

Negative Affectivity, Political Contention, and Turnout: A Genopolitics Field Experiment

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Recent genopolitics and political psychology research suggests individuals' biological differences influence political participation. The interaction between individual differences and environments has received less attention, not least because of the confound of self-selection into environments. To test the interaction between innate predispositions and an exogenous environmental influence, we conducted a field experiment during the 2010 California midterm elections. We randomly assigned subjects to receive a postcard mobilization treatment designed to induce an emotional response to the degree of political contention in the election. We tested the possibility that subjects who are genetically predisposed toward negative affectivity will be less likely to vote after treatment exposure. To our knowledge, this is the first field experiment in political science to measure genetic moderation of a treatment, and it suggests experimental approaches can benefit from the inclusion of genetically and other biologically informative covariates.

KEY WORDS: genopolitics, field experiments, mobilization, emotion

Competition between political actors and the participation of individual citizens in elections form the heart of electoral democracy (Dahl, 1971). Political contention is thought to link these two features: competition between political actors encourages the participation of individual citizens (Riker & Ordeshook, 1968). Individual political participation, in turn, is also thought to carry important benefits for citizens, including the satisfaction of influencing an election and carrying out one's civic duty. Despite the importance of these theoretical benefits, we lack consistent evidence on the relationship between political contention and political participation. In this article, we explore the idea that political contention can exert different, even contradictory, effects on the likelihood of participating in politics. While inconclusive, the pattern of findings

we report is consistent with the notion that individuals who have a genetic risk for a construct termed “negative affectivity” are more likely to withdraw from participation when the contentiousness of an election is made salient to them. By contrast, those who are less predisposed to negative affectivity may be motivated to vote by political contention.

The suggestive findings that follow in this article may have important implications for the study of political contention and its effects on political participation. In an electoral context, we conceptualize political contention as a close contest between two or more parties. As the margin between parties grows, contention is said to decrease. Closely related to this is the extent of contention: The more parties there are contesting an election with some legitimate chance of winning power, the greater the extent of political contention. The theoretical reasoning for a positive relationship between political contention and political participation is clear (Riker & Ordeshook, 1968). As political contention increases, an individual voter’s probability of being decisive in the election increases, in turn increasing their own expected benefits from voting. Despite this, there is mixed evidence that political contention measurably increases political participation. Beginning with ecological analyses, a modest relationship between the closeness of races and political participation is observed empirically (e.g., Blais, 2000; Cox & Munger, 1989; Jacobson & Kernell, 1983). However, cross-national evidence also suggests that an increase in the number of parties contesting an election is associated with lower turnout (Blais & Carty, 1990; Jackman, 1987). Survey and experimental research reveals similarly conflicting results, as some studies find that when political contention takes the form of competing political messages, political participation decreases, while experiments conducted in the laboratory have demonstrated a positive relationship between contention and participation (Levine & Palfrey, 2007). Finally, while field experimental research has found evidence that turnout can be increased via the provision of information (Nickerson, Friedrichs, & King, 2006), appeals to civic duty (Gerber & Green, 1999), or social pressure (Gerber, Green, & Larimer, 2008, 2010a), we still lack a convincing field experimental demonstration of the role of political contention itself on political participation.¹

A growing body of work has shown that innate, individual differences affect the way we think and behave politically. Recently, the study of the role of personality in political behavior has been revived with both refined measures and updated theory (Blais & Labbe-St-Vincent, 2010; Denny & Doyle, 2008; Gerber, Huber, Doherty, Dowling, & Ha, 2010b; Gerber, Huber, Doherty, Dowling, & Panagopoulos, 2009, 2013; Mondak, 2010; Mondak, Hibbing, Canache, Seligson, & Anderson, 2010; Mondak & Halperin, 2008). A genetic basis has also been established for political participation broadly (Dawes et al., 2014; Fowler, Baker, & Dawes, 2008; Funk et al., 2010; Klemmensen, Hobalt, Norgaard, Petersen, & Skytthe, 2010) and for voting specifically (Dawes & Fowler, 2009; Fowler et al., 2008; Fowler & Dawes, 2008). Other behaviors and attitudes correlated with voting also have heritable components, such as strength of partisanship (Hatemi, Alford, Hibbing, Martin, & Eaves, 2009), a sense of civic duty (Dawes et al., 2014; Klemmensen et al., 2010; Loewen & Dawes, 2012), and political interest (Dawes et al., 2014; Funk et al., 2010).²

To date, much of this work has focused on discerning the *direct* effects of genes or personality on political behavior or searching for mediating traits that explain the relationship between biological inputs and behavioral outputs. Since the underlying theory of why innate differences between people

¹ The closest we likely come to this is the work of Arceneaux and Nickerson (2009), who demonstrate through a meta-analysis of 11 previous GOTV experiments that the electoral context—namely, election salience measured by the interest level of the electorate—has an effect on which kinds of voters are most responsive to mobilization. Their study shows that lower-propensity voters are more effected by GOTV efforts in high salience elections. However, the authors do not consider the effect of contention or salience in the mobilization message itself.

