

Biology, Ideology and Epistemology: How Do We Know Political Attitudes Are Inherited and Why Should We Care?

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One of the most confirmed findings in behavioral genetics is that social attitudes are partially heritable, with studies using various methods, samples and dependent variables consistently attributing roughly 40-60 percent of population variance in such attitudes to broad sense heritability (for a review see Bouchard and McGue 2003). Political attitudes are no exception to this general rule, and several recent studies using different samples and employing behavioral genetics' standard methodological tools, report issue orientations, ideology, and strength of partisan attachment as having similar heritability coefficients (e.g. Alford, Funk and Hibbing 2005; Bell, Shermer, and Vernon 2009; Bouchard et al. 1990; Eaves, Eysenck and Martin 1989, Hatemi et al. 2009a, Martin et al. 1986; for a review see Hatemi et al. 2011). Concurrent with high heritability estimates, these studies find a large role for unshared environment but a considerably smaller role for shared environmental experience, which typically accounts for 20 percent or less (often much less) of population variance for a given trait. In many respects, these findings contradict decades of empirical work in political science treating individual differences in attitudes as environmentally determined and the environmental experiences shared by family members as particularly effective agents of political socialization (Campbell et al. 1960, Jennings and Niemi 1991, Zaller 1992).

Claims for a heritable component for political orientations, especially as an influence more powerful than agents of political socialization like family, are thus quite appropriately viewed with skepticism. It is difficult to conceive of political attitudes as being genetically informed; indeed, it is absurd to conceive of, say, a "pro-choice gene". Yet gene-political

behavior correlations, individual variation in the physiological processing of environmental information, and even pharmacological manipulation of social interactions, consistently suggest that biology plays a critical role in shaping social, economic and political attitudes and behavior (Dawes and Fowler 2009; Hatemi et al. 2011b; Kosfeld et al. 2005, Oxley et al. 2008). So while there may be no gene for a specific issue preference or ideological orientation, the biological systems built by genes seem to play an important role in mediating political attitudes. For example, individual variations in how autonomic nervous systems react to out groups may predispose people toward policy preferences on out group-related issues such as affirmative action and immigration (see Oxley et al. 2008, Smith et al. 2011, Vanman et al. 2004). Nervous systems may be conditioned environmentally, but their basic structure and function is genetically based. Thus physiology may be one means by which attitudes are mediated by genes. Similar arguments can be constructed from known correlations between genetically-based levels of neurotransmitters or hormones and social or political behavior (Johnson et al. 2006, Madsen 1985, Zak, Kurzban, and Matzer 2005).

Developing causal connections in chains that include biology and political attitudes opens up a new and still relatively untitled research field that, as suggested above, represents an important new direction for political science (Fowler and Schreiber 2008, Hibbing and Smith 2008; Funk 2010; see also Merelman 1971, Masters 1989, Carmen 1997). Before the discipline commits to such a research direction, however, it is worth asking whether the findings prompting this shift stand up to critical scrutiny.

A catalyst for this new research direction was Alford, Funk and Hibbing's (2005) analysis of a large twins data set to generate heritability estimates of political attitudes. Building from earlier studies by Martin et al. (1986) and Eaves, Eysenck and Martin (1989), they found

that approximately half the population variance in a summative measure of political attitudes, the Wilson-Patterson Index, could be attributable to broad scale heritability; only 11 percent was attributed to the twins' shared environment with the rest owing to unshared environment. A rapidly evolving literature on the heritability of political attitudes and behavior has since emerged, and a variety of different samples, methods and attitudinal and/or behavioral dependent variables has largely confirmed and extended these findings (e.g. Hatemi et al. 2007, 2009; 2010; Bell, Shermer, and Vernon 2009, Eaves and Hatemi 2008; Klemmensen et al. 2010).

Despite the consistency of the results generated by this literature, their validity has been consistently challenged on a number of key issues. Most critically, the twin and extended kinship methods employed are held to be biased toward confirming the hypothesis of a heritable basis for whatever trait is being studied. Concurrently, and especially in the case of political attitudes, critics claim that these methods are biased against the hypothesis that similarity in attitudes is driven by similarity in environments.

To fully appreciate these criticisms, it is necessary to understand how heritability estimates are generated. Most studies of the heritability of social attitudes employ a classic twin design (CTD), which seeks to decompose variation of any measured trait (a "phenotype") into its heritable and environmental components. To do this the CTD takes advantage of a naturally occurring experiment centered on twins. Dizygotic twins (DZs) come from separately fertilized eggs, sharing, on average, 50 percent of their genes. Monozygotic twins (MZs) come from a single fertilized egg that splits and thus share 100 percent of their genes. If twins share the same environment (same parents, household, broader cultural influences etc), the combination of constant environment and MZ-DZ genetic variation provides a lever to disentangle environmental versus genetic influence on a given trait. The logic is simple: If MZ pairs are

more alike than DZ pairs on the trait of interest and common environmental influences, such as being raised in the same home, by the same parents, and growing up in the same neighborhood is being held constant, greater MZ co-twin similarity can be attributed to their greater genetic similarity. The CTD thus takes twin pairs as the unit of analysis and is analogous to an ANOVA that splits variance into between- and within-group effects.

In the CTD variance is commonly partitioned into that shared between siblings, and unique variance (E), or variance that is unique to the individual. Shared variance is in turn decomposed into that attributed to genetic effects (A) and that attributed to family or shared environment (C). These ACE estimates are reported as standardized variance components; thus an A estimate of .40 indicates 40 percent of the total variance in the trait being studied is attributed to genetic influences. There are several methodological approaches to generating ACE estimators; correlation-based (the Falconer technique), regression-based (DeFries-Fulker models), and, what is arguably the mainstream approach, maximum likelihood structural equation techniques (for primers on these methods see Purcell 2008; Medland and Hatemi 2009; Smith and Hatemi 2011). The key for present purposes is not the different mechanics of partitioning variance but the crucial shared assumption on environmental similarity. The equal environments assumption, or EEA, assumes common environmental influences on the phenotype being studied are the same for MZs and DZs. If this assumption is violated, i.e. MZ twin pairs experience a different magnitude of the shared environment than DZ pairs and this difference influences the trait of interest, then CTDs will overestimate A and underestimate C.

Importantly, the assumption is *not that MZs and DZs have equal environmental experiences*. It is well known, for example, that MZs are more likely to be dressed alike, share the same bedroom, and have the same friends (Kendler et al. 1987, Loehlin and Nichols 1976).

Rather, the EEA assumes that these more similar environmental experiences do not lead to greater co-twin similarity on the specific trait being studied. Any serious violation of the EEA means in effect that broad sense heritability (A) is “stealing” variance that should be properly apportioned to common environment (C).

Political Socialization, Political Similarity, Political Science and the EEA

Though generally using individuals (as opposed to families) as the unit of analysis, and focusing on mean impacts as opposed to partitioning variance, the hypothesis that similar environments lead to similar political attitudes and/or behavior is strongly implied by a literature on political opinion and socialization that stretches back for at least half-a-century (e.g Hyman 1959, Campbell et al. 1960, Cook 1985, Zaller 1992, Sapiro 2004). Family in particular has long been seen as a primary environmental influence on political attitudes in this literature, with significant positive relationships between parents and offspring on political orientations being un-controversially interpreted as the impact of common environmental socialization rather than genes (e.g. Jennings and Niemi 1968, 1975, 1991; Hess and Torney 1967; Tedin 1974; Glass 1986; Jennings et al. 2009; Stoker and Jennings 2008; Zuckerman et al. 2007). Based on this research it is perfectly reasonable to conclude that siblings raised in similar environments will have similar political attitudes. For any given political phenotype, from the perspective of this literature, common environment (C) should have a significant influence, as should unique environment (E). Heritability (A), on the other hand, would have none or very little.

The basic expectations from the political science literature, then, are these: $C > 0$, $E > 0$ and $A = 0$. Indeed, at least three generations of political scientists studying attitudes and behavior have based their empirical and theoretical efforts, at least implicitly, on this premise. In contrast,

CTD analyses consistently report $A > C$ and $E > C$. This tension between expectations from the political science literature and behavioral genetics findings is unlikely to be explained away by different units of analysis (individual versus family) or different approaches to partitioning variance. There is a fundamental difference here in theoretical expectation of what explains variance in political traits. CTD studies all suggest heritability plays a larger role than common environment in explaining political traits; the broader political science literature implies that common environment is a primary influence and that heritability, in contrast, has little influence.

