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# Beyond genetic explanations for leadership: The moderating role of the social environment

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ABSTRACT

Leadership role occupancy has recently been shown to have a genetic basis. We extend prior research by examining the moderating effects of the social environment during adolescence on the genetic influences on leadership role occupancy at work. Utilizing a sample of male twins (89 pairs of identical and 54 pairs of fraternal twins, with a mean age of 36.5 years), we found that genetic influences are weaker for those reared in enriched environments (i.e., higher family socioeconomic status, higher perceived parental support, and lower perceived conflict with parents). For those twins who had relatively poorer social environments, genetic influences on leadership role occupancy are significantly greater. These results have important implications for early interventions on leadership development inside and outside organizations.

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In the last several decades, behavioral genetic research has firmly established that individual differences in virtually all intellectual and behavioral domains have a genetic component (see Plomin, DeFries, Craig, & McGuffin, 2003). This research has been extended to a number of phenomena in organizational settings, such as job satisfaction (Arvey, Bouchard, Segal, & Abraham, 1989), work values (Keller, Bouchard, Arvey, Segal, & Dawis, 1992), job mobility (McCall, Cavanaugh, Arvey, & Taubman, 1997), and entrepreneurship (Nicolaou, Shane, Cherkas, Hunkin, & Spector, 2008). Researchers have also examined the genetic contributions to leadership. Several studies have shown that leadership role occupancy—the extent to which individuals had occupied or are now occupying positions of formal leadership in organizational settings—has a genetic component (Arvey, Rotundo, Johnson, Zhang, & McGue, 2006; Arvey, Zhang, Avolio, & Krueger, 2007; Ilies, Gerhardt, & Le, 2004). Using quantitative behavioral genetics methods to estimate heritability (i.e., $h^2$, the proportion of the total observed variability of a variable due to genetic differences between people), Arvey et al. (2006) found, based on a sample of male twins, that about 30% of the individual differences in leadership role occupancy can be attributed to latent genetic factors (i.e., $h^2 = .30$). This estimate was replicated in an independent sample of female twins (Arvey et al., 2007).

An interesting and controversial question surrounding the nature of genetic influences on leadership is whether the genetic effects establish constraints on the effectiveness of leadership development/intervention efforts in organizations and in earlier life. Partitioning the variance in a measure of leadership into its genetic and environmental components provides little opportunity to examine the role played by environmental factors along the growth path of a potential leader. Apart from the main effects of genetics, the environment and the developmental efforts stemming from the environment could have an active influence on the extent to which one capitalizes on his or her genetic endowments for leadership. Directly relevant to this possibility are gene–environment interactions (G × E)—the notion that environments can modify the influence of an individual's genetic background, either strengthening or weakening the effects of genes on phenotypes (Plomin, DeFries, & Loehlin, 1977).

The present study examines such moderating effects of environmental factors on genetic influences on leadership. Extending the findings of Arvey et al. (2006, 2007), we investigate the moderating role of the social environment in adolescence on the genetic effects on leadership role occupancy at work. Specifically, we examine whether the heritability of leadership at work is moderated by individuals' developmental environment in adolescence.

Studying such moderating effects of the environment has been advocated as a means to advance research in both basic (e.g., Dick & Rose, 2002; Van den Oord & Rowe, 1998) and applied (Ilies, Arvey, & Bouchard, 2006) psychology. Ilies et al. (2006) have suggested that the examination of G × E interaction effects on organizational behavior constructs is a fruitful avenue for future research, and argued that organizational scholars

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should engage in such endeavors in order to advance theories on the developmental processes leading to individual differences in behavior at work.

We note at the outset that the social environmental variables examined in the current study may not be "purely environmental" or exogenous in the sense that they are independent of any genetic influences. Gene–environment correlations—the notion that genes may affect the tendency of people to select themselves into certain environments or evoke specific responses from the people around them (Scarr & McCartney, 1983)—may also be present. Consequently, environmental measures could be genetically determined to some extent (e.g., passive, active, evocative G–E correlations; Rutter, Moffitt, & Caspi, 2006; Scarr & McCartney, 1983). Previous research has found genetic influences in variables that are commonly assumed to be environmental, such as organizational climate (Hershberger, Lichtenstein, & Knox, 1994). The existence of G–E correlations may confound the results when G × E interactions are examined (Scarr & McCartney, 1983). Following prior research (e.g., Purcell, 2002), the current study uses appropriate procedures to partial out the potentially confounding effects of gene–environment correlations. The details of these procedures are reported in the method section.