² For more information on the theory and methodological approaches used in previous genopolitics studies, see Medland and Hatemi (2009); Hatemi, Dawes, Keller, Settle, and Verhulst (2011); Hatemi and McDermott (2012); Hatemi, Byrne, and McDermott (2012).

are influential suggests that these traits act in large part to shape our interpretations of the environment, the moderating effect of genes and other individual differences becomes crucial both in better understanding the mechanisms of environmental influences but also in disambiguating mean effects of environmental exposures by capturing previously unobserved variation between people. There are a handful of studies that have begun to investigate the interactive effects of genes and environmental stimuli on political attitudes (Hatemi, 2013; Settle, Dawes, Christakis, & Fowler, 2010b) and behavior (McDermott, Dawes, Prom-Wormley, Eaves, & Hatemi, 2013; Mondak et al., 2010; Settle, Dawes, & Loewen, 2010a; Weinschenk & Panagopoulos, 2014). However, as with much if not all observational work, these findings can be challenged by the possibility of endogeneity or selection. Genetic, personality, and other psychological differences may lead us into our social and contextual environments (Scarr & McCartney, 1983), and this is most especially a problem for the proximate exposures—such as friendships, discussion partners, and other social interactions—that have formed the basis for much of the interaction work so far.

To make a stronger, cleaner causal inference about the conditioning role of the environment, we require an exogenously generated manipulation of the level of contention in a political environment (for a general discussion on this causal framework, see Rubin, 2005; Gerber and Green, 2012). Field experiments have increasingly been utilized by political scientists to tackle the problem of endogeneity and unobserved heterogeneity (Gerber & Green, 2012). In this article we employ a field experiment to more cleanly test the interaction between an individual's innate predispositions and an *exogenous* environmental influence. This represents the first biologically informed *field* experiment in political science of which we are aware. Our use of an experiment in a setting outside of a laboratory increases both the ability to detect a causal relationship as well as our confidence that the effect persists in the real world.

Scholarship about the role of anxiety and neuroticism—two facets of the broader trait of negative affectivity—leads us to two hypotheses. First, *ceteris paribus*, individuals scoring high on these traits should be less likely to vote compared to those scoring low on these traits. Second, those with a high risk for negative affectivity will be more likely to abstain when exposed to greater political contention. The greater sensitivity of these subjects to the emotion-inducing stimuli in competitive elections should lead them to withdraw. To explore this possibility, we conducted an experiment on a sample of 761 registered California voters that had previously been genotyped for genetic variants associated with traits related to anxiety and neuroticism. For each of these individuals, we generated genetic risk scores for negative affectivity. This technique, which we explain in greater detail below, allows us to estimate an individual's propensity for some trait, in this case negative affectivity. Ours is the first application of this technique in political science. We pair this with a randomized intervention. Specifically, on the basis of observational pilot-study findings exploring the joint effects of stress sensitivity and contention, we designed a postcard-mobilization treatment meant to induce an emotional response to the degree of contention in an election. We delivered the treatment to 380 subjects randomly selected from our overall sample in the week prior to the November 2010 midterm elections. We find suggestive evidence that those individuals who are genetically predisposed towards negative affectivity are less likely to vote when exposed to an exogenous increase in political contention. Conversely, those with lower genetic risk for negative affectivity do not appear to respond in this way when exposed to an exogenous increase in political contention.

It is important to highlight the limitations of our study at the outset. First, the uncertainty associated with our key estimates constrains us from decisively ruling out the possibility of null effects. We suspect this is primarily due to the limited statistical power available given the size of our sample. Nevertheless, the overall pattern of effects we uncover—coefficients signed in expected directions—remains robust to a variety of specifications of the genetic risk score. Accordingly, we interpret our results with caution, characterizing them as useful and suggestive, but not definitive; we do not believe this compromises the significance of the contribution.

Moreover, we argue the research design we employ provides a template of how to conduct political field experiments utilizing biological information. We consider this to be an important dimension of the study. We introduce an important, new approach to the discipline that has the potential to further our understanding of the way that biological predispositions influence our response to exogenous environmental exposures. To undertake this demonstration, we briefly review the literature on emotions and politics, underlining the importance of heterogeneity in responses to political stimuli. We next present our hypotheses. We then describe our measure of negative affectivity, our sample, and our treatment. After presenting our results, we conclude with a discussion of the implications of our findings.

Negative Affectivity and Political Participation

For many years in the study of emotion and politics, the standing assumption was often one of homogeneity in individuals' responses to environmental influences. The result of such assumptions is that individual-level differences in personality traits that exhibit an effect for a subset of a population, or exhibit differing effects for different subsets, may go undiscovered or may lead to contradictory results. The original formulation of Affective Intelligence, the seminal theory developed by George Marcus and his colleagues to link emotions to political behavior (Marcus, Neuman, & MacKuen, 2000), is one example of such assumed homogeneity. The theory asserts a dual-processing model in which two different emotional systems regulate the response to cues in the political environment. The disposition system affects levels of enthusiasm and aversion when processing routine information, and the surveillance system affects levels of anxiety in response to novelty or threat. These subsystems in turn affect political behavior: People who report feeling anxious about candidates or political issues report higher levels of information acquisition, more interest in the campaign, and participation beyond voting (Marcus et al., 2000). Tests of the theory in the last decade demonstrate some theoretical consensus that enthusiasm and anger generally increase political participation whereas anxiety generally deflates political participation but increases interest (Huddy & Mason, 2008). However, this pattern is a far from consistent empirical finding in the literature and is not entirely consistent with the original theory (but see also Ladd & Lenz, 2008).

Such inconsistencies, we argue, may largely be a function of searching for average effects among all subjects, rather than allowing some subjects to respond to novel information strongly while others do not respond to it at all. We argue that work on the relationship between emotions and politics should consider how differences between individuals will interact with the environment to affect political behavior. Although work in psychology has shown differential response to stimuli based on trait-level genetic, psychological, and personality traits (Hariri, 2009; Norris, Larsen, & Cacciopo, 2007; Rhodes et al., 2007), the emotion and politics literature has only recently begun to consider how individual differences might affect political behavior via differences in the emotional processing of politics. Much of the recent work has focused on differences in attributes of interest to political scientists such as partisanship and political sophistication (Pyle, 2012; Searles, 2010) or level of political interest (Cryderman & Arceneaux, 2010), which, while intriguing, do not assess the underlying traits that may differ between people.

