The genetic architecture of economic and political preferences

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Preferences are fundamental building blocks in all models of economic and political behavior. We study a new sample of comprehensively genotyped subjects with data on economic and political preferences and educational attainment. We use dense single nucleotide polymorphism (SNP) data to estimate the proportion of variation in these traits explained by common SNPs and to conduct genome-wide association study (GWAS) and prediction analyses. The pattern of results is consistent with findings for other complex traits. First, the estimated fraction of phenotypic variation that could, in principle, be explained by dense SNP arrays is around one-half of the narrow heritability estimated using twin and family samples. The molecular-genetic–based heritability estimates, therefore, partially corroborate evidence of significant heritability from behavior genetic studies. Second, our analyses suggest that these traits have a polygenic architecture, with the heritable variation explained by many genes with small effects. Our results suggest that most published genetic association studies with economic and political traits are dramatically underpowered, which implies a high false discovery rate. These results convey a cautionary message for whether, how, and how soon molecular genetic data can contribute to, and potentially transform, research in social science. We propose some constructive responses to the inferential challenges posed by the small explanatory power of individual SNPs.

genoeconomics | genopolitics | GCTA

There has been growing enthusiasm for the use of molecular genetic data in social science research. This enthusiasm is based on a number of potential contributions that such research could make to social science (1–3). For example, if specific genetic markers can be identified that are associated with a behavioral trait, then such predictive markers may shed light on the biological pathways underlying that trait (3, 4). If a set of genetic markers is sufficiently predictive, then these markers could be used in social science research as control variables, as instrumental variables (5, 6; for critical perspectives, see refs. 7, 8) or, under certain conditions, as factors for identifying at-risk individuals (1–3).

The extent to which this potential of molecular genetic data will be fulfilled for a given trait hinges on the trait's "molecular genetic architecture," i.e., the joint distribution of effect sizes and allele frequencies of the causal genetic variants (9). The architecture—which is the result of evolutionary forces, including mutation, drift, and selection—determines the difficulty with which the genetic variants associated with a trait can be identified and what sample sizes will be required for gene discovery. It also determines the out-of-sample aggregate predictability that can be derived from a set of genetic markers considered jointly.

Existing studies claiming to have established genetic associations with economic and political traits typically use samples of several hundred individuals, and no such study has used a sample larger than 3,000 individuals (for a recent review, see ref. 10). An implicit assumption underlying these studies is that there exist genetic variants whose effects are large enough that they can be reliably detected in samples of this size.

In this paper, we study the genetic architecture of economic and political preferences. For these traits, we ask whether the assumption of large effects of individual genetic variants is justified. We also explore the implications of the genetic architecture for efforts to realize the potential contributions of molecular genetic data in economic and political research.

We focus on preferences because they are fundamental building blocks in the models that economists and political scientists use to predict behavior. For example, measures of risk preferences predict diverse risky behaviors, such as smoking, drinking, and holding stocks rather than bonds (11, 12). Experimentally elicited patience predicts body mass index, smoking behavior, and exercise (13). Political preferences similarly predict a wide range of political behaviors, including voting (14) and monetary campaign contributions (15), as well as campaign activities like volunteering, attending rallies, and displaying yard signs (16). Behavior genetic studies, beginning with pioneering work on social and political attitudes (17, 18), have found that some of the variation in political and economic preferences can be statistically accounted for by genetic factors (19–24).

We use a new sample of comprehensively genotyped subjects from the Swedish Twin Registry. These subjects were recently administered, as part of a survey called Screening Across the Lifespan Twin survey, Younger cohort (SALTY), a rich set of questions measuring economic and political preferences. We study four fundamental economic preferences—risk aversion, patience, trust, and fair-mindedness—and five dimensions of political preferences, derived from a factor analysis of a comprehensive battery of attitudinal items. The five attitudinal dimensions are immigration/crime, economic policy, environmentalism, feminism/equality,

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and foreign policy. We also study educational attainment because, even though it is probably more distal from basic biological processes, it is available for a larger sample of genotyped individuals. For comparability with previous work and with our other estimates, we report twin-based estimates of heritability from this new sample, and we confirm moderate (30–40%) twin-based heritability estimates for these traits. However, our main focus is on using the dense single nucleotide polymorphism (SNP) data to learn about the genetic architecture of these traits.

