OLS is AOK for ACE: A Regression-based Approach to Synthesizing Political Science and Behavioral Genetics Models

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Abstract

There is a growing interest in empirically exploring the biological underpinnings of political attitudes and behavior. Heritability studies are a primary vehicle for conducting such investigations and data sets rich in political phenotypes are becoming broadly accessible. A bottleneck exists, however, in exploiting these opportunities because they involve a statistical re-tooling for political scientists and require a conceptual shift that has substantial implications for the field's traditional theoretical models. Methodologically, most twin studies rely on structural equation models unfamiliar to political scientists. We show this methodological bottleneck is easily navigable. However, the lesser discussed shift in theoretical assumptions poses the larger problem to integrating biological elements into the study of political attitudes and behavior. To address these lacunae, we provide a detailed explication of a regression-based method, which foundations are familiar to all political scientists with even minimum quantitative training, as a suitable platform to analyze genetic influence on political attitudes and behaviors. In doing so, we provide a necessary platform for bridging important conceptual divides between political science and behavioral genetics.

Introduction

A number of recent high-profile studies in political science argue that behavior and attitudes are driven by biological as well as environmental influences (Alford, Funk and Hibbing 2005; Fowler and Dawes 2008; Hatemi et al 2010a). This incorporation of biological influences is viewed as a fundamentally new approach to systematically studying political attitudes and behavior, and potentially a harbinger of a paradigm shift (Fowler and Schreiber 2008).[1]

The central aim of this rapidly expanding literature is not to replace environmental determinism with biological determinism, but to empirically demonstrate that explanatory accounts of political attitudes and behavior must take seriously the notion that biology's causal role is greater than zero (Smith and Hibbing 2008; Hatemi and Hannagan 2008). As specific genes (Dawes and Fowler 2009; Hatemi et al 2010b), brain activation patterns (Amodio 2007), hormone and neuropetide levels (Madsen 1985; Zak et al 2005; De Dreu et al 2010), and physiological responses (Oxley et al 2008) systematically correlate with various social/political attitudes and behaviors, it seems a safe bet to assume that at least some portion of the variation in traits of key interest to the discipline has biological roots.

Behavioral genetics research has been the primary catalyst for the growing empirical exploration of biological influences on political preferences and was directly responsible for triggering a broader debate on the integration of biology into conceptual frameworks in political science (Alford, Funk and Hibbing 2005; Charney 2008; Hatemi and McDermott 2011; Fowler and Dawes 2008; Smith and Hibbing 2008, Smith et al 2010). However, integrating behavioral genetic approaches and findings into traditional conceptual frameworks to the study of attitudes and political behaviors presents three significant issues for political science: (1) Data availability,

[1] It is important to note that some political scientists have been calling for just such a shift for more than thirty years (e.g. Somit 1972; Masters et al 1984). With few exceptions, however, these calls were been largely ignored by the discipline's mainstream until relatively recently.

(2) methodological exposure, and most importantly, (3) successful synthesis of two very different theoretical frameworks.

In this paper we demonstrate that all three of these challenges are navigable. The first two challenges are minimal barriers easily hurdled by anyone with minimal quantitative training and an internet connection. We begin with a discussion of recent advances in data availability that provides specific resources for political scientists to gain access to twin data. We then follow with a review of the empirical and theoretical models in both behavioral genetics and political science through the explication of a regression based approach to twin modeling. We use this approach in a didactic manner to illustrate the theoretical distinctions between social science and biometric models. In doing so we address the third and most significant challenge, i.e. forging a comprehensive synthesis of the biometric model that frames heritability analyses with the traditional causal frameworks generated in political science. While making no pretensions that this challenge can be resolved to universal satisfaction within the confines of a single paper, we, achieve this synthesis by providing original empirical demonstrations within the regression context that has long served as basis for conceptualizing, testing and refining causal models in political science. We conclude by showing this approach has the signal advantage of clarifying key conceptual issues raised when attitudes and behaviors are treated as at least partially genetically influenced, a finding that has broad implications for how we interpret not just contemporary and future research, but how we interpret extensive existing empirical literatures in political science.

Challenge One: Getting Data

The most practical approach to investigating genetic influence on political attitudes and behaviors is to match known genetic variance among individuals with observed variance in their political traits (i.e. twin-based studies). The basic logic here is that if people more genetically similar to each other are also more similar on political (or any other observed) trait, it provides evidence that trait is genetically influenced. These sorts of heritability studies are the work horse of empirical research in behavioral genetics and are capable of providing fairly precise estimates of the degree of genetic influence on a target trait. In terms of data, all they require is a relatively large sample of twins which generally means using a population registry.

Yet while population registries have existed around the world for decades, they were rarely been employed to collect data on a broad range of political traits. A handful of behavioral geneticists have been sporadically examining the heritability of ideology (more specifically, conservatism) for four decades (Eaves and Eysenck 1974; Martin et al 1986). Historically, however, political traits were never the focus of data collection in this field. Alford, Funk and Hibbing's (2005) study on the heritability of issue attitudes, for example, relied on a 20-year-old data set (VA30K) that included few other political traits and was difficult to access. Happily, the data availability situation has changed radically in the past five years as comprehensive, politically-oriented data collected on twins have become publically accessible. For example, the Minnesota Twins Political Survey (NSF 0721378) includes hundreds of variables and is freely available for anyone to download with an internet connection (http://www.unl.edu/polphyslab/data.html). Similar data collection efforts have been completed in Australia (Hatemi et al 2010c) and Denmark (Klemmensen et al 2010; for an introduction to new political twin studies see Hatemi 2012). Combined with publically available studies that contain political traits, such as the National Longitudinal Study of Adolescent Health (http://www.cpc.unc.edu/projects/addhealth) and the National Survey of Midlife Development in the United States (http://www.midus.wisc.edu/), twin data on political traits is becoming increasingly available.

Limitations do continue to exist, even in the most easily accessible data. For example, the Minnesota Twins study, to date one of the most comprehensive survey of political traits done using a twin registry, will not include every variable desired by political scientists, and the subjects lack significant variation on potentially important socio-demographic characteristics (notably race, age and geography). Even so, there is no doubt that a wide range of twin data on political traits is now readily available from several sources, and that data sources and accessibility will continue expand. In short, while far from perfect, data availability and accessibility is no longer an obstacle to conducting heritability studies on an increasingly wide range of political behavioral and attitudinal traits.

Challenge Two: Methods

Gaining access to data is only half the battle; interpreting and executing heritability studies requires applying a particular type of statistical model to the target trait. Twin studies have been developed by psychologists and behavioral geneticists, who primarily rely on structural equation modeling (SEM) approaches to analyze twin and kinship data. The primary approach of political scientists doing twin studies has been to "pull" these methods from behavioral genetics research and direct them at political traits. Unfortunately, SEM approaches are unfamiliar to many political scientists; for the uninitiated they can be difficult to interpret let alone apply, and this unfamiliarity is exacerbated by the special type of structural model needed to analyze twin data. Learning these methods demands a significant methodological retooling for a traditionally trained political scientist (for a primer see Medland and Hatemi 2009). Practically speaking, applying SEM methods also requires competence in specialized software packages specifically built and designed for this sort of analysis (Mx and/or OpenMx). Gaining that competence may require a significant investment in addition to gaining an understanding of the technical underpinnings of the model (Mx has a native scripting language, OpenMx requires a familiarity with the R statistical language).

