health traits on voter turnout and political participation. Thus, the effects of the PGIs for cognitive performance, adventurousness (only for second-order voting), morningness (only for first-order voting), self-rated health, subjective well-being and physical activity are positive whereas the neuroticism and depression PGIs are negatively associated with voter turnout. The results for risk attitudes are less clear and suggest that individuals more prone to risky behavior are less likely to vote in first-order elections but more inclined to vote in second-order elections.

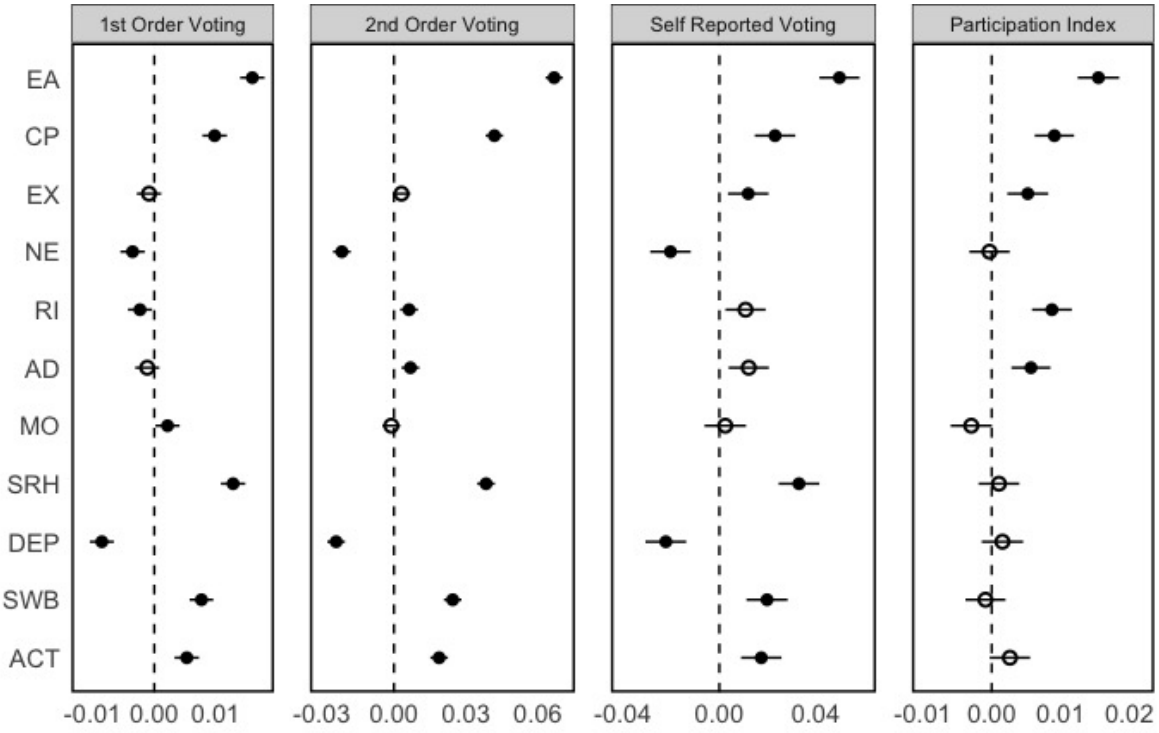
Second, the magnitudes of these effects are non-negligible. For example, a one standard deviation increase in the PGIs for cognitive performance or self-rated health increases the likelihood of voting in midterm or European Parliament elections by around 4 percentage points. Put differently, each of these two PGIs accounts for almost 1% of the variation in voter turnout in second-order elections (see Table A.5 in the Appendix).

Looking instead at the results for self-reported voting the overall pattern of estimates is very similar to the one found for the validated turnout outcomes. Concerning non-voting political participation, it is interesting to note that the few significant PGIs corroborate a common finding in previous research on personality and politics. Above all, the PGIs for extraversion, risk attitudes and adventurousness

[8] Lee et al. (2018) show that the EA PGI explain 12-13% of the variation in years of schooling which amounts to around 50% of the total SNP heritability in educational attainment.

are positively related to political acts such as contacting a politician or attending a political rally. This suggests that these traits have positive effects on participation in some circumstances, especially those involving greater amounts of sociability, but not in others (Mondak et al., 2010; Gerber et al., 2011; Lindell and Strandberg, 2018). In the Appendix (Table A.11) we report estimated effects of the PGIs on each of the acts included in the participation index separately. In addition, in the Appendix we present the results from a preliminary mediation analysis where we examine whether and to what extent the different PGIs influence political participation via their respective target traits. Overall, our analysis suggests that the effects of the PGIs are partly mediated by the traits they are constructed to predict.

Figure 1: Baseline results




*Note:* PGIs for: (E)ducational (A)ttainment; (C)ognitive (P)erformance; (EX)traversion; (NE)uroticism; (RI)sk; (AD)venturousness; (MO)rning person; (S)elf(R)ated (H)ealth; (DEP)ression; (S)ubjective (W)ell(B)eing; physical (ACT)ivity. The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). All models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genetic-relatedness matrix. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. Filled circles indicate indicates significance at the 5% level. Complete coefficient estimates together with standard errors are reported in Table A.5 in the Appendix.