As a first step, we use a new method (25–27) that uses the dense SNP data to estimate the proportion of variance in these traits that can be jointly explained by the genotyped SNPs. The technique—which we will call genomic-relatedness-matrix restricted maximum likelihood (GREML)—has been applied to height (25), intelligence (28, 29), personality traits (30), several common diseases (31), and schizophrenia (32), but never before to economic and political phenotypes. GREML provides a lower-bound estimate of narrow heritability that does not rest on the same set of assumptions relied on in twin studies. A key assumption behind GREML is that among individuals who are *not* in the same extended families, environmental factors are uncorrelated with differences in the degree of genetic similarity, or "relatedness." In this analysis, genetic relatedness is directly *estimated* from the SNP data, unlike in behavior genetic studies, where *expected* relatedness (inferred from the family pedigree) is used. (Another, distinct approach is to estimate the genetic variance from within-family variation in genetic relatedness; see ref. 33.) A common concern raised about behavior genetic studies of political and economic traits is that expected relatedness could be correlated with similarity in environmental factors that are not endogenous to genotype (as defined by Jencks; ref. 4). Because there is more random variation in the realized degree of genome sharing relative to the expected degree as the expected relatedness declines (34), environmental confounding is less likely to drive estimates that are based on realized relatedness among individuals whose expected relatedness is negligible.

Under the key assumption of no environmental confounding, an estimator for heritability can be obtained by examining how the correlation in phenotype between pairs of individuals relates to the realized genetic distance between those individuals. It would be an unbiased estimator of narrow heritability if genetic distance were calculated using all of the genetic variants that are causal for the phenotype. In practice, because the causal variants are not known, the SNPs typed on the genotyping chip are used to estimate genetic distance. Because these SNPs are only imperfectly correlated with the causal variants, relatedness with respect to the causal variants is measured with error. Consequently, the estimated relationship between phenotype and genetic relatedness is attenuated, and hence the estimator is a lower bound for narrow-sense heritability (25). Our GREML-based heritability estimates, although noisy, are on average about half the size of the twin-based heritability estimates. This gap may imply that genotyped SNPs tag about half of the genetic variation in these traits or that twin-based estimates of narrow heritability are biased upward (35, 36).

We next explore the molecular genetic architecture of the phenotypes. Specifically, we estimate heritability using relatedness measured separately by chromosome to test how evenly distributed the genetic effects are across the genome. We supplement these results by reporting findings from a standard genome-wide association study (GWAS) for each trait, in which individual SNPs are tested for association with the outcome of interest. Finally, we also perform a risk prediction exercise in which we randomly split the dataset into a discovery and a validation sample. We use a pruned set of SNPs from the discovery sample to build a predictor and then examine to what extent the predictor is correlated with the outcome in the validation sample. Similar approaches have been applied in the study of schizophrenia (37), height (38), and intelligence (28), but none of these methods have been applied to economic or political preferences. We find essentially no predictive power for the traits we study.

Our results paint a picture of economic and political preferences as highly polygenic traits for which individual SNPs explain only a small fraction of variance. The inferential challenges implied by this genetic architecture suggest—as we later discuss—that new approaches are needed to study genetic influences on preferences and behavior.

Results

As a preliminary, we computed the sibling correlations for all 10 variables (*SI Appendix*, Table S4). (Details on variable construction and materials and methods are available in *SI Appendix*.) The sibling correlations for the SALTY questions on patience (39), risk aversion*, and political preferences† have previously been analyzed and are reported to facilitate comparison with the GREML estimates. The implied heritabilities of the economic preferences are typically about 30% and the estimates for political preferences are typically around 40%.

We next estimated, for each trait, the proportion of phenotypic variation accounted for by measured SNPs, using the GREML estimator (25–27). These lower-bound heritability estimates for the nine traits are reported in Table 1. These analyses are all based on mixed-sex samples, controlling for sex, birth year, and the first 10 principal components of the genotypic data. (The fact that we observe some GREML point estimates of zero is not surprising. Because the estimator is constrained to produce a nonnegative estimate, the bound at zero will often be attained when the true population parameter is low and estimated imprecisely.)

