The Leadership Quarterly 24 (2013) 45–60

Contents lists available at SciVerse ScienceDirect

The Leadership Quarterly

journal homepage: www.elsevier.com/locate/leaqua

# Born to lead? A twin design and genetic association study of leadership role occupancy ☆

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ARTICLE INFO

*Article history:*
Received 4 February 2012
Received in revised form 20 August 2012
Accepted 21 August 2012
Available online 10 September 2012

*Keywords:*
Leadership role occupancy
Twin design
Genetic association study

ABSTRACT

We address leadership emergence and the possibility that there is a partially innate predisposition to occupy a leadership role. Employing twin design methods on data from the National Longitudinal Study of Adolescent Health, we estimate the heritability of leadership role occupancy at 24%. Twin studies do not point to specific genes or neurological processes that might be involved. We therefore also conduct association analysis on the available genetic markers. The results show that leadership role occupancy is associated with `rs4950`, a single nucleotide polymorphism (SNP) residing on a neuronal acetylcholine receptor gene (*CHRNB3*). We replicate this family-based genetic association result on an independent sample in the Framingham Heart Study. This is the first study to identify a specific genotype associated with the tendency to occupy a leadership position. The results suggest that what determines whether an individual occupies a leadership position is the complex product of genetic and environmental influences, with a particular role for `rs4950`.



## 1. Introduction

A recent special issue in this journal focused on individual difference research in leadership (Leadership and Individual Differences, Volume 23). Mapping out the debate in this area, the editorial for this issue discusses the biological foundations of leadership and asks researchers working in the field an intriguing question: "Is there a specific leadership gene?" (Antonakis, Day, & Schyns, 2012, 646). In this article we address this interesting question.

We focus on one specific aspect of leadership – leadership role occupancy – identifying a specific gene that may be determining the role occupancy, nested in the overall study of heritability of this particular conceptualization of leadership. Using data from the National Longitudinal Study of Adolescent Health (Add Health), we employ a twin study design to measure the joint effect of genes and environment on variation in leadership role occupancy. Twin studies are important because they allow us to gauge the overall

☆ We thank the editor and anonymous referees for their valuable comments. Research was supported by National Institute on Aging grant P-01 AG-031093 and National Science Foundation grant SES-0719404. This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis.

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http://dx.doi.org/10.1016/j.leaqua.2012.08.001

relative influence of genes, and the results show that the heritability of leadership role occupancy in this sample is about 24%. However, twin studies do not illuminate the specific genes or neurological processes that may possibly be involved in explaining the heritability of leadership role occupancy. Thus, we turn to the genetic markers available in the Add Health data, in order to establish whether any of them are significantly associated with leadership role occupancy. Taking into account multiple testing, we find that one of the genetic markers – the rs4950 marker residing on a neuronal acetylcholine receptor gene (*CHRNB3*) – has a significant relationship with the propensity to occupy a leadership role. Given the large number of polymorphisms on the human genome, there is a risk in reporting false positive results. We therefore further test this genetic association on another large independent sample from the Framingham Heart Study that contains similar information on leadership role occupancy and the genotype for the rs4950 genetic marker. This replication shows a similarly significant association.

This article is the first study to identify a specific genotype that is associated with occupying a leadership position. Genes – and the neurological processes that they influence – are upstream from personality factors related to the emergence of leadership. Identifying genes associated with leadership role occupancy may thus bring us closer to understanding the sources of leadership emergence. Leadership role occupancy is just one possible conceptualization of leadership, hence our results provide only the first relevant contribution to the field of the biology of leadership as recently surveyed in The Leadership Quarterly special issue on The Biology of Leadership (Volume 23, Issue 2). Although we cannot fully detail the cascade of neurological dynamics through which this particular gene may influence leadership role occupancy, we do know several things about its function and we are able to test several theories of mediation. Although none of these prove to be significant, knowing that rs4950 is potentially important for leadership role occupancy helps us to generate additional hypotheses about its function that could be tested in subsequent research and in other data sets. For example, one possibility is that the gene influences leadership role occupancy via its effect on traits of impulsivity and patience that have previously been associated with the acetylcholine receptor genes (Azam, Winzer-Serhan, Chen, & Leslie, 2002; Wonnacott, 1997). Thus, our results provide new insights into the determinants of leadership emergence and they also show how models of complex social behavior can benefit from integrating genetic variation as a possible factor.

## 2. Heritability of leadership role occupancy

Heritability of leadership role occupancy has been the focus of several studies over the last decade. Many of the studies employed the classical twin design (e.g. Arvey, Rotundo, Johnson, Zhang, & McGue, 2006; Arvey, Zhang, Avolio, & Krueger, 2007; Li, Arvey, Zhang, & Song, 2012) and find that about a third of the variance in leadership role occupancy can be explained by genetic factors. At the same time, researchers have not yet been able to identify specific genes that are associated with leadership (e.g. Antonakis et al., 2012; Chaturvedi, Arvey, Zhang, & Christoforou, 2011). But there are many reasons to believe that such genes exist. Nicolaou, Shane, Cherkas, Hunkin, and Spector (2008) describe four complementary mechanisms through which genes could influence the propensity to occupy a leadership position. First, genes may directly influence chemical reactions in the brain that affect behavior. This would imply that specific genotypes may directly predispose individuals to take on a leadership role.

Second, genes may affect the development of individual attributes affecting the predisposition to occupy a leadership position. For example, a heritable personality trait like extraversion (e.g. Bouchard & Loehlin, 2001; Evans & Leighton, 1989; Jang, Livesley, Vernon, et al., 1996; Riemann, Angleitner, & Strelau, 2006) incorporates several attributes, including sociability, gregariousness, exhibitionism (Barrick & Mount, 1991). This trait has been shown to be strongly associated with leadership (e.g. Judge, Bono, Ilies, & Gerhardt, 2002). Extraversion has also been shown to be related to the long alleles of the DRD4 exon III repeat genotype (Benjamin et al., 1996; Eichhammer et al., 2005).[1] Thus, the effect of genes on the level of extraversion in people may influence their propensity to occupy leadership roles.

Third, genes may influence people to gravitate towards specific environments, meaning environmental factors are not randomly distributed among people of different genotypes (Neale & Maes, 2002; Scarr, 1992). Thus, genes may influence the tendency of people to select into environments more favorable for a leadership role (Kendler & Eaves, 1986; Plomin, DeFries, & Loehlin, 1977). For example, intelligence and cognitive abilities have been shown to be highly correlated with leadership (e.g. Judge, Colbert, & Ilies, 2004), and genes influence how much time people spend in school (Behrman & Taubman, 1989; Tambs, Sundet, Magnus, & Berg, 1989). As a result, genes may influence the propensity to occupy a leadership role through their influence on educational attainment.

Fourth, genes may influence differential sensitivity to environmental stimuli that mediate the propensity to occupy a leadership role. This phenomenon is known as gene–environment interaction (Moffitt, Caspi, & Rutter, 2005; Plomin et al., 1977), and has been shown to be important for leadership role occupancy by Zhang, Ilies, and Arvey (2009). For example, the salience of information has been shown to be related to variation in the dopamine D4 receptor gene (Berridge & Robinson, 1998; Volkow, 2004). Problem solving and task knowledge have been associated with leadership characteristics (e.g. Judge et al., 2004). Thus, the interaction of the dopamine receptor gene with information that aids in the completion of cognitive tasks associated with leadership emergence may influence the tendency to engage in leadership roles among those who inherit the genotype that is associated with increased sensitivity to such information.

[1] It is important to note that the original association result between extraversion and the long alleles of the DRD4 exon III repeat genotype has recently been questioned after subsequent replication attempts often failed to corroborate this relationship (Munafò et al., 2008).

Although the empirical tests we conduct in this article do not differentiate between these mechanisms, they do help to establish whether or not genes play a role, and if they do, which particular genes may be involved. Antonakis (2011) identifies several issues that may further complicate the analysis of specific causal pathways linking genes and leadership emergence. By identifying candidate genes, we hope to identify potential causal pathways so that mediation analyses can be used to test these pathways in future work.

It is important to note that the relationship between traits and leadership may be complex, at least more complex than assumed in previous studies. Antonakis (2011) introduces the ascription-actuality trait theory that creates the explanatory framework for the relationship between traits and leadership emergence. Utilizing insights from behavioral economics, Antonakis (2011) differentiates between traits that really matter and traits that only seem to matter. We postulate that ascription allows a leader to emerge but it does not determine whether a particular leader becomes effective—instead, skills (and the traits determining them) explain leadership effectiveness. The relationship between traits and leadership emergence can suffer from false negatives (traits that matter for emergence are not perceived by observers as important, like the election of presidents based on intelligence). Similarly, the relationship between traits and leadership emergence can suffer from false positives. For example, traits that in reality do not matter for emergence are perceived as important by the observers and this means they indirectly influence emergence. Todorov, Mandisodza, Goren, and Hall (2005) and Antonakis and Dalgas (2009) show that when observers infer competence from the appearance of political candidates, this also affects their judgments of their capacity to lead.

## 3. Data

### 3.1. Add Health

To identify specific genes that may help to explain variation in the emergence of leaders, we use The National Longitudinal Study of Adolescent Health (Add Health) (Harris et al., 2009). Add Health was started in 1994 in order to explore the health-related behavior of adolescents in grades 7 through 12. By now, 4 waves of data collection have taken place and participating subjects are around 30 years old. The first wave of the Add Health study (1994–1995) selected 80 high schools from a sampling frame of 26,666. The schools were selected based on their size, school type, census region, level of urbanization, and percent of the population that was white. Participating high schools were asked to identify junior high or middle schools that served as feeder schools to their school. This resulted in the participation of 145 middle, junior high, and high schools. From those schools, 90,118 students completed a 45-minute questionnaire and each school was asked to complete at least one School Administrator questionnaire. This process generated descriptive information about each student, the educational setting, and the environment of the school. From these respondents, a core random sample of 12,105 adolescents in grades 7–12 were drawn plus several over-samples, totaling more than 27,000 adolescents. These students and their parents were administered in-home surveys in the first wave.