Newer work by the scholars who originally formulated the theory of Affective Intelligence (MacKuen, Marcus, Neuman, & Miller, 2010a, 2011) has explored the effect of more innate personality traits on emotion and political attitudes. This work has generally supported the idea that while innate traits may moderate people's emotional response to politics, they do not alter the fundamental operation of the dual-processing model. However, this work has primarily focused on moderation of attitudes and has not been linked directly to voting outcomes. The only published article examining behavior (Wolak & Marcus, 2007) finds that personality seems to play more of a role in the dispositional system (the activation of enthusiasm and aversion) as compared to the surveillance system

(activation of anxiety), but it does not test whether personality traits moderate the expression of emotion or interact with the expression of emotion to affect anticipated campaign participation. MacKuen, Marcus, Neuman, & Miller (2010b) explores moderation and interaction effects but notes that the analysis is preliminary because of a lack of theoretical justification for testing specific interactions between personality traits and emotion.

The work focusing on personality associations with political behavior (that does not explicitly address emotional response) has found fairly consistent support for the roles of four of the five traits in the Five Factor Model.³ Findings have been rather varied with respect to neuroticism—a trait conceptually related to threat sensitivity and trait-level anxiety. Mondak et al. (2010) find that neuroticism increases some types of political participation; other scholars find that neuroticism is inversely related to several forms of participation, including voting, contacting an official, contributing money to a candidate, and trying to persuade others to vote (Gerber, Huber, Raso, & Ha, 2009; Mondak & Halperin, 2008).

Such findings may appear contradictory, casting doubt over the role of traits like anxiety or neuroticism in political behavior. We aim to clarify the role of these traits by demonstrating how their effects might vary by political context. We study the trait-level behaviors of anxiety and neuroticism by referring to a single construct called “negative affectivity” (Watson & Clark, 1984) that can be measured using either scales of trait-level neuroticism or trait-level anxiety (Watson & Clark, 1984, p. 468). Negative affectivity “reflects pervasive individual differences in negative emotionality and self concept” (Watson & Clark, 1984, p. 465–466) and has as one of its constituent components the concept of trait-level anxiety, although individuals who score highly on measures of negative affectivity tend to report more negative affect even in the absence of any particular stressor. People who have high scores on measures of negative affectivity frequently experience subjective feelings of nervousness, tension, and worry; in a given situation, they are more likely to experience a significant level of distress and thus are likely to respond more strongly to stressful situations. They are more sensitive to social cues representing both implicit and explicit threat (Hariri, 2009). In an electoral context, we expect those with negative affectivity to withdraw from politics as levels of conflict increase. Previous research on conflict avoidance gives some indication that this may be true. Denny and Doyle (2008) suggest that their finding of an association between the personality trait of aggression and voting could be because those who dislike conflict are less likely to be interested in politics and therefore are less likely to vote.

We choose to broaden our study to the concept of negative affectivity instead of focusing solely on the more frequently studied traits of anxiety or neuroticism for two reasons, one theoretical and one methodological. First, as we describe above, the concept of negative affectivity incorporates the features of these traits that we think are most relevant for the proposed mechanism of the effect of electoral competition, suggesting that individuals who score highly on either or both anxiety or neuroticism are similarly responsive to the potential stress of political contention. The concepts of anxiety and neuroticism are complex and multifaceted, but the commonality between them—a predisposition toward negative affective states that shape response to environmental influences—is the element most important to our hypotheses. Second, the approaches used to construct the genetic risk indices necessitated a broader conceptualization of the trait. The final iteration of the risk score involved utilizing a large genome-wide association study (GWAS) that employed an item response theory analysis (van den Berg et al., 2014) of a variety of phenotypic measurements, including neuroticism items (from the NEO Personality Inventory, Eysenck Personality Questionnaire, the Eysenck Personality Questionnaire Revised Short Form, and the International Personality Item Pool inventory), harm avoidance items (from Cloninger’s Tridimensional Personality Questionnaire), and all item data for negative

³ We provide a brief summary of these findings in the the online supplementary information.

emotionality from the Multidimensional Personality Questionnaire.⁴ In the next section, we set out our hypotheses explicitly.

Hypotheses

Synthesizing the research on emotions in politics with the literature in psychology on the effects of trait-level negative affectivity, we put forward two hypotheses about the way that genetic risk toward negative affectivity may affect voter turnout.

H1: Increased genetic risk for negative affectivity reduces the likelihood of turning out to vote, all else equal.

The experience of anxiety induced by social interactions can condition responses to events precipitating future social interactions, and this conditioned sensitivity to potentially threatening social cues varies considerably among individuals (Hariri, 2009). Neurotic individuals are more likely to become anxious in the face of threats (Carver, Sutton, & Scheier, 2000; Carver & White, 1994). Because neurotic people have been shown to have stronger and more prolonged skin conductance reactivity to emotionally evocative stimuli than do people who score low on a neuroticism scale (Norris et al., 2007), one explanation for decreased political participation among those who score highly on measures of negative affectivity is that anxious people may try to avoid situations that are anxiety inducing in order to reduce their arousal (Rusting & Larsen, 1995). Therefore, if they perceive that participating in politics is generally stressful, it is likely that people sensitive to threat may withdraw from the political sphere.