If CTD studies provide reasonably accurate estimates of variance, genetic influences (and by extension biological influences more generally) clearly need to be integrated into our theorizing about the causes of political attitudes and behavior. Such integration has been resisted by critics claiming that the twin studies driving the movement to give genes and biology equal consideration with the environment fail to provide any believable evidence for the heritability of political attitudes (Joseph 2004, Charney 2008, Beckwith and Morris 2008). EEA critics do not necessarily reject the notion that political orientations might be influenced by genes or biology more generally (e.g. Suhay, Kalmoe and McDermott, 2007, 35). The central argument is that CTD studies inflate heritability estimates and do not accurately account for environmental-based sources of variance in political attitudes due to several specific shortcomings in the model. Yet these criticisms, like most of the literature questioning the validity of the EEA, are made on the basis of secondary analysis of published research, not on the basis of empirical examination of CTD assumptions on political variables. Suhay, Kalmoe, and McDermott (2007) present an extension of these arguments in a critique that is notable for its organization and comprehensive summary of the four key arguments against the EEA. We proceed by addressing each of these points in turn, drawing directly from their language.

Point 1: Similar Experiences In explaining the role of similar experiences in

undermining the equal environment assumption, Suhay, Kalmoe, and McDermott (2007, 15-16)

offer the following explanation and references.

MZ twins spend more time together and are more likely than DZ twins to have been “inseparable as children” (Joseph 2004). They are more likely than DZs to study together, have the same friends, and attend social events together (Joseph 2004; Scarr and Carter-Saltzman 1979). Because of this “inseparability,” MZ twins will tend to have more similar experiences than DZ twins...In sum, we can expect that these various life experiences, shared at a greater rate by MZ twins, will push their political attitudes in the direction of conformity.

Point 2: Mutual Influence Going on to address the role of mutual influence as a

violation of the EEA, Suhay, Kalmoe, and McDermott (2007, 16-18) argue that:

It is likely that MZs influence *each other* in the direction of conformity more so than DZs for several reasons...MZs share a much closer bond than do DZ twins (Charney 2006; Joseph 2004)...Rose and Kaprio (1988) found that MZ intra-pair similarity with respect to the personality traits “extroversion” and “neuroticism” correlated with twins’ frequency of contact as well as their age of separation; because these correlations existed among “identicals,” the observed relationships could *not* be due to more genetically “like” twins interacting more often.

Point 3: Similar Treatment: In explaining how similar treatment could complicate the

estimation of heritability in a classic twin study, Suhay, Kalmoe, and McDermott (2007, 14)

suggest that:

MZ twins can expect similar treatment in some regards based simply on the fact that they look so much alike (Moore 2001; Charney 2006). How we are perceived and treated on the basis of such attributes affects our own behavior. For example, relatively attractive people are, on average, treated better than others and are perceived as more successful, resulting in (again, on average) more sociable personalities and higher levels of achievement (Moore 2001; Fiske 2004).

Point 4: Shared Prenatal Environment Taking a decidedly more biological turn, Suhay,

Kalmoe, and McDermott (2007, 18-19) offer their fourth and final EEA critique by focusing on

shared prenatal environment:

There is yet a fourth reason why MZ twins may be more alike than DZ twins for reasons that cannot be attributed in any direct sense to genetics. MZ twins on average have more similar *prenatal* environments. Approximately two-thirds of MZ twins share a “chorion” in their

mother's womb; the other third of MZs inhabit separate chorions within the womb. On the other hand, *all* DZ twins inhabit separate chorions (Moore 2001, 57).

Testing the Social Equal Environment Assumptions

We address the four primary criticisms of twin studies listed above, focusing our empirical analysis on points 1 and 2, which are at the heart of concerns about CTD studies of political traits. Both points focus directly on factors in the family and social environment of twins that clearly link to our general notions of how the environment, through active or passive political socialization, shapes political orientations.

In conjunction with the University of Minnesota Twin Registry, the first twin study devoted primarily to political variables was recently compiled. Adult twins from the Minnesota Twin Family Registry completed a detailed survey on social and political issues, values, and behaviors. The Minnesota Twin Family Registry is a birth-record based registry containing approximately 8,000 twin pairs born in Minnesota from 1936 to 1955. The twins were recruited to the registry in middle-age, from approximately 1983 to 1990 (for details on the registry see Lykken, et al.. 1990, and Krueger and Johnson 2002). The twins in our sample were born from 1947 to 1956. No opposite sex DZ twins were recruited for our study (this is because opposite sex twins can also lead to inflated estimates of heritability in a CTD). A total of 1349 individuals completed our survey, including 596 complete twin pairs (356 MZ pairs and 240 DZ pairs). Our sample is approximately 39 percent male and 61 percent female (compared to 45 percent male and 55 percent female for the full registry in the 1947 to 1956 birth years) and consists of approximately 60 percent MZs and 40 percent DZs (compared to 52 percent and 48 percent for the full registry; higher participation by MZs than by DZs is typical). By definition, twin samples are not representative of an entire adult population (the latter obviously includes non-twins). However, with the exception of age and race (all members of Minnesota Twins Registry are

middle-aged; over 90 percent are white), our sample is broadly representative of a cross section of US adults on a broad variety of socio-demographic indicators.

Included in the survey were items specifically designed to allow empirical analysis of the EEA critique at the heart of the dispute over whether twin studies can provide reliable evidence for a genetic component to political attitudes. Point 1, similar experience, was addressed with a series of items including whether twins were dressed alike growing up, attended the same classes at school, shared the same bedroom at home, and had the same friends growing up (5 point scales coded so 1=never and 5=always). Point 2, mutual influence, was assessed using items about contact with their co-twin including how often they see their twin, how often they talk to their co-twin on the telephone, and how often they contact their twin by email (7 point scales coded so 1=less than once a year and 7=every day). These items do not exhaust all potential categories of similar experience and mutual influence (shared social experiences in adulthood, for example, could also potentially play a role) but are comprehensive enough to provide a robust empirical test of the key claims. These variables certainly allow us to assess whether, compared to DZs, MZ twins had more similar childhood experiences and have more frequent contact as adults. As we can see from Table 1, the twins do in fact exhibit differences across zygosity for most of these measures. Specifically, compared to DZs, MZ twins are significantly more likely to report similar childhood experiences and more likely to report closer contact with their twin, though many of the differences are substantively modest.

(Table 1 about here)

Do these differences in similar experience and mutual influence, as CTD critics suggest, help explain the differences in the adult political similarity of MZ and DZ twins? To answer this question we focus our analysis on a particular political trait: ideology, measured using a Wilson-

Patterson index. This choice is deliberate as the Wilson Patterson Index is the phenotype analyzed in the classic studies on the heritability of political traits (e.g. Martin et al. 1986; Alford, Funk and Hibbing 2005). Included in the survey was a Wilson-Patterson battery asking subjects to agree or disagree with 27 contemporary issues-of-the-day (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 (Cronbach's $\alpha = .85$), and ideology was measured as the mean response to all 27 items.

A straightforward approach to examining whether the greater ideological similarity of MZ twins relative to DZ twins is environmentally based is to simply look at twin correlations across variation in family and social environment. If points 1 and 2 of the EEA critique are correct, DZs should look like MZs when they have more similar social and family environments, and MZs should look like DZs when they have dissimilar social and family environments.

Column 1 of Table 2 shows simple correlations of Wilson-Patterson scores between MZ siblings at high/low levels of adult contact and similar/dissimilar childhood experiences, as well as an overall correlation that ignores variation in mutual influence and similar experience. Column 3 does the same for DZs. If points 1 and 2 of the EEA critique are correct, we should see similar patterns in these columns: MZ and DZ correlations should fall with lower levels of adult contact and dissimilar childhood experiences, and rise with higher levels of adult contact and more similar childhood experiences. Higher levels of contact/more similar childhood experiences should push DZ correlations up toward the overall correlation for MZs (.64), and lower levels of contact/more dissimilar childhood experiences should push MZ correlations down towards the overall DZ correlation (.35).