## Gene–environment interactions on leadership

For adolescents, the family environment is a critical and the most important component of the social environment in which they grow up (Bell, 1968; Zastrow & Kirst-Ashman, 1997). Family environments (e.g., levels of financial resources and parental support) are more proximal and influential than other aspects of adolescents' social environments (e.g., neighborhood, school, and peers; Bahr, Marcos, & Maughan, 1995; Brook, Nomura, & Cohen, 1989). Consequently, in the current study we focus on the family environment in adolescence and examine its impact on the genetic influences on leadership at work. It is notable that family environment can be captured in the behavioral genetics model as the shared and/or non-shared environmental effects. By definition, shared environmental effects refer to the extent to which growing up in the same family makes people similar. Non-shared-environmental factors refer to the unique environment that different siblings experience, despite growing up in the same family. For example, although parents' divorce is a "common" event to siblings, it could have unique and differential influences across different children (including twins) because of the unique ways in which each child experiences this event. In other words, parents' divorce is a common environmental factor only to the extent it provide similar/shared experience to different siblings or twins.

The moderating effects of the family environment on genetic influences have been examined for a variety of organizational behavior-related constructs such as substance use (Dick et al., 2007), verbal abilities (Asbury, Wachs, & Plomin, 2005), intelligence (Harden, Turkheimer, & Loehlin, 2007; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), and emotional stability (Jang, Dick, Wolf, Livesley, & Paris, 2005). The direction of the environment's moderating effects was found to differ across the different constructs examined. For example, supportive and affluent family environments have been shown to strengthen the effects of genetic factors on intelligence (e.g., Harden et al., 2007; Rowe, Jacobson, & Van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Conceptually, it can be argued that enriched environments may enable the underlying genetic predispositions to exert greater influence, and thus, those reared in enriched social environments show higher heritability as compared with those reared in depleted environments (Bronfenbrenner & Ceci, 1994).

In contrast, other researchers have argued for a weakening effect of enriched environments on the genetic influences on certain constructs. They contend that higher genetic influences could be fostered by adverse environments (e.g., Gottesman, 1991; Plomin & Rutter, 1998; Rende & Plomin, 1992). Supporting this argument, affluent family environments have been shown to weaken the genetic effects on verbal abilities in early childhood (e.g., Asbury et al., 2005). Conceptually, in the context of leadership, enriched environments could weaken genetic influences because, similar to verbal abilities in childhood, skills and personal characteristics that lead to leadership emergence are largely interpersonal (social) and are thus typically proactively acquired and developed through life experience (Day, 2000). (In contrast, intelligence, as an intrapersonal capacity endowment, is less likely to be influenced by social factors.) Therefore, the influence of individuals' genetic predispositions for leadership could be strengthened by deprived, conflict-ridden environments.

That is, because the development of leadership capabilities, as well as the emergence of leaders, is thought to depend upon overcoming setbacks, adversity, and crises (e.g., Bennis & Thomas, 2002; Bligh, Kohles, & Meindl, 2004; Luthans & Avolio, 2003; Masten & Reed, 2002; Osborn, Hunt, & Jauch, 2002), it can be argued that social environments characterized by adversity and interpersonal conflict allow greater influence of genetic differences on leadership potential, thus making individual differences in genetics more important in distinguishing between leaders and non-leaders. Conversely, the absence of adversity and conflict makes genetic differences associated with the effective handling of or learning from such situations less relevant, which would translate into a weaker link between genetic differences and individual differences in leadership emergence. As Bennis (1994) suggested, one of the most reliable predictors of leadership is people's capability to find meaning in negative situations and to learn from these circumstances. Bennis (1994, p. 121) argues that people's adaptive capacity is "an almost magical ability to transcend adversity, with all its attendant stresses, and to emerge stronger than before". Coutu (2002) also contends that the ability to rebound from adversity and conflict is critical to successful leaders. Therefore, the presence of adverse factors during one's early development stages could allow and even magnify the influences of differences in genetic backgrounds when predicting leadership role occupancy at work.

Thus far, we have presented two distinct conceptual arguments suggesting opposite moderating effects of the social environment on the heritability of leadership. Following theoretical and empirical work on the heritability of intelligence (e.g., Rowe et al., 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), the first argument suggests that more enriched, as opposed to more impoverished, environments would allow greater influence of genetic differences in leadership capacity, thus strengthening the heritability of leadership emergence. Second, based on leadership theory that links overcoming adversity and crises to leadership emergence (Bligh et al., 2004; Luthans & Avolio, 2003), and given the social nature of leadership skills which suggests that learning and development play a crucial role in the realization of one's leadership potential (unlike for intelligence), an opposite effect can be predicted such that more impoverished social environments like those involving interpersonal conflict would allow the greater influences of genetic differences in leadership capacities.

In sum, previous conceptual work can be interpreted to suggest both a positive and a negative moderating effect of the social environment on the heritability of leadership role occupancy. Because of the lack of specific empirical research on gene–environment interactions in the context of leadership emergence, we will not formally propose a directional moderating effect, but rather examine the direction of such interactions in an exploratory fashion. In

the following sections we describe three measures reflecting the social environments that people face in adolescence, and we then empirically examine the moderating effects of these measures on the genetic influences on leadership role occupancy at work.