Wave II (1996) was comprised of another set of in-home interviews of more than 14,738 students from the Wave I sample and a follow-up telephone survey of the school administrators. Wave III (2001–2002) consisted of an in-home interview of 15,170 Wave I participants. Finally, Wave IV (2008) consisted of an in-home interview of 15,701 Wave I participants. The result of this sampling design is that Add Health is a nationally representative study. Women make up 49% of the study's participants, Hispanics 12.2%, Blacks 16.0%, Asians 3.3%, and Native Americans 2.2%. Participants in Add Health also represent all regions of the United States.

In Wave I of the Add Health study, researchers screened for sibling pairs including all adolescents that were identified as twin pairs, half-siblings, or unrelated siblings raised together. The sibling-pairs sample is similar in demographic composition to the full Add Health sample (Jacobson & Rowe, 1998). The number of identical (MZ) and non-identical (DZ) twins who still participated in Wave IV was 1,120 (432 MZ and 688 DZ), with 872 twins (432 MZ and 440 DZ) in same sex pairs. The Add Health data have been widely used for twin studies (e.g. Fowler, Baker, & Dawes, 2008; Harris, Halpern, Smolen, & Haberstick, 2006).

Allelic information for a number of genetic markers were collected for 2,574 individuals as part of Wave III. These particular genes were chosen because they are known to affect brain development, neurotransmitter synthesis and reception, and hormone regulation. They are the dopamine D4 receptor gene (*DRD4*), the dopamine D2 receptor TaqIA (*DRD2*), the dopamine transporter gene (*SLC6A3*), the serotonin transporter 6A4 gene (*SLC6A4*), and the monoamine oxidase A-uVNTR (*MAOA*). Add Health also genotyped alleles of four single nucleotide polymorphisms (SNPs), which are variations in a single base-pair in the DNA strand. These SNPs reside on two nicotinic acetylcholine receptor genes, `rs2304297` and`rs892413` on *CHRNA6* and `rs4950` and `rs13280604` on *CHRNB3*. Details of the DNA collection and genotyping process are available on the Add Health website (Add Health Biomarker Team, 2007). Variable definitions and descriptive frequencies are given in Appendix.

### 3.2. Leadership role occupancy

In this article we utilize one possible operationalization of leadership emergence – the role occupancy – focusing on whether an individual held an office with leadership functions. Several previous studies on heritability of leadership have also used the role occupancy model (e.g. Arvey et al., 2006, 2007; Li et al., 2012; Zhang et al., 2009). As Ahlquist and Levi (2011, 19) note, this approach captures an important feature of leadership since many people lead "not by addressing a crowd *ab initio* but rather by occupying an office" (see also a similar definition in (Bass & Stogdill, 1990, 19).

Similarly to Avolio, Rotundo, and Walumbwa (2009), we are primarily interested in whether individuals are leaders (or not) and not on the type of leadership they exhibit (e.g. transformation or transactional). As such, our approach naturally follows the

suggestions in Ilies, Gerhardt, and Le (2004, 208) that genetical foundations of leadership emergence should be studied before investigations of leadership effectiveness because "emergence is the first step in the leadership process."[2]

In the Add Health data, subjects were asked the following question in Wave IV: "Thinking about your official job duties, which of the following statements best describes your supervisory responsibilities at your (current/most recent) primary job?" Answer categories included "I (supervise/supervised) other employees" and "I (do/did) not supervise anyone." We use responses to this question to construct a binary indicator of "leaders" and "followers." Those who refused to answer or marked "don't know" constitute less than 1% of respondents and were discarded for the purpose of this study. Fig. 1 shows the distribution for the genetic and twin samples. Mean-comparison tests reject the null hypothesis that these distributions are different for the MZ, DZ, and genetic sample groups.

The leadership role occupancy question that we utilize from Add Health refers to the supervisory role of the respondent. The bio-history methodology used in Arvey et al. (2006, 2007), Li et al. (2012) potentially provides for a more fine-grained measure, identifying respondents as team leaders, shift supervisors, managers, directors, presidents etc. The role occupancy measure available in the Add Health data and used here is potentially a noisy measure of leadership role occupancy, as supervision role may be at the lower end of the leadership roles thus potentially underestimating heritability of higher level leadership roles.[3] Overall, this can be viewed as adding measurement error to our estimations. Given general attenuation effect of measurement error (e.g. Carroll, Ruppert, Stefanski, & Crainiceanu, 2006) if we observe some significant effects in our estimations, the true effect can be even stronger.

## 4. Twin study

### 4.1. Methods

Twin studies compare traits, behaviors, and other outcomes (called "phenotypes") of twins who share 100% of their genetic material (identical or monozygotic twins) to those who share about 50% of the genes on which humans differ (fraternal or dizygotic twins) in order to estimate the relative importance of genetic and environmental influences. If we assume that the influence of the environment on the phenotype is the same for monozygotic (MZ) and dizygotic (DZ) twins (the "common environments" assumption), and there are no gene–environment interactions, then the variance in leadership can be decomposed into additive genetic effects (A), common or shared environmental influences (C), and unshared or unique environmental influences (E). The ACE model does not allow us to observe environmental and genetic influences directly, but it allows us to estimate these effects by observing the covariance across MZ and DZ twins.

Although the assumptions underlying the ACE model are strong, the method produces results that have been validated in numerous studies. For example, studies of twins reared apart generate similar heritability estimates to those generated by studies of twins raised together (Bouchard, 1998). More recently, Visscher et al. (2006) use the small variance in percentage of shared genes among DZ twins to estimate heritability without using any MZ twins, and they are able to replicate findings from studies of MZ and DZ twins reared together. Moreover, personality and cognitive differences between MZ and DZ twins persist even among twins whose zygosity has been miscategorized by their parents, indicating that being mistakenly treated as an identical twin by one's parents is not sufficient to generate a difference in concordance (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Scarr & Carter-Saltzman, 1979; Xian et al., 2000).

The ACE model can be formally expressed as:

$$y_{ij} = \mu + A_{ij} + C_j + E_{ij}$$

where $y$ is the measure of the phenotype, $j$ denotes the family, $i$ denotes the individual twin in the family, $\mu$ is the mean of this phenotype across all observations, $A_{ij} \sim N(0,\sigma_A^2)$ is the additive genetic component, $C_j \sim N(0,\sigma_C^2)$ is the shared environment component, and $E_{ij} \sim N(0,\sigma_E^2)$ is the unshared environment component. Notice that these assumptions imply:

$$\mathrm{Var}(y) = \sigma_A^2 + \sigma_C^2 + \sigma_E^2$$

If we further assume that the unshared environment is uncorrelated between twins ($\mathrm{COV}(E_{1j},E_{2j}) = 0$), that genes are perfectly correlated between MZ twins ($\mathrm{COV}_{MZ}(A_{1j},A_{2j}) = \sigma_A^2$), and the covariance between DZ twins who share half their genes on average is half that of identical twins ($\mathrm{COV}_{DZ}(A_{1j},A_{2j}) = \frac{1}{2}\sigma_A^2$), then we have two additional equations:

$$\mathrm{COV}_{MZ}\left(y_{1j},y_{2j}\right) = \sigma_A^2 + \sigma_C^2,$$
$$\mathrm{COV}_{DZ}\left(y_{1j},y_{2j}\right) = \frac{1}{2}\sigma_A^2 + \sigma_C^2$$

[2] Li et al. (2012) analyzed heritability of leadership role occupancy and a different conceptualization of leadership—transformational leadership. They showed that both conceptualizations are differentially heritable, although the levels of heritability are not statistically different between these two leadership manifestations. Li et al. (2012) also show that just under half of the genetic component of leadership role occupancy corresponds to the genetic factors related to transformational leadership. Thus, potential insights from our study can also be helpful in the analysis of biological foundations of leadership performance. We do not deal with this issue in current study but leave it for future work.

[3] We thank an anonymous referee for making this point.

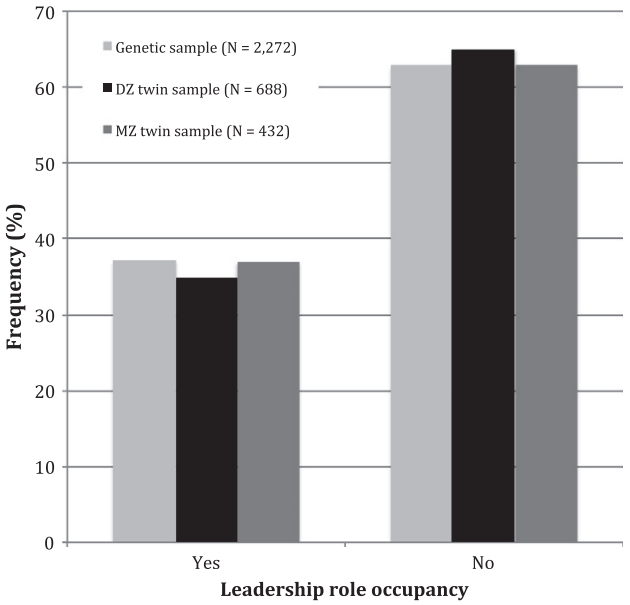


**Fig. 1.** Distribution of leadership role occupancy by genetic and twin zygosity samples.

The covariance equations reflect the fact that DZ twins share on average 50% of their genes whereas MZ twins share all of their genes. Based on these equations, we can estimate the ACE model via a random effects regression model with the $2 \times 2$ variance–covariance matrix specified as:

$$\Omega_j = \begin{bmatrix} \sigma_A^2 + \sigma_C^2 + \sigma_E^2 & R_j\sigma_A^2 + \sigma_C^2 \\ R_j\sigma_A^2 + \sigma_C^2 & \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \end{bmatrix}$$

where $R$ is the genetic relatedness of the twin pair equaling 1 for MZ twins and $\frac{1}{2}$ for DZ twins. We follow the ACE model parameterization described in Rabe-Hesketh, Skrondal, and Gjessing (2008). With leadership role occupancy operationalized as a binary variable, we estimate the model using probit. We use the variances of the random effects to generate estimates of heritability, common environment, and unshared environment.[4] Since the residual variance is fixed in a probit model, this ACE model is unidentified. Therefore, we must fix $\sigma_E = 1$. Also included in the model are standard controls for age and gender.