(Table 2 about here)

What we actually observe is a pattern of relatively stable MZ correlations. The range in column 1 runs from .45 to .77. There is certainly some variation here but it is clearly anchored around a central tendency reflected in the overall correlation of .64. MZ correlations for the most part simply do not change much. For levels of adult contact there is some separation, shifting on average from .73 at high levels of contact to .60 at low levels of contact. For similar childhoods there is virtually no change; an average of .64 for similar childhood experiences compared to .63 for dissimilar childhood experiences. MZ correlations never approach the overall DZ correlation of .35. Indeed, MZ correlations even in the *lowest* contact/dissimilar categories are greater than DZs in the *high* contact/dissimilar categories.

In contrast, DZ ideological similarities display extreme variation depending on levels of adult contact/similarity in childhood experiences. The range for DZ correlations is from roughly .17 to .67 (and the .17 correlation is insignificant). There are two points of central tendency for DZ correlations; the mean correlation for low contact/dissimilar childhood experiences is about .25, while the comparable coefficient for high contact/similar childhood experiences is about .50. Regardless of environmental similarity DZs never become as ideologically alike as MZs.

Overall, these results strongly suggest that differences in MZ-DZ ideological similarities are not based solely in environmental similarities; the MZ results clearly indicate a genetic role for ideology. However, the DZ findings may lend credence to criticisms that heritability estimates (A) from CTD studies should be taken with a grain of salt. Given MZ and DZ correlations, partitioning variance into A, C and E estimates requires nothing more than some trivial arithmetic, and we report ACE estimates for all of our MZ-DZ correlations in Table 2 (the equations for these variance components estimates are reported in the Table 2 column headings;

for a full description of this method see Medland and Hatemi 2009; Smith and Hatemi 2011). Note that our overall estimate of A is .59, i.e. on the high end of the 40-60 percent range reported by other studies. Yet there is extreme variation in the estimates of A and of C in Table 2; depending on the correlations used, A or C can account for virtually all or none of the variance in Wilson Patterson scores. This variation in the A and the C estimates is being driven almost exclusively by differences in DZs; DZ correlations in column 3 account for about 80 percent of the variation in A estimates in column 5. The variation in MZ correlations accounts for none.

What are we to make of this? The results clearly indicate that points 1 and 2 of the EEA critique cannot account for all MZ-DZ differences; genetic influence turns out to be critical to ideological similarity for both MZs and DZs. The MZ results indicate that genetic similarity leads to ideological similarity regardless of environmental variation. The crucial point emerging from the DZ results is not just that ideological similarity is a product of environmental variation, but that *genetic* variation is required for this environmental influence to manifest itself. In short, environmental similarities seem to influence similarities in political temperament but this influence is mediated by genes; this is the classic definition of a gene-environment (GxE) interaction (Kendler and Eaves 1986).

This situation potentially has important implications. Genes may indeed influence political temperaments, but if the extent of this influence is heavily dependent on family and social environments perhaps political science need not pay too much attention to genes (and perhaps biology more broadly conceived) after all. The key question is what happens to the 40 to 60 percent of variance in ideology being consistently attributed to A when the moderating effects of family and social environment are accounted for. Does A become substantively trivial?

Given the implications for both sides of the debate, we replicated the analysis presented in Table 2 on the Virginia 30K (VA30K) data used in the classic studies of political traits by Martin et al. 1986 and Alford, Funk, and Hibbing 2005. The VA30K sample is different in important ways from the Minnesota sample. It represents a different population in a different political environment (adults aged 18-98 in the 1980s). The VA30K survey has few political items but does include a Wilson-Patterson Index, though it contains a different set of issue attitudes (issues-of-the day relevant to the mid-1980s) and employs a three-point rather than the seven-point scale used in the Minnesota sample. It also has a similar set of mutual influence/similar childhood experience items. This is enough to allow a comparative test of points 1 and 2 on a separate sample. Despite differences in both the item content and the scale of responses, the two samples generate remarkably similar patterns of stability in MZ correlations and instability in DZ correlations (see Table 3). Indeed, MZ correlations are not only relatively invariant *within* the samples, they are often directly comparable *across* samples. For example, in the VA30K the overall MZ correlation for the 3-point Wilson Patterson is .65, the comparable coefficient for the Minnesota 7-point Wilson Patterson is .64. In contrast, DZ correlations are much more unstable, varying by levels of contact and childhood environment both within and across samples. The one notable exception is for similarity of childhood environment, where the Virginia 30K study results do not indicate any impact of variation in childhood environments for either MZ or DZ pairs. In other words, on this particular measure environmental variation reported in the VA30K seems to have *less* of an impact than in the Minnesota sample.

(Table 3 about here)

There are some notable differences in ACE estimates generated by the VA30K and Minnesota data. The overall A estimate from the VA30K is .43 and the C .22, compared to .59

and .05 for the Minnesota sample. There are four explanations for these differences. First is the difference in variable measurement (7- versus 3-point Wilson-Patterson scales). Second is the age difference in the samples. The Minnesota data is limited to middle-aged adults; VA30K a much broader age range (18-98). As genetic influences on social traits become stronger across the life course (Hatemi et al. 2009) and as the Minnesota data is concentrated on adults in the last third of the life course, it is not surprising that this data would generate higher A estimates. Third, these can be viewed as samples of two different populations (Minnesota versus a sample where 60% was ascertained from across the US and 40% from the greater Virginia area), and genetic and environmental influences on a given trait may vary across populations. Finally, the VA30K sample is 6 times larger.

Our concern is less with differences than similarities. Using a simple correlation-based CTD, both samples generate A estimates within the 40 to 60 percent range (albeit on different ends of that range), both show MZ ideological similarities are not particularly sensitive to differences in social or family environment, both show DZ ideological similarities are highly sensitive to these differences, and, finally, both indicate that variation in A estimates—certainly within and possibly even across samples—is due largely to environmental variation relative to DZ twins.

A Formal Test of Environmental Influence on Genetic and Environmental Paths

The analyses presented in Tables 2 and 3 offer, at best, mixed support for points 1 and 2 of the EEA critique. The invariance of MZ similarities relative to social and family environments is not at all consistent with these arguments. Yet the potential of a gene-environment interaction does leave open the possibility that the individual CTD estimates of A, C and E might be “pushed” around by environmental influences. To what extent is the variance consistently

attributed to A, C and E in CTD studies substantively moderated by family and social environment?

(Figure 1 about here)

To answer this key question we tested a series of structural equations models (SEM) on the Minnesota data. In this framework, a CTD is operationalized as a set of equations with known or assumed values (observed variances-covariances of the observed trait, genetic and environmental relationships between twins) and a set of unknown values. The object is to use the known values to estimate the unknowns, which is done iteratively using maximum likelihood estimators (for primers see Medland and Hatemi 2009, Purcell 2008). The key unknowns, of course, are the amount of total variance that can be assigned to the latent ACE components. The basic framework is presented graphically as a path model in Model A of Figure 1. Here, following the tracing rules of path analysis, total (observed) variance in trait T is simply a product of the (SEM estimated) squared path coefficients a, c and e:

$$T^2 = a^2 + c^2 + e^2 \quad (1)$$

The standardized ACE estimates are simply the right hand elements represented as a proportion of T^2 (e.g. $A = a^2/T^2$). Also portrayed in Model A is an exogenous control variable “M”; inclusion of this term allows us to “regress” out M and calculate ACE estimates from Equation 1 only after accounting for the main effect of M. This is potentially important because we can assess the impact of similar childhood environments and levels of adult contact by accounting for the variance they independently account for in ideology and see what happens to our ACE estimates. The idea is to stop A from “stealing” variance from these particular elements of C.