Yet there are methodological alternates to SEM that might be more familiar to political scientists, including a Pearson-Aitken approach (Hawke et al 2008) and Bayesian approaches (Fowler, Baker and Dawes 2008). We provide an introduction to one of these alternates, developed by Defries and Fulker (1985), that uses nothing more than basic ordinary least squares regression (OLS), i.e. a methodology familiar to any political scientist with even modest quantitative training. Defries and Fulker's OLS-based approach to twin analyses (so-called DF analysis or DF modeling) has a key set of advantages for a social scientists with no extensive tradition of including biology in its empirical analyses. DF analysis requires no new methodological training, can be conducted on any statistical software that can do regression, and, perhaps most importantly, serves as a practical and intuitive platform for empirical and theoretical synthesis: DF models can readily analyze the genetic basis of a wide variety of political phenotypes while simultaneously incorporating and extending the environmental-based body of knowledge created by political science over the past 50 years. And, as we discuss later in the paper, in doing so DF models also highlight and clarify a set of conceptual issues related to incorporating biometric theory into political science causal frameworks that has yet to be explicitly addressed by the extant literature. Apart from a working understanding of regression, then, all that is required to begin using now widely available data to examine genetic influences on political traits is basic grasp of the logic underlying biometric theory and how that framework translates into regression terms.

The Logic of Biometric Theory

Using heritability studies as a basic platform to investigate the biology of political traits requires recognizing that theory in behavioral genetics is fundamentally different from theory in political science in at least two ways. First, unlike political science which is notorious for its theoretical heterodoxy and competing causal frameworks, behavioral genetics has a universal conceptual model. Second, the explanatory target of this universal framework is fundamentally different than the explanatory targets of conceptual frameworks in political science.

In political science conceptual frameworks typically take the form of a particular set of independent variables (X) that are theoretically posited to cause a dependent variable (Y). There are some "usual suspect" variables that are reasonably consistent inclusions in vector X for models seeking to explain a given Y (e.g. education, gender). The mix of variables of X, though, can vary wildly with causal frameworks; there may be multiple competing causal frameworks for any given Y, and completely different set of causal models (packages of X) for another Y. This is in stark contrast with behavior genetics research. Rather than formulating sets of competing environmentally-based theories and testing the hypothesis that a measured variable X correlates with (and is theoretically presumed to cause) a measured variable Y, twin studies are anchored, like all of quantitative genetics, on a general biometric theory. This theory assumes that any given phenotype, i.e. any observed or measured trait, is a product of environmental and genetic factors. Assuming independent (no interactions) contributions of genes and environment to phenotype, the basic quantitative genetic model thus takes the form: P = G + E. The objective of statistical modeling from the biometric theory perspective is to uncover the genetic and environmental causes of the phenotype (P), and the central obstacle in achieving this objective is that while P is observed, G and (often) E is not.

The genetic and environmental etiology of P is most commonly estimated using what is known as the variance components approach, where the observed variance of P is partitioned into latent (unobserved) environmental and genetic factors (see Neale and Cardon 1992). Operationalization of the model is achieved by using data from related individuals, traditionally twins though extended family designs are increasingly common (see Eaves and Hatemi 2008; Hatemi et al 2010a).

By using genetically related individuals variance in P can be partitioned into variance shared by family members and variance unique to the individual (conceptually, this process is analogous to the within/between variance partition in a standard ANOVA). Variance in P can theoretically be decomposed into four components, two genetic and two environmental. The genetic components are additive genetic influences (A), or the summative influence of individual alleles, and non-additive (D) genetic influences, which represent interaction effects between genetic markers. The environmental components are shared environment (C) or all non-genetic influences that twins share, and everything non-genetic that makes them different (E). Assuming no interaction between genes and environment, then, the simplest operationalization of the basic P = G + E quantitative genetic model is: P = A + D + C + E.

However, if the data being used are restricted to twins raised together, which is often the case, the P = A + D + C + E model cannot be operationalized because C and D are confounded. This is because mathematically the exact same piece of information -- the extent to which dizygotic (fraternal, or DZ) twin trait similarity is more or less than half the monozygotic (identical, or MZ) similarity -- is used to estimate both parameters, thus C can mask D and vice versa (these confounds can be disentangled by using extended family designs; for technical details see Eaves et al 1977, Rijsdijk and Sham 2002). If data is limited to twins reared together (as it is in most twin data sets), then the practical choice is to operationalize either an ACE or an ADE model. Choosing between the two typically rests on whether non-additive genetic effects (D) or common environment (C) is seen as having the greater influence on the target trait.

The basic rule for assessing the relative influence of D versus C is to compare DZ and MZ correlations on the target trait. If the DZ correlation is less than half of the comparable correlation of MZs, non-additive genetic effects are indicated. This is because we expect the correlation of D to be perfect between MZ twin pairs, i.e. because they are genetically identical we expect them to share the same non-additive genes. So if the target trait is significantly influenced by non-additive genetic sources we expect MZ twin pairs to be much more alike on that trait than DZs who, on average, will share less than half of their non-additive genes. On the other hand, if the more dominant influence on the target environment is C rather than D we expect DZs to look much more like MZs. This is because both types of twin pairs have the same shared environment; a DZ twin has the same shared environment (e.g., school, home, income, neighborhood, etc) as their DZ sibling, just as an MZ twin has the same shared environment as their MZ sibling. Thus if the DZ correlation on the target trait is more than half of the comparable correlation for MZ twins, C is seen as the best variance component to model. If the DZ correlation is less than half of the MZ correlation, dropping C and modeling D is justified.

The P = A + C + E (ACE) model approach is, by far, the most common approach to estimating the genetic etiology of political attitudes and behaviors, and understandably so.

Elements of the common environment (e.g. family socialization) are of central interest to the study of political behavior and attitudes, so there may be good theoretical as well empirical grounds to opt for an ACE as opposed to an ADE approach. Accordingly, we limit our discussion to the operationalization of ACE models (for more on non-additive influence see Keller et al 2005).

Accurately partitioning variance of P into ACE latent factors depends on several key assumptions. In a classic twin design (CTD) utilizing data only from MZ and DZ twins these include:

1. The correlation for MZ and DZ pairs for E is zero (i.e. rE for MZs = 0, and rE for DZs = 0). This is largely true by definition: E is defined as whatever makes twins different on P.

2. The correlation for MZ and DZ pairs for C is 1. This is the equal environments assumption (EEA), which assumes that shared environments affect MZ and DZ pairs equally, at least as it relates to the phenotype being studied (see discussion above). While a particular target of critics of heritability studies in political science (see Charney 2008; Beckwith and Morris 2008), a series of empirical studies conclude the assumption is warranted for political traits (for more on the EEA see Hatemi et al 2009a; Littvay 2012; Smith et al 2012). Regardless, we are not seeking to resolve the debate over the tenability of this assumption here. We simply note the importance of the assumption to the modeling approach (if the EEA is violated A is over- and C is under-estimated).