The likelihood functions in genetic models often present computational challenges for maximum likelihood approaches because they contain high-dimensional integrals that cannot be evaluated in closed form and thus must be evaluated numerically. This has prompted the increasing use of Bayesian methods, implemented using Markov Chain Monte Carlo (MCMC) algorithms, to estimate the variance components in ACE models.[5] This is the approach we take here. We choose vague prior distributions to ensure they do not drive our results. For the thresholds, we use a mean-zero normal distribution with variance $10^6$ and for the precision parameters associated with $\sigma_{A_2}$, $\sigma_E^2$ and $\sigma_C^2$ we use a Pareto distribution with shape parameter equal to 1 and scale parameter equal to 0.001 which is the equivalent of putting a uniform (0,1000) prior on the variances.[6] We began sampling from the joint posterior distribution after convergence was established using the Brooks and Gelman (1998) statistic (values of less than 1.1 on each parameter indicate convergence). For all of the models, the burn-in period was 100,000 iterations and the chains were thinned by 100.

In addition to estimating an ACE model, we estimated all of the possible submodels to compare model fit. These include an AE model, which assumes only heritability and common environment, a CE model, which assumes only common and unshared environment, and an E model. If a submodel fits better than the general ACE model, this suggests the variance or variances not included in the submodel should not be included. To compare the submodels we used the deviance information criterion (DIC), a Bayesian method for model comparison analogous to the Akaike Information Criterion (AIC) in maximum likelihood estimation. Models with smaller DIC are considered to be superior (Gelman, Carlin, Stern, & Rubin, 2004).

### 4.2. Twin results

The first step in assessing the potential role of genetic factors is to compare the correlation among MZ twins to that of DZ twins. For leadership role occupancy, the bootstrapped Spearman's rank correlation for MZ twins is 0.357 (95% CI = 0.122, 0.565)

[4] They are defined as $\frac{\sigma_A^2}{\sigma_A^2+\sigma_C^2+\sigma_E^2}$, $\frac{\sigma_C^2}{\sigma_A^2+\sigma_C^2+\sigma_E^2}$, and $\frac{\sigma_E^2}{\sigma_A^2+\sigma_C^2+\sigma_E^2}$ respectively.

[5] For a detailed discussion of Bayesian ACE models, we refer to van den Berg, Beem, and Boomsma (2006).

[6] A Pareto distribution has proven to work well for variance components in genetic models (Burton et al., 1999; Scurrah, Palmer, & Burton, 2000).

and for DZ twins is 0.033 (95% CI = −0.249, 0.285). The difference in correlations is significant ($p = 0.029$, one sided). These correlations are suggestive that genetic factors play a significant role. In turn, the results of the ACE model are presented in Table 1.

Table 1 shows that the ACE model, controlling for gender and age, yields a heritability estimate of 24% (95% CI = 6%, 46%). The estimate for common environment is 10% (95% CI = 1%, 28%) and the estimate for unshared environment is 66% (95% CI = 49%, 82%).[7,8] Fig. 2 shows the 95% credible area of the joint estimates. The figure shows that a substantial degree of leadership emergence appears to be heritable and that shared environment plays a relatively small role in the model.

We also examine submodels in Table 1. The AE model yields a heritability estimate of 32% (95% CI = 11%, 50%). The DIC value is slightly smaller for the ACE model, suggesting that the model that best explains variance between twins is one composed of heritability, shared and unshared factors. Whether we take the ACE or the AE model, it remains that a substantial degree of leadership role occupancy can be explained by genetic variation.[9]

## 5. Genetic association

Because Add Health collected a number of specific genetic markers, it presents us with a unique opportunity to move beyond a twin study design and to consider specific genotypes that may be associated with leadership role occupancy. Below, we introduce some basic concepts in genetics, our genetic association research design, and we present results for the genotypes that are available in the Add Health data. These genes have been previously associated with a number of cognitive functions and behaviors and therefore are candidate genes in our genetic study of leadership role occupancy.

### 5.1. Basic concepts in genetics

Human DNA is composed of an estimated 21,000 genes that form the blueprint for molecules that regulate the development and function of the human body. Genes are distinct regions of human DNA found in the 23 pairs of chains, or chromosomes, that make up all human DNA. Almost all cells in a human contain the same inherited DNA chains that develop from the moment of conception.

Individuals inherit one half of their DNA from each parent, with one copy of each gene coming from the mother and one copy from the father. Some genes come in different versions, known as "alleles." Each parent has two separate copies of an allele at each "locus," or location, on the chromosome, but each sperm or egg cell contains only one of these alleles. Thus, a child has a 50% chance of receiving a particular allele from a particular parent. For example, suppose that at a given locus there are two possible alleles, A and B. If both parents are "heterozygous" at that locus, meaning they each have an A and a B allele (AB or BA—order is irrelevant), then a given offspring has a 25% chance of being "homozygous" for A (AA), a 25% chance of being homozygous for B (BB) and a 50% chance of being heterozygous (AB or BA). If an individual is heterozygous at a locus, a "dominant" allele may impose itself on the "recessive" allele, and the expression of the latter allele will not be observed.

Genes transcribe proteins that begin a cascade of interactions that regulate bodily structure and function. Many of the observable traits and behaviors of interest, referred to as phenotypes, are far downstream from the original genotypes present in DNA. While in some cases one allele can single-handedly lead to a disease (such as sickle cell anemia, Huntington's disease, and cystic fibrosis), the vast majority of phenotypes are "polygenic," meaning they are influenced by multiple genes (Mackay, 2001; Plomin, DeFries, McClearn, & McGuffin, 2008), and are also – crucially – shaped by a multitude of environmental forces. As a result, association models between genotypes and phenotypes are an important first step, but they are not the end of the story. It is also important to investigate the extent to which genetic associations are moderated by environmental factors and other genes.

### 5.2. Candidate genes

Research in behavioral genetics often starts with "candidate" genes that are known to influence behaviors or processes in the body that are related to the phenotype of interest. For leadership role occupancy and most other social behavior phenotypes, this means focusing on genes that affect brain development, neurotransmitter synthesis and reception, hormone regulation, and transcriptional factors (Benjamin et al., 2007; Damberg, Garpenstrand, Hallman, & Oreland, 2001). The particular selection of genes in Add Health were chosen because "[T]hese candidate genes have been reported to be associated with individual differences in behavior related to mental health; are reported to be functional, exonic, in promoter regions, or affect gene expression; are expressed in the brain; and have *prima facie* involvement in neurotransmission" (Add Health Biomarker Team, 2007).

[7] The maximum likelihood estimation is executed with the Mx program as described in Neale and Maes (2002).

[8] The Brooks & Gelman values associated with model convergence for the ACE model are 1.01, 1.00, and 1.00 for the A, C, and E components respectively.

[9] Based on DIC, an ADE model that estimates additive and non-additive genetic variation as well as unique environmental variation fits the data best (DIC = 1006). Coventry and Keller (2005) conclude that the combined value of additive (A) and non-additive (D) genetic variation from model based on only twin data, rather than including additional family members (e.g. parents, non-twin siblings, spouses, and children), should be considered an estimate of broad-sense heritability. Further, they suggest that the same is also true for estimates of additive genetic variation when non-additive genetic variation is not modeled (ACE or AE models). Therefore, we choose to present the more general ACE model that includes common environment with the caveat that the heritability estimate should be considered an overall measure of genetic effects.

**Table 1**
Summary of model results. Note: The ACE model consists of additive genetic factors (A), shared or common environmental factors (C), and unshared environmental factors (E). The model includes 184 MZ and 162 DZ complete same-sex twin pairs. It is estimated with controls for age and gender.

| Model | Heritability | Shared environment | Unshared environment | DIC |
|---|---|---|---|---|
| ACE | 24 (6, 46) | 10 (1, 28) | 66 (49, 82) | 1013.0 |
| AE | 32 (11, 50) | | 68 (50, 89) | 1014.0 |
| CE | | 23 (6, 38) | 77 (62, 94) | 1023.1 |
| E | | | | 1035.3 |

The dopamine D4 receptor (*DRD4*) and dopamine D2 receptor TaqIA (*DRD2*) genes encode dopamine receptor subtypes that are activated by the neurotransmitter dopamine. Genetic variation in these genes has been associated with neurological disorders such as schizophrenia, attention deficit disorder, and also the behavioral traits of novelty-seeking (Benjamin et al., 1996; Ebstein et al., 1996), extraversion (Eichhammer et al., 2005), and risk-taking (Dreber et al., 2009). Though doubt has been cast on the original association result between extraversion and the long alleles of the `DRD4 exon III` repeat genotype as subsequent replication attempts have often failed to corroborate this relationship (Munafò, Yalcin, Willis-Owen, & Flint, 2008).

The Dopamine Transporter gene (*SLC63*) encodes a protein in the membrane of neurons that allows for the re-uptake of dopamine that remains in the synaptic cleft between neurons. Genetic variation in the promotor region of this gene – that regulates the amount of protein produced – has been associated with a number of cognitive and neuropsychiatric deficits (Fuke et al., 2001).

The serotonin transporter gene (*SLC6A4*[10]) encodes a transporter in the cell wall that absorbs excess serotonin into the pre-synaptic neuron in parts of the brain that influence mental states (Canli & Lesch, 2007). *SLC6A4* has been studied for at least a decade and much is known about the way different versions of this gene influence transcription, metabolism, and signal transfers between neurons, all of which may influence personality. In particular, the less transcriptionally efficient alleles of this gene have been reported to moderate the influence of life stress on depression (Caspi et al., 2003); and the more transcriptionally efficient alleles have been linked to the selective processing of positive emotional stimuli (Fox, Ridgewell, & Ashwin, 2009) and subjective well-being (De Neve, 2011; De Neve, Christakis, Fowler, & Frey, in press). These genetic associations are tentative and require further replication before they can be considered anything more than suggestive.

The *MAOA* gene encodes monoamine oxidase A, an enzyme that degrades neurotransmitters such as serotonin, dopamine, and epinephrine (adrenaline) in parts of the brain that influence cognitive ability and behavior. Genetic variation in MAOA has been associated with impulsivity (McDermott, Tingley, Cowden, Frazetto, & Johnson, 2009; Meyer-Lindenberg et al., 2006; Passamonti et al., 2006) and, more recently, with borrowing behavior (De Neve & Fowler, 2010).

