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The Effect of 9/11 on the Heritability of Political Trust¹

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Abstract

Following the attacks of September 11, 2001, a rally effect led to a precipitous rise in political trust. However, the increase in political trust concealed a simultaneous decline among a smaller portion of the population. This paper examines the psychological mechanisms underlying these heterogeneous attitudes towards government and shows that a biosocial model best explains the observed patterns of response. The interplay of genetic and environmental factors of political trust reveals the stable but dynamic nature of heritability: genetic influences of political trust increased immediately following 9/11 but quickly decayed to pre-9/11 levels.

Keywords

political trust; rally effect; 9/11; biopolitics

Trust in government is dynamic, displaying both a long-term downward trend (Hetherington, 1998; Levi and Stoker, 2000) and short-term responses to events. Among the latter, a number of rally-around-the-flag surges in political trust have been observed. These surges have followed crises such the Reagan assassination-attempt (Ostrom and Simon, 1989) and the Persian Gulf War (Parker, 1995). Examined at the individual level, however, rallies show substantial heterogeneity with some citizens becoming less trustful of government even as the majority shows increased levels of trust (Huddy et al., 2005). This paper illuminates one source of such heterogeneous effects—those rooted in genetics—by examining variation in political trust before and after the attacks of September 11, 2001. To better understand the heterogeneous responses, *rally* and *regress* effects, I explore political trust at a genetic level. Specifically, I advance a two-pronged argument: 1) the expression of trust in government is partially heritable and partially environmental, and 2) the rally effect can be explained by changes in the underlying genetic and environmental etiology of trust in government due to threatening environmental stimuli, a form of *gene-environment interaction*.

To evaluate these claims, I first argue that the extant literature on political trust, social trust, and personality provide a plausible basis for hypothesizing that political trust is substantially heritable. Second I argue that a plausible mechanism of gene expression is via emotions of fear and anger. Third, I outline a model—consistent with the first two arguments—of gene

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expression that implies a gene-environment interaction. I then show that this model yields specific, empirical, and testable expectations. These expectations are then evaluated using a dataset of twin and sibling pairs. Since some respondents were interviewed before and some after the attacks of 9/11, I can analyze the impact of threat, genes, and their interaction on political trust within the context of a natural experiment.

The Heritability of Trust

Political trust is “a basic evaluative orientation toward the government” (Hetherington, 1998, pg. 791) and in recent decades has undergone a long-term decline in America. Concern over the consistently low levels of trust since the early 1980s has resulted in scholarship that seeks to explain this decline (e.g., Hetherington, 1998; Levi and Stoker, 2000). One predominant explanation is that a low level of trust is more likely when economic concerns are at the fore and high a level of trust is more likely when international concerns are at the fore (Hetherington and Rudolph 2008). Such an explanation is consistent with the long-term decline in and stable low levels of trust since the end of the Cold War and the subsequent shift in focus from international issues to economic issues. Still, there is considerable variation in trust at the *individual* level, and scholarship has often characterized this aspect of political trust as a function of attitudes, including partisanship (Citrin, 1974), satisfaction with government performance (Miller, 1974), interpersonal trust (Abramson & Inglehart, 1970), personal life satisfaction (Inglehart, 1988), perceived government effectiveness (Hetherington 1998), diffuse support for other societal institutions (Caldeira & Gibson, 1995), and political values such as norms of democracy, commitment to liberty, and political efficacy (Caldeira & Gibson, 1992).

I argue that trust in government is partially heritable and provide evidence of this possibility by focusing on its relationship to social trust and agreeableness. Observational research using twins has identified a heritable component for an array of attitudes (Alford et al., 2005; Verhulst, Hatemi, and Eaves, 2012), including social trust (Oskarsson et al., 2012; Sturgis et al., 2010). Corroborative evidence of a biological basis to social trust has developed in the field of neuroscience. Social trust levels have been shown to fluctuate in response to oxytocin, a neuropeptide that regulates social behavior in animals, including attachment and affiliation behaviors (Zak, 2005). Experimental results find that oxytocin-treated participants exhibited more trusting behavior during a trust game than the control group (Kosfeld et al., 2008; Baumgartner et al., 2008). This is important because a link between social and political trust has been consistently documented in survey research (Brehm and Rahn, 1997; Yamagishi and Yamagishi, 1994), although the precise nature of this linkage remains unclear (Levi and Stoker, 2000). The fact that social trust and political trust are related raises the possibility of a more general and heritable propensity to trust or at a minimum a heritable component to political trust.