*Family socioeconomic status*

Socioeconomic status (SES) describes a family's ranking on a hierarchy according to access to or control over some combination of valued commodities such as wealth, power, and social status (Mueller & Parcel, 1981). SES often serves as an overall measure of the level of resources available to adolescents when they grow up (McLoyd, 1998). Following the previous arguments, high SES (and thus high levels of resources and enriched environment) could either allow greater impacts of genetics in influencing leadership emergence (as is the case for intelligence) or weaken the effect of individual differences associated with their genetic backgrounds on leadership potential. The reasoning here is that those with high SES experienced less adversity compared to those with low SES, because low SES can be considered an indicator of adverse social environments during the developmental years.

*Perceived parental support and perceived conflict with parents*

We also examine the moderating role of two direct measures of family environment when adolescents were growing up: perceived parental support and perceived conflict with parents. High parental support in adolescence, similar to high levels of family SES, may reflect a supportive and enriched environment in which individuals can fully develop their genetic leadership potential because enriched family environments can promote the development of skills that allow individuals to more efficiently capitalize on future leadership opportunities in life. This suggests that parental support would positively influence the heritability of leadership emergence.

Conversely, in enriched social environments, abundant resources may play a greater role than individuals' genetic endowments in promoting leadership emergence in later life. Moreover, individuals in depleted parental environments do not have such luxuries and have to rely upon their genetic endowments to develop their leadership potential; thus, the genetic potential for leadership would be more influential in such an environment. Following this line of thinking, the parental support that individuals experienced during adolescence would negatively influence the genetic effects on leadership emergence such that the heritability of role occupancy at work would be lower for those who experienced high levels of parental support.

As noted, the presence of interpersonal conflict is a feature of the social environment thought to be important for the development of leadership skills. Because we study the role of the social environment in adolescence, we focus on conflict with parents as a particularly salient form of interpersonal conflict during these formative years. As was the case with the other two environmental constructs, if harmonious family environments characterized by little interpersonal conflict provide the nutrients for the realization of genetic differences in leadership capacities, conflict should negatively influence the heritability of leadership emergence, such that the heritability would be higher for those who experienced low conflict with their parents in adolescence.

On the other hand, as discussed in the introduction, managing conflicts or crises, as well as learning from such experiences, is important for leadership (e.g. Bligh et al., 2004; Luthans & Avolio, 2003; Mumford, Friedrich, Caughron, & Byrne, 2007; Osborn et al., 2002), and the presence of interpersonal conflict in adolescence may therefore allow greater influence of genetic differences in individuals' conflict management abilities that predict leadership in later life. This view predicts that conflict with parents positively influences the heritability of leadership emergence. Next we describe a study that tests such opposite predictions for the moderating effects of the three environmental variables on the heritability of leadership role occupancy at work.

## Methods

*Sample and procedures*

The sample consists of a cohort of identical (i.e., monozygotic or MZ) and fraternal (i.e., dizygotic or DZ) twins drawn from the Minnesota Twin Registry (see Lykken, Bouchard, McGue, & Tellegen, 1990). The Registry used public birth records to identify various cohorts of twins born in the state of Minnesota. For the present investigation, we utilized a series of surveys conducted on a cohort of male twins born between the years 1961 and 1964 (a cohort of 1116 twins).

The current study uses data from three surveys conducted at different time points. When the twins were first recruited to the Registry (in 1995, when they were, on average, at the age of 32), 885 of them completed a Background Questionnaire and provided demographic information and measures on their family background including family socioeconomic status. The response rate was 79.3% for this survey. In 1997, this cohort received a Parental Environmental Questionnaire assessing their retrospective perceptions of parental environment when they were adolescents including perceived parental support and perceived conflict with parents. A total of 682 of the twins completed this questionnaire, yielding a response rate of 34.2% for this survey. In 2000, the same cohort received a survey on their leadership activities, and a total of 650 completed this survey, yielding a response rate of 58.2%. Due to the response rates to the various surveys, analyses have slightly different sample sizes when different parental environment variables were examined. In particular, sample size is 286 (89 pairs MZ twins and 54 pairs DZ twins) when perceived parental support and perceived conflict with parents were examined as potential moderators of genetic influences on leadership. Sample size is 340 (103 pairs of MZ and 67 pairs of DZ twins) when SES was tested as a moderator. Among the 286 twins, all are white males with a mean age of 36.5 ($SD$ = 1.09), and 74.4% were married or living with a partner. With regard to education levels, 60.9% of them have high school or higher educational attainment.

Data based on the leadership questionnaire have been published elsewhere (i.e., Arvey et al., 2006) in which the existence of genetic influences on leadership role occupancy was investigated. Although the dependent variable in the current study is the same as that in Arvey et al. (2006), we ask very different research questions in this study (related to the moderating role of family environments). Moreover, the various measures assessing environmental characteristics were drawn from survey instruments not used in prior research reports on leadership using this database.