3. The correlation for genetic similarity (A) is 1.0 for MZs and (on average) .5 for DZs. This reflects expectations about population levels of genetic relatedness; MZs develop from a single fertilized egg and share 100 percent of their structural DNA. DZs develop from separate eggs and, like any other set of siblings, share an average of 50 percent of their DNA. The DZ average is based on an assumption of random mating. If individuals pick mates on the basis of phenotypic similarity (assortative rather than random mating) and the phenotype is genetically influenced, then DZs will be more genetically similar than assumed and A will be under-

estimated. Though attracting less attention from twin study critics, there are good reasons to assume that people *do* assortatively mate on the basis of political phenotypes (Alford et al 2010; Eaves and Hatemi 2008; Hatemi et al 2010a). If so, genetic influence on political traits is at least as likely (and probably more likely) to be underestimated as to be overestimated. [2]

Given twin data and these assumptions, it is possible to partition observed phenotypic variance in P into the latent A, C and E components. While the logic is universal, a number of different methods can be employed to generate the estimates for A, C and E. The simplest is the Falconer approach, which employs correlations—i.e. standardized co-variance/variance ratios--between MZ and DZ twins (Holzinger 1929; Falconer 1960). The correlation on a given phenotype for MZ twins (rMZ) is a function of what makes them similar, i.e. A and C. Under the assumptions of the twin model, MZ twins share all A and all C (r=1.0 for A and C), logically then, rMZ = A + C. DZs, on the other hand, share all of C, but only half of A. So, rDZ = A/2 + C. Given this, simple algebraic manipulation can isolate A by subtracting the equations for MZ the and DZ:

$$
\begin{aligned}
rMZ - rDZ \quad &= A - A/2 + C - C \\
&= rMZ - rDZ = A/2 \\
&A = 2(rMZ - rDZ)
\end{aligned}
$$

C can similarly be isolated, C = rMZ – A, or C=2rDZ – rMZ. As A, C and E represent proportions of variance they must sum to 1, so A + C + E = 1. Thus, a direct way to estimate E is to simply subtract A + C from 1; as A + C = rMZ, it follows that E = 1 – rMZ. Thus the observed variance in P is portioned into the latent components ACE and E.

The Falconer approach is an intuitive and straightforward method for calculating reasonable estimates of A, C and E (this is the method employed by Alford, Funk and Hibbing in

[2] These are not the only assumptions of the model. Classically there is also an assumption of no gene-environment interaction (which would require adding a GxE term), though these are increasingly being explicitly modeled. The external validity of finding from twin studies also rests on the assumption that twins are representative of the general population. For a more extensive introduction to the logic and assumptions of twin studies see Medland and Hatemi 2009.

their highly cited 2005 article). Yet this approach has important statistical limitations, notably the inability to test the significance of the estimates. How confident can we be that A, C and E represent meaningful estimates? As Falconer (1989, 175) points out, while it is a relatively simple matter to estimate genetic parameters from correlation coefficients, "it is perhaps more important to test whether the parameters are non-zero."

It was to achieve these ends, and also to incorporate explicit modeling of covariates, sex differences, unlike sex twin pairs, gene-environment interaction effects, multiple phenotypes, extended kinships, and specific genetic loci that more sophisticated statistical techniques were developed to decompose the variance in P. In behavioral genetics, the mainstream approach is structural equation modeling (SEM) that uses maximum likelihood estimators of ACE. The SEM approach operationalizes a CTD as a set of equations with known values that include observed variances-covariances on the observed trait, and assumed values taken from the expected genetic and environmental relationships between twins discussed above. The equations also include the latent parameters for A, C and E and the objective is to iteratively converge on a set of ACE estimates that have the highest probability of being true given the known and assumed values. Virtually all heritability analyses published in political science use either the Falconer or SEM approach (e.g. Alford, Funk and Hibbing 2005; Hatemi et al 2010a; Hatemi et al 2010b; Hatemi et al 2010c). The SEM approach addresses many of the shortcomings of the Falconer approach. It allows for statistical testing of ACE estimates, can include and generate point estimates for covariates and generally provides a much more robust and flexible platform for quantitative analysis in a CTD. The SEM approach, however, comes with a set of potentially important drawbacks for political science. These have little to do with its statistical capabilities (which are considerable), but rather with the investment required to competently execute these techniques, extract clear inference from what can easily become highly complex models, and effectively communicate the results to a political science audience unfamiliar with the necessary methodological and inferential context. At a minimum, this approach requires not just a familiarity with SEM, but the particular development and application of such techniques in behavioral genetics, as well as expertise in specialized software packages (Mx and OpenMx are probably the most widely used software packages in behavioral genetics, both were developed specifically for behavioral genetics applications, and, practically speaking, both require learning a scripting language). Finally, the SEM approach to twin models was developed specifically to operationalize the biometric model; this is not to say that political scientists cannot or should not learn these techniques. There are excellent primers on this approach, including at least one written specifically for political scientists (Medland and Hatemi 2008; see also Carey 2003).

. There is, however, an alternate approach to operationalizing a CTD that avoids the limitations of the Falconer approach, retains most of the advantages of the SEM approach, requires little in the way of methodological retooling, and makes for an elegant and intuitive platform for operationalizing traditional political science conceptual models, primarily because it uses the same methodological framework for testing models that political scientists have employed for decades.

The DeFries-Fulker Model

DeFries and Fulker (1985) developed a multiple regression approach to analyzing twin data (DF analysis or a DF model) that is simple, flexible, and provides unbiased estimates of genetic and environmental parameters equivalent to those generated by the maximum likelihood SEM approach (for a technical proof of why this is so see Rodgers and McGue 1994; see also Cherney, DeFries and Fulker 1992, Cherney et al 1992; Labuda, DeFries and Fulker 1986). Not only is this approach intuitive for anyone familiar with OLS regression, extensions of the model are capable of producing parameter estimates for specific environmental variables (not just umbrella estimates of C and E) that have particular relevance for political science.

The basic DF regression model takes the following form:

(1) $Y_1 = a + b_1Y_2 + b_2R + b_3 (R*Y_2) + e$

Y_1 is the phenotypic measure of one twin, Y_2 represents the phenotypic measure of the co-twin and R represents the coefficient of genetic relatedness (1.0 for MZs and .5 for DZs). By employing Y_2 as an independent variable the model is employing one sibling's score on Y to predict the co-sibling's score, thus Y_2 logically captures what makes twins similar. Under the logic of the quantitative genetic model such similarities can be due to environmental or genetic influences. The interaction term $R*Y_2$ captures the latter; it is simply a measure of how much of that similarity is conditioned on the sibling's level of genetic relatedness. With $R*Y_2$ accounting for variance reflecting genetic influence, Y_2 thus isolates the shared environmental influences that make twins more similar on the target trait. It should make intuitive sense that Y_2 and $R*Y_2$ capture, respectively, the concepts of C and A in the basic genetic model.