## Within-family analysis

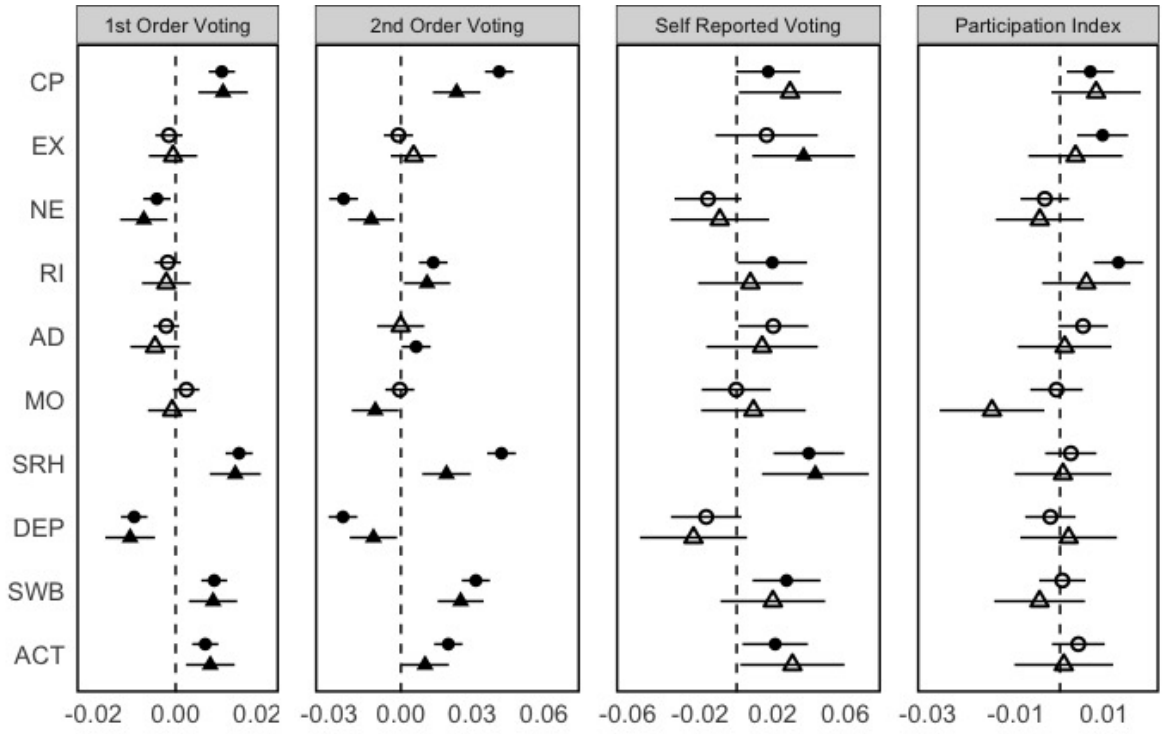
We next assess the extent to which the associations between the health and psychological trait PGIs reported in Figure 1 can be considered causal in nature. As discussed above, the PGI effects may be confounded by population stratification despite the fact that the previous analysis included controls for a set of principal components of the genetic-relatedness matrix and restricted the samples to individuals of European descent. Moreover, the between-family estimates displayed in Figure 1 may capture both direct genetic effects (effects of individuals' health- and psychological trait-linked genes on their political behavior) and indirect effects via genetic nurture.

In order to identify and account for bias due to any remaining population stratification and genetic nurture, we restrict our analysis to sibling pairs and run sibling-fixed effects models. The coefficient estimates and the 95% confidence intervals are presented in Figure 2. To simplify interpretation, we display the between-family effects in this restricted sample as circles and the estimates from models in which we include fixed effects for each sibling pair as traingles.

Looking first at the results for validated first- and second-order voting in the first two panels, the significant estimates in the sibling fixed-effects models in which we only make use of random within-family variation in the PGIs strongly suggest that many of the psychological and health PGIs are causally related to voter turnout. However, comparing the between- and the within-family estimates, there is some indication of confounding in the models for voting in second-order elections. Thus, to a certain degree, the between-family estimates (circles) may pick up effects of genetic nurture and population stratification. In accordance with previous studies on the effects of the EA PGI on various outcomes, we hypothesize that the bulk of the difference between the between- and within-family estimates is due to genetic nurture effects (Kong et al., 2018; Selzam et al., 2019; Dawes et al., 2021) (although as previously mentioned, the within-family estimates may also be "overcorrected" toward zero due to attenuation bias in within-family models).

The within-family results for self-reported voting and the political participation index are less precise. The estimates for the political participation index follow the pattern for second-order voting in that the magnitude of the between-family coefficients are larger than the corresponding within-family coefficients in most cases. On the other hand, there is no clear pattern of smaller effects when comparing the significant between-family coefficients for self-reported voting to the corresponding sibling fixed-effects estimates. Thus, the results do not suggest that the between-family estimates are inflated due to population stratification or genetic nurture.

Figure 2: Within-family results




*Note:* PGIs for: (C)ognitive (P)erformance; (EX)traversion; (NE)uroticism; (RI)sk; (AD)venturousness; (MO)rning person; (S)elf(R)ated (H)ealth; (DEP)ression; (S)ubjective (W)ell(B)eing; physical (ACT)ivity. The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1). Circles denote PGI effects from between-family models. Triangles denote PGI effects from family-fixed effects. All between-family models include controls for sex, birth-year dummies, interaction between sex and the birth-year dummies, sample fixed effects, and the first ten principal components of the genetic-relatedness matrix. The within-family models include controls for sex and sample fixed effects. Standard errors, shown in parentheses, allow for clustering at the family level. The significance tests are adjusted for false discovery rate within each column using the Benjamini-Hochberg procedure. Filled circles/triangles indicate indicates significance at the 5% level. Complete coefficient estimates together with standard errors are reported in Table A.6 in the Appendix.

## Discussion

This study makes several empirical contributions to the political behavior literature. Above all, it improves our understanding of how genes are related to political participation. Past work based on twin studies has demonstrated that individual differences in the propensity to vote and engage in other political acts can be partly explained by genetic variation. However, twin studies cannot make individual quantitative predictions about the specific sources of genetic and environmental variance. The departure point for this study was a number of psychological and health traits that have been shown to be related to political participation in previous research. In contrast to previous studies on psychological and health traits and participation, we made use of polygenic indices constructed to capture the genetic variation in ten of these psychological and health traits. Based on over 40,000 individuals in four samples from the US and Sweden we showed that *genes linked to these traits* predict voter turnout and engagement in non-electoral political acts. The significant associations we found between the PGIs and our outcome measures, especially based on the within-family models, represent strong evidence of a molecular genetic association with political participation. Furthermore, the estimated effects are substantially meaningful; for example, a one-standard deviation increase in the PGIs for cognitive performance and self-rated health is associated with a 4 percentage points increase in the likelihood of voting in a second-order election.