*Measures*

*Leadership role occupancy*

Participants' responses to two questions were used to assess their leadership role occupancy at work. The first question asked them to indicate, on a checklist, whether they had held positions at work that would be considered managerial or supervisory in nature (e.g., work group leader, director, vice-president, etc.). The positions were coded according to the organizational hierarchy (e.g., we assigned 7 points for "President", 6 points for "Vice-President", etc.). The second question asked the individuals to indicate

the number of work-related professional associations in which they served as a leader. These two measures used the bio-history approach, which is a well-known and acceptable procedure in assessing autobiographical or historical events among individuals (Mumford & Stokes, 1992). Eleven follow-up phone calls were made to verify the information participants provided on these two questions, and no errors were detected. The two scores were standardized and summed to form a composite measure of leadership role occupancy. The same measure was used in Arvey et al. (2006), who reported detailed information on the psychometric properties of this composite measure. These authors also provide conceptual and empirical support for the validity of this measure to assess leadership role occupancy.

*Socioeconomic status (SES)*

SES was measured as a composite score of the father's occupational status and the mother's level of attained education, which are the two most frequently used indexes of SES (Ensminger & Fothergill, 2003). Education level was assessed as the number of years completed at school, ranging from 0 to 21. Occupational status was measured using the Hollingshead Index of Social Position (Hollingshead, 1975), which rates occupations (rather than jobs) on a 7-point scale where 1 = higher executives, proprietors of large concerns, and major professionals, and 7 = unskilled employees. This occupational status measure was reverse coded so that higher scores represent higher status occupations. The two measures were standardized and then summed to present SES. The coefficient alpha was .85. Similar indicators of SES were used in previous research such as Kennedy (1992) and Otto and Atkinson (1997).

*Perceived parental support*

Perceived parental support was measured using the Parent Environment Questionnaire (PEQ; Elkins, McGue, & Iacono, 1997). The PEQ is a 42-item self-report inventory and was developed for assessing different aspects of parent–child relationships. The parental support scale in PEQ consists of 12 items for father and mother separately, assessing the extent to which the parent–child relationship is characterized by support and closeness. Each item was answered on a 4-point scale (4 = definitely false, and 1 = definitely true). Sample items include "I talked about my concerns and my experiences with my parent" and "My parent comforted me when I was discouraged or have had a disappointment". Responses were scored such that higher scores indicate higher support from the parent. The internal consistency reliability was .91 for father and .88 for mother. The scores for both parents were averaged to represent perceived parental support and the overall internal consistency reliability was .93.

*Perceived conflict with parents*

Perceived conflict with parents was measured using the conflict scale in the PEQ (Elkins et al., 1997) which consists of 12 items for father and mother separately. This scale assesses the extent to which the parent–child relationship is characterized by disagreement, tension, and anger. Sample items include "My parent often criticized me" and "There were often misunderstandings between this parent and myself". Responses were scored such that higher scores indicate a higher level of perceived conflict with the parent. The internal consistency reliability was .91 for father and .90 for mother. The scores for both parents were averaged to represent perceived conflict with parents, with an overall internal consistency reliability of .93.

To ensure that the self-reported measures on perceived parental support and perceived conflict with parents are measuring distinct constructs, we conducted confirmatory factor analysis on the items. To maintain a favorable indicator-to-sample-size ratio, we randomly divided the 12 items for each of the two variables into four parcels before entering them into the CFA. For both mother and father, the hypothesized two-factor models show a better fit ($\chi^2$ = 49.9 and 91.3, *df* = 19 and 19, CFI = .99 and .97, TLI = .98 and .96, RMSEA = .06 and .06 for mother and father, respectively), than the model in which all the items loaded on a single factor ($\chi^2$ = 478.2 and 622.0, *df* = 20 and 20, CFI = .78 and .78, TLI = .68 and .69, RMSEA = .23 and .26 for mother and father, respectively). After father and mother data were combined, the hypothesized four-factor model shows a better fit ($\chi^2$ = 392.8, *df* = 94, CFI = .95, TLI = .94, RMSEA = .07) than the two-factor model in which perceived parental support and conflicts with parents are combined to load onto a factor for mother and another factor for father ($\chi^2$ = 1143.5, *df* = 99, CFI = .77, TLI = .72, RMSEA = .18). Based on these results we believe the two self-reported family environmental variables are distinct from each other.

*Analyses*

All analyses were conducted using the statistical software Mplus (Muthén & Muthén, 1998–2007). The three environmental variables were first examined to assess the genetic influences, if any, on each variable. Most environmentally induced variables also reflect genetic variability (Plomin & Bergeman, 1991), a phenomenon referred to as gene–environment correlations (G–E). G–E correlations may complicate the study of G × E interaction because the existence of G–E correlations makes it difficult to obtain a pure measure of the environment, since the measure itself may be genetically influenced (Rutter et al., 1997). We used a strategy offered by previous researchers to deal with G–E correlations while testing for G × E (Purcell, 2002), i.e., we directly test a bivariate moderation model that incorporates the proposed moderator and the dependent variable.