Essentially then, the DF model straightforwardly operationalizes the standard quantitative genetic model: $P = A + C + E$. In fact, the unstandardized regression coefficient b_1 is an unbiased estimate of C and b_3 represents an unbiased estimator of A (appropriate proofs can be found in Rodgers and McGue 1994; Labuda, Defries and Fulker 1986; for a particularly lucid non-technical description of why this so see Turkheimer et al 2005; 1223). These estimates of A (b_3) and C (b_1) can be tested for significance using the usual t tests, and R-squares can be used to assess impact on model fit. Given estimates of A and C, it is trivial to estimate E. In terms of standardized variance, P=1 and if $P = A + C + E$, then $E= 1 – (A + C)$. The coefficient for the R term (b_2) estimates the mean difference between twins and can be viewed as a basic test of the equal environments assumption (Rodgers and McGue 1994, 260). The coefficient b_2 captures the mean difference in Y between MZs and DZs after accounting for the impact of A and C. This term is frequently statistically insignificant, and if so b_2R is often neither reported nor interpreted. The intercept has its usual interpretation as an estimate of the dependent variable when all independent variables are zero.[3]

[3] The original DF formulation presented here is still widely employed, even though a number of extensions and refinements have been proposed. The best known of the latter is probably Rodgers and Kohler's (2005)

This model specification and interpretation should be intuitive to anyone familiar with multivariate regression, but there is one issue outside of basic OLS analysis that needs to be raised. This is centered on a data entry question: Which twin's score should be entered as the dependent variable (Y_1) and which should be used as the base measure of common environment (Y_2)? The typical approach to avoid this problem is double entering the data so that twins are represented on both sides of the equation (i.e. the data is stacked so that twin1 is regressed on twin2 and vice versa). Double entry obviously doubles the N which means standard errors have to be adjusted for the correct degrees of freedom. The standard correction is to multiply the standard errors by the square root of the degrees of freedom of the double-entered vector by the degrees of freedom of a the single-entered vector. In other words, multiply the standard errors by the square root of 2. [4] Double entry can be avoided by random assignment within each twin pair, i.e. randomly designating twins as Y_1 or Y_2.

Since its development, DF analysis has been widely used in behavioral genetics research as a robust platform to operationalize the biometric model and, importantly for our purposes, to simultaneously analyze conceptual models that include specific components of C and E (for examples see Boardman et al 2008, Rodgers et al 1994, Beaver et al 2009).

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reformulation, which is based on centering the data. Centering allows the R parameter and the intercept to be dropped, thus directly modeling P = A + C. One potential concern with DF models that might occur to some readers is the possibility that as regression coefficents the parameters representing A and C might be statistically significant yet greater than 1.0 or less than zero. While this is mathematically possible, it is an unlikely occurrence. More possible (though not particularly probable) are confidence intervals that would extend beyond the 1.0/zero boundaries. Such results would likely be caused when most of the variance in P is being driven by either A or C and/or when twins are systematically dissimilar on a trait (e.g. if twins who are liberal generally and systematically tended to have co-twins who are more conservative). In any case, in terms of substantive inference the story would almost certainly be the same as that taken from a maximum likelihood approach constraining estimates to the boundaries of 1.0 and zero, i.e. either all the variance (an estimate of 1.0 or greater than 1.0) or none of the variance (zero or less than zero) is attributed to A, C or E. Regardless of statistical approach estimates are just that, i.e. estimates, so inference in such scenarios would be more conservatively interpreted as "most of the variance" or a "trivial amount of variance" rather than all or none.

[4] Alternate approaches to correcting for standard errors include clustering standard errors on twin-pairs using a GEE, random effects or a Huber-White approach. . See Kohler and Rodgers (2001) for a more in-depth discussion of the degrees of freedom issues raised in DF models.

Within political science, individual-level causal models of attitudes and behaviors are readily captured in a simple regression model; generically it takes the following form:

(2) $Y = a + \sum b_i X_i \ + e$

Y is the attitude or behavior, X is a vector of theoretically specified independent variables, and regression methods and their various extensions are the primary quantitative tools used to analyze such models. Hypothesis testing is focused on H_0: $b_i = 0$, and if the null is rejected and theoretical expectations upheld, the parameter b is interpreted as a point estimate of the causal relationship between X and Y. X and Y are measured variables (survey responses, self-reports or lab or field observations). For the vast majority of empirical studies of political attitudes and behavior employing this generic model there is an implicit assumption that Y has no heritable or genetic component. Y is viewed as a function of environmental forces (education, socio-economic status and so forth) and the handful of biologically-based variables that do make a regular appearance in the vector X (notably race and sex) are interpreted through an environmental lens. For example, sex-based differences in political attitudes and behaviors are interpreted as products of differing socialization patterns and cultural expectations for males and females, not as products of the sex-based biological differences (e.g. Hatemi et al 2010d).

The theoretical basis of conceptual models operationalized by variants of Model 2 is clearly very different from the universal biometric theory that underpins the specification of the DeFries-Fulker model in Equation 1. Yet despite these different theoretical anchors, as both are regression models applied to individuals they are easy to synthesize into single modeling framework. Simply add Equation 1 to Equation 2:

(3) $Y_1 = a + b_1 Y_2 + b_2 R + b_3\ (R{*}Y_2) + \sum b_i X1_i \ + e$

From a purely technical standpoint, this is trivial. DF models are now widely and routinely extended to include all manner of covariates that take the form of specifically measured environmental variables (Boardman et al 2008 and Beaver et al 2009 provide public policy-related examples relevant to political science). Given the appropriate data, any model formulated within the framework of Equation 2 can be added to Equation 1, thus allowing a readily accessible and highly flexible platform for incorporating genetic factors into studies of political attitudes and behavior. Thus, given the appropriate data, any environmentally-grounded theoretical model of political traits ever developed in political science (Equation 2) can be tested in conjunction with a model of genetic influence (Equation 1) with a simple regression model (Equation 3).

As a purely methodological exercise, then, synthesizing the biometric model into traditional political science causal frameworks is simple and straightforward. All that is needed is appropriate data, (now readily available) a basic grasp of the logic of biometric model (summarized above), and a working familiarity with garden variety OLS regression (a quantitative skill set virtually universal among traditionally trained political scientists). In short, the data and methodological barriers to synthesizing the basic biometric model and traditional political science conceptual frameworks are very low. The conceptual and theoretical implications of this synthesis, however, need to be carefully thought through.

The theoretical distinction made by shifting from Equation 1 (a traditional biometric model) and Equation 2 (a traditional political science model) to the combined model in Equation 3 is of central importance. Essentially the same theoretical distinction is made by scholars using SEM methods to analyze genetic influences on political attitudes and behavior, but the critical implications often remain unaddressed in favor of explicating the methodological foundations. Explicating the DF approach as we do here provides an ideal vehicle to highlight and clarify these issues. The key conceptual issue to be grasped here is that the *methodological synthesis does not automatically bring theoretical synthesis*. Indeed, the results of any given application of Equation 3 can be interpreted through two distinct theoretical lenses; that of behavioral genetics and that of social science. These different perspectives can lead to very different inferences from identical results. This question of inference is critically important because the implications drawn from particular theoretical platform drive not simply how we interpret the results of a given study, but how we might re-interpret the results of an existing and extensive empirical literature in political science. To fully grasp these potentially far-reaching implications, it is important to understand how models 1 and 2 look very different depending on theoretical perspective; these perspectives turn out to have critical implications for how model 3 is interpreted.

The View From the Biometric Model. From the perspective of biometric theory Equation 1 obviously makes perfect sense (see discussion above). Equation 2 has no theoretical relevance at all. It assumes away genetic influence on a target trait (i.e. the whole explanatory purpose of biometric theory), making it essentially a model of the error term in Equation 1. This is especially the case as much of the empirical substance of political science theories are unique (to the individual) environment influences; level of education, income, partisan intensity, ideology and the like. Such causal frameworks may certainly include shared environmental aspects; for example, parental ideology in a study of political socialization. For the most part, though, viewed through the lens of the biometric model Equation 2 boils down to P=E, with C included now and then and A ignored completely. The key point here is that from the view of biometric theory, models like Equation 2 perversely consign everything of theoretical relevance to the error term.