Research on the relationship between attitudes and personality, specifically agreeableness, provides additional bases to hypothesize the heritability of political trust. It has traditionally been assumed that personality predicts attitudes (e.g., Gerber et al., 2010; Mondak et al., 2010); however, an alternative model that emphasizes a common biological basis of personality and attitudes has garnered attention recently (Verhulst et al., 2010). In this

model, called the common causes model, biological and environmental factors are shown to be the source of the correlation between personality and political trust. Research consistently identifies a heritable component to agreeableness (Bouchard & Loehlin, 2001), and a logical extension of this work would suggest that the correlation between agreeableness and political trust (Anderson, 2010) may in fact be a consequence of a common cause—including a genetic one, which is, at a minimum, suggestive of a heritable component to political trust.

The Rally Effect

While political trust levels have declined to low and rather stable levels in recent years (Hetherington, 1998), short-term spikes in political trust—known as rally-around-the-flag effects—are often observed during times of domestic or international crises. I argue that these short-term fluctuations in political trust are due to changes in the underlying genetic and environmental etiology of trust. Rally effects are patriotic sociopolitical responses to sudden, dramatic, and international conflict (Lambert, Schott, & Scherer, 2011; Mueller, 1970) and have been observed following the Iran hostage crisis (Callaghan & Virtanen, 1993), the Reagan assassination-attempt (Ostrom & Simon, 1989), the Persian Gulf War (Parker, 1995), and more. Perhaps the most studied rally effect is the one that occurred after the attacks of September 11, 2001. Survey research has repeatedly shown a spike and decay in presidential approval (Huddy, Feldman, Taber, & Lahav, 2005) and trust in government (Perrin & Smolek, 2009) following 9/11. The microfoundations of the rally effect are not yet fully understood, with several models offered to explain when rallies will and will not occur (Hetherington & Nelson, 2003; for a review of the rally effect see Lambert et al., 2011).

Nonetheless, reactions to the attacks of 9/11 were not uniform among the population. Individuals understood the attacks differently: some experienced anger while others experienced anxiety (Huddy et al., 2005; Lerner et al., 2003; Skitka, 2004).² It is important to make a distinction between these reactions because attitudes moved in opposite directions for these groups. Those who experienced *anger* were significantly more likely rally by showing more approval of the president and expressing less political tolerance, for example, while those who experienced *anxiety* were more likely to regress by showing less approval of the president and expressing more political tolerance (Huddy et al., 2005; Skitka, 2005). A similar rally-regress pattern has also characterized levels of political trust. Perrin and Smolek (2009) estimate that 45% of the population rallied after 9/11, while about 25% of the population regressed. As a result of this heterogeneity, the observed net change in political trust can fail to capture the regress effect and, therefore, underestimate the degree of support offered by individuals who rally. While Huddy and colleagues identify some factors that explain the heterogeneity of response, including age, education, gender, income, authoritarianism, and geographic and temporal proximity to 9/11, much of the variation remains unexplained. I argue that the pattern of heterogeneous responses is suggestive of a gene-environment interaction.

²Huddy and colleagues (2005) use the terms perceived threat and anxiety, while Lerner and colleagues (2003) as well as Skitka and colleagues (2004) use the terms anger and fear. These studies reach similar conclusions about the emotional appraisal of 9/11, and as such, I see anger and perceived threat as well as fear and anxiety as functionally equivalent terms. However, I will primarily use the terms anger and anxiety for purposes of discussion.

A gene-environment interaction (GxE) is said to exist when genes are not expressed under some conditions but are expressed under other conditions (Verhulst & Hatemi, 2013). I propose that the sudden and dramatic attacks of 9/11 stimulate the expression of genes related to stress responsiveness. If this argument is correct then the changes in variation--moving from a pre-conflict level to the rally effect to the decay of the rally effect--should be reflected in the degree to which genes and the environment explain political trust across these contexts. Specifically, I argue that genes influence responses to stress, including the emotions used to appraise threatening events, which then influence political trust levels.³