If an environmental variable is genetically influenced, we use bivariate moderation models (Purcell, 2002) to examine the extent of G–E association between the genetic factors influencing the moderator and the leadership variable. That is, a series of nested models are compared and the most parsimonious model is chosen to examine the necessity of using such bivariate models. The lack of common paths linking the moderator and the dependent variable can ensure that the environmental/moderator variable is independent of the genetic factors associated with the dependent variable. In these cases, the moderating tests based on the more parsimonious univariate moderation models are unbiased (Plomin et al., 1977; Purcell, 2002).

*Univariate analyses on the potential moderators*

We first conducted tests to examine whether the three potential moderators are purely environment-induced. Specifically, we conducted univariate quantitative genetic analyses to estimate the latent genetic components as well as environmental components in the variances of the moderators. The univariate models aim at partitioning the observed between-individual variance in a specific behavioral construct into additive genetic (A), shared environmental (C), and non-shared (E) environmental variances. The non-shared environmental variance represents residual variance not explained by either of the other two sources, along with measurement error. In particular, the equation predicting leadership role occupancy ($T_i$) for twin $i$ in each twin pair is

$$T_i = \mu + aA + cC + eE, \quad (1)$$

where *A, C, E* are independent latent factors with variance 1 and mean zero, *a, c, e* are their corresponding coefficients to be estimated, and $\mu$ represents the intercept. Based on Eq. (1), the variance of $T_i$ is decomposed into three components $a^2$, $c^2$, and $e^2$, and the heritability can be estimated as $h^2 = a^2/(a^2 + c^2 + e^2)$, which represents the proportion of total variance that is due to genetic factors.

In the two-group structural equation models, the genetic factors between the two twins correlate at $r = 1.0$ in the identical twins group because identical twins share 100% of their genes. For the fraternal twins group the genetic factors correlated at $r = .5$ because, on average, fraternal twins share 50% of their genes. The correlations between common environmental factors are, by definition, 1.0 for both groups. The potential moderators were separately analyzed using the univariate model based on Eq. (1).

*Univariate moderation analyses*

The method of variance decomposition in Eq. (1) reflects an averaged estimate on the heritability of a construct because the standard univariate ACE model assumes that the A, C, and E components are fixed over the entire population from which the sample is drawn. Following previous research (e.g., Turkheimer et al., 2003), our moderation analyses incorporated moderators to Eq. (1) such that the *A*, *C*, and *E* effects are made dependent on the moderator variable. The magnitude of the variance components varies as a continuous function of the moderator variable. The equation predicting leadership role occupancy for twin *i* in each twin pair is now

$$T_i = \mu + \beta_M M_i + (a + \beta_X M_i)A + (c + \beta_Y M_i)C + (e + \beta_Z M_i)E, \qquad (2)$$

where $M_i$ represents the moderator variable and $\beta_M$ represents the main effect of the moderator on the mean of the dependent variable; *a*, *c*, and *e* are the main effects; and $\beta_X$, $\beta_Y$, and $\beta_Z$ represent the moderating coefficients for genetic, shared environmental, and non-shared environmental variance, respectively.

Fig. 1 illustrates the conceptual path diagram for the moderated model. Application of standard path-tracing rules to the path model results in the structural equation specified by Eq. (2). The heritability estimate is still the proportion of total variance that is due to genetic factors, but is now a non-linear function of *M*, rather than a population-average estimate for all the participants.

$$h^2|M = \frac{(a + \beta_X M)^2}{(a + \beta_X M)^2 + (c + \beta_Y M)^2 + (e + \beta_Z M)^2}. \qquad (3)$$

To facilitate model convergence, perceived parental support and perceived conflict with parents were transformed to standardized scores before conducting moderation analyses. In all the univariate, bivariate, and moderation analyses, we compared series of nested models in order to choose the best-fitting models (based on varies fit indexes) to examine the moderating effects.

*Bivariate moderation analyses*

If an environmental variable shows significant genetic influence (i.e., a significant $h^2$), we conduct analyses using bivariate moderation models recommended by Purcell (2002). Fig. 2 provides an illustration of the bivariate moderation model in which the moderator variable is decomposed into its genetic and environmental variance components and tested for gene–environment interaction in the presence of gene–environment correlation (Purcell, 2002).

In this model, the paths expressing the genetic and environmental influences on the moderator variable are considered constant. The common paths, rather than $a_M$, $c_M$, $e_M$, are the focus of our investigation. Using the genetic factors as an example, both the common genetic effect between the moderator and leadership and the unique genetic effects on leadership are partitioned into a mean part that is independent of the moderator ($a_c$ and $a_u$, respectively), and a part that is a linear function of the moderator ($\beta_{Xc}M$