The View from Traditional Political Science Causal Frameworks: From this perspective, Equation 2 obviously makes sense (see discussion above). Equation 1 has no theoretical relevance at all. It seems to be built around getting a point estimate of A (i.e. the coefficient b_3); this makes no sense because the causal impact of A on political attitudes and behaviors has been, pretty much universally, assumed to be either zero or so trivial as not worth investigating. Included in the model is an environmental measure of more potential relevance to traditional political science causal frameworks, but it is extremely limited. First, it only captures a particular element of the environmental causes of political behaviors and attitudes: the environment shared by siblings. Second, it provides no basis for testing specific elements of that environmental dimension that might be theoretically relevant. Rather than specific environmental variables for, say, parental attitudes or political culture, there is an omnibus variance estimate (i.e.

C, or b_1) that does little for political scientists theoretically. Indeed, Equation 1 seems to be mostly a model of the error term in Equation 2. This is because the variables used to construct most individual-level theories of political behavior are considered unique environment (E) and are not modeled at all. The key point here is that from the view of traditional political science causal frameworks, models like Equation 1 perversely consign everything of theoretical relevance to the error term.

Combining Theoretical Perspectives: Synthesizing the theoretical legacies of Equations 1 and 2 into Equation 3 is thus not as straightforward as the methodological integration. There are two basic approaches here. The first is from a political science perspective, which sees Equation 3 primarily as incorporating a biological covariate (A) into Equation 2. The second is a behavioral genetics perspective, which sees Equation 3 as mainly a means to decompose the C and E estimates from Equation 1 into their particular sub-components. Both perspectives have implications for each other and for how to interpret empirical analysis of political traits.

To take the political science perspective first, Equation 3 can be treated as a simple extension of Equation 1. The only difference is the addition of two covariates and an interaction term. This does not change how the variables in vector X are interpreted; assuming all regression assumptions are met, then traditional hypothesis testing ($H_0 = b_i = 0$) for individual variables is done using the usual t or z tests, and if this null is rejected the usual interpretation applies, i.e. a one unit shift in X is associated with a b_i change in Y. The novelty of Equation 3 from a traditional political science (i.e. Equation 1) perspective is only that A is being explicitly modeled rather than sent off to the error term. Variables in vector X are thus treated as estimates independent of genetic effects on the dependent variables. Interpreting Equation 3 in this fashion seems intuitive—even obvious—to a political scientist. Still, the potential implications of doing this are far reaching. For the most part it is largely unknown how popular or accepted causal frameworks of political attitudes and behavior perform once genetic influence is accounted for. It is quite possible that significant portions of the accepted wisdom generated by empirical studies framed in terms of Equation 2 will not replicate in Equation 3. If A is non-zero and non-trivial for political traits, as an ever-growing list of studies assure us, it implies that traditional causal frameworks have made a fundamental error. Indeed, it suggests empirical models in political science have been theoretically miss-specified for at least a half-century. Given that, the inferences taken from those models needs to be re-examined and re-tested within the causal framework of Equation 3.

From the behavioral genetics perspective, the theoretical synthesis just sketched raises a number of red flags. Indeed, from this perspective it is not at all clear how—or even if—the variables of central theoretical interest to political scientists in vector X should be interpreted. This is because those variables muddy the central purpose of the quantitative genetic models, i.e. disentangling the environmental and genetic etiology of Y. Remember, the key purpose of the quantitative genetic model is to accurately parse variance into A, C and E components. It is not at all clear how the variables in X can be interpreted in terms of C or E terms and, especially, whether their impact is independent of genetic influence. From a behavioral genetics perspective we are *not* simply isolating A as a covariate to test a theoretical model; indeed in employing variants of Equation 3 we may end up defeating the entire purpose of the quantitative genetic model. Rather than cleanly parsing variance into A, C and E, Equation 3 can create significant difficulties in inferring whether phenotypic variance is genetically or environmentally influenced.

To understand why, consider the b_1Y_2 term; a key assumption of the biometric model is that this captures the complete impact of C. What if, however, we include specific measures of C in the vector X? Such specific measures of C may be of considerable theoretical interest to political scientists. For example, a study of individual political attitudes or behavior might want to include family socialization terms such as parental party identification, parental ideology, or whether the family regularly discussed politics. If, as is typical in political science, we assume that these are purely environmental variables causally related to Y, we can interpret them as such. However, *this is an assumption not something being empirically tested*.

This assumption may or may not be true. Consider a variable in Equation 3 like parental ideology, which from a traditional political science perspective is viewed as a common agent of socialization for siblings raised in the same family. In other words, it is a specific measure of C. If this is indeed the case, when we add a variable like parental ideology to Equation 3 the omnibus C term, i.e. b_1, should decrease. This is because this term captures *all* of C. Yet variables assumed to be specific measures of C may in reality include genetic not just environmental influences. It turns out that treating a variable like political ideology as purely environmental--a non-controversial assumption for a large literature in political science—upsets the logic justifying the interpretation of Equation 1, and by extension the interpretation of Equation 3. After all, according to an expanding number of studies political ideology *is* genetically influenced (for a review see Hatemi et al 2011). Yet here we *must* treat it as a (shared) environmental variable. This is because the logic of the general quantitative genetics model demands that parental ideology is treated solely as a property of the twins' environment, not as a part of their genetic inheritance. Indeed, even if ideology is 100 percent heritable, all the variance in the parental ideology variable is by the assumptions of the model being assigned to C rather than to A. This because there is no variance on parental ideology within twin pairs; the correlation for DZ and MZs on parental ideology is the same, i.e. rDZ = rMZ = 1; by the second assumption of the CTD listed above, this is by definition C. In short, from the perspective of behavioral genetics we include in the model a variable empirically known to be genetically influenced yet treat it is a purely environmental variable.

A number of studies have pointed out that interpreting a specific measure of C in vector X as independent of genetic influence is logically flawed (for in-depth treatments of this issue see Purcell and Koenen 2005; Turkheimer et al 2005). It is worth repeating here that there is no technical barrier to including such environmental terms; just a significant issue of how to interpret them. Simply put, some "environmental" variables of central interest to political science theories may actually include genetic (A) as well as environmental (C) effects.

At least in the abstract, specific measures of E should be easier to include in the vector X without contamination from genetic effects. This is because in a base DF model these impacts are already consigned to the error term. Specific measures of E, then, simply take systematic variance out of e in Equation 2 and put it into X in Equation 3. Conceptually this does not involve "stealing" variance from the estimates of A and C. A number of refinements have been suggested for specifying elements of the non-shared environment, the best known coming from Rodgers et al (1994), who formulated the following extension of the base DF model:

(4) $Y_1 = a + b_1Y_2 + b_2R + b_3\,(R{*}Y_2) + b_4ENVDIF + b_5\,(R{*}ENVDIFF) + e$

Equation 4 is simply Equation 3 with the X vector specified to include two variables, a difference score (ENVDIF) that represents the difference between twins on a measured independent variable (X1 – X2), and an interaction term between this difference score and the measure of genetic relatedness. ENVDIF is designed to tap into differences between twins that are relevant to Y. This is an intuitive approach to getting at E; the base DF model controls for all of A and C, and ENVDIF picks up differences between twins on a measured variable to account for additional variance in Y. Those differences, however, do not have to be all environmental; they could also be due to nonshared genetic influences. This is tested by the R*ENVDIF term, which accounts for whether the differences are genetically mediated or not. If R*ENVDIF is significant, nonshared genetic influences are presumed to play a role; if it is insignificant they are not and this term can be dropped from the model.