H2: Individuals with a greater risk for negative affectivity will be more likely to abstain from voting when treated with a mobilization message that makes political contention salient, all else equal.

Affective intelligence theory and the subsequent empirical work it spawned suggest that induced emotion can affect political behavior (Brader, 2005; Civettini & Redlawsk, 2009; Valentino, Brader, Groenendyk, Gregorowicz, & Hutchings, 2011; Valentino, Gregorowicz, & Groenendyk, 2009; Valentino, Hutchings, Banks, & Davis, 2008). For people who are already predisposed toward heightened emotional activation, triggering them to think about an election may have the effect of heightening their emotional response relative to those people who are more emotionally stable. If the treatment activates negative emotions, these individuals may seek to reduce this effect by disengaging with politics.⁵ Accordingly, we expect negative affectivity risk to depress turnout among our treatment group. Those with less of a genetic predisposition towards negative affectivity should, by contrast, be mobilized by political contention.

Measuring Negative Affectivity

Interest in the genetic basis of negative affectivity is increasing. Over three dozen articles have been published in the last decade that have sought to find genetic polymorphisms associated with one or more of the measures that comprise the construct of negative affectivity. Genes are sections of DNA,

⁴ Earlier iterations of the creation of the risk score involved searching broadly for all genome-wide association studies for our traits of interest, instead of using one large GWA meta-analysis study. Using the more inclusive construct of negative affectivity permitted us to include more GWA studies and thus more information in the construction of the original index.

⁵ We emphasize that our hypothesis is a departure from the expectations of previous political science literature that suggests a uniform *mobilization* response to our treatment if individuals are motivated to seek out additional information about the candidates or issues at stake.

most of which code for the production of specific proteins. DNA contains numerous variants and the different versions of these variants are called “alleles.” A genetic polymorphism exists when there is more than one allele present in the population. Genetic polymorphisms which consist of a change in a single nucleotide of DNA are called “single nucleotide polymorphisms” (SNPs). Within a given genomic region, there can be many (and sometimes hundreds) of SNPs which can potentially be associated with a trait of interest. For a more thorough review of basic genetics concepts, see Hatemi et al. (2012).

There have been two general approaches to finding these associations. The first is hypothesis driven and relies on our current understanding of the neurochemical pathways associated with negative affectivity to identify genes that are theoretically linked to these traits, such as the 5HTTLPR (Hariri, 2009), BDNF (Chen et al., 2006), and COMT genes. These hypothesized relationships can then be empirically tested. An alternative approach is atheoretical and employs the tool of a genome-wide association study to identify which SNPs, out of the hundreds of thousands of DNA base pairs genotyped, are most closely associated with a given trait. Because of the large number of tests run in GWAS analyses, it is standard to adjust the p -value to be very small in order to reduce the number of false positive associations. However, this simultaneously risks increasing the number of false negatives, which may contribute to the fact that GWAS results have been very hard to replicate.

Both of these approaches face serious challenges. A single SNP rarely accounts for more than 1% of the variance in a given trait (Gibson, 2010), even in a trait that we know is under strong genetic influence, such as height (Visscher, 2008). The difficulty in finding genetic polymorphisms that account for the genetic variation between people in a trait has been called the “missing heritability” problem (Manolio et al., 2009) and is a major challenge to scholars interested in genetic association studies. Furthermore, there is measurement error when trying to detect a genetic association between any single SNP and a trait or behavior of interest. One of the key problems in replicating the findings of GWAS research in association studies is that the SNPs that are associated with a trait of interest in a GWAS may not contribute to causing the trait itself, but instead may be a proxy (i.e., be correlated with) the variation in the genome that contributes to the biochemical pathway that results in the trait.

A relatively recent and more advanced technique that has been used to address this problem is the creation of *genetic risk scores*.⁶ A genetic risk score approach is used to account for the measurement error problems that are endemic for association studies with single SNPs because a risk score is better able to detect the underlying variation in the genome by summarizing and aggregating the information across the genome instead of relying on potentially noisy measurement from single SNPs.⁷ Thus, departing from previous work in political science that has incorporated measurement of genetic constructs using the techniques described above, we create a genetic risk index for negative affectivity instead of testing individual SNPs. This approach is new and, to our knowledge, its use in this article is its first application within political science.⁸

In order to construct our *genetic risk scores*, we utilized the results of a recent GWAS meta-analysis of traits related to negative affectivity⁹ based on a sample of approximately 63,000

⁶ For a good review of the problems motivating the development of these risk scores—also called multilocus profiles of genetic risk, or index or scale approaches—see McCrae, Scally, Terracciano, and Abecasis (2010), Belsky et al. (2013), Wray et al. (2014), and Dudbridge (2013).

⁷ Intuitively, these indexes should be thought of like any other survey index. Related items are employed to differentiate individuals on some underlying trait or value. While a much larger number of items could be proposed, the quality of the index is not principally a function of the number of items but how well these items recover the underlying trait.

⁸ We wish to note that we have adapted the approach outlined in Wray et al. (2014) and refer the interested reader to that article (particularly the sections on pp. 1070–1072) for a more comprehensive explanation of the technique.