For economic preferences, only one of the four variables, trust, is significant, with the point estimate suggesting that the common SNPs explain over 20% of phenotypic variation (P = 0.047). The remaining effects are lower, in one case zero, and not statistically distinguishable from zero. For political preferences, two of the five GREML estimates for the derived attitudinal dimensions are statistically significant, and one is marginally significant: 0.203 (P = 0.079) for immigration/crime, 0.344 (P = 0.012) for economic policy, and 0.354 (P = 0.009) for foreign policy. Keeping in mind that the GREML estimates are noisy and are lower bounds, taken as a whole they are consistent with low-to-moderate heritabilities for these traits. The cumulative effect of the SNPs is much more precisely estimated for educational attainment because this phenotype is available for all of the genotyped individuals in the sample. For this phenotype, our estimate is 0.158 (P = 0.004).

To help interpret our findings, we also report retest reliabilities for the preference measures using data from 491 respondents who answered the survey twice. We find that the political preferences were on average measured more reliably than the economic preferences. This finding is consistent with previously reported retest reliabilities for the risk questions* and the political preferences† on the SALTY survey. The reliabilities are given in the bottom two rows of Table 1. The GREML estimates are noisier (and the P values tend to be higher) for phenotypes with lower retest reliabilities.

We also conducted the GREML analyses separately by chromosome (as in refs. 40 and 28). Between conventionally unrelated individuals, realized relatedness is random and independent across chromosomes, and the expected relatedness measured from any chromosome is zero. If the genetic variation that predicts a trait were uniformly distributed across the genome, rather

*Beauchamp JP, Cesarini D, Johannesson M (2012) The Psychometric Properties of Measures of Economic Risk Preferences. Working Paper, Harvard University, New York University, and Stockholm School of Economics.

†Oskarsson, et al. (2010) Do Genes Mediate the Relationship between Personality and Ideology? Uppsala University Working Paper.

Table 1. GREML estimates of narrow heritability

	Economic preferences					Political preferences					
	Education	Risk	Patience	Fairness	Trust	Imm./crime	Econ. policy	Environ.	Femin./equal.	Foreign policy
$V(g)/V(P)$	0.158	0.137	0.085	0.000	0.242	0.203	0.344	0.000	0.000	0.354
SE	0.061	0.152	0.148	0.150	0.146	0.147	0.150	0.148	0.147	0.149
P value*	0.004	0.186	0.285	0.500	0.047	0.079	0.012	0.500	0.500	0.009
N	5,727	2,327	2,399	2,376	2,410	2,368	2,368	2,368	2,368	2,368
Chrom.	0.442	0.118	–0.195	–0.111	0.460	0.118	0.496	–0.311	0.247	0.462
P value†	0.039	0.601	0.385	0.623	0.031	0.601	0.019	0.159	0.268	0.030
ρ_{retest}‡		0.71 (0.66–0.76)	0.40 (0.27–0.52)	0.57 (0.49–0.65)	0.63 (0.57–0.69)	0.86 (0.84–0.89)	0.85 (0.81–0.87)	0.62 (0.53–0.69)	0.78 (0.74–0.82)	0.70 (0.65–0.75)
N_{retest}‡		475	483	469	482	471	471	471	471	471

GREML estimates for the 10 variables are reported. We estimated the matrix of genetic relatedness after omitting one twin per pair and then restricted the analyses to individuals whose relatedness did not exceed 0.025 in absolute value. Chrom. (chromosome) shows the estimated correlation between chromosomal length (measured in centimorgan) and the proportion of variation explained by relatedness estimated from that chromosome. Imm., Immigration; Econ., Economic; Environ., Environmentalism; Femin./equal., Feminism/equality.

*One-tailed likelihood ratio test of the hypothesis that the proportion of variation explained by common SNPs on the autosomes is zero.

†Test of the null hypothesis that, across the 22 autosomes, the correlation between chromosomal length and the proportion of variation explained by the chromosome is zero.