Even here, though, the ENVDIF term represents environmental impacts independent of genetic impacts only the basis of underlying assumptions. The idea of subtracting one twin's score from the other (X1-X2) is to expunge A and C from the measure and just leave E. If we consider the independent variable X in terms of the basic genetic model, X1 can be thought of as a phenotype like any other, i.e. X1 = A + C + E; subtracting the co-twin's score (X2) can be thought of as removing the common environmental sources of variation in X1, i.e. C (the logic is essentially the same as using Y_2 to account for all of C in Y_1 in the base DF model). Subtraction of X2 from X1 may remove C, but does not necessarily remove A. In short, even though there is a separate estimate of A included in Equation 4 (b_3), the model does *not* test whether ENVDIF is a purely environmental impact independent of genetic influence.[5] We can assume A = 0 for X1 and that ENVDIF is a purely unique environmental measure, but, again, this is an assumption and not something empirically tested by the model.[6]

***Synthesis is Methodologically Easy, Theoretically Controversial*:** The bottom line is that DF analysis involves nothing more using regression methods to control for all of A and all of C for a given phenotype. This OLS platform can be readily extended to include all manner of covariates, including those that constitute theoretically specified causal models of political attitudes and behavior. The only technical barriers to doing so are those that apply to regression generally and the easily dealt with issue of inflated degrees of freedom due to data double entry. DF models can easily incorporate a wide range of covariates, and serve as an easily accessible platform for political scientists to combine traditional models of political attitudes and behaviors (Equation 2) with a basic quantitative genetic model (Equation 1).

The big issue for using a regression framework to synthesize the biometric model with political science is interpretation, which is driven by theoretical perspective. Political scientists and behavioral geneticists could easily interpret the same models very differently because of the differing assumptions and causal frameworks brought to the analysis. Most significantly, a researcher can assume added covariates are purely environmental and interpret them accordingly; political scientists seem highly likely to do exactly this given the disciplines' overwhelming history of environmental determinism. Again, however, this is an assumption and not something the model actually tests. In practice, a wide range of variables political scientists might include in the vector X in Equation 3 could combine elements of A as well as C and E. This means the

[5] Purcell and Koenen (2005; 497) do suggest a reformulation of the DF Model that can test the effect of measured E variables independent of genetic effects, though this has yet to be widely employed.
[6] It is important to re-emphasize that the essential issues here are not unique to DF models. Interpreting results for environmental variables as independent of any genetic influence is dependent more on the assumptions made by the analysis than the particular statistical approach.

estimates of A and C, b_1 and b_3, may change with the addition of these covariates. Such potential "remixing" of variance is a significant and potentially troubling issue from the standpoint of the quantitative genetic model; after all, the whole purpose of the latter is to disentangle A, C and E from each other.

From a political science standpoint, however, this may be less of a concern. Equation 3 is a significant departure from Equation 1 only in that it explicitly controls for genetic sources of variance in Y_1; adding covariates via vector X does not change that, it is simply that some of these additional variables might include elements of A (or C). Given Equation 1, however, it should be clear that empirical studies in political science have been quite comfortable ignoring the genetic components of variance in their measures; indeed, historically if such genetic components were seen anywhere, it was as trivial elements of the error term. What Equation 3 does from a political science standpoint is pull A out of the error term and make it explicit, even though some of the variance belonging to A are likely to be picked up by the variables included in vector X. From a traditional political science standpoint, the novelty of Equation 3 is that A is included at all. How do traditional political science explanations of attitudes and behavior hold up when such genetic influences are explicitly controlled for? This is the question Equation 3 can help answer.

DF Models of Political Attitudes and Behavior: An Empirical Example

In the next section of this paper we provide two examples of how Equation 3 can be used as a platform for synthesizing traditional political science empirical analysis with that of behavioral genetics. Importantly, these examples also highlight some the conceptual issues discussed above. Our data are drawn from the Minnesota Twins Political Survey, the first twin survey devoted entirely to political attitudes and behaviors. This data set includes 596 complete twin pairs (356 MZs, 240 DZs).[7]

[7]The data employed in this project were collected with the financial support of the National Science Foundation in the form of SES-0721378, PI: John R. Hibbing; Co-PIs: John R. Alford, Lindon J. Eaves,

We analyze two dependent variables created from this survey, one attitudinal and one behavioral. The former is a modified Wilson Patterson Index; this is a summative measure of political attitudes originally designed to tap conservatism (Wilson and Patterson 1968). We use this measure deliberately as it is arguably the most widely used measure of political attitudes employed in heritability studies (for a review see Bouchard and McGue 2003). The Wilson Patterson battery included in the Minnesota survey asked subjects to agree or disagree with 27 issues (everything from gun rights to gay marriage, and from evolution to increased military spending). Each item was coded so that the theoretical response range was -3 (strongly agree with liberal stand on the issue) to +3 (strongly agree with conservative stand on the issue). These items had high internal consistency (chronbach's alpha = .85), and our attitude measure as the mean response to all 27 items.

Our second dependent variable was a measure of political participation. This was a summative index of seven dichotomously coded items asking whether subjects were registered to vote, had ever attended a political rally, had ever campaigned for a candidate or cause, had ever contributed money to a political campaign or cause, had ever contacted a public official, belonged to a political group, or ever held an elected office. The chronbach's alpha for these items was approximately .73 and a factor analysis of the items resulted in a single dimension with an eigenvalue > 1.0 that accounted for approximately 36 percent of the variance. In short, this measure seems to be a reliable general index of behaviors associated with political participation.

Following the basic approach represented in Equation 2, for each dependent variable we specified a basic causal model intended to be representative of the explanatory approach used in political science to explain attitudes and behaviors. Both models included three basic demographic measures widely employed as standard control variables in political science models: sex (a dummy variable where 1=male), income (a six item index where 1=under $20,000 and

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Carolyn L. Funk, Peter K. Hatemi, and Kevin B. Smith, and with the cooperation of the Minnesota Twin Registry at the University of Minnesota, Robert Krueger and Matthew McGue, Directors.

6=$100,000 or more), and education (a six item index where 1=did not graduate high school and 6=professional or graduate training or degree). To these basic controls we added a variable designed to be representative of the sorts of specific theoretical interests political scientists might have relative to political attitudes and behaviors.

For the Wilson Patterson Index we included a measure of family political attitudes. This item asked, “Which of the following best describes your family’s political views,” and had five response options ranging from 1=almost all liberals to 5=almost all conservatives. A number of studies in political science argue that political attitudes are socialized and that a particularly effective source of this socialization is family (e.g. Campbell et al 1960; Jennings and Niemi 1968, 1975). If we take an environmentally determinist perspective, then, our hypothesis is that family political views will be a significant, positive predictor of political attitudes.

For our model to explain the participation index, we included a measure of family socialization into politics. This item asked, “During the time you were growing up, how often did you and your family members (other than your twin) discuss politics,” and had four response options ranging from 1=all the time to 4=not at all. From an environmentally determinist perspective, the basic theoretical expectation is that those who are socialized by their family to treat politics as important—or at least worthy of frequent discussion—are more likely to view politics as something important and worth participating in as an adult.