The rally effect emerges from anger, while the regress effect emerges from feelings of anxiety (Huddy et al., 2005; Lerner et al., 2003). Anger differs from anxiety in that the latter is characterized by feelings of uncertainty, lack of control, and personal vulnerability. Anxious individuals also tend to be risk-averse, while those who are angry seek out retaliation. While these emotional appraisals have only been linked to policy preferences (e.g., Huddy et al., 2005; Lerner et al. 2003), it seems likely that they also underpin the change in political trust. Anger has been shown to *decrease* generalized trust because it is characterized by negative valence and the belief that someone else is in control of the situation (Dunn & Schweitzer, 2005). Rally effects are different, however, than stimuli studied previously. This is because the perpetrator of the threat is not the same as the object of trust. Because the government is viewed as a means for retaliation, anger may actually *increase* political trust. Anxiety shares a negative valence with anger, but differs because it is characterized by low or uncertain situational control—or the belief that the outcome of a situation is beyond any particular person's control; the uncertainty of situational control uniformly depresses trust (Dunn & Schweitzer, 2005). In instances of sudden, dramatic, and international conflict, the salience and intensity of emotional appraisal, particularly the feelings of anger or anxiety, become the primary bases for evaluating government.

The second part of my argument is that genes should account for more variation in political trust immediately after 9/11, because emotional responses to threat are influenced by genetic predispositions. Research on psychobiological indicators of stress provides evidence for the heterogeneous responses identified earlier: reactions to threat-related stimuli fall into effortful and distressful dimensions in which some individuals respond with increased vigilance and attention, while others respond with fear, anxiety, and loss of control (Lundberg & Frankenhaeuser, 1979). Insights can also be gained from the study of traumatic events and the development of symptoms of post-traumatic stress disorder (PTSD). Research consistently shows that PTSD symptoms, including the avoidance and arousal symptom clusters, have a heritable component (Stein et al., 2002; True et al., 1993). To the extent that post-9/11 anger corresponds to the effortful and arousal components and post-9/11 anxiety corresponds to distressful or avoidance components then it is plausible that responses to 9/11 are at least partially genetic.

³Ultimately I can only test the relationship between genes and trust due to data limitations; however, I think it is useful to articulate how such an empirical observation might come to be. In the section below, I provide evidence to support the genes-emotions-trust linkage but do so in reverse order, starting with the relationship between emotions and trust and then moving to the relationship between genes and emotions.

Twin Studies

A twin study design reveals how variance in trust in government can be partitioned between genetic and environmental factors. Variation in phenotype—the observed trait of interest—can be explained by comparing the similarity of monozygotic (identical) twins to dizygotic (fraternal) twins. Since monozygotic (MZ) twin pairs have identical genes while dizygotic (DZ) twin pairs only share half of the genetic makeup on average, a twin study design focuses on the differences in genes between these twin types while simultaneously modeling the effects of the environment. If political trust is heritable, concordance among MZ twin pairs will be greater than among DZ twin pairs.

Environmental factors can be further divided into two categories: shared environment and nonshared environment. The shared environment is all non-genetic factors that make members of a twin pair more similar to one another while the nonshared environment is all non-genetic factors that make members of a twin pair dissimilar.⁴ The twin study design accounts for the influence of the environment since shared environment is equal for all twin pairs while nonshared environment has no commonality within a twin pair. This component of twin studies relies on the equal environments assumption, which posits that the MZ and DZ twin pairs have comparable environments (Plomin et al., 2001). Generally, this assumption has been found to be tenable for psychological traits (Medland & Hatemi, 2009). Together, variation in phenotype is the additive relationship between genes (A), shared environment (C), and nonshared environment (E). This relationship is commonly referred to as the ACE model. This study extends the traditional twin study design in two ways: 1) by including non-twin sibling pairs in the analysis, and 2) by adding a gene-environment interaction term.

Sibling Pairs

First, full siblings, half siblings, and biologically unrelated siblings are included in the analysis. These non-twin sibling pairs have varying degrees of genetic similarity, but still share all of the environmental influences which make them similar to each other (i.e., shared environment) and none of the environmental influences which make them different (i.e., nonshared environment), much like MZ and DZ twins. Full siblings share approximately half of their genetic makeup, half siblings share a quarter, and unrelated siblings share none of their genes. Non-twin sibling pairs bolster the study in two ways (Posthuma & Boomsma, 2000). First, the added observations increase the sample size; the increased statistical power allows for more complex models to be employed beyond the basic ACE model. Second, greater variation in the genetic component of sibling pairs allows the models to better disentangle familial effects (i.e., genes and shared environment).