The nicotine acetylcholine (*nACh*) receptors encoded by *CHRNA6* and *CHRNB3* are located throughout the central nervous system and are studied for their influence on impulsive behavior as they have been shown to modulate dopamine release in the midbrain (Azam et al., 2002; Wonnacott, 1997). Dawes and Loewen, 2010 report evidence for an association between the *CHRNA6* gene and political behavior, suggesting that it is mediated by patience.

### 5.3. Family-based association methods

Genetic association studies test whether an allele or genotype occurs more frequently within a group exhibiting a particular phenotype than those without the phenotype. A significant association can mean one of three things: (1) the allele itself influences leadership role occupancy; (2) the allele is in "linkage disequilibrium" with an allele at another locus that influences role occupancy; or (3) the observed association is a false positive signal due to "population stratification."[11]

Population stratification occurs because groups may have different allele frequencies due to their genetic ancestry. Leadership role occupancy in these groups may be the product of their environments, alleles other than the one of interest, or some unobserved reason. For example, two groups may not have mixed in the past for cultural reasons. Through the process of local adaptation or genetic drift, these groups may develop different frequencies of a particular allele. At the same time, the two groups may also develop divergent behaviors that are not influenced by the allele but solely by the environment in which they live. Once these two groups mix in a larger population, simply comparing the frequency of the allele to the observed behavior would lead to a spurious association.

There are two main research designs employed in association studies that address the population stratification issue differently, case–control and family-based designs. Case–control designs compare the frequency of genotypes among subjects that exhibit a phenotype of interest to subjects who do not. As a result, case–control designs are vulnerable to population stratification if either group is especially prone to selection effects. A typical way to control for this problem is to include controls for the race or ethnicity of the subject or to limit the analysis to a specific racial or ethnic group.

[10] The *SLC6A4* gene has several other names, including HTT, 5HTT, and SERT.

[11] Given our data, we cannot differentiate between 1 and 2. In order to do so, we would need additional genetic information about loci in close proximity to the locus of interest. Thus, a significant association means that either a particular SNP, or one likely near it on the same gene, significantly influences a predisposition to occupy a leadership position.

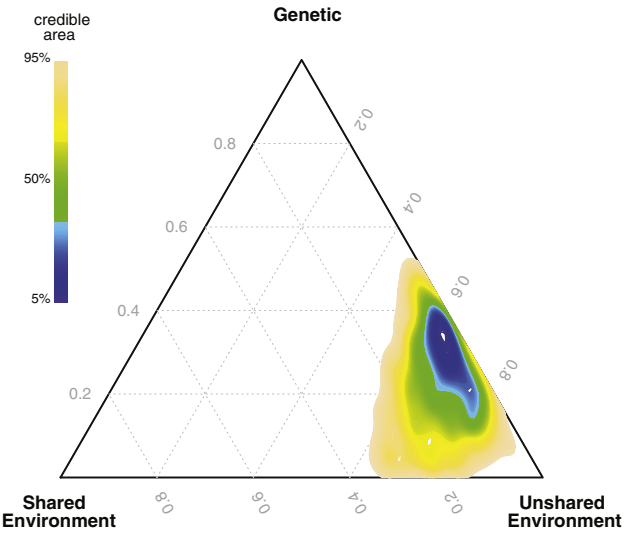


**Fig. 2.** The heritability of leadership role occupancy. Note: Ternary plot shows the posterior distribution of estimated components of total variance in an ACE model of leadership role occupancy among Add Health subjects. The plot represents the proportions of three variance components (genetic, shared environment, and unshared environment) that sum up to one. A point in the center indicates all three components contribute equally, whereas a point at a vertex indicates that a single component fully explains the variance. Colors indicate credible areas calculated by using 2,000 posterior draws to estimate a three-dimensional kernel density. The blue areas indicate the highest density regions with the most credible estimates, while the beige areas contain 95% of the draws (i.e., the probability that the true estimates lie outside the colored region is 0.05). Mean heritability is estimated to be 24%.

Family-based designs eliminate the problem of population stratification by using family members, such as parents or siblings, as controls. A variance-components based association analysis for sibling pairs was first suggested by Boehnke and Langefeld (1998) and Spielman and Ewens (1998) and is defined as:

$$y_{ij} = \beta_0 + \beta_b G_{bj} + \beta_w G_{wij}$$
$$G_{wij} = g_{ij} - G_{bj}$$
$$G_{bj} = \frac{\sum_i g_{ij}}{n_j}$$

where $y_{ij}$ is the dependent variable of interest for individual $i$ in family $j$, $n_j$ is the number of family members, $g_{ij}$ is the genotypic score which equals the number of alleles (0, 1, 2), $G_{bj}$ is the expected genotypic score, and $w_{ij}$ is an individual Õs deviation from the expected genotypic score. A positive value for $G_{wij}$ means that an individual inherited an excess number of copies of an allele from his or her parents.

By decomposing genotype scores into between-family (b) and within-family (w) components, it is possible to control for spurious results due to population stratification because only the coefficient on the between-family variance ($a_b$) will be affected. The association result is determined by the coefficient on the within-family variance ($\beta_w$) which, in essence, shows whether the allele frequencies among siblings with the phenotype differ significantly from the frequencies among their siblings without the phenotype. While a family-based design is very powerful in minimizing Type I error (false positives), it reduces the power to detect true associations, and is thus more prone to Type II error or false negatives (Xu & Shete, 2006). Of course, when genotype data for siblings are available – as is the case in Add Health – then a family-based test produces the more robust results.

Here we use logit to perform the family-based tests for genetic association:

$$P\left[Y_{ij} = 1 \middle| Z_{kij}, U_j\right] = \text{logit}\left(\beta_0 + \beta_w G_{wij} + \beta_b G_{bj} + \beta_k Z_{kij} + U_j + \epsilon_{ij}\right)$$

where $i$ and $j$ index subject and family respectively. For the respective Add Health genes, $G_w$ is the within-family variance component of the individual's genotype (measured as subject genotype minus their family's mean genotype score), $G_b$ is the between-family variance component of the individual's genotype (measured as their family's mean genotype score), $Z_k$ is a matrix of variables to control for the age and gender differences of the Add Health sample, $U$ is a family random effect that controls for potential genetic and environmental correlation among family members, and  is an individual-specific error. The coefficients are reported as odds ratios; therefore the null hypothesis is that $\beta_w = 1$ (that is, this particular genetic variation within a family does not significantly alter the odds of being employed in a leadership position).

### 5.4. Association results

Table 2 presents the results of the family-based tests for genetic association between the available genotypes and leadership role occupancy. These models include standard controls for age and gender. A family-based design reduces the risk of omitted variable bias and the possibility that results are spuriously associated with genetic ancestry.[12]

The results in Table 2 indicate that the rs4950 SNP on the *CHRNB3* gene is statistically significant. However, our results remain vulnerable to multiple testing and need to be adequately corrected. After the Bonferroni correction[13] for multiple testing, rs4950 remains significantly associated with leadership.[14] The rs4950 SNP produces a within-family variance component that has a large positive odds ratio. In fact, as compared to the family average, having one more rs4950 A allele (instead of a G allele) increases the odds of being in a leadership role by approximately 50%. It is worthwhile noting that this genetic effect is comparable with the well-studied effect of gender that we also observe in our measure of leadership role occupancy. In line with the behavioral genetics literature we keep to a basic set of control variables (gender and age) and the family-based model to account for population stratification.[15]

We also test the relationship between rs4950 and leadership role occupancy examining the differences across the levels of this sample. Tabulated frequencies in Table 3 show that for individuals with both A alleles on rs4950, 40% occupy leadership roles. In contrast, for individuals with both G alleles about 30% are in leadership positions. About a third of our population has both an A and G allele on rs4950, and these individuals exhibit no meaningful difference from the average frequency of occupying a leadership position. In summary, individuals with two rs4950 A alleles are proportionally more likely to be in a leadership role in comparison to individuals with one or two G alleles, suggesting a possible dominance effect. These differences across levels are significant ($\chi^2 = 8.50, p = 0.014$).

Other variables may mediate the relationship between the genotype we have identified and leadership role occupancy (Baron & Kenny, 1986). We might also expect genes to contribute to variation in socio-economic or psychological factors (Bowles & Gintis, 2002), which in turn would affect the propensity to be in a leadership role. In order to test for potential mediators, we regress a number of variables common to the leadership literature and available in Add Health separately on rs4950 along with the standard controls for race, age, and gender. Table 4 presents relevant results of these estimations. We find that rs4950 is not significantly associated with any of these variables and can thus rule out the notion that these variables act as mediators.

### 5.5. Replication in the Framingham Heart Study

Specific genotypes usually only account for a very small amount of the variance in complex social behaviors, which means the tests often have low power. As a result, it is very important to replicate results in independent samples (Beauchamp et al., 2011; Charney & English, 2012). Here, we use the Framingham Heart Study (FHS), a population-based, longitudinal, observational cohort study that was initiated in 1948 to prospectively investigate risk factors for cardiovascular disease. Since then, the FHS has come to be composed of four separate but related cohort populations: (1) the "Original Cohort" enrolled in 1948 ($N = 5209$); (2) the "Offspring Cohort" (the children of the Original Cohort and spouses of the children) enrolled in 1971 ($N = 5124$); (3) the "Omni Cohort" enrolled in 1994 ($N = 508$); and (4) the "Generation 3 Cohort" (the grandchildren of the Original Cohort) enrolled beginning in 2002 ($N = 4095$). Published reports provide details about sample composition and study design for these cohorts (Cupples & D'Agnostino, 1988; Kannel, Feinleib, McNamara, Garrison, & Castelli, 1979).

Out of the 14,428 members of the three main cohorts of the FHS, a total of 9,237 individuals have been genotyped (4,986 women and 4,251 men). FHS makes available a data set of expected genotypes for all 2,543,887 SNPs in the European ancestry HapMap sample that was computed from the 550,000 observed SNPs from an Affymetrix array using the program MACH (for information on how this data set was constructed, see De Bakker (2008)). These data contain the same rs4950 SNP that we analyze in Add Health.