It is important here to note that a PGI does not fully capture the genetic variation in its target trait. Table A.2 in the Appendix reports the predictive capacity of the PGIs and the heritability based on all genotyped SNPs, the so called SNP heritability, for the ten psychological and health traits used in this study. The SNP heritability is the upper theoretical limit for the predictive capacity based on all genotyped SNPs. The average SNP heritability for the ten traits is equal to 12.4% with a low of 5.1% (risk attitudes) and a high of 23.2% (cognitive performance). The corresponding predictive capacities (measured as the incremental $R^2$) of the PGIs vary between 2.4% (the PGI for risk attitudes) and 10.7% (the PGI for cognitive performance) with an average amounting to 5.0%. Consequently, our results represent a lower bound on the effect of genes associated with the ten psychological and health traits on voter turnout and political participation. Ongoing larger GWA studies will provide us with more accurate polygenic indices in the near future.

Our study is also meant to serve as an example of how genetic information can be practically integrated into empirical political science research. A central task for future research on political behavior should be to continue along this path and increase our understanding on how the interplay between genetic and social factors accounts for individual differences in political traits. In our view, such an enterprise will ultimately make significant contributions to the study of political behavior. We argue that the use of polygenic indices can play a crucial role in such an endeavour. The increasing availability of powerful polygenic indices linked to different social and psychological traits will greatly aid political scientists who are interested in integrating genetic information into their own empirical work. As described in the

Appendix, several PGIs connected to a number of datasets of interest for social and political scientists are already available. Moreover, given the rapid development in genomic research and the decreasing costs of genotyping, we expect that PGIs will be available in a growing number of datasets that include information on political traits in the near future.

In the empirical section of the study we provided an example of how PGIs can be very useful for researchers with a primary interest in understanding the genetic underpinnings of political traits. For one thing, within-family estimates of PGI effects provide us with credible causal evidence of effects of genetic markers linked to certain traits on political traits. However, polygenic indices are not simply a means of detecting genetic influence. As way of conclusion we will therefore explicate and exemplify how PGIs can be integrated into political behavior research in ways useful to political scientists who do not care about genes per se.[9]

We anticipate that the eventual contributions of integrating PGIs into political behavior research will fall into several categories. First, while rising standards for causal inference have prompted scholars of political behavior to employ a variety of approaches (e.g. instrumental variables regressions and RCTs) in their search for causal antecedents of political traits, these approaches often have important limitations and may not be possible for some research questions. Thus, there is a (growing) need for better ways of directly controlling for possible confounders in standard correlational research designs. By including PGIs as control variables, researchers studying political behavior can reduce potential bias due to genetic confounding (as well as account for residual variance and thus lower the standard errors associated with estimates of nongenetic parameters of interest).

As an illustration of this, we present results in the Appendix (Table A.13) showing that the effects of the health and psychological traits on voter turnout and political participation decrease by, on average, 11% when controlling for the other PGIs examined in this paper. As more precise PGIs become available, this effect size reduction will only increase (Becker et al., 2021).

Second, PGIs can be used to enrich political socialization research and help us peer into the black box of the family. A case in point is parent-child relations. We know from a longstanding research tradition that children resemble their parents along a number of political attitudes and behaviors (Jennings, Stoker and Bowers, 2009). However, correlations between biological relatives are etiologically ambiguous since parents transmit both a rearing environment and a set of genes to their children. One way of disentangling social from genetic intergenerational transmission is to study *genetic nurture effects*. For example, showing that parental genotype (as measured by PGIs) is related to offspring traits conditional on offspring genotype strongly suggests a role of environmental factors affected by heritable characteristics of the parents. Moreover, PGIs can help us study how children influence their parents and siblings or actively evoke their environments. It has been reported that having sisters causes young men to be more likely to express conservative viewpoints (Healy and Malhotra, 2013) and that having a first-born

[9]The following empirical examples were not pre-registered.

daughter leads to higher levels of support for gender-equality policies and candidates among the fathers (Oswald and Powdthavee, 2010; Sharrow et al., 2018). By conditioning on the genotypes of parents or siblings, it is possible to study how the random draw of genetic predispositions for a host of traits also affects parental and sibling behavior.

In the Appendix (Table A.14) we provide an empirical example of the latter kind. More precisely, we test if a person's political participation is associated with his/hers sibling's genetic predispositions for the traits in focus in this study. The pattern of results in these sibling-correlation models is striking. There are no signs of sibling genetic nurture effects on voting in first-order elections. The models for second-order voting, however, show consistent and relatively strong effects of the sibling's PGIs on the propensity to vote. This difference in results for first- and second-order voting is interesting and suggests that the lower-salience and information-poor environment in the typical second-order election will increase the relative importance of the social context - here understood as the predispositions of one's siblings - over individual resources in determining an individual's decision to vote.

Third, PGIs can be a powerful source of latent heterogeneity, providing measures of moderating traits that may otherwise be difficult or impossible to capture, for example in studies of environmental interventions to increase voter turnout (Imai and Strauss, 2011; Kam and Trussler, 2017) or when investigating whether certain reforms exacerbate or mitigate existing inequalities (Larsen, 2019). As previously discussed, PGIs have properties that many other such measures lack, such as safeguarding against reverse causation and providing the possibility of using within-family variation to plausibly identify causal effects. This type of interplay between genetic and social factors, often called *gene-environment interactions* (GxE) is widely believed to be pervasive for behavioral traits (Conley and Rauscher, 2013).

As a simple but concrete example, in section A.2.8 in the Appendix we show that educational attainment, in accordance with a diathesis-stress hypothesis (Monroe and Simons, 1991; Colodro-Conde et al., 2018), acts as a significant dampener on the negative influence of the depression PGI on voter turnout. The negative influence of genetic susceptibility to depression appears to be driven completely by those at the low end of the education distribution, while at the top end of the distribution the relationship in fact disappears.

To summarize, our study provides an example of how genetic information can be practically integrated into empirical political science research. While twin studies have shined a light on the link between genes and political traits, they are limited in what they can tell us. Rather than a latent measure of genetic factors, polygenic indices are individual-level, direct measures of the genetic propensity for a trait that can be used to build and test new theories as well as refine existing ones. Large GWA studies are increasingly being conducted for social behaviors and outcomes, many of which are relevant for political scientists. In addition, there is a growing availability of samples that include information on both PGIs for different relevant traits and measures of political attitudes and behavior. Our hope is that scholars will integrate both genetic and social factors in models of political behavior in order to more fully understand and

account for the processes that give rise to individual differences in political attitudes and behavior. As such, a research agenda along these lines can also aid in developing more effective policies that deal with the underlying causes and consequences of persistent inequalities in political behavior.