‡These rows show the estimated retest correlations (with 95% confidence intervals) and sample sizes for the retest correlations. For sample descriptions, see *SI Appendix*.

than being concentrated in a particular location, then greater realized relatedness from any given chromosome will predict greater phenotypic similarity, and this association will be stronger from longer chromosomes because longer chromosomes make up a larger fraction of the genome than shorter ones. Table 1 shows the estimated correlation between chromosomal length, measured in centimorgans, and the fraction of variance explained by the estimates of realized relatedness estimated using only data from one chromosome. The correlation is positive for 7 of 10 phenotypes, significantly so ($P < 0.05$) in four cases. Positive correlations have previously been reported for height (40), cognitive ability (28), and schizophrenia (32) and have been interpreted as evidence that the trait is highly polygenic with causal variants distributed across the genome.

Next, we tried to identify individual SNPs that predict economic and political preferences. For none of the 10 traits did we identify any SNPs that pass the conventional genome-wide significance threshold of $P < 5 \times 10^{-8}$ (41). In fact, no single SNP attains a P value lower than 10^{-7} for any of the 10 traits. The standard diagnostic for population stratification (i.e., ethnic confounding) in GWAS is inflated test statistics in the Q-Q plot (e.g., ref. 42); there is no evidence of inflated test statistics across the traits, with estimated λs (43) in the range of 0.987 (environmentalism) to 1.014 (educational attainment). While this diagnostic check suggests that our controls for population structure worked well, it is somewhat surprising that there is no systematic tendency for the λs to be larger than 1, given that some inflation is expected under a polygenic model even without any stratification (44). However, more inflation is expected in a larger sample, and λs have not been much larger in other analyses with comparable sample sizes to ours (28). In *SI Appendix*, we provide details on the full set of SNPs with P values $<10^{-5}$ for the nine preference measures, but we are skeptical that any of these associations will be replicable.

Finally, we examined the aggregate, out-of-sample predictive power of the SNPs. Following ref. 37, we first estimate the regression coefficient for each SNP in a discovery sample, composed of a randomly drawn 90% of the sample. From this set of coefficients, we form a prediction equation on the basis of a pruned set of 111,957 markers that includes only SNPs that are approximately in linkage equilibrium (to avoid double counting SNPs that are correlated with other SNPs). In a validation sample composed of the remaining 10%, we evaluate the correlation between individuals' predicted phenotype and observed phenotype. Although the correlation between the predicted and observed phenotype is positive, as expected, in 7 cases out of 10, we do not find quantitatively appreciable out-of-sample predictability for any of the traits: For most phenotypes, the explanatory power of the predictor is well below $R^2 = 0.1\%$. These results are reported in *SI Appendix*.

Discussion

The data reported here reveal a number of descriptive facts about the heritability and genetic architecture of political and economic preferences. First, we estimate sibling correlations for several traits, some of which have never before been studied in large samples, and we confirm that there is a robust separation of the monozygotic (identical twin) and dizygotic (fraternal twin) correlations. We obtain heritability estimates that are consistent with typical estimates previously reported for both political attitudes (19) and economic preferences (20, 23, 24), as well as educational attainment (45, 46). Overall, these results are consistent with the hypothesis that, for each of the 10 phenotypes, there exists a moderate correlation with genetic factors. None of the sibling correlations are adjusted for measurement error. A plausible conjecture is that the lower heritabilities of the economic preferences relative to the political preferences result from attenuation bias due to greater measurement error in economic preferences, as evidenced by the lower test–retest reliabilities of the economic preference measures.

Second, our molecular-genetic–based estimates of heritability partially corroborate the twin-based estimates and suggest that molecular genetic data *could* be predictive of preferences if the causal variants were known. When we estimate the cumulative effect of genotyped SNPs using GREML (25–27), we find that the estimated heritabilities are lower than the twin-based estimates, but the overall pattern of results suggests that point estimates are generally nonzero and, for the better measured variables, statistically distinguishable from zero. Because there is a lively debate regarding whether twin studies of political behavior have established that there is heritable variation in these traits (35), we note that our evidence for heritability is based on different assumptions than twin studies.