Accounting for the main effect of an exogenous control variable, however, does not directly model gene-environment interaction. Model B in Figure 1 extends Model A to do exactly this. Here, we are not simply accounting for the main effect of M , but examining how it independently moderates the paths of a , c and e :

$$T^2 = (a + \beta_a M)^2 + (c + \beta_c M)^2 + (e + \beta_e M)^2 \quad (2)$$

Equation 2 treats each of the a , c and e paths like an individual regression equation. The individual a , c and e terms are analogous to intercepts and the betas are slopes estimating the additional impact of the moderator variable. We get standardized ACE estimates in the same fashion as from Equation 1, but those estimates will now take into account the moderating effect of M on each of these paths. Equation 2 allows us to do two important things. First, it allows us to statistically test GxE (see Purcell 2002 for technical details). Note that Equation 1 is nested in Equation 2, i.e. Equation 1 is nothing more than Equation 2 with the betas set to zero. Thus we can set β_a to 0 and test whether M is actually altering the contribution to A using standard goodness of fit statistics. We can do the same for C and E . Second, we can generate point estimates of the impact of M . Just as in a regression equation, we can simply plug in a value for M and see how it changes contribution of the latent component to observed variance.

To operationalize Models A and B using the Minnesota sample, we first created two moderator variables by conducting separate factor analyses on our three mutual influence questions and four similar experience questions reported in Table 1 (i.e. we used all available variation in the 5- and 7-point items for the factor analysis, not the dichotomous high/low cuts presented in Table 2). The idea here was to capture all the common variance from our measures of mutual influence and similar experience and enter them as controls in our structural equations model. Each analysis reported a single factor that accounted for approximately 50 percent of the

variance. These factors were normally distributed around zero. These two factors—one for mutual influence and one for similar experience—constitute the M terms portrayed in Models A and B.

With these moderator variables in hand we simply operationalized Model B using the Mx statistical software package and began systematically dropping terms (i.e. constraining them to zero) until we ended up with the most basic version of Model A portrayed in Equation 1, i.e. a classic twin design that includes no exogenous control variable and no moderator variables on the ACE paths. Using this basic Model A analysis, the standardized, maximum likelihood estimates (ACE) calculated from Equation 1 are as follows:

$$Var(T) = .60 + .04 + .36$$

In other words, a classic twin design model applied to the Minnesota sample estimates that 60 percent of the variance in Wilson-Patterson scores is attributable to broad sense heritability and less than five percent to shared environment. The 95 percent confidence intervals around these estimates are A (.37-.69), C (0-.25), E (.30-.42). In and of themselves, these results are not that interesting; they simply replicate the findings of other studies using different samples that consistently report A at 40 to 60 percent and C at 20 percent or less. The real question is what happens when we allow mutual influence and similar experience to moderate these estimates. Are these relatively high A (and low C) estimates masking a significant GxE interaction anchored in the family and social environmental dimensions central to points 1 and 2 of the EEA critique?

Our analysis provides a mixed answer. The fully specified Model B results in the following (unstandardized) estimates for Equation 2:

$$Mutual\ Influence\ Moderator: T^2 = (.63 + .01M)^2 + (.21 - .11M)^2 + (.48 + .10M)^2$$

$$\text{Similar Experience Moderator: } T^2 = (.50 + .01M)^2 + (.16 - .02M)^2 + (.39 + .03M)^2$$

To test the statistical significance of the moderator terms we used the goodness of fit of the full model as a baseline to compare nested models when these terms were constrained to zero (goodness of fit is measured using a -2 log-likelihood, which is distributed as a chi-square and can be used in an analogous fashion to employing a joint F test to assess terms in a regression equation). The results of these significance tests are reported in Table 4. The key information in this table is in the last column, which reports whether constraining specific terms to zero results in a model that is a significantly worse fit than the full model. Statistically insignificant probabilities ($p > .05$) indicate dropping the term has no impact on model fit and that the term can be treated as zero. Note that these tests indicate that the A and C moderators (β_a and β_c from Equation 2) are statistically insignificant. There is a main effect for similar experience, but this is not the case for mutual influence; i.e. mutual influence does not independently account for *any* significant amount of variance in Wilson-Patterson scores.

(Table 4 about here)

Still, our analysis is picking up some evidence of GxE. For example, the A and C moderator terms for similar experience do come close to significance ($p = .07$ and $.08$). Giving the maximum statistical benefit of the doubt, we treated all the moderator terms as real and calculated how they changed the ACE estimates. In other words, we plugged in different values of M in Equation 2 to see how the overall estimates from Model A (a CTD) might change with variation in social and family environment. So, for example, to calculate the estimate of A if mutual influence is high, say, two standard deviations above the mean, we take our results for the heritability component reported above and put in a value of 2 for M (this is because our M terms are already standardized factor scores). This results in: $(.63 + .01*2)^2 = .43$. To standardize the

estimate, we simply divide .43 by total variance (in this case .92) which gives us .47. Thus the model estimates that A, i.e. the proportion of variance in Wilson Patterson scores attributable to genetic influence, is .47. To calculate genetic influence when mutual influence levels are very low, we can replace 2 with -2, i.e. a factor score two standard deviations below the mean (this results in an A estimate of .57). Even if the moderator term, and thus the difference in A, is not statistically significant, A estimates that are substantively different from those reported by Model A would provide at least some hint that failing to account for environmental influence results in inflated heritability estimates of political temperament in a CTD. In Table 5 we report ACE estimates for Model A (no moderators) and for Model B with different values of mutual influence and similar environment moderators. To generate these estimates we entered mean mutual influence/similar experience scores as a value for M in Equation 2 (-1 to 1.0), as well as scores for two standard deviations below this mean and two standard deviations above the mean.

(Table 5 about here)

These estimates strongly suggest that the overall estimates generated by a classic twin design are not unduly biased because they fail to control for similar experience or mutual influence. The lowest we could push the A estimate is .47, which is the heritability estimate if the mutual influence value M is set at two standard deviations above the mean. Approximately six percent of our sample is that far out in the tail on our variable of mutual influence. What this means is that even if we treat mutual influence as significant (which in our model it is not), and even if we assume an entire population where mutual influence is extremely high, the model still estimates heritability accounts for about 50 percent of the variance in political attitudes. We did manage to push the C estimate outside of the 95 percent confidence intervals generated by the un-moderated classic twin design (Model A) reported above. This again, however, relied on

ignoring the statistical insignificance of the mutual influence moderator and assuming an extreme value on this element of the social environment (two standard deviations below the mean). These rather heroic assumptions managed to push the estimate of C to .31.

What this analysis shows is that while gene-environment interactions may indeed play a role in driving political orientations, the umbrella estimates of heritability generated by a standard, no-frills CTD (Model A) are quite robust. Genetic influence may be moderated by various elements of family or social environments, but even out in the tails of this environmental influence the heritability estimate of political temperament remains non-zero, non-trivial and within the 40 to 60 percent range generated by standard CTDs.

Taking Stock and Comparing Politics to Educational Attainment

The overall pattern of our results is not as simple as often portrayed by either side of the EEA debate. If the greater genetic relatedness of MZ twins was irrelevant to their observed greater ideological similarity then MZ and DZ twins should display substantially reduced correlations when environmental similarity or contact is reduced. In addition, the correlations for MZ and DZ pairs should be approximately equal across levels of environmental similarity and contact. As Table 2 and Table 3 clearly demonstrate, this is not the case. In contrast, if environmental similarity and adult contact were of absolutely no consequence for political ideology then the political similarity of DZ twins should remain unaltered across the high and low categories of environmental similarity and adult contact. In fact, as Table 2 and 3 report, there are robust differences for DZ co-twin correlations, suggesting environmental factors have interactive rather than direct impacts. We find some evidence of such an impact in our data,

though nothing to suggest that the typical heritability estimates generated by CTD studies of political traits are seriously or misleadingly inflated.

One way to summarize the overall pattern of our analyses is to say that at high levels of genetic similarity, environmental factors play a small role in explaining political similarity. At more typical levels of genetic similarity, environmental factors contribute more to political similarity. Our analyses are thus consistent with a susceptibility model, i.e. the heritable underpinnings of adult ideology operate largely by creating varying levels of susceptibility to specific sorts of ideological appeals. MZ cotwin correlations in this view vary less with environmental differences not because the individual twins lack susceptibility to environmental influence, but instead because *they share precisely the same susceptibility to environmental influence*. DZ twins in contrast vary in degree of shared genetic heritage (GWAS show a range of similarity among DZ twins running from .38 to .62—see Visscher et al. 2006) and this genetic variability interacts with environmental variability to produce variations in the phenotype.