We freely concede that the models we construct are under-specified from a number of theoretical viewpoints in political science, and that there are numerous other causal candidates for inclusion on the right-hand side of our regression equations. Our purpose here, however, is less to test a specific theoretical specification than to demonstrate the utility of a particular analytic method for testing *any* theoretically justified combination of independent variables. At a minimum, our models include a set of “usual suspect” control variables common to most variants of Equation 2, and one variable that can be confidently defended as being theoretically justified from a traditional, environmentally determinist political science perspective.

To operationalize the basic DF model we double enter the data as described above, using one twin's attitudinal/behavioral score to predict the co-twin's score. The coefficient of genetic relatedness is constructed from twin zygosity as classified by the Minnesota Twin Registry, such that R=1.0 for MZs and R=.50 for DZs.

Results

Tables 1 and 2 report the results of three models of political attitudes and behaviors. Model 2 in both tables is a straightforward operationalization of Equation 2; the results can be interpreted in exactly the same fashion as countless other examples of empirical studies in political science. We have not adjusted standard errors for model 2; for illustrative purposes we are simply using survey results from approximately 890 individuals in exactly the same fashion countless researchers in political scientists have used data from sources such as the American National Election Survey (ANES). The only difference is that half of our sample is related to the other half.

TABLES 1 AND 2 ABOUT HERE

The fact that our sample is one of related siblings, however, seems to have little impact on the substance of the results, which hold few surprises for political scientists. Sex and education have statistically significant relationships with attitudes and behavior, with males being slightly more associated with conservative attitudes and rates of participation. Higher levels of education are associated with more liberal attitudes and higher rates of participation. The coefficient for income is substantively trivial and statistical insignificant. The basic demographic controls, then, show pretty much the same relationships with attitudes and participation that we would expect from any large representative sample of American adults.

The variables of more theoretical interest also perform as we might expect. Family political views are strongly associated with adult political attitudes (the standardized beta for this variable is .39, the highest of any variable in model 2). Frequency of political discussion in the developmental environment is strongly associated with participation (standardized beta -.29;

again, the highest of any variable in the model); as frequency of family discussion of politics declines so do levels of political participation. If we take the traditional environmentally determinist view of political science and stop our analysis with model 2 in Table 1 and Table 2, we would interpret the results as strong evidence that family socialization plays a key role in determining political attitudes. In doing so, we implicitly we assign genetic influences on our dependent variable to the error term; by assumption model 1 treats A=0.

While model 2 in both tables represents a simple, applied example of Equation 2, model 1 is a straightforward application of Equation 1. The latter are DF models with adjusted standard errors. These analyses suggest that the assumption that A=0 is not necessarily warranted. This is especially the case in Table 1, which echoes a fairly extensive list of empirical studies reporting that the observed variance in political attitudes as measured by the Wilson Patterson Index is primarily attributable to genetic inheritance and unique environment. The coefficient for shared environment is small and statistically insignificant.

Model 1 in Table 2, however, shows a different pattern than general political orientations. A significant, non-zero portion of observed variance in political participation is attributed to genetic influence. Here, however, A accounts for only about 35 percent of the variance—considerably less than the comparable estimates for the Wilson Patterson Index. Environmental influences seem to have a considerably larger role in determining political participation. The C estimate is still statistically insignificant, but it is roughly double of the comparable estimate for the Wilson Patterson Index. If we limit our interpretation to the ACE results of model 1, it would appear that unique environment accounts for the lion's share of variance in political participation; E is estimated as .54 if we include C (1 - .35 + .11), and .65 if we treat it as zero.

The DF models suggest that political attitudes and, to a somewhat lesser extent, political behavior, have significant, non-zero genetic influences. They also suggest that there are powerful environmental forces that account for significant proportions of the observed phenotypic variance, but that these are not likely to be environmental experiences shared by twins. What the

DF models do not tell us, however, is what political science has historically been most interested in, i.e. the exact nature of those environmental influences.

Model 3 seeks to address this latter issue by combining Models 1 and 2. Table 1's combined model shows family political views and education continuing to be significant predictors for Wilson Patterson scores in the presence of explicit genetic controls. Interestingly, sex is now an insignificant variable. While we can reasonably infer education and family political views help determine political attitudes, we should exercise caution in interpreting these results. The results for education and family political views are real enough; the question is whether they should be interpreted as estimates *independent* of genetic influence. We may have a priori theoretical reasons for assuming this independence, but this is not being tested by the model. Indeed, if anything the model suggests this assumption is not warranted.

Note that the coefficient representing A decreases in model 3 compared to model 1. When modeling discrete elements of ACE independently, we implicitly assume that the environmental variables transferred from model 2 are purely environmental; however these elements are certain to interact in some manner; observing the A estimate shifting from .59 to .44 hints that this assumption may not be correct. Actually, this is not that surprising. If political attitudes are heritable, then within family political views logically have some genetic basis. A number of studies already report that educational attainment is genetically influenced (e.g. Tambs et al 1989). From the perspective of behavioral genetics, the results in model 3 may present problems because there is a reasonable chance that the estimates do not reflect clean partitions into genetic and environmental variance components. This possibility could be explored with more complex models that that specify gene-environment interactions. However, from the perspective of political science, this may be of considerably less theoretical importance. What model 3 clearly tells us is that political attitudes are at least partially inherited, that education and family political views shape political attitudes, and that both of these latter variables are probably not purely environmental. Those findings, in and of themselves, have highly significant implications for political science. At a minimum these are: (1) The assumption of A=0 implicit in equation 2, i.e. as assumption in bedded in 50 years of empirical analysis of political attitudes, is incorrect. (2) Our ability to manipulate variables to achieve socially desirable ends—e.g. increasing education levels as a means to promote civic engagement—may be more limited that commonly assumed, because these variables are partially innate.

Table 1 also provides a baseline statistical case for using Equation 3 model specifications. The adjusted r-squares for model 1 and model 2 are roughly comparable, accounting for about 25 to 30 percent of variance. Compare that with the .39 adjusted r-square for model 3. The combined model explains roughly a third more of the variance. This extra explanatory horsepower is, undoubtedly, coming from the fact that model 3 is explicitly seeking to investigate whether elements of what the other two models consign to the error term are, in fact, systematic.

The combined model 3 of political behavior in Table 2 tells a somewhat different story from the combined model of political attitudes in Table 1. Again, sex is insignificant, while education and the independent variable of theoretical interest remain significant in the presence of explicit genetic controls. A big difference, however, is that now the A coefficient is also insignificant. Following the traditional interpretation guidelines of regression, we can therefore treat the estimate for additive genetic influence as equivalent to zero. This, however, *does not mean political participation is not influenced by genes.* We can make this case theoretically, but to do so we have to make some assumptions, specifically that the other significant variables in the model, i.e. education and political interest are purely environmental. If we are not willing to make that assumption, we need to recognize that the genetic influence on participation may be working through the covariates of education and family discussion. Multivariate cholesky decomposition (structural model), is often used to test these very circumstances. For example, in an exploration of voting behavior, Hatemi et al (2007) found that all of the genetic variance on vote choice could be accounted for the genetic variance on correlated attitudes. The same can be said for political interest; a basic Falconer estimate based on this data set calculates A is roughly .23 for political interest. So even though the A coefficient is, in effect, zero and the only significant variables are (in a traditional political science sense) environmental, we cannot conclude political participation is a wholly environmental phenomena free of genetic influence.