Previous papers on height (25), intelligence (28, 29), personality traits (30), and several diseases (31, 32) have found that the SNP-based heritability estimates are between one-quarter and one-half the size of twin-study estimates. One interpretation of the gap is that genotyped SNPs tag less than half the additive genetic variation in those traits (which would occur if causal variants are imperfectly correlated with SNPs on the SNP arrays, e.g., because their allele frequencies are lower than those of the SNPs). The gap may also reflect an upward bias in twin-based estimates of narrow heritability due to environmental confounding (35) or nonadditive variation (36).

Do economic and political preferences parallel other phenotypes in having SNP-based heritabilities that are half or less the magnitude of the twin-study estimates? If so, it would suggest that economic and political preferences have a similar genetic architecture, a similar degree of bias in twin-based estimates, or both. Because the economic and political preference measures have twin-based heritabilities around 0.30 (20) and 0.40 (19), respectively, the hypotheses of one-half magnitude would be GREML point estimates of around 0.15 and 0.20. Our evidence, considered in its entirety, is not inconsistent with these hypotheses, but the point estimates are quite noisy. An alternative approach is to examine the number of statistically significant associations. For economic preferences, if the SNP-based heritability parameter in the population is 0.15, and if sample estimates have a SE of 0.15 (as suggested by Table 1), then our power to statistically reject the null hypothesis of zero heritability in a one-sided test at the 5% level is about 26%. For political attitudes, if we assume a SNP-based heritability parameter in the population of 0.20, and we assume a SE of 0.15 (again as suggested by Table 1), then the corresponding statistical power is about 38%. If the traits are independently distributed, this calculation implies that for the nine preference variables, we should expect to observe 2.9 significant associations at the 5% level. In fact, we observe three significant associations at the 5% level and one more at the 8% level. The results, therefore, are close to what one would expect under the hypothesis that the SNP-based heritability estimates are about half the magnitude of the twin-based estimates.

Third, our analysis of individual SNPs does not reveal any associations that are significant at the conventional threshold of genome-wide significance required in genetic association studies. This is unsurprising in light of the accumulating evidence that the effects of common variants on complex outcomes are small (47), especially in the context of social science traits (1, 2). *SI Appendix*, Fig. S1 displays power calculations, given the SALTY sample size, for detecting true associations across a range of effect sizes as measured by the R^2. For the preference measures, the study was well powered to detect individual markers that explain at least 1.25% of trait variation at a nominal significance level of 10^{-7}. No single SNP in our sample attains this level of significance—the lowest P value we observe is 1.1×10^{-7}. Moreover, 1.25% is an upper bound to the effect sizes we can rule out because: first, because 1.1×10^{-7} is the smallest of many millions of P values we estimated, it almost surely capitalizes on chance to some extent and overstates the strongest genetic association in our data [the well-known "winner's curse" in statistical inference (48)]; and second, for many of the variables, the lowest observed P value was considerably higher than 10^{-7}. To illustrate our statistical power another way, if across the nine preference measures there are a total of 10 independently distributed SNPs each with R^2 of 0.75% or larger, our study had statistical power greater than 90% to detect at least one of them—and yet we found none. We conclude that it is unlikely that many common polymorphisms with such effect sizes exist. *SI Appendix*, Fig. S1 shows that the study was even better powered for educational attainment. Hence our failure to detect associations at these levels of significance indicates that true associations between common SNPs and economic and political phenotypes are likely to have very small effect sizes. [Whereas the survey measures we use here are common in economics (e.g., ref. 11), it is also common in economics to measure preferences using laboratory tasks that attach financial incentives to performance (49). As we explain in *SI Appendix*, our conclusion that the effect of individual SNPs on preferences are very small would hold even if measures of preferences were much more reliable than those we use here.] Of course, our evidence does not rule out the possibility that there exist rare variants with large effects on these phenotypes because sufficiently rare variants will have low correlations with the genotyped SNPs. Because such variants would be rare, however, a large sample would be required to detect them, as well.