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# Supplementary Appendix

## A.1 Data

Table A.1 displays the question wordings for all items used to construct the political participation measures in each of the four samples or cohorts used in this study. Table A.2 lists all polygenic indices included in the PGI repository described in Becker et al. (2021) and used in this study. The 10 PGIs included in the study are in **boldface**. The criteria for being included in the study are as follows: i) a health or psychological target trait and ii) an incremental $R^2 \geq 2\%$. Finally, Table A.3 shows all cohorts included in the PGI repository. The four samples included in the study are in **boldface**. The criteria for being included in the study are as follows: i) relevant political outcomes should be included; and ii) the sample should be family-based/include sibling pairs. It should be noted here that several of the remaining repository cohorts include phenotypic data of relevance to political behavior researchers, for instance the English Longitudinal Study of Ageing UK (ELSA) and Dunedin Multidisciplinary Health and Development Study New Zealand.

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

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

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

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

STR: Researchers interested in using STR data must obtain approval from the Swedish Ethical Re-

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

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

| | |
|---|---|
| | **Minnesota Center for Twin and Family Research (MTFS)** |
| Political participation | N/A |
| Turnout - self-stated | I vote in national or state elections is "not true at all", "not very true", "pretty true", or "very true". |
| Turnout - high stake | 1996, 2000, 2004, 2008, 2012, 2016 and 2020 elections |
| Turnout - low stake | 1994, 1998, 2002, 2006, 2010, 2014 and 2018 elections. |
| | **National Longitudinal Study of Adolescent to Adult Health (Add Health)** |
| Political participation | Question wording: "Which of the following things have you done during the last 12 months?" Response alternatives: i) Contributed money to a political party or candidate; ii) Contacted a government official regarding political or community issues; iii) Run for a public office; iv) Run for a non-public office; v) Attended a political rally or march. Political participation is an additive index including the 3 subitems that are also present in the other datasets (contacted gov't official, contributed, attended rally/march/demonstration), rescaled to the 0-1 range. |
| Turnout - self-stated | Question wording: "How often do you usually vote in local or statewide elections?" Response alternatives: i) Never; ii) Sometimes; iii) Often; iv) Always. The turnout scale will be rescaled to the 0-1 range. |
| Turnout - high stake | N/A |
| Turnout - low stake | N/A |
| | **Swedish Twin Registry (STR)** |
| Political participation | Question wording: "During the last five years, have you done any of the following to express your political opinions?" Response alternatives: i) Contacted a politician personally, or in writing, or some other way; ii) Contacted a public sector official; iii) Made financial contributions; iv) Participated in a protest action or demonstration; v) Boycott, for example certain goods; vi) Signed a petition. Political participation is an additive index including the 3 subitems that are also present in the other datasets (contacted gov't official, contributed, attended rally/march/demonstration), rescaled to the 0-1 range. |
| Turnout - self-stated | N/A |

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

| | |
|---|---|
| Turnout - high stake | Validated turnout data from the national elections in 1970, 1994, 2010, and 2018. Turnout is measured as the per-individual average turnout across all 4 elections. |
| Turnout - low stake | Validated turnout data from the European Parliament elections in 2009 and 2019. Turnout is measured as the per-individual average turnout across the 2 elections. |
| | **Wisconsin Longitudinal Study (WLS)** |
| Political participation | NA |
| Turnout - self-stated | Which statement best describes your decision to vote in the November 2002 election? i) I did not vote in the election in November 2002 ii) I thought about voting in November 2002 but did not iii) I usually vote but did not vote in November 2002 iv) I am sure I voted at the polls in the election in November 2002 v) I am sure I voted by absentee ballot in November 2002. Which statement best describes your decision to vote in the November 2008 election? i) I did not vote in the election in November 2008 ii) I thought about voting in November 2008 but did not iii) I usually vote but did not vote in November 2008 iv) I am sure I voted at the polls in the election in November 2008 v) I am sure I voted by absentee ballot in November 2008. |
| Turnout - high stake | 2000, 2004, 2008, and 2012 elections |
| Turnout - low stake | 2002, 2006, and 2010 elections. |

Table A.2: Polygenic indices in the PGI repository

| **Target trait** | **Incremental $R^2$** | $h^2_{SNP}$ |
|---|---|---|
| *Anthropometric* | | |
| Body mass index (BMI) | 17.03% | 24.26% |
| Height | 36.20% | 46.60% |
| *Cognition and education* | | |
| Alzheimer's | 0.22% | 4.62% |
| Childhood reading | 1.14% | 6.90% |
| **Cognitive performance** | **10.73%** | **23.18%** |
| Educational attainment | 7.27% | 11.04% |
| Highest math | 7.85% | 15.10% |
| Self-reported math ability | 8.47% | 14.62% |
| *Fertility and sexual development* | | |
| Age at first birth | 6.98% | 19.58% |
| Age first menses (women) | 9.88% | 19.65% |

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Table A.2: Polygenic indices in the PGI repository

| | | |
|---|---|---|
| Age voice first deepened (men) | 0.79% | 9.63% |
| Number ever born (men) | 0.58% | 4.40% |
| Number ever born (women) | 1.72% | 7.96% |
| *Health and health behaviors* | | |
| Alcohol - misuse | 1.62% | 9.83% |
| Allergy - cat | 0.67% | 9.61% |
| Allergy dust | 0.49% | 8.17% |
| Allergy - pollen | 0.87% | 11.03% |
| Asthma | 1.72% | 5.91% |
| Asthma/eczema/rhinitis | 3.37% | 8.18% |
| Attention deficit hyperactivity disorder | 4.09% | 22.84% |
| Cannabis use | 1.46% | 8.25% |
| Cigarettes per day | 3.49% | 11.07% |
| COPD | 0.36% | 2.64% |
| **Depressive symptoms** | **3.08%** | **7.22%** |
| Drinks per week | 2.17% | 5.46% |
| Eczema | 0.07% | 1.03% |
| Ever smoker | 5.43% | 8.73% |
| Hayfever | 2.59% | 7.63% |
| Migraine | 1.76% | 5.97% |
| Nearsightedness | 7.53% | 16.57% |
| **Physical activity** | **3.95%** | **15.13%** |
| **Self-rated health** | **5.48%** | **9.34%** |
| *Personality and well-being* | | |
| **Adventurousness** | **3.51%** | **8.14%** |
| Agreeableness | 0.67% | 8.58% |
| Cognitive empathy | 0.77% | 10.33% |
| Conscientiousness | 0.87% | 9.81% |
| Delay discounting | 0.21% | 7.48% |
| **Extraversion** | **3.88%** | **19.78%** |
| Left out of social activity | 1.90% | 5.79% |
| Life satisfaction - family | 1.04% | 7.17% |
| Life satisfaction - finance | 0.99% | 7.16% |
| Life satisfaction - friends | 1.14% | 7.61% |
| Life satisfaction - work | 0.58% | 7.55% |
| Loneliness | 0.86% | 3.99% |
| **Morning person** | **7.76%** | **15.86%** |
| Narcissism | 1.23% | 4.69% |
| **Neuroticism** | **5.67%** | **12.61%** |
| Openness | 1.44% | 11.17% |
| Recharge by socializing | 0.73% | 3.43% |
| Religious attendance | 1.28% | 5.17% |