The FHS also asked 3,540 participants in the offspring cohort a series of questions about their work life. Of these, 1,860 reported working at least 40 hours a week outside the home and responded to a series of questions about their relationships with their boss, co-workers, and their "immediate subordinate" (defined on the survey as "the person directly below you"). As an example, one question asked subjects if the subordinate "is a person you can rely on to carry his/her load." Respondents who answered at least one question about the subordinate are coded as being in a leadership role, while those who answered "does not apply" to all 7 questions about subordinates are coded as not being in a leadership role. According to this method of ascertainment, 58.6% of the sample indicated that they had a subordinate. This question is different from the question asked in Add Health that we used to construct our leadership role occupancy variable. The FHS derived measure is noisier, and potentially

[12] Population stratification turns out to be a real issue in the case of *CHRNB3* given that the between-family variance component on rs4950 is significant. For this reason, we should be cautious about the interpretation of any results produced using the alternative, case–control method.

[13] The Bonferroni approach requires a more stringent significance level equal to the standard significance threshold divided by the number of tests. Here this would imply a $p$-value threshold of $0.05/9 = 0.006$.

[14] We note that this candidate gene approach does not require a Bonferroni corrected $p$-value threshold of $p < 10^{-7}$ as would be standard in genome-wide association studies (GWAS) where hundreds of thousands of SNPs are tested in a single analysis without *a priori* hypotheses.

[15] The rs4950 genotype is robust to model specifications that incorporate additional control variables that are more common in the leadership literature (Big 5 personality traits, IQ measure, and height). For specific variable descriptions please see summary statistics in Appendix.

**Table 2**
Family-based models of association between the Add Health genotypes and leadership role occupancy. Note: Logistic odds ratios and *p*-values for the within and between-family variance components. The last two columns present results for both SNPs on the CHRNB3 gene. The interpretation of the main result is that, as compared to the family average, having one more `rs4950` A allele (instead of a G allele) increases the odds of being a leader by approximately 50%. Variable definitions are presented in the Appendix.

| | | DRD4: r7 | DRD2: a2 | SLC6A3: r10 | MAOA: High | SLC6A4: Long | rs2304297:C | rs892413:A | rs4950:A | rs13280604:A |
|---|---|---|---|---|---|---|---|---|---|---|
| Within | OR | 1.20 | 0.88 | 1.17 | 0.91 | 1.12 | 1.00 | 1.02 | 1.50 | 1.38 |
| | *p*-value | 0.351 | 0.453 | 0.371 | 0.414 | 0.432 | 0.977 | 0.899 | 0.006 | 0.057 |
| Between | OR | 0.87 | 1.04 | 0.90 | 1.08 | 0.88 | 1.08 | 1.16 | 1.16 | 1.11 |
| | *p*-value | 0.096 | 0.619 | 0.238 | 0.204 | 0.071 | 0.294 | 0.045 | 0.041 | 0.152 |
| Male | OR | 1.33 | 1.35 | 1.33 | 1.34 | 1.34 | 1.32 | 1.37 | 1.35 | 1.40 |
| | *p*-value | 0.001 | 0.001 | 0.001 | 0.001 | 0.001 | 0.003 | 0.000 | 0.001 | 0.000 |
| Age | OR | 1.06 | 1.06 | 1.06 | 1.06 | 1.06 | 1.05 | 1.06 | 1.06 | 1.06 |
| | *p*-value | 0.018 | 0.023 | 0.017 | 0.017 | 0.037 | 0.047 | 0.039 | 0.031 | 0.035 |
| Intercept | OR | 0.14 | 0.13 | 0.15 | 0.12 | 0.18 | 0.17 | 0.17 | 0.16 | 0.15 |
| | *p*-value | 0.001 | 0.000 | 0.001 | 0.000 | 0.004 | 0.003 | 0.002 | 0.002 | 0.002 |
| *N* | | 2251 | 2250 | 2254 | 2238 | 2250 | 2079 | 2155 | 2126 | 2026 |
| Pseudo $R^2$ | | 0.007 | 0.006 | 0.006 | 0.006 | 0.007 | 0.005 | 0.008 | 0.009 | 0.009 |

contains higher levels measurement error. With the standard effects of measurement error in nonlinear models (see e.g. Carroll et al., 2006), we do not expect the FHS replication results to be as strong as in the Add Health results presented above.[16]

We merged the gene and work data and conducted an association test using a logit regression with a general estimating equations (GEE) approach to account for within-family correlation of errors. As shown in Model 1 in Table 5, this association is statistically significant and in the expected direction. In Model 2, we include additional controls for age and gender. To control for population stratification in the absence of family clustered data we resort to including the first ten principal components of a singular value decomposition of the subject-genotype matrix in the regression (Price et al., 2006).[17] Once again, the replicated association is statistically significant—the results in Table 5 suggest that having one more `rs4950` A allele (instead of a G allele) increases the odds of occupying a leadership role by approximately 25%.

### 5.6. rs4950 and leadership role occupancy

Given that we have identified an association in two independent samples, we now consider the relationship between this candidate gene and leadership role occupancy. The nicotine acetylcholine (`nACh`) genes encode receptors on sending and target neurons that play a critical role in the neurotransmission process. `nACh` receptors are made up of a combination of two different protein subunits, $\alpha$ and $\beta$. There are 8 different $\alpha$ ($\alpha 2-\alpha 7$, $\alpha 9-\alpha 10$) and four different $\beta$ subunits ($\beta 2-\beta 4$). The triggering, closure, and desensitization of `nACh` receptors are each influenced by the $\alpha$ and $\beta$ subunits of which they are comprised (Dani & Bertrand, 2006).

Researchers studying impulsive behavior have focused on presynaptic `nACh` receptors, and the genes that code for them, because they have been shown to modulate dopamine release in the midbrain (Azam et al., 2002; Wonnacott, 1997). Dopamine in the *substantia nigra compacta* (`SNc`) plays a critical role in positive reinforcement associated with learned behavior as well as established stimulus response habits (Meyer, Yoshikami, & McIntosh, 2008). The `SNc` also supplies dopamine to the striatum, an area of the brain that has been demonstrated to be highly active during laboratory tests of delay discounting (Hariri et al., 2006).

Two particular subunits that have been shown to influence dopamine release are $\alpha 6$ and $\beta 3$ (Cui et al., 2003; Meyer et al., 2008). The *CHRNA6* gene encodes $\alpha 6$ receptor subunits and the *CHRNB3* gene encodes $\beta 3$ subunits. *CHRNA6* and *CHRNB3* are associated with impulsive behaviors like nicotine initiation and dependence (Feng et al., 2004; Hoft et al., 2009; Li et al., 2005; Schlaepfer, Hoft, & Ehringer, 2008) and alcohol abuse (Hoft et al., 2009). These behaviors are considered to be impulsive because they privilege immediate small rewards over long-term health benefits. Moreover, they are correlated with impulsivity and patience as measured by the delay discount task (Bickel, Odum, & Madden, 1999; Madden, Petry, Badger, & Bickel, 1997).

[16] Attenuation bias is just one possibility, but more generally measurement error effects in nonlinear models can move estimates in any direction. For details see Carroll et al. (2006).

[17] The first principal component of a set of variables is the linear combination of the variables with the coefficients chosen to capture as much of the sample variation as possible. The second principal component is obtained analogously, but subject to the constraint that it seek to capture whatever variation is remaining after the first principal component has been applied and so on. We used the scores of each individual on each of the 10 first principal components as control variables in the main regression specification; in effect, these 10 values capture common variation across the population structure, and thus offer at least a partial control for population stratification. In fact, ancestral origin can usually be determined with just the first two components (Novembre et al., 2008). Because principal component analysis assumes independent observations, we did not use our entire (family-based) FHS sample to construct the principal components. Instead we used a subsample of 2,507 unrelated individuals to calculate the principal components of the genotypic data and then projected the other individuals in the sample onto those principal components, thus obtaining the loadings of each individual on each of the top 10 principal components.

**Table 3**
Relationship between leadership role occupancy and rs4950 genotype.

| | rs4950 | | | |
|---|---|---|---|---|
| Leader | Both G alleles | G & A allele | Both A alleles | Total |
| No | 192 | 507 | 641 | 1,340 |
| | 69.6% | 63.8% | 60.4% | 62.8% |
| Yes | 84 | 287 | 421 | 792 |
| | 30.4% | 36.2% | 39.6% | 37.1% |
| Total | 276 | 794 | 1,062 | 2,132 |
| | 100% | 100% | 100% | 100% |

Scientific research is not at a stage yet where it can fully detail the cascade of neurological dynamics through which this particular SNP may influence leadership role occupancy. However, taken together, the reported findings suggest that the *CHRNA6* and *CHRNB3* genes may play a role in the regulation of behavior because of the central role of the dopamine system. As noted earlier, the dopamine system has also been shown to significantly influence sensitivity to information stimulus (Berridge & Robinson, 1998; Volkow, 2004), which increases the ability to deal with cognitive tasks associated with a leadership role. Recent studies in neurobiology have provided more direct evidence for the link between dopamine and leadership (Lee et al., 2009), and separately for the hormone oxytocin (Nowack, 2009), which has been shown to interact with dopamine to mediate socio-affiliative behaviors (Baskerville & Douglas, 2010).

## 6. Discussion and conclusion

### 6.1. Discussion

Our study takes a first step towards providing new insights into the fundamental origins of leadership emergence by studying genetic variation as a possible source of leadership role occupancy. First, using a classical twin design, we estimated that about a quarter of the variation in leadership role occupancy is heritable. This is a slightly more conservative estimate of the heritability of leadership role occupancy as compared to previously reported heritability estimates of about a third (e.g. Arvey et al., 2006, 2007). Second, this is the first research to identify a specific genotype that is significantly associated with occupying a leadership position. Using data collected by the Add Health study we found that individuals with two A alleles for the rs4950 SNP are significantly more likely to occupy leadership positions as compared to individuals with one or two G alleles. This result held after correction for multiple testing and was obtained using family-based association tests designed to avoid false positives due to population stratification. Furthermore, using data from the Framingham Heart Study we were able to replicate the association, lending further support to this genetic association finding.