The ACE model, and its extension to include non-twin sibling pairs, can be depicted as a set of covariance equations. The first equation below shows variation in individual phenotype as an additive function of variation in genes, shared environment, and nonshared environment. The other equations below depict the expected covariance for each type of sibling pair:

⁴Additionally, the unshared environment contains all the measurement error.

monozygotic twin pair (MZ), dizygotic twin pair (DZ), full sibling pair (FS), a half sibling pair (HS), and an unrelated sibling pair (US).

$$\begin{aligned}\sigma^2_P &= \sigma^2_A + \sigma^2_C + \sigma^2_E \\ COV_{MZ} &= \sigma^2_A + \sigma^2_C \\ COV_{DZ} &= 1/2\sigma^2_A + \sigma^2_C \\ COV_{FS} &= 1/2\sigma^2_A + \sigma^2_C \\ COV_{HS} &= 1/4\sigma^2_A + \sigma^2_C \\ COV_{US} &= \sigma^2_C\end{aligned}$$

For monozygotic twins, the covariance is a function of these first two components, as genes and shared environment act to make monozygotic twins similar to one another. Covariation among dizygotic twins and full siblings are also depicted, with the variation in genes weighted by $1/2$ to account for the reduced genetic similarity of these sibling pairs. Likewise the genetic source of half sibling covariation is weighted by a factor of $1/4$. Unrelated sibling pairs do not share genes; the term for genetic influence is dropped from their covariance calculation. The shared environment, which makes siblings similar, does not contribute to the covariation in a twin pair and is therefore not depicted in any of the equations above.

Gene-Environment Interaction

In the second extension of the traditional twin study design, a gene-environment interaction term is included so that the influences on political trust can be compared across contexts. Gene-environment interactions are common in the study of psychological traits, but are less frequently found in political science (for an exception, see Hatemi, 2013). The full model for this analysis, derived from the gene-environment interaction model developed by Purcell (2002), is presented in Figure 1 as a set of structural equations. Traditional gene-environment interaction designs add only one moderating variable to the ACE model. However, this study requires two moderators to capture the shock and decay effect of 9/11 on political trust responses. The dichotomous coding of interview day as pre- or post-9/11 models a permanent shift in the estimates; the actual day of interview permits modeling the shock and decay.⁵ The moderating variables are represented by M_P and M_{SD} .⁶ They have direct and indirect effects on political trust scores. The direct effects are the β_P and β_{SD} on the paths leading from M_P and M_{SD} to the outcome variable as well as the intercept, μ . The moderating effects can be seen in the paths from A, C, and E to the outcome variables. The moderated effects are represented as beta coefficients: β_U , β_V , and β_W moderating paths a, c, and e for M_P , and β_X , β_Y , and β_Z moderating paths a, c, and e for M_{SD} .⁷ Consistent with past twin research the model includes two control terms, referred to as definition terms, to account for the age and sex of respondents (Medland & Hatemi, 2009); these definition terms are not depicted in Figure 1.

⁵More information on the specific coding of these two variables is included in the following section.

⁶Subscript “P” for permanent shift and “SD” for shock and decay

⁷Additionally, the direct effects of the moderating variables on political trust account for gene-environment correlation (rGE), because the variance in the phenotype due to the moderating variable is now estimated as a separate term. Gene-environment correlation occurs when “different genotypes are selectively exposed to different environments” (Plomin et al., 2001). In this instance, a non-zero rGE is unlikely because of the exogenous nature of the 9/11 attacks.

If the moderating effects of the post-9/11 environment are significant then the total variance in phenotype will differ across levels of the moderator. Correspondingly, the absolute and proportional variance explained by A, C, and E will also differ at each value of the moderator. If the moderating effects are not significant then the absolute and proportional variance explained by A, C, and E should be constant across different values of the moderator. As is typical in structural equation modeling, the full model is tested first and then parameters are systematically dropped to see if any nested models fit the data significantly better. The model with the fewest parameters and without a decrement in fit is the preferred model. All model fitting was completed using the OpenMx package in R (Boker et al., 2011).