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Table A.2: Polygenic indices in the PGI repository

| | | |
|---|---|---|
| Religious belief | 0.64% | 7.01% |
| **Risk tolerance** | **2.45%** | **5.13%** |
| **Subjective well-being** | **3.01%** | **7.68%** |

Table A.3: Samples in the PGI repository

| **Sample** | **Country** |
|---|---|
| Dunedin Multidisciplinary Health and Development Study | New Zealand |
| English Longitudinal Study of Ageing | UK |
| Environmental Risk Longitudinal Twin Study | UK |
| Estonian Genome Center, University of Tartu | Estonia |
| Health and Retirement Study | US |
| **Minnesota Center for Twin and Family Research** | **US** |
| **National Longitudinal Study of Adolescent to Adult Health** | **US** |
| **Swedish Twin Registry** | **Sweden** |
| Texas Twin Project | US |
| UK Biobank | UK |
| **Wisconsin Longitudinal Study** | **US** |

## A.2 Auxiliary results and robustness checks

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

### A.2.1 PGI cross-correlations

Figure A.4 displays bivariate correlations for the eleven PGIs used in this study. A number of mostly expected clusters can be noted. First, the PGIs for educational attainment and cognitive performance are positively correlated (Okbay, Beauchamp, Fontana et al., 2016; Lee et al., 2018). Second, as reported in Okbay, Baselmans, De Neve et al. (2016) subjective well-being, depressive symptoms, and neuroticism are genetically correlated. Third, genetic predispositions for risk attitudes are positively correlated with predispositions for adventurousness and, to a lesser extent, extraversion. Finally, the PGI for self-rated health seems to be moderately strongly correlated with the PGIs for the remaining three health-trait PGIs: depression, subjective wellbeing, and physical activity.

Table A.4: PGI cross-correlations

| Variables | EA | CP | EX | NE | RI | AD | MO | SRH | DEP | SWB | ACT |
|---|---|---|---|---|---|---|---|---|---|---|---|
| EA | 1.000 | | | | | | | | | | |
| CP | 0.447 | 1.000 | | | | | | | | | |
| EX | 0.003 | -0.058 | 1.000 | | | | | | | | |
| NE | -0.107 | -0.090 | -0.144 | 1.000 | | | | | | | |
| RI | 0.068 | -0.029 | 0.227 | -0.187 | 1.000 | | | | | | |
| AD | 0.119 | -0.033 | 0.243 | -0.137 | 0.487 | 1.000 | | | | | |
| MO | -0.034 | -0.078 | 0.038 | -0.050 | 0.026 | 0.066 | 1.000 | | | | |
| SRH | 0.361 | 0.190 | 0.047 | -0.237 | 0.092 | 0.188 | 0.126 | 1.000 | | | |
| DEP | -0.184 | -0.113 | -0.054 | 0.522 | -0.069 | -0.072 | -0.079 | -0.399 | 1.000 | | |
| SWB | 0.155 | 0.073 | 0.148 | -0.388 | 0.197 | 0.156 | 0.115 | 0.370 | -0.456 | 1.000 | |
| ACT | 0.180 | 0.011 | 0.082 | -0.061 | 0.124 | 0.172 | 0.096 | 0.290 | -0.139 | 0.159 | 1.000 |

*Note:* PGIs for: (E)ducational (A)ttainment; (C)ognitive (P)erformance; (EX)traversion; (NE)uroticism; (RI)sk; (AD)ventouresness; (MO)rning person; (S)elf(R)ated (H)ealth; (DEP)ression; (S)ubjective (W)ell(B)eing; physical (ACT)ivity. The polygenic indices are standardized within each sample and birth year (mean=0, s.d.=1).

### A.2.2 Full regression results

In the main text we present the results using graphs. The corresponding regression tables are presented below. That is, Figure 1 in the main text is based on the estimates displayed in Table A.5 whereas the estimates displayed in Figure 2 are found in Table A.6.

Table A.5: Baseline results

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

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

Table A.6: Within-family results

| | 1st order | | 2nd order | | Self-reported | | Participation | |
|---|---|---|---|---|---|---|---|---|
| | BF | WF | BF | WF | BF | WF | BF | WF |
| CP | 0.011*** | 0.012*** | 0.041*** | 0.023*** | 0.017* | 0.029 | 0.007** | 0.008 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.009] | [0.014] | [0.003] | [0.005] |
| EX | −0.002 | −0.001 | −0.001 | 0.005 | 0.017 | 0.037** | 0.010*** | 0.004 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.009] | [0.014] | [0.003] | [0.006] |
| NE | −0.005** | −0.008** | −0.024*** | −0.012** | −0.016 | −0.009 | −0.003 | −0.005 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.009] | [0.014] | [0.003] | [0.005] |
| RI | −0.002 | −0.002 | 0.014*** | 0.011** | 0.020* | 0.008 | 0.013*** | 0.006 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.015] | [0.003] | [0.005] |
| AD | −0.002 | −0.005 | 0.006** | −0.000 | 0.020 | 0.014 | 0.005 | 0.001 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.016] | [0.003] | [0.005] |
| MO | 0.003 | −0.001 | −0.000 | −0.011** | −0.000 | 0.009 | −0.001 | −0.015 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.015] | [0.003] | [0.006] |
| SRH | 0.016*** | 0.015*** | 0.042*** | 0.019*** | 0.040*** | 0.043** | 0.002 | 0.001 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.015] | [0.003] | [0.006] |
| DEP | −0.010*** | −0.011*** | −0.024*** | −0.012** | −0.017 | −0.024 | −0.002 | 0.002 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.015] | [0.003] | [0.006] |
| SWB | 0.009*** | 0.009*** | 0.031*** | 0.025*** | 0.028** | 0.020 | 0.001 | −0.005 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.010] | [0.015] | [0.003] | [0.005] |
| ACT | 0.007*** | 0.008** | 0.020*** | 0.010* | 0.021* | 0.031 | 0.004 | 0.001 |
| | [0.002] | [0.003] | [0.003] | [0.005] | [0.009] | [0.016] | [0.003] | [0.006] |
| $\overline{Y}$ | 0.919 | 0.919 | 0.667 | 0.667 | 0.560 | 0.560 | 0.080 | 0.080 |
| $N$ | 20,488 | 20,402 | 20,402 | 22,866 | 1,660 | 1,660 | 4,206 | 4,206 |