⁹ De Moor et al. (2015) label this underlying trait as “neuroticism,” but their phenotypic measurement encompasses the set of traits that comprise negative affectivity. To measure these traits in the subjects used in the meta-analysis, the authors used item response theory analysis to recover a score for each subject based on his or her answers to one or more batteries. These include the neuroticism items from the NEO Personality Inventory, Eysenck Personality Questionnaire, the Eysenck Personality Questionnaire Revised Short Form, and the International Personality Item Pool

individuals (De Moor et al., 2015).¹⁰ Of the 6,949,614 SNPs analyzed as part of the meta-analysis, 526,645 are contained in our sample of genotyped SNPs.¹¹ To construct the scores, we followed the procedure suggested by Wray et al. (2014). Based on a set of independent SNPs,¹² we estimated two genetic risk scores:

$$\text{GRS 1} = \sum_i \hat{\alpha}_i \text{SNP}_{ij}^{P \leq 0.1}, \quad (1)$$

$$\text{GRS 2} = \sum_i \hat{\alpha}_i \text{SNP}_{ij}^{P=1}, \quad (2)$$

where the $\hat{\alpha}$ s come from the De Moor et al. (2015) meta-analysis. GRS 2 is based on all of the independent SNPs in our sample that overlap with the meta-analysis, and GRS 1 is based on only those SNPs that achieve a p -value of 0.1 or smaller in the meta-analysis.

Sample

The overall universe from which the pool of subjects included in our field experiment was extracted included approximately 1,100 California residents.¹³ The data used for extraction were previously collected by researchers at the University of California San Diego School of Medicine. Participants were recruited from southern California by access to a population birth record-based twin registry (Cockburn, Hamilton, Zadnick, Cozen, & Mack, 2002) as well as by newspaper advertisement (Zhang et al., 2004). The protocol for this larger study was approved by the UCSD Institutional Review Board, and each subject gave written informed consent for participation in the Twin and Family Study run by Daniel O'Connor and his colleagues. As part of their informed consent, subjects agreed to being recontacted by the study (including receiving surveys). We considered our study covered by the original informed consent and thus did not apply for separate IRB approval for our study design or treatment postcard.

To create the sampling frame for the field experiment, we checked all addresses using the U.S. Postal Service's Mail Update procedure. We removed all individuals with undeliverable addresses and those who no longer resided in California. We identified a total of 761 individuals from the original sample who were listed in the April 2009 California State Registrar of Voters file. We then randomly assigned a subset of these respondents—a total of 380 people—to receive the treatment. Using a combination of full name, birthday, address information, and phone number, we were able to match all 761 people in the sample in the voter file after the November 2010 election in order to determine whether each person had voted. Balance tests on the treatment and control groups reveal no major differences in demographics or genetic information (Table 1). We also provide summary statistics for each covariate by experimental group in Table 2.

In the sampling frame, 293 subjects in the treatment group and 290 subjects in the control group were previously genotyped on 592,312 SNPs using the Illumina 610 Quad genotyping array. Genetic

inventory. These also include all item data for harm avoidance from Cloninger's Tridimensional Personality Questionnaire, and all item data for negative emotionality from the Multidimensional Personality Questionnaire (De Moor et al., 2015, p. 643).

¹⁰ The summary statistics of the meta-analysis necessary to construct the score are publicly available for download at <http://www.tweelingenregister.org/GPC/>. The sample we analyze here was not part of the GWA study.

¹¹ Strand ambiguous SNPs and indels (the insertion or deletion of nucleotide bases in a person's DNA) have been excluded from all analysis.

¹² We pruned the SNPs using PLINK's (Purcell et al., 2007) p -value informed clumping procedure (with an LD threshold of $r^2 < 0.2$ across 500kb).

¹³ For a detailed description of the original sample, see Zhang et al. (2004).

Table 1. Balance Tests for Treatment and Control Groups

	Treatment		Control		<i>p</i> -value
	Mean/Prop	<i>SD</i>	Mean/Prop	<i>SD</i>	
Voted (Nov. 2008)	0.88	0.32	0.91	0.29	0.25
Age	50.39	16.56	51.95	17.27	0.29
Male	0.29	0.46	0.31	0.47	0.60
PC1	0.00	0.03	-0.00	0.04	0.40
PC2	0.00	0.03	-0.00	0.04	0.20
PC3	0.00	0.04	-0.00	0.03	0.52
PC4	0.00	0.03	0.00	0.03	0.86
PC5	0.00	0.03	0.00	0.02	0.46
PC6	-0.00	0.03	-0.00	0.02	0.90
PC7	0.00	0.03	0.00	0.03	0.96
PC8	-0.00	0.03	0.00	0.03	0.59
PC9	0.00	0.03	0.00	0.03	0.73
PC10	-0.00	0.02	0.00	0.04	0.04
Genetic Index 1	-0.01	0.97	0.01	1.03	0.75
Genetic Index 2	-0.01	0.97	0.01	1.03	0.79

Note. *p*-values indicate whether or not the control and treatment groups are significantly different on the characteristic of interest. PC1-PC10 are dimensions from our principal component factor analysis for population stratification.

risk scores were calculated for all subjects for whom we had complete information for all SNPS in the index.¹⁴

An important consideration when conducting genetic analysis is that ethnic groups have different allele frequencies due to their genetic ancestry. This is known as *population stratification*. These differences in genetic ancestry may lead to false-positive results if not accounted for.¹⁵ The two most popular ways to account for the confounding effect of population structure are to either apply principal-component analysis (PCA) (Price et al., 2006) or multidimensional-scaling (MDS) (Li and Yu, 2008) methods to genome-wide data (Wang et al., 2009). The estimated principal components or MDS dimensions capture the underlying population and thus can be included as controls to correct for population stratification in subsequent analyses (Wang et al., 2009). To control for genetic-background heterogeneity in all of our regressions, we estimated principal components using the software tool GCTA (Yang, Lee, Goddard, & Visscher, 2011) including all autosomal SNPs available in the data. We also performed the analysis including the top 10 principal components as covariates in the association analyses.