Genes – and the neurological processes that they encode – are upstream from personality factors related to leadership emergence that have been identified thus far in the literature. Identifying genes associated with leadership role occupancy takes us a step closer to understanding the biological sources of leadership emergence. At the same time, we remain cautious and note that other independent replication studies are needed to corroborate this novel association result. Furthermore, we cannot currently pinpoint a precise causal pathway that connects the rs4950 SNP on the *CHRNB3* gene to leadership role occupancy. The rs4950 SNP may directly influence role occupancy, but could also affect the development of traits that, in turn, affect a predisposition to take on a leadership position. Moreover, the rs4950 SNP may influence the tendency of people to select into environments more favorable for a leadership role or the rs4950 SNP may result in a differential sensitivity to environmental stimuli that mediate the propensity to occupy a leadership role. While our analyses rule out some potential mediators and gene–environment interactions, we remain largely agnostic on the different pathways that may relate the rs4950 SNP to leadership role occupancy. The task is made more difficult by the fact that leadership role occupancy is also a complex product of environmental influences and gene–environment interactions and it is likely polygenic in nature. Specific evaluation of causal pathways is further complicated by the complex relationship between traits and leadership emergence (Antonakis, 2011). In future work, we plan to explore the effects of rs4950 on various characteristics of leadership emergence by investigating the effect of the neuronal acetylcholine receptor genes (nAChRs) on the dopamine system which, in turn, is strongly related to personality traits and cognitive behavior (Lee et al., 2009).

Past research has shown just how important leadership can be for organizational (e.g. Antonakis, Cianciolo, & Sternberg, 2004; Bennis & Townsend, 2005), political (e.g. Schofield, 2006), and economic (e.g. Jones & Olken, 2005) outcomes. We also know that leadership is partly hereditary (e.g. Arvey et al., 2006, 2007; Chaturvedi, Zyphur, Arvey, Avolio, & Larsson, 2012; Chaturvedi et al., 2011; Johnson, Vernon, Harris, & Jang, 2004). However, there are still many questions about the individual differences of leaders, including the biological underpinnings of the differences (e.g. Antonakis, 2011; Antonakis et al., 2012). While we learnt a lot about the biology of leadership (e.g. Lee, Senior, & Butler, 2012; Senior, Lee, & Butler, 2011), we are only now starting to look at the level of genotype. Our contribution to the field is to show that genetic variation can help to explain variation in leadership role occupancy, identifying a specific gene that may drive the role occupancy. We believe that this will inform existing literature and

**Table 4**
Assessing potential mediators. Note: Shown are the coefficients on rs4950 measured in linear regression models for the following dependent variables respectively: income, job, education, religiosity, welfare, medication, smoking, alcohol, IQ, height, and the Big 5 personality traits. All regressions also include race, age, and gender controls.

| rs4950: A | | | |
|---|---|---|---|
| Dependent variable | Coeff. | SE | *p*-Value |
| Income | 819.5 | 1330.6 | 0.538 |
| Job | 0.007 | 0.016 | 0.646 |
| Education | 0.008 | 0.081 | 0.917 |
| Religiosity | −0.031 | 0.030 | 0.303 |
| Welfare | −0.009 | 0.007 | 0.192 |
| Medication | 0.008 | 0.016 | 0.602 |
| Smoking | −0.012 | 0.018 | 0.503 |
| Alcohol | 0.000 | 0.042 | 0.993 |
| IQ | 0.457 | 0.476 | 0.338 |
| Height | 0.343 | 0.288 | 0.234 |
| Openness | −0.078 | 0.087 | 0.370 |
| Conscientiousness | −0.028 | 0.092 | 0.764 |
| Extraversion | −0.076 | 0.107 | 0.478 |
| Agreeableness | −0.021 | 0.085 | 0.807 |
| Neuroticism | −0.050 | 0.093 | 0.587 |

guide future research into the biological foundations of leadership emergence. The results of our study can also inform future research into genetic determinants of leadership effectiveness.

### 6.2. Implications for practice

As suggested by Antonakis (2011, 272) research into genetic determinants of leadership is "very fundamental in nature and does not have immediate practical utility...." The results in our study are unlikely to have any direct implications for assessment and selection (and even if they did, privacy concerns would likely render the information unusable). Instead, we think this research is useful for understanding how leadership emerges and how we might be able to adapt environmental factors to improve leadership ability. Additional research is needed to ascertain the role of rs4950 (and many other genotypes) in shaping leadership emergence, and other possible genetic factors influencing leadership effectiveness.

Our work additionally should draw attention to the ethical issues surrounding the use of genetic tests for leadership selection and assessment. Human resource managers should be aware of the ethical problems that genetic profiling introduces into standard human resource management procedures. It is unlikely that individuals will want to be screened in this way, and the potential for violation of privacy means that we should seriously consider extending current protections against genetic discrimination from health care to employment. Given that genetic factors do not explain most of the variance in leadership emergence, our main suggestion for practice is that this research may help in the identification of specific environmental factors that can help in the development of leadership skills.

**Table 5**
GEE logit models of association between rs4950 and leadership role occupancy. Note: Variable definitions are in the Appendix. Standard errors (SE) and *p*-values are also presented.

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| | OR | SE | *p*-Value | OR | SE | *p*-Value |
| rs4950 "A" alleles | 1.25 | 0.14 | 0.04 | 1.25 | 0.14 | 0.04 |
| Male | | | | 1.62 | 0.19 | 0.00 |
| Age | | | | 1.00 | 0.00 | 0.55 |
| Principal component 1 | | | | 1.00 | 0.00 | 0.21 |
| Principal component 2 | | | | 5.45 | 13.4 | 0.03 |
| Principal component 3 | | | | 232 | 3,086 | 0.09 |
| Principal component 4 | | | | 5.7E+9 | 1.8E+10 | 0.76 |
| Principal component 5 | | | | 1.23 | 4.43 | 0.95 |
| Principal component 6 | | | | 3.74 | 19.3 | 0.01 |
| Principal component 7 | | | | 6,905 | 33,489 | 0.07 |
| Principal component 8 | | | | 0.01 | 0.04 | 0.14 |
| Principal component 9 | | | | 7.61 | 27.7 | 0.58 |
| Principal component 10 | | | | 0.02 | 0.07 | 0.26 |
| Intercept | 0.99 | 0.18 | 0.97 | 0.77 | 0.27 | 0.46 |
| *N* | 1543 | | | 1543 | | |
| Pseudo $R^2$ | 0.002 | | | 0.02 | | |

### 6.3. Conclusion

The results we report here suggest that what determines whether people occupy leadership positions may be a complex product of genetic and environmental influences. But this is just the beginning. To better understand the causal mechanisms underlying leadership role occupancy, future research should investigate whether the genetic variant rs4950 and the neuronal acetylcholine receptor genes affect alternative measures of leadership emergence, leadership types, and personality traits that are essential components of leadership emergence and effectiveness. It should also focus on the way these and other genetic variants interact with contextual influences to jointly shape leadership. But most importantly, future work should not assume that the environment is all that matters. If we really want to understand leadership and its effect on organizational, institutional, economic, and political outcomes, we must study both nature and nurture.

## Appendix

### *Variable definitions*

| Variable name | Variable definition |
|---|---|
| Leadership role occupancy | Dummy variable constructed from the answer to the question "Thinking about your official job duties, which of the following statements best describes your supervisory responsibilities at your (current/most recent) primary job?" The dummy variable takes 1 when supervising—or having supervised—other employees. |
| DRD4 | Number of r7 alleles (0, 1, or 2) as opposed to r4 alleles. |
| DRD2 | Number of a2 alleles (0, 1, or 2) as opposed to a1 alleles. |
| SLC6A3 | Number of r9 alleles (0, 1, or 2) as opposed to r10 alleles. |
| MAOA | Number of "High" alleles (0, 1, or 2) as opposed to "Low" alleles. |
| SLC6A4 | Number of "Long" alleles (0, 1, or 2) as opposed to "Short" alleles. |
| rs2304297 | Number of C alleles (0, 1, or 2) for this SNP on *CHRNA6* (as opposed to G alleles). |
| rs892413 | Number of A alleles (0, 1, or 2) for this SNP on *CHRNA6* (as opposed to C alleles). |
| rs4950 | Number of A alleles (0, 1, or 2) for this SNP on *CHRNB3* (as opposed to G alleles). |
| rs13280604 | Number of A alleles (0, 1, or 2) for this SNP on *CHRNB3* (as opposed to G alleles). |
| Principal Component 1–10 | Individual loading for each individual on the 10 principal components associated with the 10 largest eigenvalues of a singular value decomposition of the subject-genotype matrix. |

### *Summary statistics*

| | Mean | Std Dev | Min | Max |
|---|---|---|---|---|
| Leadership role occupancy | 0.37 | 0.48 | 0 | 1 |
| DRD4: r7 | 0.43 | 0.59 | 0 | 2 |
| DRD2: a2 | 1.47 | 0.64 | 0 | 2 |
| SLC6A3: r10 | 1.54 | 0.60 | 0 | 2 |
| MAOA: High | 1.18 | 0.86 | 0 | 2 |
| SLC6A4: Long | 1.14 | 0.72 | 0 | 2 |
| rs2304297: C | 1.31 | 0.71 | 0 | 2 |
| rs892413: A | 1.37 | 0.70 | 0 | 2 |
| rs4950: A | 1.35 | 0.71 | 0 | 2 |
| rs13280604: A | 1.33 | 0.73 | 0 | 2 |
| Age | 29.1 | 1.7 | 25 | 34 |
| Income | 34,632 | 38,284 | 0 | 920,000 |
| Job | 0.78 | 0.41 | 0 | 1 |
| Education | 5.67 | 2.19 | 1 | 13 |
| Religiosity | 2.54 | 0.88 | 1 | 4 |
| Welfare | 0.04 | 0.21 | 0 | 1 |
| Medication | 0.61 | 0.49 | 0 | 1 |
| Smoking | 0.43 | 0.49 | 0 | 1 |
| Alcohol | 0.92 | 1.28 | 0 | 6 |
| IQ | 98.48 | 17.08 | 7 | 122 |
| Height (cm) | 169.87 | 10.13 | 122 | 226 |
| Openness | 14.50 | 2.45 | 4 | 20 |
| Conscientiousness | 14.64 | 2.69 | 4 | 20 |
| Extraversion | 13.22 | 3.06 | 4 | 20 |
| Agreeableness | 15.24 | 2.41 | 4 | 20 |
| Neuroticism | 10.45 | 2.74 | 4 | 20 |

## References


Add Health Biomarker Team. (2007). Biomarkers in wave iii of the add health study. http://www.cpc.unc.edu/projects/addhealth/files/biomark.pdf

Ahlquist, J., & Levi, M. (2011). Leadership: What it means, what it does, and what we want to know about it. *Annual Review of Political Science*, *14*, 1–24.