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

### A.2.3 Baseline results in separate samples

In the main text we present results based on a pooled sample. To check whether the estimates are unduly driven by any of the subsamples we have re-estimated the baseline models from Table A.5 in each of STR, WLS, MTFS and Add Health samples. The results are reported in Tables A.7 through A.10. For convenience we also display the estimates from Table A.5 in the first column of each table. Finally, the last column in each table reports whether or not (using F-tests) the coefficient estimates vary significantly across the subsamples.[1]

The estimated PGI effects on first-order voting, self-reported voting and political participation do not seem to vary systematically across the different samples. There are some exceptions to this rule – for instance, the positive effects of the PGIs for risk attitudes and adventurousness on the participation index are driven by the STR sample. Moreover, the associations between voting in first-order elections and the PGIs for cognitive performance (strongest in STR), risk attitudes (strongest in MTFS) and self-rated health (strongest in WLS) differ significantly across the three samples. However, the overall pattern is one of consistency. Looking instead at Table A.8, the results for second-order voting stick out and the pattern is rather clear: The PGI effects are, in most cases weaker in the WLS sample compared to the STR and MTFS samples. The most obvious difference between these samples is the age composition of the study participants. The individuals in the WLS sample are significantly older than the individuals in the other two samples (see Table 1 in the main text). We speculate here that experience and habit are more important for the decision to vote than genetic predispositions among older citizens. Moreover, this may be especially so in second-order elections, which tend to be viewed as less important by voters, parties, and the media and thus present a more information-poor electoral environment for citizens to navigate. More research is, of course, necessary to substantiate these assertions.

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

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

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

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

Table A.8: Baseline results - second-order voting

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

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

Table A.9: Baseline results - self-reported voting

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

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

Table A.10: Baseline results - participation index

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

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

### A.2.4 Baseline results for separate participation items

In Table A.11 we present the baseline estimates for models using as outcomes the three separate items included in the political participation index – whether the respondents contacted a government official, contributed money to a political cause, and attended a political rally or march during the last 12 months (Add Health)/5 years (STR).[2] For convenience we also display the estimates for the index from Table A.5 in the first column of the table. The last column reports whether or not (using Wald tests) the coefficient estimates vary significantly across the three outcomes.[3]

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

[2]The separate analyses for each participation item were not pre-registered.

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

Table A.11: Baseline results - separate participation items

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

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

### A.2.5 Mediation Analysis

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

With these caveats in mind, Table A.12 presents coefficient estimates from a simple mediation analysis. We restrict the mediation analysis in two ways. First, we need information on the target traits to test for mediation.[4] Second, we only include results for PGIs that are significantly related (at the 0.05 level) to an outcome when restricting the sample to individuals for which we have information on the corresponding target trait. In the end, this leaves us with five PGI–target trait combinations for the turnout in first-order elections, seven PGI–target trait combinations for turnout in second-order elections, eight PGI–target trait combinations for self-reported voting, and three PGI–target trait combinations for the political participation index.[5]

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

[4]For a detailed description of the availability of relevant target trait information and how these traits are measured in each of the four samples we refer to two separate pre-analysis plans posted on the OSF website on April 14, 2021: `https://osf.io/gnv9t/`, and `https://osf.io/vrd75/`.

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

The results in Table A.12 show that the effects of the PGIs on the four outcome measures shrink somewhat when controlling for the respective target trait. The decrease in PGI effect magnitude is modest. On average, the target traits account for around 25% of the corresponding PGI effect. However, there is a great deal of variation in the degree to which the the PGI effects decrease when controlling for the corresponding target trait. As we would expect, the target traits that are measured using validated multi-item batteries (e.g. cognitive performance, extraversion and depression) account for a larger share of the PGI-effects compared to the target traits that are measured using only a single item (e.g. risk attitudes, self-rated health, subjective well-being, and physical activity).