Data and Measures

The data come from the *third wave* of the National Longitudinal Study of Adolescent Health (Add Health), a four-wave panel study that includes a large sample of sibling pairs (Harris & Udry, 2008). The first wave of data was collected in 80 high schools (grades 7-12), or a corresponding feeder school for the high schools that did not include seventh grade, for a total of 132 schools. A sample of students from each school was administered an in-school questionnaire (N = 90,118). A portion of these respondents and a second sample of students were then selected to answer family-wide in-home questionnaires in this and three subsequent waves. Respondents who were given the family-wide in-home questionnaire include the genetic sample, which is comprised of monozygotic twins, dizygotic twins, full siblings, half siblings, and unrelated siblings. Zygosity was determined by self-report of perceived zygosity and confusability of physical appearance. Unrelated siblings include adopted-adopted sibling pairs, adopted-biological sibling pairs, and step-sibling pairs. The final sample for this study includes 2,061 sibling pairs (4,122 *individual* respondents) interviewed in 2001-2002 as part of Wave III of the Add Health data collection (53% female with a median age of 22).⁸ It is important to note that the sibling pairs in Add Health are nationally representative along important demographic dimensions (Harris, 2005).

Respondents in Wave III of Add Health were asked if they agreed or disagreed with the statement "I trust the federal government." Responses were reverse coded so that higher scores correspond to more trust (1 = strongly disagree and 5 = strongly agree).⁹ The mean value of trust in government is significantly different between pre- and post-9/11 responses. A t-test indicates that the average pre-9/11 political trust score of 3.10 is significantly lower than the average post-9/11 trust score of 3.30. A 0.2 change in the mean corresponds to an 8.9% increase in those that reported slight or strong trust in the federal government after 9/11. A homogeneity test of trust scores shows no significant differences in the variance or mean across sibling type. The means are also the same for males and females, although there

⁸The sample is drawn from a population of young adults with an age range of 18-26. If, in fact, genetic influences are revealed more clearly with age (e.g., Hatemi et al., 2009), then an otherwise similar study of adults might be expected to show a stronger genetic effect. Parsing out the impact of age on the influences on political trust is a particularly relevant task given an aging population and the record low levels of political trust in the United States.

⁹There were similar questions asking about trust in state and local government, but these were not used. Examining trust in the federal government makes the most sense because rally effects occur after "sudden, dramatic, and *international military conflict*" (Lambert et al. 2011), in which the federal government is the primary institution that responds to the perpetrators of the conflict. Additionally, an examination of trust in the federal government is consistent with past research on rally effects (e.g., Baker and Oneal, 2001; Chanley, 2002; Mueller, 1970).

is slightly lower variance in political trust for females compared to males; sex and age are included as control variables in the model.

The moderating variable is based on a natural survey experiment that occurred during the study. In the sibling sample for this study, 982 of the 4,122 respondents were interviewed before 9/11 (about 24%), and the balance after. Given that a larger portion of the sample was interviewed after 9/11, the data allow for broad group comparisons (those interviewed before 9/11 and those interviewed after 9/11) and post-treatment considerations (the number of days between 9/11 and the interview). Since twins were interviewed at different times, it is possible that two siblings will differ on these moderating effects; these siblings are referred to as discordant sibling pairs.

In the analysis, two variables capture the moderating effect. The first is binary and indicates whether the respondent was interviewed before 9/11 (coded as 0) or after 9/11 (coded as 1). The binary variable captures any shift in the genetic and environment influences from 9/11 (i.e., the long-term effects of 9/11 on the influences of trust). The second variable is continuous and captures the shock and decay effects following 9/11 (i.e., the short-term changes in estimates as a consequence of 9/11). Pre-9/11 respondents are coded 0, while

post-9/11 respondents are coded as $\frac{1}{\sqrt{\text{days}}}$, where interview days values range from 1 (i.e., interviewed on 9/11) to 222 (i.e., interviewed 221 days after 9/11).¹⁰ Figure 2 shows the distribution of the interview days for the sample.