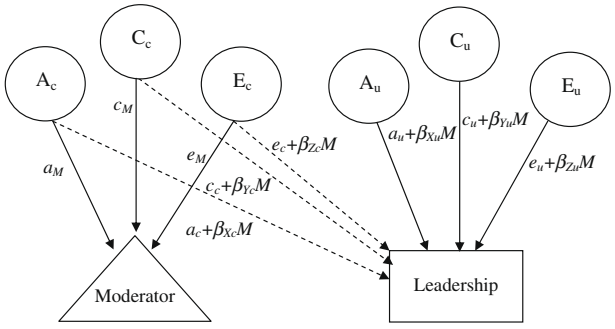


**Fig. 2.** Bivariate moderation model for testing G × E interaction in the presence of G–E correlations (for one twin only). $\beta_X$, $\beta_Y$, and $\beta_Z$ are coefficients of the moderator for the genetic, shared- and non-shared environmental paths, respectively. Subscript *M*, *c*, and *u* refer to the coefficients of the paths associated only with the moderator, those common to the moderator and the leadership variable, and those unique to the leadership variable, respectively.

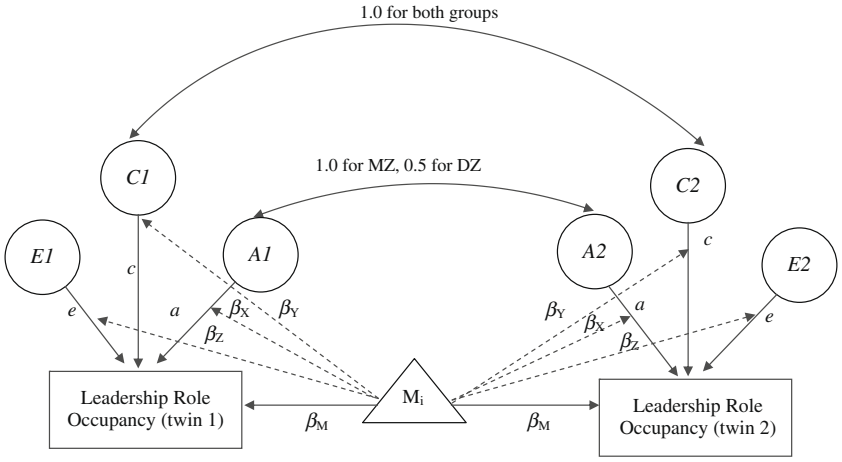


**Fig. 1.** Univariate moderation model for testing G × E interaction. The latent variable A, represented in a circle, indicates additive genetic influences on leadership role occupancy. C represents common (shared) environmental influences, and latent E represents unique environmental influences, which are uncorrelated between the twins. The triangle indicates the mean for the moderator and is necessary when modeling raw data. The standard paths *a*, *c*, and *e*, indicating the magnitude of effect of each latent variable on the dependent variable, each include a $\beta$ term, which indicates the impact of a potential environmental moderator *M* on each of these genetic and environmental influences.

**Table 1**
Means, standard deviations, and individual-level correlations of the variables.

| | *N* | Mean | *SD* | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|---|
| 1. Socioeconomic status | 366 | .00 | .77 | (.85) | | | |
| 2. Perceived parental support | 304 | 3.07 | .56 | .21*** | (.93) | | |
| 3. Perceived conflict with parents | 304 | 1.75 | .54 | −.11* | −.61*** | (.93) | |
| 4. Leadership role occupancy | 366 | .01 | .79 | .02 | .03 | .05 | – |

Reliability is reported along the diagonal in parentheses.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

and $\beta_{XtiM}$, respectively). Similar configurations are specified for the shared and non-shared environmental factors.

The moderator can take the same value or different values for the two twins in a pair (Purcell, 2002). The extent to which the moderator variable moderates the variance in leadership is measured on all six paths. The nested models were derived by successively fixing the moderation and/or main effect coefficients to zero. As Purcell (2002) contends, the genetic effects on the moderator only matters when these genetic factors are shared with the dependent variable under investigation. If the common paths' main effect and moderation coefficients can be fixed to zero without reducing the model fit, the bivariate moderation models can be reduced to the more parsimonious univariate moderation models.

To establish convergent validity, we also conducted analyses on each of the two measures for the composite variable of leadership role occupancy, i.e., leadership positions at work and leadership positions in work-related associations. Highly similar results were found for these two indicators as compared with results for the composite measure. We thus only report the results for the composite measure below.

## Results

Table 1 provides the sample sizes, means, standard deviations, and individual-level correlations of the variables of this study. Perceived parental support was significantly correlated with perceived conflict with parents ($r = -.61$, $p < .001$). None of the three environmental variables was significantly correlated with leadership role occupancy. The absence of significant correlations between the leadership variable and the environmental moderators provides preliminary support for the lack of confounding effects from gene–environment correlations.

Table 2 provides a comparison between MZ twins and DZ twins on the key variables in this study. MZ twins do not differ from DZ twins in terms of the mean levels on SES, perceived parental support, perceived conflict with parents, and leadership role occupancy. Levene's tests show that MZ and DZ twins have equal variances on the four variables. To further explore the data structure, we calculated twin correlations for the leadership variable at different levels of the three moderators. High and low conditions of a particular moderator were determined as above and below the mean, respectively. As shown in Table 3, the difference between MZ and DZ correlations are larger when SES is low, perceived parental support is low, or perceived conflict with parents is high. These correlations provide preliminary results on the moderating effects of the three moderator variables.