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

Table A.12: Results - mediation

| | 1st order | | 2nd order | | Self-reported | | Participation | |
|---|---|---|---|---|---|---|---|---|
| CP PGI | 0.007*** | 0.002 | 0.031*** | 0.014*** | 0.022*** | 0.008* | 0.009*** | 0.004** |
| | [0.001] | [0.002] | [0.002] | [0.003] | [0.005] | [0.005] | [0.002] | [0.002] |
| CP Trait | | 0.016*** | | 0.066*** | | 0.060*** | | 0.020*** |
| | | [0.002] | | [0.003] | | [0.005] | | [0.002] |
| % mediated | 66.0% | | 56.9% | | 64.2% | | 55.3% | |
| *N* | 23,328 | | 23,279 | | 6,626 | | 8,884 | |
| EX PGI | | | 0.006** | 0.003 | 0.012*** | 0.006 | 0.005*** | 0.003** |
| | | | [0.003] | [0.003] | [0.004] | [0.004] | [0.002] | [0.002] |
| EX Trait | | | | 0.028*** | | 0.050*** | | 0.021*** |
| | | | | [0.002] | | [0.004] | | [0.002] |
| % mediated | | | 49.6% | | 47.7% | | 42.3% | |
| *N* | | | 21,913 | | 7,749 | | 14,273 | |
| NE PGI | −0.004** | −0.004*** | −0.017*** | −0.016*** | −0.020*** | −0.016*** | | |
| | [0.001] | [0.001] | [0.002] | [0.002] | [0.004] | [0.004] | | |
| NE Trait | | −0.005*** | | −0.005** | | −0.037*** | | |
| | | [0.001] | | [0.002] | | [0.004] | | |
| % mediated | 10.2% | | 2.3% | | 22.5% | | | |
| *N* | 28,404 | | 28,087 | | 7,748 | | | |
| RI PGI | | | | | 0.011*** | 0.011*** | 0.009*** | 0.007*** |
| | | | | | [0.004] | [0.004] | [0.001] | [0.001] |
| RI Trait | | | | | | −0.009* | | 0.016*** |
| | | | | | | [0.005] | | [0.002] |
| % mediated | | | | | -5.8% | | 20.5% | |
| *N* | | | | | 7,777 | | 14,218 | |
| SRH PGI | 0.011*** | 0.009*** | 0.035*** | 0.031*** | 0.033*** | 0.024*** | | |
| | [0.001] | [0.001] | [0.002] | [0.002] | [0.005] | [0.005] | | |
| SRH Trait | | 0.016*** | | 0.024*** | | 0.047*** | | |
| | | [0.002] | | [0.003] | | [0.005] | | |
| % mediated | 19.1% | | 9.4% | | 28.5% | | | |
| *N* | 30,443 | | 30,439 | | 5,633 | | | |
| DEP PGI | −0.007*** | −0.005*** | −0.019*** | −0.016*** | −0.023*** | −0.016*** | | |
| | [0.002] | [0.002] | [0.003] | [0.003] | [0.005] | [0.005] | | |
| DEP Trait | | −0.013*** | | −0.022*** | | −0.050*** | | |
| | | [0.002] | | [0.003] | | [0.005] | | |
| % mediated | 21.9% | | 13.4% | | 30.2% | | | |
| *N* | 20,058 | | 20,061 | | 5,633 | | | |
| SWB PGI | 0.006*** | 0.005*** | 0.018*** | 0.016*** | 0.020*** | 0.019*** | | |
| | [0.002] | [0.002] | [0.003] | [0.003] | [0.004] | [0.004] | | |
| SWB Trait | | 0.008*** | | 0.020*** | | −0.022*** | | |
| | | [0.002] | | [0.003] | | [0.005] | | |
| % mediated | 13.8% | | 12.8% | | -5.2% | | | |
| *N* | 20,662 | | 20,673 | | 7,773 | | | |
| ACT PGI | | | 0.014*** | 0.013*** | 0.016*** | 0.016*** | | |
| | | | [0.003] | [0.003] | [0.005] | [0.005] | | |
| ACT Trait | | | | 0.008*** | | 0.003 | | |
| | | | | [0.003] | | [0.005] | | |
| % mediated | | | -4.0% | | 0.5% | | | |
| *N* | | | 16,230 | | 5,633 | | | |

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

### A.2.6 Confounding

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

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

Table A.13: Results - confounding

| | 1st order | | 2nd order | | Self-reported | | Participation | |
|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| CP Trait | $0.017^{***}$ | $0.015^{***}$ | $0.069^{***}$ | $0.061^{***}$ | $0.062^{***}$ | $0.053^{***}$ | $0.021^{***}$ | $0.018^{***}$ |
| | [0.002] | [0.002] | [0.002] | [0.003] | [0.005] | [0.005] | [0.002] | [0.002] |
| % confounded | 10.8% | | 11.9% | | 14.7% | | 13.2% | |
| $N$ | 23,328 | | 32,279 | | 6,626 | | 8,884 | |
| EX Trait | $0.008^{***}$ | $0.007^{***}$ | $0.028^{***}$ | $0.026^{***}$ | $0.050^{***}$ | $0.049^{***}$ | $0.022^{***}$ | $0.020^{***}$ |
| | [0.002] | [0.002] | [0.002] | [0.002] | [0.004] | [0.004] | [0.002] | [0.002] |
| % confounded | 2.9% | | 6.9% | | 3.2% | | 5.5% | |
| $N$ | 21,903 | | 21,913 | | 7,749 | | 14,273 | |
| NE Trait | $-0.005^{***}$ | $-0.005^{***}$ | $-0.006^{***}$ | $-0.004^{*}$ | $-0.039^{***}$ | $-0.034^{***}$ | $0.006^{***}$ | $0.006^{***}$ |
| | [0.001] | [0.001] | [0.002] | [0.002] | [0.004] | [0.004] | [0.002] | [0.002] |
| % confounded | 9.9% | | 32.1% | | 14.1% | | 4.7% | |
| $N$ | 28,404 | | 28,087 | | 7,748 | | 13,880 | |
| RI Trait | | | $0.015^{***}$ | $0.014^{***}$ | | | $0.017^{***}$ | $0.016^{***}$ |
| | | | [0.003] | [0.003] | | | [0.001] | [0.001] |
| % confounded | | | 10.8% | | | | 7.5% | |
| $N$ | | | 21,247 | | | | 14,218 | |
| SRH Trait | $0.017^{***}$ | $0.016^{***}$ | $0.028^{***}$ | $0.022^{***}$ | $0.052^{***}$ | $0.044^{***}$ | | |
| | [0.001] | [0.001] | [0.002] | [0.002] | [0.005] | [0.005] | | |
| % confounded | 9.2% | | 20.9% | | 14.7% | | | |
| $N$ | 30,443 | | 30,439 | | 5,633 | | | |
| DEP Trait | $-0.014^{***}$ | $-0.013^{***}$ | $-0.024^{***}$ | $-0.020^{***}$ | $-0.052^{***}$ | $-0.047^{***}$ | | |
| | [0.002] | [0.002] | [0.003] | [0.003] | [0.005] | [0.005] | | |
| % confounded | 7.2% | | 15.7% | | 9.9% | | | |
| $N$ | 20,058 | | 20,061 | | 5,633 | | | |
| SWB Trait | $0.008^{***}$ | $0.008^{***}$ | $0.022^{***}$ | $0.020^{***}$ | $0.023^{***}$ | $0.022^{***}$ | | |
| | [0.002] | [0.002] | [0.003] | [0.003] | [0.004] | [0.004] | | |
| % confounded | 6.1% | | 5.9% | | 5.4% | | | |
| $N$ | 20,662 | | 20,673 | | 7,773 | | | |
| ACT Trait | $0.005^{***}$ | $0.005^{**}$ | $0.009^{***}$ | $0.007^{**}$ | | | | |
| | [0.002] | [0.002] | [0.003] | [0.003] | | | | |
| % confounded | 13.9% | | 23.8% | | | | | |
| $N$ | 16,225 | | 16,230 | | | | | |