Results

As a preliminary inspection of a possible genetic influence, I report intra-class correlations of political trust by sibling in Table I. The intra-class correlations provide initial evidence for both genetic and environmental factors for trust in government. If there is any genetic basis to political trust then the intra-class correlation should be highest for MZ twins and lowest for unrelated siblings. Correlation are reported for each sibling type, broken down by pairs interviewed before 9/11, pairs interviewed after 9/11, and discordant pairs (i.e., one is pre-9/11 and the other is post-9/11). The overall strength of correlation for trust in government generally corresponds to the similarity in genetic makeup for each type of sibling pair. The correlation between MZ twins is greater than the correlation between DZ twins and between full siblings, which is greater than the correlation between half-siblings, etc. This cascading trend seen overall and among pairs interviewed after 9/11, while not perfect, is suggestive of a genetic component. The discrepancy between DZ twins and full siblings, whom both share half their genes, is one of the anomalies in this trend, but this anomaly is not statistically significant.

I calculate the simple ACE decomposition separately for interviews before and after the attacks of 9/11, not accounting for any shock or decay after 9/11. This analysis, reported in Figure 3, reveals that genetic and environmental influences of trust are different depending

¹⁰Other functional forms of the decay effect, such as days, one over days, one over the cube root of days, or one over the fourth root of days, were tested using a non-genetic sample to determine which best captured changes in post-9/11 trust levels; these did not fit the data as well. Nonetheless, the results are robust to the choice of functional form.

on when respondents were interviewed, thereby confirming the trends seen in the intra-class correlations. Shared environment has a small impact in the pre-9/11 variation, whereas it accounts for almost no variation following 9/11. The influence of the nonshared environment also decreases after 9/11, while the genetic influence substantially increases.¹¹

A full moderation analysis that includes the shock and decay effects of 9/11 accounts for dynamic trends in the genetic and environmental influences on political trust. Using model fitting techniques typical of structural equation models, I find that the best fitting model is the one in which the permanent shift parameter for genes is dropped. That is, this model allows me to drop a parameter while not also forgoing a decrement in fit when compared to the full model ($\chi^2 = -0.98$, $p < 0.01$). The estimated coefficients for this model are reported in Table II. The square of the a , c , and e estimates represent the variance components of genes, shared environment, and nonshared environment when both moderators equal zero. Put in other words, the main effects of a , c , and e are the impact of genes and environment on pre-9/11 trust scores. The effects for all three are significant. The effects of the moderators and control variables are also reported in Table II. The permanent shift term for genes is not listed because this parameter was dropped from the model.

While the estimated coefficients of the permanent shift terms are not statistically significant, the shock and decay interaction terms are significant for genes and nonshared environment. The opposite signs of β_X and β_Z reflect the increased influence of genes post-9/11 and the corresponding decrease in the non-shared environment. This indicates that 9/11 had a moderating effect on the impacts of genes and nonshared environment on the expression of political trust, as predicted by the gene-environment interaction hypothesis. The size of the permanent shift terms indicates the degree to which the impact of environmental influences on political trust changed from pre-9/11 to post-9/11 levels in the long-run; the statistical insignificance of these terms shows that the estimated changes in shared and nonshared environmental influences are indistinguishable from zero, suggesting no permanent changes. However, the insignificance of these terms does not signify a lack of any change. As noted before, the shock and decay interaction parameters are significant and indicate a sharp short-term change in genetic and nonshared environmental influences immediately following 9/11.

The change in variance over time can be seen in Figure 6, which plots the absolute values of the variance components on the y-axis for the days surrounding 9/11.¹² Values plotted at time 0 and earlier represent the variation in pre-9/11 estimates. The difference between the values at time 0 (pre-9/11) and time 1 (9/11) is the size of the structural break, which is the effect of the shock and decay on the genetic and environmental factors underpinning political trust on 9/11. In terms of variation, the size of the structural break for genetic influences is positive and indicates that genes account for more variation after 9/11. The structural break for nonshared environment is negative; it accounts for almost no variation in political trust on 9/11 and in the days immediately following. This change is temporary,

¹¹It is important to note that the graph depicts the absolute variation due to genes and environment; as a consequence, the variance components do not add up to one, instead they reflect the total variation in political trust. More specifically, the sum of the pre-9/11 variance components equals the variation in pre-9/11 political trust scores; the same is true of the post-9/11 variance components.

¹²The total variation on any given day does not sum to one because the plotted values are the absolute variation explained by genetic and environmental influences.

however, as the nonshared environmental influence returns to its pre-9/11 levels over time as indicated by the decay effect. Moreover, the insignificance of the permanent shift terms indicates that the influences of genes and environment on political trust should asymptotically reach their pre-9/11 levels, given that no further events cause additional shock.