Fourth, the results from our prediction exercise show that a standard polygenic risk score estimated in our sample has negligible out-of-sample predictability. This finding does not in any way contradict the results from the GREML analysis. GREML uses the measured SNPs to estimate realized relatedness between individuals, and given the large number of SNPs in a dense SNP array, realized relatedness can be estimated relatively precisely. In contrast, estimating a prediction equation that can predict well out of sample requires precise estimates of the effects of individual SNPs. In the limit of an infinite sample, it would be possible to perfectly estimate the effects of individual SNPs and thereby construct a polygenic risk score whose predictive power reaches the theoretical upper bound that is estimated by GREML. The smaller the discovery sample used to estimate the prediction equation, the noisier are the estimates of the individual SNP effects, and hence the lower will be the out-of-sample predictive power of the polygenic risk score that is constructed on the basis of these estimates. Evidently a discovery sample of 2,900 individuals (about 90% of 3,200) is far too small to obtain predictive power for standard measures of economic or political preferences.

These findings fit well with an emerging consensus in medical genetics that genetic variants that individually explain a substantial share of the variation in complex traits are unlikely to exist. If anything, the problem is likely to pose an even greater challenge in the social sciences because the phenotypes are usually several degrees removed from genes in the chain of biological causation (1–3). Our results suggest that much of the "missing heritability" (50)—the gulf between the cumulative explanatory power of common variants identified to date and the heritability estimated in behavior genetic studies—for social science traits reflects the fact that these traits have a complicated genetic architecture, with most causal variants explaining only a small fraction of the phenotypic variation. If so, then large samples will be needed to detect those variants.

Turkheimer (51) famously proposed three "laws of behavior genetics": first, all human behavioral traits are heritable; second, the proportion of variance attributable to common environment is smaller than the proportion attributable to genes; and third, a large portion of individual differences is explained by factors other than common environment and genes. We believe that there is accumulating evidence in favor of a fourth "law" regarding the molecular genetic architecture of behavioral traits: Genetic variants that are common in a population have very small individual

effects on behavioral traits. If true, this law would help explain the repeated failure to replicate initially promising candidate gene findings with large effect sizes (29, 52, 53), as well as the failure to date of genome-wide association studies to discover genetic variants associated with behavioral traits even in samples numbering tens of thousands of individuals (54). There is direct evidence for such an architecture for intelligence (28, 29), personality (30), and now economic and political preferences. Like Turkheimer's three laws, this fourth law is a summary of patterns of empirical results, not a theoretical necessity, so it could fail to hold in some specific cases, but we conjecture that it will generalize to most other behavioral phenotypes.

Our conclusions have a number of implications for research at the intersection of genetics and social science. There has recently been an explosion of reported associations in samples that are very small by the standards of medical genetics. Such studies are only adequately powered if the genetic marker's population R^2 for the trait is considerably larger than the upper bounds established by the GWAS findings reported here. Our findings, based on a sample an order of magnitude larger than most existing studies, suggest that adequate power actually requires a sample size that is yet another order of magnitude larger even than ours (1, 2, 55).

Published genetic associations with economic and political traits, even if statistically significant, should be approached with caution for two reasons. First, because most published studies are dramatically underpowered, the probability that an association study will detect a true signal is vanishingly small; hence, when a significant association is observed, Bayesian calculations indicate that the posterior odds that it is a true association are low (1, 2, 52). For example, Benjamin et al. (1) show that under reasonable assumptions about genetic effect sizes on economic traits, an observed association that is statistically significant at the 5% level in a sample of a few hundred individuals constitutes virtually no evidence in favor of a true effect—yet such reported associations are typical of most published genetic studies of social science traits. Second, publication bias—the tendency for findings, as opposed to nonfindings, to be selectively reported by researchers and selectively published by journals—are magnified in genetic association work because the typical dataset has many behavioral measures and many genetic markers (56). Testing for gene–environment interactions further compounds the problem of multiple hypothesis testing (57).