A susceptibility perspective also highlights the possibility that there may be an underlying genetic component to environmental similarity. The analyses reported thus far are deliberately structured in a manner which assumes the critics position; i.e. that environmental similarity leads to ideological similarity. Yet it is also possible that ideological similarity leads to environmental similarity. This especially could be the case with adult contact. In other words, if liberals prefer the company of liberals over conservatives, and vice versa, twins who are more alike ideologically may be more likely to maintain a high level of contact with their co-twin. If, as twin studies suggest, there is a genetic predisposition towards ideology, this in turn raises the possibility that there is a genetic component underlying the environmental variation reported by twins. This latter view already has considerable empirical support. For example, Posner et al.

(1996) using several thousand twins pairs in a longitudinal sample, found that while there is some evidence for contact leading to similar Wilson Patterson scores, there is a considerable stronger case for similar Wilson Patterson scores leading to higher levels of contact.

Unfortunately our data on political ideology are not longitudinal and thus provide no leverage to empirically test the causal direction at issue here. We can, however, gain some insight by considering an alternative trait: highest level of educational achievement.

We found that cotwin correlations on education follow the same patterns as those reported for ideology in Table 2. Specifically, MZ correlations for highest level of educational achievement are in the .6 to .7 range and vary only slightly by level of contact. Also, as was the case in Table 2, the DZ correlations for highest level of educational achievement are elevated for twins who see each other or speak to each other frequently, and substantially diminished for DZ pairs who have less frequent contact. As noted above, for political ideology this configuration of results suggests that for DZ twins, where there is sufficient genetic variation for a gene/environment interaction to emerge, more frequent adult contact leads to both more similar adult political environments and the potential for mutual persuasion and reinforcement. The same argument cannot be made for highest level of educational attainment, as this has already been determined well before the contact levels reported by our 50-plus year old subjects. This pattern is expected, however, if both level of contact and educational achievement are being driven at least in part by genetic similarity.¹

¹ It should be noted that nothing in this analysis speaks to the broader and more contentious debate on the heritability of educational attainment and its correlates (such as IQ), and inter-group socioeconomic differences. Our point is simply that MZ similarities in educational attainment do not vary much with environmental differences, while DZ similarities in educational attainment do vary with such environmental differences. Our analysis says nothing about the heritability of educational attainment by race, income or any other socio-demographic variable (for varying perspectives on this debate see Bouchard et al 2003, Turkheimer 2003).

Points 3 and 4: Biological Equal Environment Assumptions

To this point, we have confined our concern to points 1 and 2 of the four point general critique of the equal environments assumption. Points 1 and 2 concern similarities in the social environment and as such are consistent with traditional social science explanations for political attitudes and behaviors. Points 3 and 4, in contrast, focus on biological environments which are less often considered in the existing literature.

The similar treatment criticism assumes that at least some of the increased similarity in the ideology of MZ twins is a reflection of the fact that they are on average more similar in appearance than DZ twins. This is an intriguing claim and there is some basis for such an expectation. Numerous studies suggest physical appearance relates to how people are treated socially and economically, that the appearance of political candidates (especially racial and gender attributes) can shape electoral preferences and voting behavior, and that physical characteristics such as upper body strength in males may lead to preferences for certain political outcomes (Sell et al. 2009; Heflick and Goldenberg 2009; Mobius and Rosenblat 2006; Rosenberg et al. 1986). There is even some preliminary evidence tying beauty to ideology; Berrgren et al. (nd) found that conservative political candidates tend to be better looking and that conservatives generally respond more to physical beauty in the political realm. It thus seems not unreasonable to suggest that physical appearance shapes treatment by others, and that the socialization effect of such treatment may have some influence on political temperament.

Even if this is so, however, whatever variation in ideological similarity can be attributed to physical appearance has an indisputably genetic basis. Even critics of twin studies concede that MZ twin pairs look more alike than DZ twin pairs because of their closer genetic relationship. The argument in point three then is not that genes have a substantively trivial role in

determining political ideology, simply that this role is indirect. Still, while the link from genes to physical appearance is well established, evidence for the link from physical appearance to political ideology is less extensive. If critics of twins studies are correct, what we are talking about here is not simply whether better looking people are more likely to get jobs, dates, votes, viewers, or social deference, but whether physical appearance systematically creates an environment that socializes individuals into a particular set of political attitudes. We are aware of no such study with this specific empirical focus, but DZ twins, like the population in general, vary substantially in their appearance so this thesis could conceivably be tested empirically. Yet, even if the thesis that physical appearance plays a role in socializing political beliefs is proven correct, it does nothing to contradict the belief that individual genetic variation is unrelated to political orientations. Indeed, it confirms the opposite. If physical appearance is shown to determine ideology, this simply shifts the debate from whether genes influence political traits to how they influence political traits.

The final challenge raised by critics that we address here is the most biological in nature. It attributes the heightened similarity between MZ twins relative to DZ twins to the fact that the majority (roughly two-thirds) of MZ twins are also monozygotic twins (i.e. in utero they develop within the same fetal sac) while all DZ twins are not. This presupposes that the shared fetal sac of MZ twins represents a substantial shared environment and that this heightened similarity extends directly or indirectly to adult political orientations. We know of no existing studies of chorionicity and political attitudes or behaviors, though there have been a small number of chorionicity studies related to other traits. The logic of the studies is quite straightforward. If the MZ- DZ difference can be accounted for by the monozygotic-dichorionic difference, then the approximately one third of MZ twins that are also dichorionic

will resemble DZ twins with whom they share that trait more closely than MZ monozygotic twins. Studies have linked chorionicity to some personality characteristics (Reed et al., 1991; Sokol et al., 1995) and some cognitive abilities (Jacobs et al., 2001; Melnick et al., 1978). The closest thing to political orientation investigated in these sorts of studies is variation in pro-social behavior, which was found to be clearly related to zygosity but unrelated to chorionicity (Hur 2007). Still, if chorionicity accounts for a substantial share of the differences in political attitudes and behavior that twin studies attribute to genes, the implications for mainstream political science explanations of those traits are profound. The implication is that in utero environments are potentially more important determinants of political orientations than are shared childhood environments and socialization (i.e., that we are not born into the world as blank political slates). That brings critics of twin studies into agreement with proponents on a key issue, namely the importance of biological precursors of adult ideology that are present at birth, and strengthen rather than weaken our call to re-examine the epistemological foundations of the discipline.

From the perspective of twin studies, however, there are at least two key limitations of the chorionicity argument. First, while it can account for similarity in MZ twins it cannot account for any of the similarity in DZ twins. Second, chorionicity cannot account for any of the correlation between parents and offspring. Genetic heritability is clearly a more parsimonious explanation here as it simultaneously accounts for moderate similarity between DZ twins, moderate similarity between parents and offspring, and substantially higher similarity between MZ twins. Still, given the lack of empirical evidence, the influence of chorionicity on political orientations must remain an open question.

Conclusion

The validity of challenges to twin studies should be addressed as empirical issues rather than debating points; therefore, we have tested with original data two of the four main critiques and found them wanting. Our broader examination of the EEA critique indicates that even if it is ultimately discovered that some of the criticisms of twin studies are empirically valid, the evidence from those studies, combined with molecular, physiological, neurological and other studies provides good reasons for political science to take biology (not just genes) seriously as a basis for explaining attitudes and behaviors. As a discipline, we can safely ignore biology only if humans are political blank slates at birth and neither genes nor downstream biology plays any role in adult political orientations. Interestingly enough, in this we are in perfect agreement with Suhay, Kalmoe, and McDermott (2007) and also with Charney's recent writing (2010). Though raising questions about whether twin studies convincingly demonstrate genetic influence on political traits, Suhay, Kalmoe, and McDermott wholeheartedly endorse a broad role for genetics in accounting for political traits. As they conclude:

In critiquing the twin study method used by Alford et al. and highlighting the important contribution of familial socialization, we do not mean to propose an environmental determinist argument, however. The development of human traits, including ideological beliefs and political attitudes, is influenced by a number of factors: genes; other biological elements within the human organism; the physical environment, natural and manmade; and a host of micro- and macro-social environments. (2007, 35)

We fully agree. We part company only in the sense that, for the reasons detailed in this study, we see the EEA critique as conceptually and empirically providing little substantive constraint on inferences that genes and thus biology influence political orientations. The debate on exactly how much of an influence and through what processes is important and ongoing. Genetic influence may turn out to differ across various traits (attitudes versus behaviors for example) and the impact of EEA violations could potentially also vary across those traits. Our analyses, however, strongly support the inference that nothing in the EEA critique credibly

supports a claim that genetic influence on political temperament is non-zero or non-trivial. Additionally, our analyses find nothing to suggest that heritability estimates from CTD studies on political traits are misleading or unduly biased. At a minimum, the undisputed patterns of ideological similarity between MZ and DZ twins do not seem to be fully explained by the mechanisms proposed by EEA critics. Our analyses provide little reason to expect Points 1 and 2, individually or in combination, to provide a wholly environmental explanation for observed MZ-DZ differences in ideological similarity. Points 3 and 4 *are* arguments that genes and biology influence political orientation; we might be skeptical about some of the hypotheses arising from these latter two points, but are willing to be persuaded if the empirical evidence accumulates.