Antonakis, J. (2011). Predictors of leadership: The usual suspects and the suspect traits. In A. Bryman, D. Collinson, K. Grint, B. Jackson, & M. Uhl-Bien (Eds.), *Sage handbook of leadership* (pp. 269–285). Thousand Oaks: Sage.

Antonakis, J., Cianciolo, A. T., & Sternberg, R. J. (2004). *The nature of leadership.* Sage Publications, Inc.

Antonakis, J., & Dalgas, O. (2009). Predicting elections: Child's play! *Science*, *323*(5918), 1183.

Antonakis, J., Day, D., & Schyns, B. (2012). Leadership and individual differences: At the cusp of a renaissance. *The Leadership Quarterly*, *23*, 643–650.

Arvey, R., Rotundo, M., Johnson, W., Zhang, Z., & McGue, M. (2006). The determinants of leadership role occupancy: Genetic and personality factors. *The Leadership Quarterly*, *17*(1), 1–20.

Arvey, R., Zhang, Z., Avolio, B., & Krueger, R. (2007). Developmental and genetic determinants of leadership role occupancy among women. *Journal of Applied Psychology*, *92*(3), 693–706.

Avolio, B., Rotundo, M., & Walumbwa, F. (2009). Early life experiences as determinants of leadership role occupancy: The importance of parental influence and rule breaking behavior. *The Leadership Quarterly*, *20*, 329–342.

Azam, L., Winzer-Serhan, U., Chen, Y., & Leslie, F. (2002). Expression of neuronal nicotinic acetylcholine receptor subunit mrnas within midbrain dopamine neurons. *Journal of Comparative Neurology*, *444*, 260–274.

Baron, R., & Kenny, D. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*(6), 1173–1182.

Barrick, M., & Mount, M. (1991). The big five personality dimensions and job performance: A meta-analysis. *Personnel Psychology*, *44*, 1–26.

Baskerville, T. A., & Douglas, A. J. (2010). Dopamine and oxytocin interactions underlying behaviors: Potential contributions to behavioral disorders. *CNS Neuroscience and Therapeutics*, *16*, 92–123.

Bass, B., & Stogdill, R. (1990). *Bass & Stogdill's handbook of leadership: Theory, research, and managerial applications.* Free Press.

Beauchamp, J., Cesarini, D., Johannesson, M., van der Loos, M., Koellinger, P., Groenen, P., et al. (2011). Molecular genetics and economics. *Journal of Economic Perspectives*, *25*(4), 57–82.

Behrman, J., & Taubman, P. (1989). Is schooling 'mostly in the genes'? nature–nurture decomposition using data on relatives. *Journal of Political Economy*, *97*(1425–1446).

Benjamin, D. J., Chabris, C. F., Glaeser, E. L., Gudnason, V., Harris, T. B., Laibson, D. I., et al. (2007). Genoeconomics. In M. Weinstein, J. W. Vaupel, & K. W. Wachter (Eds.), *Biosocial surveys*. Washington, D.C.: The National Academies Press.

Benjamin, J., Li, L., Patterson, C., Greenberg, B., Murphy, D., Hamer, D., et al. (1996). Population and familial association between the d4 dopamine receptor gene and measures of novelty seeking. *Nature Genetics*, *12*(1), 81–84.

Bennis, W., & Townsend, R. (2005). *Rethinking leadership: Strategies to empower the organization.* New York: Quill.

Berridge, K., & Robinson, T. (1998). What is the role of dopamine in reward: Hedonic impact, reward learning, or incentive salience? *Brain Research Reviews*, *28*(3), 309–369.

Bickel, W., Odum, A., & Madden, G. (1999). Impulsivity and cigarette smoking: Delay discounting in current, never, and ex-smokers. *Psychopharmacology*, *164*(4), 447–454.

Boehnke, M., & Langefeld, C. (1998). Genetic association mapping based on discordant sib pairs: The discordant-alleles test. *American Journal of Human Genetics*, *62*, 950–961.

Bouchard, T. (1998). Genetic and environmental influences on adult intelligence and special mental abilities. *Human Biology*, *70*(2), 257–279.

Bouchard, T., & Loehlin, J. (2001). Genes, evolution, and personality. *Behavior Genetics*, *31*(3), 243–273.

Bowles, S., & Gintis, H. (2002). The inheritance of inequality. *Journal of Economic Perspectives*, *16*(3), 3–30.

Brooks, S. P., & Gelman, A. (1998). General methods for monitoring convergence of iterative simulations. *Journal of Computational and Graphical Statistics*, *7*, 434–455.

Burton, P. R., Tiller, K. J., Gurrin, L. C., Cookson, W. O., Musk, Q. A. W., & Palmer, L. J. (1999). Genetic variance components analysis for binary phenotypes using generalized linear mixed models (glmms) and gibbs sampling. *Genetic Epidemiology*, *17*, 118–140.

Canli, T., & Lesch, K. (2007). Long story short: The serotonin transporter in emotion regulation and social cognition. *Nature Neuroscience*, *10*(9), 1103–1109.

Carroll, R. J., Ruppert, D., Stefanski, L. A., & Crainiceanu, C. M. (2006). *Measurement error in nonlinear models: A modern perspective* Number 105 in Monographs on Statistics and Applied Probability (2nd edition). Boca Raton, FL: Chapman and Hall/CRC.

Caspi, A., Sugden, K., Moffitt, T., Taylor, A., Craig, I., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-htt gene. *Science*, *301*, 386–389.

Charney, E., & English, W. (2012). Candidate genes and political behavior. *American Political Science Review*, *106*, 1–34.

Chaturvedi, S., Arvey, R., Zhang, Z., & Christoforou, T. (2011). Genetic underpinnings of transformational leadership: The mediating role of dispositional hope. *Journal of Leadership and Organizational Studies*, *20*(10), 1–11.

Chaturvedi, S., Zyphur, M., Arvey, R., Avolio, B., & Larsson, G. (2012). The heritability of emergent leadership: Age and gender as moderating factors. *The Leadership Quarterly*, *23*, 219–232.

Coventry, W. L., & Keller, M. C. (2005). Estimating the extent of parameter bias in the classical twin design: A comparison of parameter estimates from extended twin-family and classical twin designs. *Twin Research and Human Genetics*, *8*(3), 214–223.

Cui, C., Booker, T., Allen, R., Grady, S., Whiteaker, P., Marks, M., et al. (2003). The beta3 nicotine receptor subunit: A component of alpha-conotoxin mii-binding nicotinic acetylcholine receptors that modulate dopamine release and related behaviors. *Journal of Neuroscience*, *2*(35), 11045–11053.

Cupples, L., & D'Agnostino, R. (1988). Survival following initial cardiovascular events: 30 year follow-up. In W. B. Kannel, P. A. Wolf, & R. J. Garrison (Eds.), *The Framingham Study: An epidemiological investigation of cardiovascular disease* (pp. 88–2969). Bethesda, MD: National Heart, Lung and Blood Institute.

Damberg, M., Garpenstrand, H., Hallman, J., & Oreland, L. (2001). Genetic mechanisms of behavior: Don't forget about the transcription factors. *Molecular Psychiatry*, *6*(5), 503–510.

Dani, J., & Bertrand, D. (2006). Nicotine acetylcholine receptors and nicotinic cholinergic mechanisms of the central nervous system. *Annual Review of Pharmacology and Toxicology*, *47*, 699–729.

Dawes, C. T., & Loewen, P. J. (2010). The chrna6 gene, patience, and voter turnout. Available at SSRN: http://ssrn.com/abstract=1494509

De Bakker, P. (2008). Imputation in the framingham heart study. Mimeo: Harvard Medical School.

De Neve, J. -E. (2011). Functional polymorphism (5-httlpr) in the serotonin transporter gene is associated with subjective well-being: Evidence from a U.S. nationally representative sample. *Journal of Human Genetics*, *56*(6), 456–459.

De Neve, J. -E., & Fowler, J. H. (2010). Credit card borrowing and the monoamine oxidase A (MAOA) gene. Available at SSRN: http://ssrn.com/abstract=1457224

De Neve, J. -E., Christakis, N. A., Fowler, J. H., & Frey, B. S. (in press). Genes, economics, and happiness. *Journal of Neuroscience, Psychology, and Economics*.

Dreber, A., Apicella, C. L., Eisenberg, D. T., Garcia, J. R., Zamore, R. S., Lum, J. K., et al. (2009). The 7r polymorphism in the dopamine receptor d4 gene (drd4) is associated with financial risk taking in men. *Evolution and Human Behavior*, *30*, 85–92.

Ebstein, R. P., Novick, O., Umansky, R., Pirelli, B., Osher, Y., Blaine, D., et al. (1996). Dopamine d4 receptor exon iii polymorphism associated with the human personality trait of novelty seeking. *Nature Genetics*, *12*, 78–80.

Eichhammer, P., Sand, P. G., Sooertebecker, P., Langguth, B., Zowe, M., & Hajak, G. (2005). Variation at the drd4 promoter modulates extraversion in Caucasians. *Molecular Psychiatry*, *61*, 33–51.

Evans, D., & Leighton, L. (1989). Some empirical aspects of entrepreneurship. *The American Economic Review*, *79*(3), 519–535.