Our conclusions regarding the molecular genetic architecture of economic and political preferences also have implications for whether, how, and how soon molecular genetic information can contribute to, and potentially transform, research in social science. One possibility is that genetic associations may shed light on biological pathways through which precursors, such as preferences, lead to important behaviors and outcomes. More speculatively, such insights may also help inspire the development of new theoretical constructs that are more closely aligned with the underlying biology than the existing concepts such as "risk aversion" or "patience" that we study here (1, 3). Contributions such as these require the identification of specific genetic variants that correlate robustly with behavior. As discussed above, the results reported here suggest the need to analyze samples which are several orders of magnitude larger than those presently used in this sort of research if such markers are to be successfully identified. Unfortunately, even if these markers are eventually identified, our quantitative results suggest that many of them will only explain a tiny share of variance. Moreover, it is possible that such markers will be too far removed from the behavioral trait in the chain of causation to elucidate the biological pathway.

Another potential contribution to social science, already being actively pursued (e.g., 5, 6), is the use of genetic markers as instrumental variables in (nongenetic) empirical work. In order for the gene-as-instrument to be valid, not only must the marker be robustly associated with the "endogenous regressor," but *all* of the behaviors associated with that marker must be understood. Otherwise, if the marker has pleiotropic effects, then the exclusion restriction assumption could be violated, invalidating the instrumental variable application. As more is understood about genetic pathways, researchers will be in a better position to assess the plausibility of the exclusion restriction assumption on a case-by-case basis.

A different potential use of molecular genetic data in social science would be as control variables for genetic heterogeneity in (nongenetic) empirical work, to reduce the variance of the error term and shrink the SEs of coefficient estimates. For such an application to have practical utility, the markers that are selected as controls need to explain a nonnegligible share of the variation. Similarly, use of genetic data to target interventions requires that the aggregate predictive power of a set of genetic variants for the trait be sufficiently large. As we have shown here, given presently attainable sample sizes, the use of genetic data to predict economic and political traits does not appear to be feasible. It is likely that extremely large—perhaps impractically large—samples will be required. (Were it the case that economic behaviors, or their precursors in the form of various preferences, traits, and skills, could be predicted from molecular genetic information, it would raise a host of ethical questions about whether and how such information should be used. The potential benefits we have emphasized here must be carefully weighed against the costs, for example, discrimination based on genotype or the breakdown of insurance markets due to adverse selection.)

In summary, our molecular-genetic–based estimates of heritability partially corroborate the twin-based estimates and suggest that molecular genetic data could, in principle, be predictive of preferences. Our other results, however, suggest that excitement about the practical usefulness of molecular genetic data in social science research needs to be tempered by an appreciation that much of the heritable variation is likely explained by a large number of markers, each with a small effect in terms of variance explained. As a consequence, for economic and political preferences, much larger samples than currently used will be required to robustly identify individual SNP associations or to generate sizeable predictive power from many SNPs considered jointly.

Rather than being destructive to the enterprise of incorporating genetic data into social science, our conclusions regarding the molecular genetic architecture of economic and political preferences can help guide research in more productive directions. First, researchers could obtain very large samples that contain both genetic and social science data. Second, to minimize attenuation bias due to error in measurement and thereby maximize power for any given sample size, researchers could formulate more reliable measures of economic and political phenotypes. Third, researchers could focus on behavioral phenotypes that are more biologically proximate. One example is smoking, a behavior for which large, replicated associations have been found with SNPs in the nicotinic acetylcholine receptor gene *CHRNA3* (58). For such biologically proximate phenotypes, it is more likely that there exist genetic markers whose associations will have nontrivial effect sizes and clearer causal interpretations.

Materials and Methods

Beginning in December 2010, a total of 9,836 Swedish twins who passed initial laboratory-based quality controls were genotyped using the Illumina HumanOmniExpress BeadChip genotyping platform. We applied standard quality controls to the genetic data. In all our GWAS analyses, we controlled for sex, birth year, and the first 10 principal components of the genotypic data, and we adjusted the SEs for nonindependence within family. We computed the GREML estimates using the publicly available GCTA software (26). Before computing the matrix of genetic relatedness for the SALTY sample, we dropped one twin per pair, always the twin with a larger number of missing phenotypes. We used a relatedness threshold of 0.025. For our prediction exercise, we randomly split the sample into a 90% training sample to construct the genetic score and a 10% validation sample to examine its predictive accuracy. See *SI Appendix* for all details on the sample and methods.

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