Treatment

Subjects assigned to the treatment group were mailed the postcard shown in Figure 1 on October 20, 2010, ensuring that they received it within the week prior to Election Day. The purpose of our treatment was to heighten subjects' awareness of the degree of contention in the upcoming races. None of the mailed cards were returned as undeliverable, which is typical for a mailing with addresses that have been validated and a subject pool that has been previously contacted. This suggests all treatment mailings were successfully delivered to subjects in the treatment group, though it is conceivable

¹⁴ For MZ twin pairs, only one individual was genotyped and the information was used for both twins. Standard GWAS quality-control measures were applied using plink v1.0.7 leaving 559,400 genotyped SNPs.

¹⁵ For example, voter turnout in different ethnic groups may be affected by their environments, alleles other than the ones included in our index, or some unobserved factor. Once these ethnic groups mix in a larger population, simply comparing the index to the number of voters would reveal an ultimately spurious relationship.

Table 2. Summary Statistics for Treatment and Control Groups

	Treatment		Control	
	Range	<i>N</i>	Range	<i>N</i>
Voted (Nov. 2008)	0–1	293	0–1	289
Age	20–95	257	20–91	258
Male	0–1	293	0–1	290
PC1	–.15–.02	293	–.16–.02	288
PC2	–.30–.09	293	–.32–.09	288
PC3	–.38–.03	293	–.38–.03	288
PC4	–.16–.19	293	–.06–.19	288
PC5	–.30–.08	293	–.29–.09	288
PC6	–.26–.26	293	–.26–.09	288
PC7	–.38–.13	293	–.37–.12	288
PC8	–.38–.32	293	–.38–.31	288
PC9	–.08–.36	293	–.16–.36	288
PC10	–.23–.04	293	–.23–.36	288
Genetic Index 1	–2.84–2.33	293	–3.20–2.21	288
Genetic Index 2	–2.81–2.17	293	–3.45–2.06	288

that some subjects failed to be treated. Thus, we report intent-to-treat effects in the analyses that follow, noting that these are the more conservative estimates of the treatment effects we detect. Taking contact rates into account would only magnify the estimated treatment effects (Angrist, Imbens, & Rubin, 1989).

We note three features about this treatment. First, it clearly primes contention by noting the likelihood of close races, using idioms such as “up for grabs,” “a toss up,” and “intense.” Second, it clearly identifies the action which voters should take to address the contention, namely voting. Third, the postcard is nonpartisan and contains substantial source credibility. It was sent by the “UCSD Politics Survey,” for which recipients had completed a long survey in the months prior

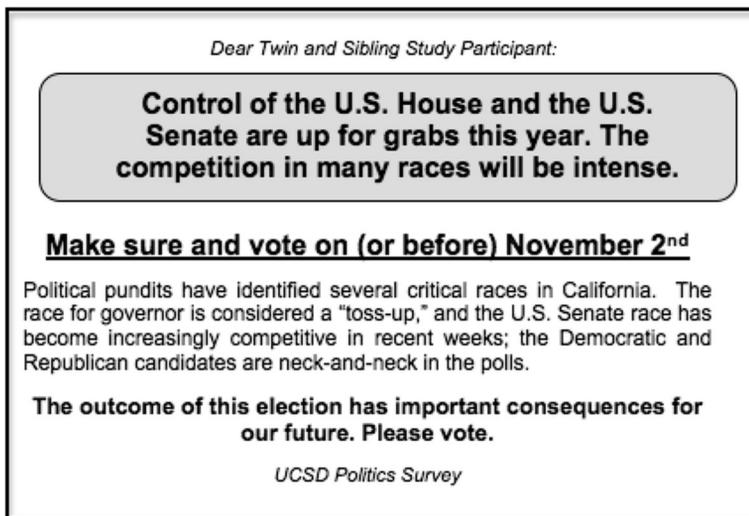


Figure 1. Treatment mailer.

and had been compensated for their participation. Accordingly, suspect subjects would have been attentive to the treatment.

To examine the internal validity of our treatment and ensure it was likely to induce anxiety, we performed two separate manipulation checks.¹⁶ This manipulation checks are described in greater detail in our online supplementary information. The first demonstrates that our treatment generates an emotional response, in particular anxiety and enthusiasm, but not anger. Our second demonstrates that subjects who read our treatment messages were more likely to report feeling anxiety than those who read a generic election message or a placebo related to recycling.

We note one final qualification regarding our treatment. Because the administered treatment is coming as a part of a medical study, subjects in treatment may have felt that their voting was being monitored. As a result, our treatment may have had the effect of exerting social pressure (Gerber et al., 2008, 2010a; Panagopoulos, 2010) at the same time as it increased anxiety about the outcome of the election. Accordingly, any result could be the effect of social pressure rather than contention triggering negative affectivity. While this is a possibility, we think two considerations argue against this claim. First, the substantial literature on social pressure generally suggests a positive effect from social pressure; the work does not suggest that exposure to social pressure should *decrease* turnout among some individuals. However, this is a clear theoretical expectation for those with negative affectivity in treatment. Moreover, the results we present below somewhat bear out this expectation. Second, as our second manipulation check shows, anxiety can be increased by mentioning the contentiousness of an election without any accompanying monitoring and social pressure messaging. Accordingly, we claim that our mailer had the effect of increasing anxiety and likely without a perfectly correlated increase in social pressure.

Results

Our analysis begins by examining whether our measure of genetic risk is directly related to the probability of turning out to vote. According to our first hypothesis, those with a greater genetic risk for negative affectivity should be less likely to participate. As a first step, we consider the effects of genetic risk in the absence of the treatment by examining only those respondents in the control condition. As Table 3 shows, there is no apparent direct effect of negative affectivity on turnout, although we note the sign of the coefficient suggests a negative, overall impact.