Before we formally test the moderation effects, we estimated whether the environmentally induced moderator variables (perceived parent support and perceived conflict with parents) are free of genetic influences and then examined potential confounding effects of G–E correlations. Since the family socioeconomic status variable has the same value for the two twins in a pair, the SES variable cannot have any genetic influence. Thus, SES is excluded from the analysis of genetic influence on the moderator variables. A series of nested model testing was conducted, and the best-fitting model was chosen based on chi-square change and a variety of fit indexes. As Table 4 shows, AE models fit the data for perceived parental support and perceived conflict with parents. The genetic components for the perceived parental support variable and perceived conflict with parents variable were estimated at .64 and .56, respectively, both with 95% confidence intervals excluding zero. Thus, we found significant genetic influences on perceived parental support and perceived conflict with parents. Based on these findings, we then proceed to conduct bivariate moderation analyses on the effects of perceived parental support and perceived conflict with parents on leadership role occupancy. It is notable that Table 4 also reports the univariate model estimates on leadership role occupancy based on the current data set. Similar results have been reported in Arvey et al. (2006) using a slightly larger sample of the twins.

Bivariate moderation analyses were conducted to examine the extent of G × E interactions in the potential presence of G–E correlations. Table 5 reports the model fit for a series of nested models for both moderators. In Model 3 for perceived parental support, the moderation and main effect coefficients for the common A, C, and E paths can be fixed at zero without causing worse fit as compared to the full model. Thus, the bivariate moderation model can be reduced to a univariate moderation model without biasing the results. This univariate A, C, and E moderation model can be further reduced to a model with only A and E paths (Model 5). Similar results were obtained for perceived conflict with parents. Consequently, although perceived parental support and perceived conflict with parents are genetically influenced, there is little evidence for G–E correlation between the genetic factors that

**Table 2**
Comparison between MZ and DZ twins.

| Variable | Means | | | *SD* | | |
|---|---|---|---|---|---|---|
| | MZ | DZ | *t*-Value | MZ | DZ | Levene's test on equal variances (*F*-value) |
| Socioeconomic status | .09 | −.05 | 1.79 | .75 | .75 | .05 |
| Perceived parental support | 3.09 | 3.02 | 1.12 | .56 | .55 | .001 |
| Perceived conflict with parents | 1.70 | 1.82 | −1.91 | .53 | .55 | 1.12 |
| Leadership role occupancy | .06 | −.03 | 1.17 | .79 | .78 | 1.04 |

None of the *t*-values or *F*-values were significant at $p < .05$. MZ and DZ refer to monozygotic and dizygotic twins, respectively.

**Table 3**
Twin correlations for the leadership variable at difference levels of the moderator.

| Moderator | # of pairs (MZ/DZ) | Twin correlations | |
|---|---|---|---|
| | | MZ | DZ |
| *Socioeconomic status* | | | |
| Low | 55/36 | .19 | .05 |
| High | 48/31 | .40 | .29 |
| *Perceived parental support (within-pair average)* | | | |
| Low | 34/28 | .36 | .18 |
| High | 55/26 | .22 | .12 |
| *Perceived conflict with parents (within-pair average)* | | | |
| Low | 57/25 | .13 | .15 |
| High | 32/29 | .51 | .06 |

High and low conditions were above and below the mean, respectively, for the particular moderator under study. We computed twin correlations using intraclass correlation coefficients (ICC[1]).

predict these two variables and the genetic factor predicting leadership role occupancy. We used the more parsimonious univariate moderation models for the subsequent moderating analyses concerning the effects of perceived parental support and perceived conflict with parents.

Table 6 provides the fit indexes for the univariate moderation models for each of the moderator variables. Similar to the nested models comparison in Table 4, we conducted model comparisons among a series of nested moderation models. The full ACE model was based on Eq. (2). In the CE models the path coefficients $a$ and $\beta_X$ were fixed at zero, and in the AE models the path $c$ and $\beta_Y$ were fixed at zero. As Table 6 shows, the AE moderation models exhibit the best fit for each of the three moderating variables. In particular, the −2 Log-likelihood change between the ACE model and the CE model was 9.23 ($\Delta df = 2$, $p < .01$), 12.06 ($\Delta df = 2$, $p < .001$), and 64.33 ($\Delta df = 2$, $p < .001$) for perceived parent support, perceived conflict with parents, and SES, respectively. The AE models show insignificant changes in −2 Log-likelihood and demonstrate the lowest values for AIC and sample-size adjusted BIC.