Twin studies within political science consistently indicate that 40 to 60 percent of the variation in adult political orientations is heritable. The notion that this variance being attributed to genes is in reality entirely due to a combination of non-political early childhood environmental similarity (i.e. being dressed alike or sharing the bedroom), the mutual influence of a sibling, pre-partum, in utero environment, physical appearance, and the seemingly inherent tendency to treat others differently based on these (biologically-based) appearances strikes us as extremely unlikely. But even if it is, key elements of this argument—in utero environment, the genetics of physical appearance—automatically bring its adherents into agreement with our central point: That political science must increase the absorption of biological variation into its epistemology. Critics and proponents of twin studies are in general agreement that a wholly non-biological paradigm for the acquisition of political attitudes will unnecessarily limit progress. Our analysis here certainly reinforces that conclusion while also advancing empirical support for a model of gene/environment interaction that gives both nature and nurture their due.

References

- Alford, John R., Carolyn L. Funk, and John R. Hibbing. 2005. "Are Political Orientation Genetically Transmitted?" *American Political Science Review*, 99(2): 153-168.
- Beckwith, Jon and Corey Morris. 2008. "Twin Studies of Political Behavior: Untenable Assumptions?" *Perspectives on Politics*. 6: 785-792.
- Bell, Edward, Julie Aitken Shermer, and Philip A. Vernon. 2009. "The Origins of Political Attitudes and Behavior." *Canadian Journal of Political Science* 42 (4): 855-79.
- Berrgren, Niclas, Henrik Jordahl, Panu Poutvaara. "The Right Look: Conservative Politicians Look Better and Their Voters Reward It." Unpublished manuscript.
[Http://ideas.repec.org/p/sol/wpaper/2013-76228.html](http://ideas.repec.org/p/sol/wpaper/2013-76228.html)
- Bouchard, Thomas Jr. and Matt McGue. 2003. "Genetic and Environmental Influences on Human Psychological Differences." *Journal of Neurobiology*. 54: 4-45.
- Campbell, Angus, Philip Converse, Warren Miller, and Donald Stokes. 1960. *The American Voter* New York: John Wiley and Sons.
- Carmen, Ira. 1997. "Biopolitics: The Newest Synthesis?" *Genetica*. 99: 173-184.
- Charney, Evan. 2008. "Genes and Ideologies." *Perspectives on Politics*. 6: 299-319.
- Charney, Evan. 2010. "Epigenetics and the Genetic Explanations of Political Behavior." Paper presented at the Annual Meeting of the Midwest Political Science Association, Chicago, April 2010.
- Cook, Timothy. 1985. "The Bear Market in Political Socialization and the Costs of Misunderstood Psychological Theories." *American Political Science Review*. 79: 1079-1093.
- Dawes, Christopher and James Fowler. 2009. "Partisanship, Voting, and the Dopamine D2 Receptor Gene." *The Journal of Politics*. 71: 1157-1171.
- Eaves, LJ, and Eysenck HJ. 1974. "Genetics and the Development of Social Attitudes." *Nature*. 249: 288-289.
- Eaves, LJ and PK. Hatemi. 2008. "Transmission of attitudes toward abortion and gay rights: Parental socialization or parental mate selection?" *Behavior Genetics* 38:247-256.
- Falconer, D.S. 1960. *Introduction to Quantitative Genetics*. Edinburgh: Oliver and Boyd.

- Fowler, James, Laura A. Baker, and Christopher T. Dawes. 2008. "Genetic Variation in Political Participation." *American Political Science Review* 102 (May): 233-48.
- Fowler, James and Christopher Dawes. 2008. "Two Genes Predict Voter Turnout." *Journal of Politics*. 70: 579-594.
- Fowler, James and Darren Schreiber. 2008. "Biology, Politics, and the Emerging Science of Human Nature." *Science*. 322: 912-914.
- Funk, Carolyn L. 2010 "Connecting the Social and Biological Bases of Public Opinion. In Robert Y. Shapiro and Lawrence R. Jacobs, eds., *The Oxford Handbook of American Public Opinion and Media*. Oxford University Press
- Glass, Jennifer, Vern Bengston and Charlotte Chorn Dunham. 1986. *American Sociological Review*. 51: 685-698.
- Hatemi, Peter, John Alford, John Hibbing, Nicholas Martin and Lindon Eaves. 2009a. "Is There a 'Party' In Your Genes?" *Political Research Quarterly*. 61: 584-600.
- Hatemi, Peter, Carolyn Funk, Hermine Maes, Judy Silberg, Sarah Medland, Nicholas Martin and Lindon Eaves. 2009b. "Genetic Influences on Political Attitudes over the Life Course." *Journal of Politics* 71 (July): 1141-58.
- Hatemi, Peter K., Katherine I. Morley, Sarah E. Medland, Andrew C. Heath, and Nick G. Martin. 2007. "The Genetics of Voting: An Australian Twin Study." *Behavior Genetics* 37:435-448.
- Hatemi, Peter, John Hibbing, Sarah Medland, Matthew Keller, John Alford, Kevin B. Smith. 2010. "Not By Twins Alone: Using the Extended Family Design to Investigate Genetic Influence on Political Beliefs." *American Journal of Political Science* 54 (July): 798-814.
- Hatemi, Peter K. Dawes C, Forest-Keller A, Settle J, and B Verhulst. 2011. "News from the Political Front: Genetics of Social Attitudes" *BioDemography* (forthcoming).
- Hatemi, Peter K., N. Gillespie, L. Eaves, B. Maher, B. Webb, A. Heath, S. Medland, D. Smyth, H. Beeby, S. Godon, G. Montgomery, G. Zhu, E. Byrne, N. Martin.. 2011b "Genome-Wide Analysis of Political Attitudes." *Journal of Politics* 73(1):1-15
- Heflick, Nathan A. and Jamie L. Goldenberg. 2009. "Objectifying Sarah Palin: Evidence that Objectification Causes Women to be Perceived as Less Competent and Less Fully Human." *Journal of Experimental Social Psychology*. 45: 589-601.
- Hess, Robert and Judith Torney. 1967. *The Development of Political Attitudes in Children*. Chicago: Aldine.
- Hibbing, John R. and Kevin B. Smith. 2008. "The Biology of Political Behavior." *The Annals of the American Academy of Political and Social Science*. 617; 6-14.