Discussion

Prior research on the antecedents of political trust has often focused on its correlation with other attitudes. Diverging from the attitude-predicts-attitude approach of past studies, I demonstrate that trust in government is partially heritable. This suggests that future research should revisit, reanalyze, and reinterpret these older studies in light of biosocial perspectives on political trust. More complex biosocial models can investigate whether political trust shares a common genetic influence with these traits or if the observed relationships are driven solely by a common environment. Thus, past research offers a robust starting point for investigating mechanisms that explain why and how genes and the environment influence political trust.

More recently, research has identified a genetic basis for *social* trust (Sturgis et al., 2010) and cooperative behavior (Cesarini, 2008), both of which have implications for political trust. Personality traits are also partially heritable (Bouchard & Loehlin, 2001; Bouchard & McGue, 2003) and structure affective response to stimuli (Gilboa & Revelle, 1994). Common genetic and environmental influences between personality, group identity, and attitudes have been identified (Weber, Johnson, & Arceneaux, 2011) and this may provide insights for understanding political trust. In particular, the agreeableness dimension of personality has been linked to political trust (Mondak & Halperin, 2008). To the extent that political trust emerges from either interpersonal trust or personality, particularly the agreeableness dimension, the heritability of political trust could be explained by one or both of these factors. Nonetheless, other research argues that personality and attitudes, potentially including political trust, are distinct traits at both the genetic and environmental levels (Alford & Hibbing, 2007; Verhulst et al., 2012). Future research should be designed to adjudicate among these and other potential mechanisms, including some of the attitudinal and personality correlates studied previously.

I also provided evidence that the rally effect can be explained by changes in the underlying genetic and environmental etiology of trust in government due to threatening environmental stimuli. A threat-related stimulus—the attacks of 9/11—moderated the expression and variation in trust in government in accordance with the observed rally effect. The influence of the nonshared environment decreased immediately after the attacks of 9/11, while the effect of genes increased. This analysis suggests that genes had a “sorting” effect on responses to the attacks, such that individuals either experienced differing levels of anger and anxiety, which then accounted for the variation in political trust immediately following 9/11.

The rate at which the genetic and environmental influences on political trust revert back to pre-9/11 levels is consistent with research on responses and recoveries to traumatic events.

In one systematic study, researchers showed the most dramatic declines in the prevalence of PTSD occurred within the first couple months after a traumatic event, including events such as 9/11 (Neria et al., 2008). Of course, rates of decline are likely to depend on the relationship between the respondent and the traumatic events—first responders or victims are more likely to experience higher, more severe, and longer stress levels than are individuals who experience traumatic events indirectly, such as through a new broadcast or by knowing someone involved (Neria et al., 2008). These findings are also consistent with the decline in perceived threat and anxiety identified by Huddy and colleagues (2005); in their study, the decline was nonlinear and extended through the New Year—a rate of decline consistent with the models reported here.

In this paper, I considered just one type of rally effect: responses to sudden, dramatic, and international conflict (Lambert et al., 2011). Other types of rally effects should be considered in future research. Some instances of international conflict may not be negatively valenced, as was the case with the 9/11 attacks. The killing of Osama Bin Laden, for example, was accompanied by positively valenced emotional appraisals. There were reports of celebrations throughout the US after the media broke news about his death.¹³ Still, these celebrations may conceal a multivalenced, heterogeneous aspect to this event, in which the celebrations are characterized by feelings of happiness even as others experience feelings of outrage at a government-coordinated killing.¹⁴

Rally effects might also arise following natural disasters. Natural disasters are sudden, dramatic, and sometimes international in scope, but would not be classified as conflict. Whether they also evoke feelings of anger and fear is unclear; it is likely that individuals also experience feelings of distress, sadness, and helplessness. Responses to Hurricane Katrina, including a sense of sympathy, for example, were largely correlated with whether victims were identified as members of the respondent's in-group (Cuddy, 2007). When rally effects occur in a comparative context, which is often the case with natural disaster, the range of emotional experiences may be shaped by cultural norms (Reddy, 2001). For example, Japanese may have emotionally appraised the tsunamis crisis in 2011 differently than Americans emotionally appraised Hurricane Katrina, especially given the documented differences in emotional appraisal and emotion regulation between these cultures (Mesquita & Albert, 2007). Given the possibility for different types of rallies, this study should be seen as a starting point for understanding how macro events shape emotional appraisals and the subsequent short-term fluctuations in political trust and policy preferences.