Feng, Y., Niu, T., Xing, H., Xu, X., Chen, C., Peng, S., et al. (2004). A common haplotype of the nicotine acetylcholine receptor alpha 4 subunit gene is associated with vulnerability to nicotine addiction in men. *American Journal of Human Genetics*, *75*, 112–121.
Fowler, J. H., Baker, L. A., & Dawes, C. T. (2008). Genetic variation in political participation. *American Political Science Review*, *101*(2), 233–248.
Fox, E., Ridgewell, A., & Ashwin, C. (2009). Looking on the bright side: Biased attention and the human serotonin transporter gene. *Proceedings of the Royal Society B*, *276*, 1747–1751.
Fuke, S., Suo, S., Takahashi, N., Koike, H., Sasagawa, N., & Ishiura, S. (2001). The vntr polymorphism of the human dopamine transporter (dat1) gene affects gene expression. *Pharmacogenomics Journal*, *1*(2), 152–156.
Gelman, A., Carlin, J. B., Stern, H. S., & Rubin, D. B. (2004). *Bayesian data analysis.* New York: Chapman and Hall.
Hariri, A., Brown, S., Williamson, D., Flory, J., de Wit, H., & Manuck, S. (2006). Preference for immediate over delayed reward is associated with magnitude of ventral striatal activity. *Journal of Neuroscience*, *26*(51), 13213–13217.
Harris, K. M., Halpern, C. T., Smolen, A., & Haberstick, B. C. (2006). The national longitudinal study of adolescent health (add health) twin data. *Twin Research and Human Genetics*, *9*(6), 988–997.
Harris, K., Halpern, C., Whitsel, E., Hussey, J., Tabor, J., Entzel, P., et al. (2009). The national longitudinal study of adolescent health: Research design. http://www.cpc.unc.edu/projects/addhealth/design
Hoft, N., Corley, R., McQueen, M., Huizinga, D., Menard, S., & Ehringer, M. (2009). Snps in chrna6 and chrnb3 are associated with alcohol consumption in a nationally representative sample. *Genes, Brain and Behavior*, *8*(6), 631–637.
Hoft, N., Corley, R., McQueen, M., Schlaepfer, I., Huizinga, D., & Ehringer, M. (2009). Genetic association of the chrna6 and chrnb3 genes with tobacco dependence in a nationally representative sample. *Neuropsychopharmacology*, *34*(3), 698–706.
Ilies, R., Gerhardt, M., & Le, H. (2004). Individual differences in leadership emergence: Integrating meta-analytic findings and behavioral genetics estimates. *International Journal of Selection and Assessment*, *12*(3), 207–219.
Jacobson, K., & Rowe, D. (1998). Genetic and shared environmental influences on adolescent BMI: Interactions with race and sex. *Behavior Genetics*, *28*(4), 265–278.
Jang, K., Livesley, W., & Vernon, P. (1996). Heritability of the big five personality dimensions and their facets: A twin study. *Journal of Personality*, *64*, 577–592.
Johnson, A., Vernon, P., Harris, J., & Jang, K. (2004). A behavior genetic investigation of the relationship between leadership and personality. *Twin Research and Human Genetics*, *7*(1), 27–32.
Jones, B., & Olken, B. (2005). Do leaders matter? National leadership and growth since World War II. *The Quarterly Journal of Economics*, *120*(3), 835–864.
Judge, T., Bono, J., Ilies, R., & Gerhardt, M. (2002). Personality and leadership: A qualitative and quantitative review. *Journal of Applied Psychology*, *87*(4), 765–779.
Judge, T. A., Colbert, A. E., & Ilies, R. (2004). A meta-analysis of the relationship between intelligence and leadership. *Journal of Applied Psychology*, *89*, 542–552.
Kannel, W., Feinleib, M., McNamara, P., Garrison, R., & Castelli, W. (1979). An investigation of coronary heart disease in families. *American Journal of Epidemiology*, *110*, 281–290.
Kendler, K., & Eaves, L. (1986). Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry*, *143*(3), 279.
Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics*, *23*, 21–27.
Lee, N., Senior, C., & Butler, M. (2012). Leadership research and cognitive neuroscience: The state of this union. *The Leadership Quarterly*, *23*, 213–218.
Lee, N. J., Senior, C., Butler, M. J. R., Powell, J., Phillips, M., & Surguladze, S. (2009). Dopamine, empathy, and the neurobiology of leadership. *British Psychological Association Annual Conference, Brighton*.
Li, W. -D., Arvey, R., Zhang, Z., & Song, Z. (2012). Do leadership role occupancy and transformational leadership share the same genetic and environmental influences? *The Leadership Quarterly*, *23*, 233–243.
Li, M., Beuten, J., Ma, J., Payne, T., Lou, X., Garcia, V., et al. (2005). Ethnic- and gender-specific association of the nicotinic acetylcholine receptor alpha4 subunit gene (chrna4) with nicotine dependence. *Human Molecular Genetics*, *14*, 1211–1219.
Mackay, T. (2001). The genetic architecture of quantitative traits. *Annual Review of Genetics*, *35*, 303–339.
Madden, G., Petry, N., Badger, G., & Bickel, W. (1997). Impulsive and self-control in opioid-dependent patients and non-drug-using control participants: Drug and monetary rewards. *Experimental and Clinical Psychopharmacology*, *5*(3), 256–262.
McDermott, R., Tingley, D., Cowden, J., Frazetto, G., & Johnson, D. D. (2009). Monoamine oxidase a gene (MAOA) predicts behavioral aggression following provocation. *Proceedings of the National Academy of Sciences of the United States of America*, *106*(7), 2118–2123.
Meyer, E., Yoshikami, D., & McIntosh, M. (2008). The neuronal acetylcholine receptors alpha4* and alpha6* differentially modulate dopamine release in mouse striatal slices. *Journal of Neurochemistry*, *105*, 1761–1769.
Meyer-Lindenberg, A., Buckholtz, J., Kolachana, B., Hariri, A., Pezawas, L., Blasi, G., et al. (2006). Neural mechanisms of genetic risk for impulsivity and violence in humans. *Proceedings of the National Academy of Sciences of the United States of America*, *103*(16), 6269–6274.
Moffitt, T., Caspi, A., & Rutter, M. (2005). Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry*, *62*(5), 473–481.
Munafò, M. R., Yalcin, B., Willis-Owen, S. A., & Flint, J. (2008). Association of the dopamine d4 receptor (drd4) gene and approach-related personality traits: Meta-analysis and new data. *Biological Psychiatry*, *63*, 197–206.
Neale, M., & Maes, H. (2002). *Methodology for genetic studies of twins and families.* Kluwer Academic Press.
Nicolaou, N., Shane, S., Cherkas, L., Hunkin, J., & Spector, T. (2008). Is the tendency to engage in entrepreneurship genetic. *Management Science*, *54*(1), 167–179.
Novembre, J., Johnson, T., Bryc, K., Kutalik, Z., Boyko, A. R., Auton, A., et al. (2008). Genes mirror geography within Europe. *Nature*, *456*, 98–101.
Nowack, K. (2009). *The neurobiology of leadership: Why women lead differently than men.* Paper presented at Life09—Congerso Internacional de Liderazgo Femenino, September 16–18, Barcelona, Spain.
Passamonti, L., Fera, F., Magariello, A., Cerasa, A., Gioia, M., Muglia, M., et al. (2006). Mao-a genetic variations influence brain activity associated with inhibitory control: New insight into the neural correlates of impulsivity. *Biological Psychiatry*, *59*(4), 334–340.
Plomin, R., DeFries, J., & Loehlin, J. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*(2), 309–322.
Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (5th ed.). New York, NY: Worth Publishers edition.
Price, A. L., Patterson, N. J., Plenge, R. M., Weinblatt, M. E., Shadick, N. A., & Reich, D. (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature Genetics*, *38*, 904–909.
Rabe-Hesketh, S., Skrondal, A., & Gjessing, H. (2008). Biometrical modeling of twin and family data using standard mixed model software. *Biometrics*, *64*, 280–288.
Riemann, R., Angleitner, A., & Strelau, J. (2006). Genetic and environmental influences on personality: A study of twins reared together using the self-and peer report NEO-FFI scales. *Journal of Personality*, *65*(3), 449–475.
Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, *63*(1), 1–19.
Scarr, S., & Carter-Saltzman, L. (1979). Twin method: Defense of a critical assumption. *Behavior Genetics*, *9*, 527–542.
Schlaepfer, I. R., Hoft, N. R., & Ehringer, M. A. (2008). The genetic components of alcohol and nicotine co-addiction: From genes to behavior. *Current Drug Abuse Review*, *1*(2), 124–134.
Schofield, N. (2006). *Architects of political change: Constitutional quandaries and social choice theory.* Cambridge University Press.
Scurrah, K. J., Palmer, L. J., & Burton, P. R. (2000). Variance components analysis for pedigree-based censored survival data using generalized linear mixed models (glmms) and gibbs sampling in bugs. *Genetic Epidemiology*, *19*(127–148).
Senior, C., Lee, N., & Butler, M. (2011). Organizational cognitive neuroscience. *Organizational Science*, *22*(3), 804–815.
Spielman, R., & Ewens, W. (1998). A sibship test for linkage in the presence of association: The sib transmission/disequilibrium test. *American Journal of Human Genetics*, *62*(450–458).

Tambs, K., Sundet, J., Magnus, P., & Berg, K. (1989). Genetic and environmental contributions to the covariance between occupational status, educational attainment, and IQ: A study of twins. *Behavior Genetics*, *19*(2), 209–222.
Todorov, A., Mandisodza, A., Goren, A., & Hall, C. (2005). Inferences of competence from faces predict election outcomes. *Science*, *308*(5728), 1623–1626.
van den Berg, S., Beem, L., & Boomsma, D. (2006). Fitting genetic models using Markov Chain Monte Carlo algorithms with bugs. *Twin Research and Human Genetics*, *9*(3), 334–342.
Visscher, P., Medland, S., Ferreira, M., Morley, K., Zhu, G., Cornes, B., et al. (2006). Assumption-free estimation of heritability from genome-wide identity-by-descent sharing between full siblings. *PLoS Genetics*, *2*, e41.
Volkow, N. (2004). Mediation of saliency in health and addiction. *157th Annual Meeting of the American Psychiatric Association, New York*.
Wonnacott, S. (1997). Presynaptic nicotinic ach receptors. *Trends in Neuroscience*, *20*, 92–98.
Xian, H., Scherrer, J. F., Eisen, S. A., True, W. R., Heath, A. C., Goldberg, J., et al. (2000). Self-reported zygosity and the equal-environments assumption for psychiatric disorders in the Vietnam era twin registry. *Behavior Genetics*, *30*, 303–310.
Xu, H., & Shete, S. (2006). Mixed-effects logistic approach for association following linkage scan for complex disorders. *Annals of Human Genetics*, *71*(2), 230–237.
Zhang, Z., Ilies, R., & Arvey, R. (2009). Beyond genetic explanations for leadership: The moderating role of the social environment. *Organizational Behavior and Human Decision Processes*, *110*(2), 118–128.