We next check for a main effect of the treatment, which we take to be a test of the general argument that greater political contention leads to higher turnout. There is no apparent main effect of the treatment. Seventy percent of subjects assigned to receive the mailer voted, compared to 69.1% who voted in the control condition.¹⁷ To confirm this comparison of means, we also performed a logistic regression of turnout on treatment condition controlling for age, gender, ethnicity, and prior voting in the November 2008 election. This is reported in the first column (“Base Model”) of Table 4. The results suggest no statistically significant main effect for the treatment, though the coefficient is positively signed.

Given our limited sample size, as well as the relatively high salience of the election, we did not anticipate that our mailer would necessarily have a direct effect. Furthermore, our theory argues for heterogeneity in the treatment response, based on levels of negative affectivity. Pooling all respondents together within each treatment group may wash out any treatment effects. We therefore turn to analyzing whether the mailer was more influential for those who score high on our negative affectivity

¹⁶ Both studies received separate ethical reviews from the relevant partner universities.

¹⁷ The magnitude of this effect is in line with average treatment effects reported for mail treatments in previous studies Green and Gerber (2008). Additionally, and perhaps unsurprisingly given the nature of our sample, we also observe that the overall turnout rate for subjects in the control condition in our study was slightly higher than the overall proportion of registered voters in California (60%) who turned out to vote in the November 2010 elections.

Table 3. Effect of Genetic Risk on Voting in Absence of Treatment

	SNPs <i>p</i> <=0.1	All SNPs
GRS 1	-0.15 (0.19)	
GRS 2		-0.14 (0.20)
<i>N</i>	255	255
LogLik	-101.02	-101.10
Pseudo <i>R</i> ²	0.27	0.27
Wald $\chi^2(5)$	53.49	53.65
<i>p</i> > χ^2	0.00	0.00

Note. Robust standard errors in parentheses. Dependent variable is coded 1 if subject voted in the November 2010 election in California, 0 otherwise. Robust standard errors in parentheses. All models include covariates for gender, age, ethnicity, and prior voting in the November 2008 election (not shown; available from the authors by request). **p* < .05; ***p* < .01; ****p* < .001 for a two-tailed test.

risk index. This affords us a clean test of our second hypothesis, namely that the treatment should decrease turnout among those with a higher genetic risk for negative affectivity, while increasing it among those subjects absent this risk.

Table 4 presents our results. The effects in columns 2 and 4 (the models without an interaction term) suggest that genetic risk scores for negative affectivity decrease the probability of turnout, although these effects do not reach conventional levels of significance for either genetic risk score.

Table 4. Results of a Separate Logistic Regression

	Base Model		SNPs		All SNPs
		Excludes Interaction	<i>p</i> <=0.1 Includes Interaction	Excludes Interaction	Includes Interaction
Treatment	0.04 (0.23)	0.03 (0.23)	0.03 (0.23)	0.03 (0.23)	0.03 (0.23)
GRS 1		-0.14 (0.12)	-0.12 (0.15)		
Treatment*GRS 1			-0.05 (0.21)		
GRS 2				-0.13 (0.12)	-0.11 (0.16)
Treatment*GRS 2					-0.03 (0.21)
<i>N</i>	512	512	512	512	512
LogLik	-230.11	-229.40	-229.38	-229.56	-229.55
Pseudo <i>R</i> ²	0.18	0.18	0.18	0.18	0.18
Wald $\chi^2(5)$	70.68	69.17	69.84	60.04	69.91
<i>p</i> > χ^2	0.00	0.00	0.00	0.00	0.00

Note. Dependent variable is coded 1 if subject voted in the November 2010 election in California, 0 otherwise. Robust standard errors in parentheses. All models include covariates for gender, age, ethnicity, and prior voting in the November 2008 election (not shown; available from the authors by request). GRS1 refers to “Genetic Risk Score 1”; GRS2 refers to “Genetic Risk Score 2” (see text for details). **p* < .05; ***p* < .01; ****p* < .001 using two-tailed tests.

This provides some qualified support for our first hypothesis, namely that those with more negative affectivity risk are more likely to disengage from politics.¹⁸ A more nuanced picture emerges when we moderate the effect of the treatment through the genetic risk score. The interaction terms in Table 4 are both negative, but neither reaches conventional levels of significance.¹⁹

Taken together, these results suggest that there may be heterogeneity in the treatment effects we detect. For voters low in genetic risk, exposure to political contention could act as a motivator and may increase their probability of turning out. However, as risk increases, sensitizing them to political contention decreases their probability of voting below what is expected for subjects in the control condition. Given the uncertainty associated with the estimates we report above, we urge caution in the interpretation of the results we report. Nevertheless, these represent unbiased estimates of the likely effects; our best guess, given the preponderance of the evidence we present, suggests that political contention does not have the same effect (or even direction of effect) for all voters.

Discussion and Conclusion

Political contention, thought to be a central motivation for participation, also holds the potential to trigger negative emotions such as anxiety. While some individuals enjoy and are motivated by conflict, others find themselves turning away. We should expect those whose reaction to such contention is emotionally negative to disengage from politics and expect those who react positively to further engage. While our study was conducted in the context of an American congressional election, we do not expect that our results hinge on the particulars of this type of election. There are multiple aspects of political contention that may be potentially stressful to voters, whether the number of choices (i.e., parties or candidates), the closeness between candidates, campaign rhetoric and communications about contention, or even candidates' ideological dispersion and uncertainty over that dispersion; which aspects matter most in which contexts is a question that merits future study, and one that also invites work in contexts outside of the United States.