Finally, one of the most important findings of this study is the dynamic nature of heritability. The influence of genes on attitudes is not static; this study shows that macro political events can moderate the genetic influences of political behavior. That heritability estimates change across contexts is not new; research in psychology has repeatedly shown that gene-environment interactions are ubiquitous. Most prior studies, however, only examine interpersonal interactions. This is particularly true in political science, where gene-

¹³The Washington Times published an article titled "Osama bin Laden's Death Sparks Celebrations in D.C., N.Y.C." in which they document the rioting and sense of elation that occurred following the news of Bin Laden's death (Noble and Somers, 2011).

¹⁴Fox News published an article titled "World: Bin Laden's Death Sparks Relief, Outrage" in which they report heterogeneous responses to the death of Bin Laden, which include feelings of relief, satisfaction, and outrage (Fox News, May 2, 2011).

environment interactions have been limited to proximate life events or social interactions (e.g. Hatemi, 2013). Few studies draw on national or global events to explain changes in genetic and environmental influences on phenotypic expression, despite their obvious influence on behaviors and attitudes¹⁵ Macro-level gene-environment interaction studies are useful to political scientists because they demonstrate how genetic underpinnings of behaviors change in response to political events or policy outputs. Future studies should continue to examine the moderating influence of proximate life events but also start incorporating macro political events and institutions.

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¹⁵See Boardman and colleagues (2011) for another example of a macro gene-environment interaction study in the public health field; they examine the effect of the surgeon general's cigarette warning on the heritability of smoking.

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Figure 1.
The ACE model

Figure 2.
About 76% of the Survey Interviews Occur after 9/11

Note: The output in this figure is based on the nested model in which the decay parameters are constrained to be zero. The shock parameters, based on a dummy variable indicating whether the respondent was interviewed before or after 9/11, reflect the change between the pre-9/11 variance and the post-9/11 variance. The absolute variation from genetic influence increases after 9/11; the environmental influences decrease following 9/11. Regardless, environmental influences always account for a majority of the variation.

Figure 3.

Genetic influences increase and environmental influences decrease after 9/11.

Figure 4. The non-shared environment accounts for the most variation before 9/11 and genes account for the most variation after 9/11.

Table I

Intra-class correlations in trust differ by sibling type and interview date.

	Monozygotic Twins	Dizygotic Twins	Full Siblings	Half Siblings	Unrelated Siblings	Overall
Pre-9/11 Pairs	0.21 (45)	0.32 (46)	0.20 (129)	0.00 (26)	0.22 (20)	0.20 (266)
Post-9/11 Pairs	0.40 (141)	0.32 (209)	0.15 (571)	0.06 (188)	0.04 (234)	0.18 (1,343)
Discordant Pairs	0.36 (28)	0.04 (60)	0.13 (205)	−0.11 (61)	−0.18 (98)	0.10 (452)
Overall	0.36 (214)	0.27 (315)	0.16 (905)	0.03 (275)	0.05 (352)	0.16 (2061)

Note: sample size in parentheses

Table II

The shock-decay interactions with genes and the non-shared environment are significant, indicating that political trust changes in the presence of threat.

	Parameter	Influence	B	s.e.
Main Effects	a	Genes	0.42 *	0.12
	c	Shared environment	0.26 *	0.13
	e	Nonshared environment	0.92 *	0.07
Permanent Shift Effects	β_U	Permanent $\times$ Genes	-	-
	β_V	Permanent $\times$ Shared environment	-0.13	0.17
	β_W	Permanent $\times$ Nonshared environment	0.03	0.06
Shock and Decay Effects	β_X	Shock-decay $\times$ Genes	1.09 *	0.56
	β_Y	Shock-decay $\times$ Shared environment	-0.19	0.57
	β_Z	Shock-decay $\times$ Nonshared environment	-1.34 *	0.50
Main Effects	β_S	Permanent shift moderator	0.17 *	0.05
	β_D	Shock-decay moderator	0.71 *	0.18
	μ	Intercept	2.16 *	0.40
Controls	β_{AGE}	Age	0.05 *	0.02
	β_{SEX}	Sex	0.15 *	0.05

Note:

* $p < 0.5$