What is relatively safe to infer from the results reported in Tables 1 and 2 is that there is a larger, direct influence of A on attitudes compared to behavior. While there may be a heritable component to behavior (or at least the behaviors in our participation index), these seem to be more likely to be mediated through environmental variables. Or at least through variables that political scientists would consider predominantly environmental.

Comparing the DF Regression Approach to Structural Equation Modeling

Finally, to demonstrate that DF models produce results comparable to those produced by SEM, maximum likelihood approaches commonly employed in behavioral genetics, we compare ACE estimates for Wilson Patterson scores generated by both methods.

TABLE 3 ABOUT HERE

As can be seen from Table 3, the DF and the SEM approach provide identical estimates for ACE components. Both methods are also amenable to model fitting, i.e. to systematically dropping model parameters to see whether estimates for specific variance components are significantly different from zero. For the DF model this simply means dropping the variable associated with A or C and using an F test to see whether the reduced model differs significantly from the full model. As Table 3 reports, both the SEM and the DF approach report an AE model is the most parsimonious fit to the data, and both approaches generate virtually identical estimates of the A and E variance components parameters.

In summary, when using a sample of only same-sex twin pairs, where basic assumptions testing criteria are met, (see Medland and Hatemi 2009), the basic univariate analysis reported in Table 3 shows that a simple, ordinary least squares regression approach using statistical software familiar to most political scientists produces results that are virtually identical to SEM in all essential respects.

Conclusion

A rapidly developing research agenda in political science is seeking to empirically explore the biological underpinnings of political attitudes and behavior. Heritability studies are a primary vehicle for conducting such investigations and data sets rich in political phenotypes are becoming broadly accessible. This presents an opportunity to integrate biological correlates of political attitudes and behavior into more traditional political science theoretical frameworks. Conducting such syntheses, however, requires overcoming three challenges: (1) Gaining access to appropriate data, (2) Gaining the statistical tools to exploit that data, and (3) Understanding the theoretical implications for interpretation and inference of such a synthesis. What we have attempted to do in this paper is demonstrate the first two challenges are easily dealt with. Data on a broad range of political traits is now readily available. We have tried to make clear that most political scientists with even minimum quantitative training possess already the necessary methodological skill set to do univariate ACE analyses. The basic regression approach of DeFries-Fulker models provides non-biased estimates of A, C and E that are comparable to structural equation, maximum likelihood estimates, and are readily amenable to simple statistical tests (F and t) of the robustness of these estimates. DeFries-Fulker models, in short, are in no way a better or worse methodological approach than structural equation/maximum likelihood approaches. They are an alternate means, suitable for specific analyses. And, for political scientists a more intuitive means—to generate what in all essential respects are identical and statistically testable parameter estimates in univariate models of continuous traits. OLS really is AOK to do an ACE.

The final challenge, is the theoretical perspective on how the new synthesis of equation 1 (the biometric model) and equation 2 (traditional political science models) that drives how these models are interpreted and what their implications are for the broader literature on political attitudes and behaviors. The OLS platform can help clarify important conceptual differences between the analytic focus of political scientists and behavioral geneticists, but it does not necessarily resolve them. The latter have a universal causal model for targeted dependent variables and the analytic goal is to correctly apportion observed variance in that dependent variable into the mostly unobserved independent variables A, C and E. In contrast, political scientists have a wide and constantly evolving set of theoretical models, characterized by a range of independent variables. The analytic goal here is not to apportion variance into a handful of universal components, but to identify the specific elements of those components. Empirically and theoretically this means constructing causal models that specify particular—i.e. observed and measured--elements of E (and sometimes C); the potential role of A has been largely ignored by vast literatures on attitudes and behavior, implicitly consigned to the error term.

Thus, while a regression platform can be readily employed as a vehicle for synthesis, differing theoretical foundations create a significant inferential caveat. This is that caution must be exercised in treating other variables in the model as wholly independent of genetic influence. This is a central concern of behavioral genetics, and appropriately so given the analytic focus of the discipline. Yet while political scientists should certainly heed such caution, the inferential problem is perhaps not quite such an obstacle. The vast majority of empirical studies of attitudes and behavior in political science already interpret the impact of statistically significant variables, at least implicitly, as independent of genetic influence. From the perspective of political science, simply treating genetic influence seriously, i.e. incorporating it as a specified element of our empirical models is a significant change. Moreover, it is a change that offers the possibility of significantly increasing the theoretical insights and explanatory power of our statistical models.

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Table 1: Determinants of Political Attitudes

Variable	Model 1 (Equation 1)	Model 2 (Equation 2)	Model 3 (Equation 3)
Sex		.17* (.05)	.09 (.06)
Income		-.01 (.01)	-.00 (.02)
Education		-.15* (.01)	-.11* (.02)
Family Political views		.33* (.02)	.21* (.03)
A	.59* (.14)		.44* (.15)
C	.05 (.09)		.04 (.12)
Constant	.27 (.10)	-.228* (.12)	-.06 (.19)
N	1181	898	896
Adj. R-Square	.29	.26	.39

* = p < .05
Unstandardized coefficients (standard errors) reported. Standard errors for model 1 and 3 are adjusted for degrees of freedom; standard errors for model 2 are unadjusted. Not reported is coefficient of genetic relatedness (R) in models 2 and 3. R is insignificant (p < .05) in both models. Estimate for E in model 1 is .36 (1 – (.59 + .05) if the insignificant parameter C is treated as substantive, and .41 if C estimate is treated as zero.

Table 2. Determinants of Political Participation

Variable	Model 1 (Equation 1)	Model 2 (Equation 2)	Model 3 (Equation 3)
Sex		.20* (.10)	.14 (.14)
Income		.06 (.03)	.06 (.05)
Education		.34* (.04)	.28* (.05)
Family Discussion of Politics		-.62* (.06)	-.50* (.09)
A	.35* (.15)		.24 (.16)
C	.11 (.13)		.08 (.13)
Constant	2.36* (.42)	2.82* (.28)	2.62* (.58)
N	1143	895	877
Adj. R-Square	.16	.23	.28

* = $p < .05$
Unstandardized coefficients (standard errors) reported. Standard errors for model 1 and 3 are adjusted for degrees of freedom; standard errors for model 1 are unadjusted. Not reported is coefficient of genetic relatedness (R) in models 2 and 3. R is insignificant ($p < .05$) in both models. Estimate for E in model 1 is .54 (1 – (.35 + .11) if insignificant C parameter is treated as substantive, .65 if C parameter is treated as zero.

Table 3: Comparing DF and SEM ACE Estimates for Wilson-Patterson Index

Model	A	C	E	R^2/χ^2	$F/\Delta\ \chi^2$	Df	P
Full							
DF	.59	.05	.36	.296			
SEM	.59	.05	.36	2614.55			
AE							
DF	**.65**		**.35**	**.296**	**.378**	**1178**	**.53**
SEM	**.64**		**.35**	**2614.75**	**.20**	**1**	**.60**
CE							
DF		.52	.48	.275	35.3	1179	.00
SEM		.52	.47	2644.07	29.56	1	.00

Bolded rows indicate results of models with best fit to the data.