Our approach supports an understanding of electoral politics in which contention has potentially contradictory effects and in which these effects are conditional upon individual differences. Our suggestive results thus contribute to the already rich literature on emotions and politics (e.g., Marcus et al., 2000; Huddy & Mason, 2008) and personality and politics (e.g., Gerber et al., 2010b, 2013).

Our results suggest that a genetic risk for negative affectivity may not in and of itself discline a person to participate politically. However, this predisposition and its associated traits do appear to heighten a person's sensitivity to their political environment. This heterogeneity in response to political contention suggests that we consider more deeply the merits of various mobilization tactics, including voter registration drives and efforts to increase turnout. These heterogenous effects also recommend a more careful consideration of contextual effects in the study of personality and political behavior. More broadly, they suggest that we look not only for emotional responses to politics that are consistent across all individuals (e.g., Marcus et al., 2000). We should also search out multidirectional

¹⁸ We note that the estimates for models that exclude covariates are substantively similar; details are available from the authors upon request.

¹⁹ In earlier iterations of this article we used an alternate method for constructing genetic risk scores. The same principles apply as in the construction of this score, but instead of using the results of a single, large meta-analysis GWA study, we constructed the score based on a literature search to identify all genetic variants that have been associated with our traits of interest in a series of smaller GWA studies, a total of 120 SNPs. Using that approach, we found that the risk indices did negatively and significantly interact with the treatment, with a larger substantive effect than we report here. However, with the advent of large genetic consortiums that allow for sophisticated meta-analyses, the methods used to construct this initial score are considered less appropriate, and we therefore report as our main results the scores generated from the most advanced technique possible at the time of submission of this article. Further details are available from the authors upon request. We thank an anonymous reviewer for recommending this change and providing helpful suggestions for its implementation.

and heterogenous effects. Individuals vary, perhaps deeply. Such variance conditions how they experience the political world.

In addition to our notion of genetically based heterogeneity, we suggest two further innovations of this article. It is the first article to our knowledge to implement an exogenous, *field experimental* treatment on a genetically informative sample. Indeed, to our knowledge this is among the first field experiments run in behavior genetics generally, though behavioral geneticists have recognized the value of the experimental approach (van IJzendoorn et al., 2011). We thus harness the inferential advantages of field experiments to test hypotheses that could be subject to confounds in a merely observational study. Accordingly, our approach might act as a template for future research. We should note that it is common for medical researchers to maintain large panels of respondents with enormous amounts of individual-level biological and psychological data. Researchers can look to these panels for opportunities to run biologically informed field experiments like ours. While such studies could be difficult and complex, they also hold out the promise of cleaner inferences about gene-environment interactions. Second, in contrast to early genopolitics studies that have relied on single genetic variants, we constructed a genetic risk index. Such indices include a substantially larger amount of information about an individual's susceptibility to some trait and allow us to better guard against chance findings and the effects of population stratification. This is an important innovation, and one that could likewise be put in practice by other researchers. The field of behavioral genetics is advancing rapidly, and international consortiums of scientists have joined forces in order to create massive samples on which to test for genetic associations with phenotypic traits. With the expanding scope of traits for which we have the information available to generate genetic risk scores, the range of questions open to social scientists likewise expands. To date, there have been large GWA studies initiated on important phenotypes of interest to scholars of political behavior—such as educational attainment, subjective well-being, risk aversion, and trust²⁰—opening the door to new research questions.

Our work has limitations. First, replication will be important to address whether these results are generalizable. We found a consistent negatively signed coefficient on the interaction between the treatment and the genetic risk score of negative affectivity using multiple derivations and approaches to calculating the score, but in the final approach outlined in this article, the key interaction coefficients were not statistically significant. Replication of our experiment on a larger sample is necessary to test whether the suggestive relationships we uncover here can be detected in studies with increased statistical power. It is important to note that while replication is important, we are hampered by limited data. Therefore, we hope to establish an important baseline against which we or other enterprising researchers could situate replications. Relatedly, our genetic risk index is derived from a broad concept of negative affectivity which incorporates many different clinical definitions and measurements of negative emotionality and neuroticism susceptibility. Future work would benefit from more refined hypotheses about what specific aspects of a disposition toward anxiety or neuroticism are most strongly theoretically and empirically related to turnout. Third, our results are derived from a relatively limited sample and a single intervention. By contrast, much of the most important field experimental work on mobilization has effectively leveraged both large samples and multiple treatments. Larger, more diverse samples and multiple treatments should be employed in the future. Experiments in a laboratory setting might offer additional leverage in understanding how individual traits interact with experimental stimuli.

The experimental revolution in mobilization offers compelling evidence that people can respond dynamically to their environments. Our results demonstrate the potential of incorporating genetic measures of individual differences into an experimental framework. Studying average relationships between a treatment and a behavioral outcome misses the point that our emotional, cognitive, and behavioral responses are heavily dependent on biological predispositions. Understanding why

²⁰ See <http://ssgac.org> and <http://www.tweelingenregister.org/GPC/> for more information.

individuals respond differently to the same stimuli offers the potential of understanding in which contexts contention will increase turnout and when it can be expected to decrease it. By taking individual differences seriously, we can better understand how different individuals experience and behave within their political environment.

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Supporting Information

Additional supporting information may be found in the online version of this article at the publisher's website:

Contextualizing Big Five Effects

Manipulation Checks

Figure 1: Weighted Proportion of Total Emotional Expression for Mailer Compared to Other Political Stimuli