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

### A.2.7 Sibling models

In the main text we show that the estimated effects of the health- and psychological trait PGIs on the participation outcomes decrease somewhat in magnitude when controlling for family fixed effects, especially so for second-order turnout and the participation index. Moreover, we argue that this pattern of results suggests confounding due to genetic nurture rather than population stratification or assortative mating. That is, part of the reason for why we observe an association between a PGI and an outcome is due to the fact that a large share of the genetic markers summarized by the PGI will also be present in the individual's relatives and cause them to behave in certain ways with downstream effects on the individual in question. In this section we present further results corroborating this interpretation and at the same time illustrate the usefulness of the PGI approach for studying family socialization and peer effects.

The bulk of previous studies on genetic nurture focus on genes linked to educational attainment. Most of these studies examine the intergenerational transmission in educational attainment. Kong et al. (2018) demonstrate that an educational attainment PGI based on non-transmitted parental genes is significantly related to offspring education and that nearly 30% of the association between the PGI and education among offspring is accounted for by parental genetic nurture effects. These results were subsequently replicated by Bates et al. (2018). Corroborating these findings, Belsky et al. (2018), Liu (2018), and Willoughby et al. (2019) report that a parental education PGI is related to offspring education also when controlling for offspring's own PGI. Finally, Selzam et al. (2019) compare within- to between-family PGI predictions of education and cognitive traits and find that the between-family estimates are substantially greater. Much of this within- and between-family difference disappears when controlling for family socio-economic status, suggesting that a large share of the between-family PGI prediction is due to genetic nurture effects.

Extant research also provides some evidence of horizontal genetic nurture between siblings. Above all, Cawley et al. (2020) show that a person's educational attainment is correlated with their sibling's PGI for educational attainment, controlling for their own PG for educational attainment.

In Table A.14 we follow the approach taken by Cawley et al. (2020) and report results from models restricted to sibling pairs and including both subjects' (ego) and siblings' (alter) PGIs as predictors. We also restrict the analysis such that we only include results for PGIs that are significantly related (at the 0.05 level) to an outcome in between-family models in the sibling sample (see the estimates in the odd-numbered columns in Table A.6).

Looking first at the results for first-order and self-reported turnout there are no signs of sibling genetic nurture effects. The estimates are inconsistently signed and never statistically significant. Turning instead to the results for second-order voter turnout the results are much clearer and strongly suggest the presence of genetic nurturing. The estimated effects of the alter PGIs on second-order voting are non-negligible in magnitude and always consistent with the sign of the effects of the ego PGI. On average the

alter PGI effect amounts to 46% of the corresponding ego PGI effect. The same pattern of consistently signed and non-negligibly sized sibling effects is also found for the political participation index. However, likely due to the smaller sample size none of these estimates are less precisely estimated.

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

Table A.14: Sibling models

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

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

### A.2.8 Gene-by-environment interaction

As our last applied example of relevant uses of PGIs, we present a simple case of a gene-by-environment interaction, the example being the interaction between a depression PGI and educational attainment for voter turnout. Previous research has documented consistent negative effects of depressive symptoms on political participation (Ojeda, 2015; Ojeda and Pacheco, 2019; Landwehr and Ojeda, 2021; Gerber et al., 2011; Ojeda and Slaughter, 2019; Engelman et al., 2021), a picture that is confirmed by our main PGI results. Meanwhile, the so-called diathesis-stress model of depression claims that it is the combination of vulnerability (diathesis) with an environmental stressor that triggers actual depressive episodes (Monroe and Simons, 1991; Colodro-Conde et al., 2018). Educational attainment, in this context, thus acts as a possible proxy for a variety of stressors induced by different socioeconomic environments.

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

As evidenced by the significant and positive interaction terms in columns 1 and 2 in Table A.15, the negative effect of the depression PGI on turnout is moderated by educational attainment. The marginal effects plots (Fig. A.1) further reveal that the negative effect of the depression PGI is entirely driven by the lower end of the education distribution and disappears at the higher end. Thus, it may be that education (or variables associated with education) acts as a dampener on the negative turnout effects of having a genetic susceptibility to depression.

As a robustness check, the same interaction is tested with PGIs for related traits (neuroticism and self-rated health) in columns 3–6.

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

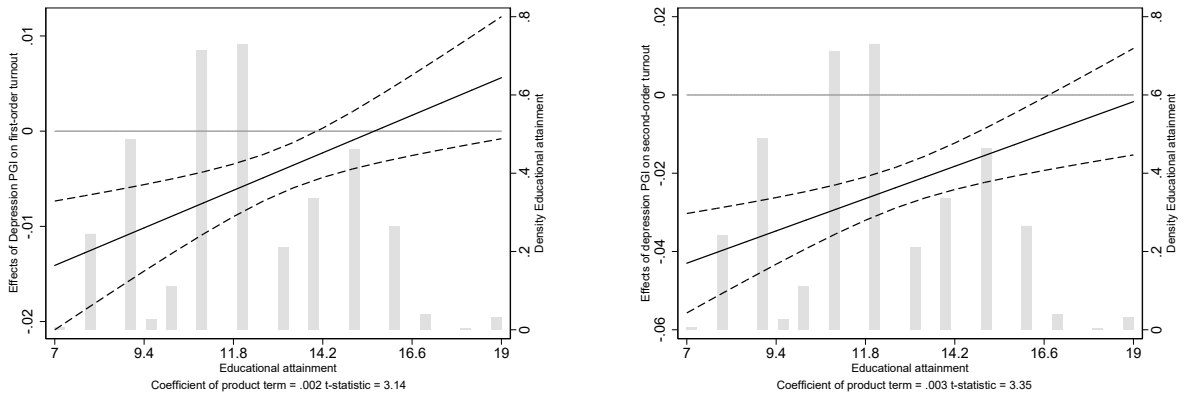

Table A.15: GxE interaction models

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

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

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