- Hyman, Herbert. 1959. *Political Socialization*. New York: Free Press.
- Jennings, M.Kent and Richard Niemi. 1968. "The transmission of Political Values from Parent to Child." *American Political Science Review*. 62: 169-183.
- Jennings, M. Kent and Richard Niemi. 1991. "Issues and Inheritance in the Formation of Party Identification." *American Journal of Political Science*. 35: 970-988.
- Jennings, M. Kent, Laura Stoker, and Jake Bowers. 2009. "Politics Across Generations: Family Transmission Reexamined." *The Journal of Politics* 71 (3): 782-799.
- Johnson, D, R. McDermott, J. Cowden, E. Barrett, R. Wrangham, and S. Rosen. 2006. "Male Overconfidence and War." *Proceedings of the Royal Society of London (Biology)*. 273: 2513-2520.
- Joseph, Jay. 2004. *The Gene Illusion: Genetic Research in Psychiatry and Psychology Under the Microscope*. New York: Algora
- Kendler, K.S., A.C. Heath, N.G. Martin and L. J. Eaves. 1987. "Symptoms of Anxiety and Symptoms of Depression. Same Genes, Different Environments?" *Archives of General Psychiatry*. 44: 451-457.
- Klemmensen, Robert, Sara Binzer Hobolt, Asbjørn Sonne Norgaard, Inge Petersen, and Axel Skythe. 2010. "The Determinants of Political Participation." Paper presented at the Annual Meeting of the Midwest Political Science Association, Chicago, April 2010.
- Kosfeld, Michael, Markus Heinrich, Paul Zak, Urs Fischbacher and Ernst Fehr. 2005. "Oxytocin Increases Trust in Humans." *Nature*. 425: 673-676.
- Krueger, Robert F. and Wendy Johnson. 2002. The Minnesota Twin Registry: Current Status and Future Directions. *Twin Research*, 5(5): 488-492.
- Langton, Kenneth P. 1967. "Peer Group and School and the Political Socialization Process." *American Political Science Review*. 61: 751-758.
- Loehlin, J.C. and R.C. Nichols. 1976. *Heredity, Environment, and Personality*. Austin, TX: University of Texas Press.
- Lykken, D.T., T.J. Bouchard, Jr., M. McGue, and A. Tellegen. 1990. The Minnesota Twin Family Registry: some Initial Findings. *Acta Geneticae Medicae et Gemellogica*, 39: 35-70.
- Madsen, Douglas. 1985. "A Biochemical Property Relating to Power Seeking in Humans." *American Political Science Review*. 79: 448-457.

Martin, Nicholas G., Lincon J. Eaves, Anthony C. Heath, R. Jardine, L. M. Feingold, and Hans J. Eysenck. 1986. "Transmission of Social Attitudes." *Proceedings of the National Academy of Sciences* 15 (June): 4364-68.

Masters, Roger. 1989. *The Nature of Politics*. New Haven: Yale University Press.

Medland, Sarah and Peter Hatemi. 2009. "Political Science, Biometric Theory, and Twin Studies: A Methodological Introduction." *Political Analysis*. 17: 191-214.

Merelman, Richard M. 1971. "The Development of Policy thinking in Adolescence." *American Political Science Review* 65 (4): 1033-47.

Mobius, Markus and Tany Rosenblat. "Why Beauty Matters." *The American Economic Review*. 96: 222-235.

Oxley, Douglas R., Kevin B. Smith, John R. Alford, Matthew V. Hibbing, Jennifer L. Miller, Mario Scalora, Peter K. Hatemi, and John R. Hibbing. 2008. "Political Attitudes Vary With Physiological Traits." *Science*. 321: 1667-1670.

Purcell, Shaun. 2002. "Variance Components Models for Gene-Environment Interaction in Twin Analysis." *Twin Research*. 5: 554-571.

Rosenberg, Shawn, Lisa Bohan, Patric McCafferty, and Kevin Harris. 1986. "The Image and the Vote: The Effect of Candidate Presentation on Voter Preference." *American Journal of Political Science*. 30: 108-127.

Sapiro, Virginia. 2004. "Not Your Parents Political Socialization: Introduction for a New Generation." *Annual Review of Political Science*. 7: 1-23.

Scarr, Sandra and Louise Carter-Saltzman. 1979. "Twin Method: Defense of a Critical Assumptions." *Behavior Genetics*. 9: 527-542.

Sell, Aaron, Leda Cosmides, John Tooby, Daniel Sznycer, Christopher von Rueden and Micahel Gurven. 2009. "Human Adaptations for the Visual Assessment of Strength and Fighting Ability from the Body and Face." *Proceedings of the Royal Society B*. 276: 575-584.

Settle, Jaime, Christopher Dawes, James Fowler. "The Heritability of Partisan Attachment." *Political Research Quarterly*. 63 (3): 601-13.

Smith, Kevin, Douglas Oxley, Matthew Hibbing, John Alford and John Hibbing. 2011. "Disgust Sensitivity and the Neurophysiology of Left-Right Political Orientations." *PLoS One*. Forthcoming.

Stoker, Laura, and M. Kent Jennings. 2008. "Of Time and the Development of Partisan Polarization." *American Journal of Political Science*. 52 (3): 619-635

Suhay, Elizabeth, Nathan Kalmoe and Christa McDermott. 2007. "Why Twin Studies are Problematic for the Study of Political Ideology: Rethinking Are Political Orientations Genetically transmitted?" Paper presented at the annual meeting of the International Society of Political Psychology, Portland, OR, July 4-7, 2007.

Tedin, Kent. 1974. "The Influence of Parents on the Political Attitudes of Adolescents." *American Political Science Review*. 68: 1579-1592.

Turkheimer, Eric, Andreana Haley, Mary Waldron, Brian D'Onofrio, and Irving I. Gottesman. 2003. "Socioeconomic Status Modifies Heritability of IQ in Young Children." *Psychological Science*. 14: 623-628.

Vanman, Eric J., Jessica Saltz, lauri Nathan and Jennifer Warren. 2004. "Racial Discrimination by Low-Prejudiced Whites: Facial movements as Implicit Measures of Attitudes Related to Behavior." *Psychological Science* 15: 711-714.

Visscher, Peter M., Sarah E. Medland, Katherine I. Morley, Gu Zhu, Belinda K. Cornes, Grant W. Montgomery, and Nicholas G. Martin. 2006. "Assumption-Free Estimation of Heritability from Genome-Wide-by Descent Sharing between Full Siblings." *PLoS Genetics* 2 (3): 316-24.

Wilson, GD and JR Patterson 1968. "A New Measure of Social Conservatism." *British Journal of Social and Clinical Psychology* 7: 264-69.

Zak, Paul, Robert Kurzban and William Matzer. 2005. "Oxytocin is Associated With Human Trustworthiness." *Hormones and Behavior*. 48: 522-527.

Zaller, John. 1992. *The Nature and Origins of Mass Opinion*. New York: Cambridge University Press.

Zuckerman, Alan S., Josip Dasović, and Jennifer Fitzgerald. 2007. *Partisan Families: The Social Logic of Bounded Partisanship in Germany and Britain*. Cambridge: Cambridge University Press.

Table 1: Similar Experience and Mutual Influence By Zygosity

Variable	MZ mean	DZ mean	Difference of means test
<i>Similar experience</i>			
When you were growing up, how often did you and your twin ...			
Share the same bedroom at home	4.74	4.55	4.89*
Have the same friends	4.11	3.61	10.54*
Dress alike	3.31	2.80	8.66*
Attend the same classes at school	3.51	3.18	5.28*
<i>Mutual influence</i>			
How often do you usually see your twin?	3.62	3.20	5.78*
How often do you usually talk to your twin on the telephone?	4.53	4.00	7.72*
How often do you usually contact your twin electronically (through text messages, e-mail, Facebook, or other devices)?	3.24	3.00	2.52*

Means, t-score reported

* < .05, 2-tailed test

N = 712 MZ (356 complete twin pairs)

N=480 DZ (240 complete twin pairs)

Similar experience questions measured on a 5 point scale where 1=never, 5=always, mutual influence questions measured on a 7 point scale where 1=less than once a year, 7=every day

Table 2: Heritability of Ideology by Mutual Influence and Similar Experiences

Attitude Item	Correlation (r)				Heritability (A)	Shared Env. (C)	Unshared Env. (E)
	MZ (1)	N (2)	DZ (3)	N (4)	2*(MZ-DZ) (5)	(2*DZ)-MZ (6)	1-MZ (7)
See your twin?							
Once a month or more	0.722	176	0.426	77	0.592	0.131	0.277
several times a year or less	0.633	144	0.327	124	0.612	0.021	0.367
Talk to your twin on the telephone?							
Once a week or more	0.720	195	0.581	78	0.278	0.442	0.28
Once a month or less	0.580	158	0.226	120	0.696	-0.128	0.425
Communicate with twin text/email?							
Once a week or more	0.769	80	0.382	36	0.774	-.005	0.231
Once a month or less	0.582	203	0.268	146	0.628	-.046	0.418
Mean r high contact	0.737		0.463		0.548	0.189	0.263
Mean r low contact	0.597		0.273		0.648	-0.051	0.403
Attended Same Classes at School							
Always/usually	0.6	137	0.533	54	0.134	0.466	0.4
Sometimes/rarely/never	0.69	114	0.274	98	0.832	-0.142	0.31
Dressed alike when growing up?							
Always/usually	0.637	101	0.666	18	-0.058	0.695	0.363
Sometimes/rarely/never	0.684	147	0.346	152	0.676	0.008	0.316
Shared the same bedroom at home?							
Always	0.667	262	0.481	141	0.372	0.295	0.333
usually/sometimes/rarely/never	0.711	42	0.170*	41	1.082	-0.371	0.289
Had the same friends growing up?							
Always/usually	0.673	255	0.463	101	0.420	0.253	0.327
Sometimes/rarely/never	0.455	32	0.165*	72	0.58	-0.125	0.545
Mean r similar childhood	0.644		0.535		0.218	0.423	0.356
Mean r dissimilar childhood	0.635		0.238		0.794	-0.159	0.365
Mean r high contact/similar childhood	0.684		0.504		0.36	0.324	0.316
Mean r low contact/dissimilar childhood	0.619		0.253		0.732	-0.113	0.381
WP 7 point index all	0.644	354	0.349	237	0.59	0.054	0.356

*not significant .05 level, all other coefficients significant .01 level.

Table 3: Comparison of Results from the Minnesota and Virginia 30K Studies

	Correlation				Heritability (A) 2*(MZ-DZ)	Shared Env. (C) (2*DZ)-MZ	Unshared Env. (E) 1-MZ
VA30K Wilson/Patterson Index (3 point)	MZ	n	DZ	n			
Mean correlation high contact	0.66		0.48		0.36	0.30	0.34
Mean correlation low contact	0.57		0.34		0.46	0.12	0.43
Minnesota Wilson/Patterson Index (7 point)							
Mean correlation high contact	0.74		0.46		0.55	0.19	0.26
Mean correlation low contact	0.60		0.27		0.64	-0.05	0.41
VA30K Wilson/Patterson Index (3 point)							
Mean correlation similar childhood	0.64		0.44		0.41	0.23	0.36
Mean correlation dissimilar childhood	0.67		0.44		0.46	0.21	0.33
Minnesota Wilson/Patterson Index (7 point)							
Mean correlation similar childhood	0.64		0.53		0.23	0.42	0.36
Mean correlation dissimilar childhood	0.64		0.24		0.79	-0.16	0.36
VA30K Wilson/Patterson Index (3 point)							
Wilson/Patterson Index all cases	0.65	2107	0.43	1384	0.43	0.22	0.35
Minnesota Wilson/Patterson Index (7 point)							
Wilson/Patterson Index all cases	0.64	354	0.35	237	0.59	0.05	0.36

Table 4: Significance Tests For Gene-Environment Interactions

Model	-2LL	df	$\Delta\chi^2$	p
<i>Mutual Influence Moderator</i>				
Full model (Model B)	2493.484	1141		
Drop Main Effect	2495.849	1142	2.365	.12
Drop A moderator	2493.564	1142	0.08	.77
Drop C moderator	2495.089	1142	1.605	.20
Drop E moderator	2520.322	1142	26.838	.00
No moderator on any path (Model A)	2528	1144	34.516	.00
<i>Similar Experience Moderator</i>				
Full model (Model B)	2501.801	1139		
Drop Main Effect	2508.468	1140	6.667	.00
Drop A moderator	2504.816	1140	3.015	.08
Drop C moderator	2504.975	1140	3.174	.07
Drop E moderator	2503.784	1140	1.983	.16
No moderation on any path (Model A)	2512.061	1142	10.26	.01

Full model is Model B from Figure 1. Goodness of fit statistics indicate impact on model fit as environmental moderator terms in model (mutual influence or similar experience) are constrained to zero. Insignificant results ($p > .05$) indicate dropping term has no impact on model fit and can be treated as zero.

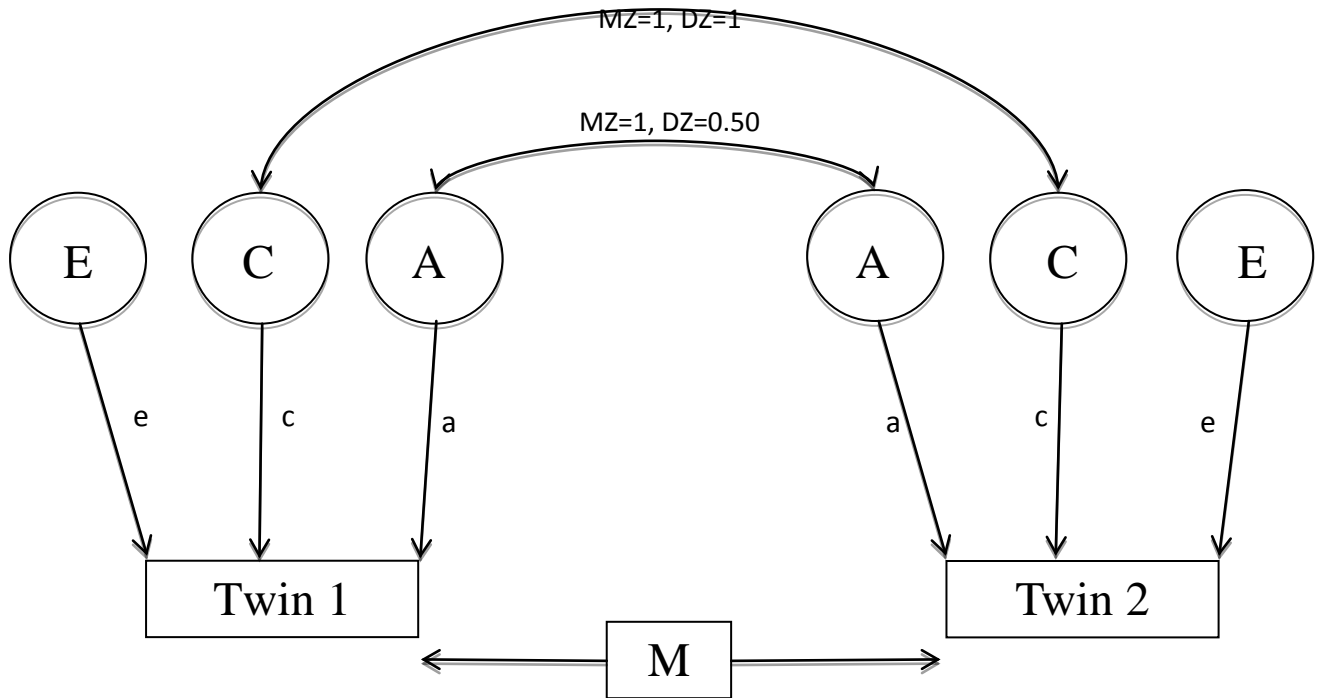
Table 5: ACE Estimates From Model A (Classic Twin Design) and Model B (GxE)

Model		A	C	E
No moderation	(Model A)	.60	.04	.36
Low Mutual Influence	(Model B)	.57	.31	.12
Mean Mutual Influence	(Model B)	.58	.07	.35
High Mutual Influence	(Model B)	.47	.00	.53
Low Similar Experience	(Model B)	.59	.12	.28
Mean Similar Experience	(Model B)	.58	.06	.35
High Similar Experience	(Model B)	.57	.02	.41

Cell values represent proportion of total population variance in Wilson-Patterson scores attributed to heritability (A), common environment (C) and unique environment (E). No moderation (Model A) estimates generated with no control or moderation terms. All other estimates calculated using estimated path betas from moderators (Model B in Figure 1) and the following mutual influence/similar experience values: Low = 2 standard deviations below the mean High = 2 standard deviations above the mean. Approximately six percent of our sample have mutual influence/similar experience scores in the high/low categories..

Figure 1: Path Models of Classic Twin Design and Gene-Environment Interaction

Model A (Classic Twin Design) = $T^2 = a^2 + c^2 + e^2$



Model B (GxE) = $T^2 = (a + \beta_a M)^2 + (c + \beta_c M)^2 + (e + \beta_e M)^2$