To ensure that our findings are not the results of non-normality or some extreme data points in our dependent variable, we used two methods to transform the leadership variable and re-ran the analyses. First, we log-transformed the leadership variable so that the transformed variable shows less skewness (skewness = .05). Second, and independent from the first method, we trimmed the leadership variable so that data points beyond the range of ±2 *SD* were set to the boundary values. Moreover, bias-corrected bootstrapping (with 500 replications) was used in all the analyses to provide 95% confidence intervals for the estimates, which is an advantageous method compared to the normality-based confidence intervals (e.g., Taylor, MacKinnon, & Tein, 2008). These two approaches provided results highly similar to those based on our original operationalization, showing the robustness of the results.

**Table 4**
Estimates and fit indexes for the univariate models on perceived parental support, perceived conflict with parents, and leadership.

| Variables | Sample size[a] | Model | Variance components | | | Fit indexes | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | *A* | *C* | *E* | $\chi^2$ (*df*) | $\Delta\chi^2$ | CFI | TLI | RMSEA |
| Perceived parental support | 96/56 | ACE | .64 (.52, .75) | .00 (.00, .11) | .36 (.25, .48) | 2.02 (6) | – | 1.00 | 1.03 | .00 |
| | | CE | – | .50 (.38, .62) | .50 (.38, .62) | 12.68 (7) | 10.66*** | .89 | .97 | .10 |
| | | AE [c] | .64 (.52, .75) | – | .34 (.25, .48) | 2.02 (7) | .00 | 1.00 | 1.03 | .00 |
| | | E | – | – | 1.0 (1, 1) | 53.96 (8) | 51.96*** | .08 | .77 | .26 |
| Perceived conflict with parents | 96/56 | ACE | .56 (.42, .69) | .00 (.00, .03) | .44 (.31, .58) | 12.35 (6) | – | .82 | .94 | .08 |
| | | CE | – | .41 (.28, .55) | .59 (.45, .72) | 19.65 (7) | 7.3** | .63 | .90 | .14 |
| | | AE [c] | .56 (.42, .69) | – | .44 (.31, .58) | 12.35 (7) | .00 | .85 | .96 | .05 |
| | | E | – | – | 1.0 (1, 1) | 46.31 (8) | 33.96*** | .00 | .72 | .23 |
| Leadership role occupancy[b] | 109/74 | ACE | .32 (.17, .47) | .00 (.00, .06) | .68 (.53, .82) | 8.04 (6) | – | .86 | .95 | .06 |
| | | CE | – | .25 (.12, .38) | .75 (.63, .88) | 11.24 (7) | 3.2 | .78 | .93 | .07 |
| | | AE [c] | .32 (.18, .47) | – | .68 (.53, .82) | 8.04 (7) | .00 | .93 | .98 | .04 |
| | | E | – | – | 1.00 (1, 1) | 23.88 (8) | 15.84*** | .00 | .73 | .14 |

* $p < .01$.
** $p < .01$.
*** $p < .001$.
[a] Sample size is expressed as number of MZ/DZ twin pairs. Modeling fitting was based on variance–covariance matrices between the two twins of a pair.
[b] The results on leadership role occupancy have been reported in Arvey et al. (2006) using a slightly larger sample.
[c] Indicates the best-fitting model.

**Table 5**
Fit statistics for the bivariate moderation models.

| Moderator | −2LL (*df*) | $\Delta\chi^2$ ($\Delta df$) | *p*-Value | AIC | Sample-size adjusted BIC |
|---|---|---|---|---|---|
| *Perceived parental support* | | | | | |
| (1) All parameters free | 1384.88 (551) | – | – | 282.88 | 198.81 |
| (2) Fix common A, C, and E moderation coefficients: $\beta_{Xc}$, $\beta_{Yc}$, and $\beta_{Zc}$ | 1386.42 (554) | 1.54 (3) | .67 | 278.42 | 196.89 |
| (3) Fix common A, C, and E main effect coefficients: $a_c$, $c_c$, and $e_c$ | 1386.43 (557) | 1.55 (6) | .96 | 272.43 | 194.21 |
| (4) Fix unique C moderation coefficient $\beta_{Yu}$ | 1386.44 (558) | 1.56 (7) | .98 | 270.44 | 193.32 |
| (5) Fix unique C main effect coefficient $c_u$ | 1386.44 (559) | 1.56 (8) | .99 | 268.44 | 192.42 |
| *Perceived conflict with parents* | | | | | |
| (1) All parameters free | 1347.75 (551) | – | – | 245.75 | 180.24 |
| (2) Fix common A, C, and E moderation coefficients: $\beta_{Xc}$, $\beta_{Yc}$, and $\beta_{Zc}$ | 1348.58 (554) | .83 (3) | .84 | 240.58 | 177.97 |
| (3) Fix common A, C, and E main effect coefficients: $a_c$, $c_c$, and $e_c$ | 1351.41 (557) | 3.66 (6) | .72 | 237.41 | 176.70 |
| (4) Fix unique C moderation coefficient $\beta_{Yu}$ | 1351.41 (558) | 3.66 (7) | .82 | 235.41 | 175.80 |
| (5) Fix unique C main effect coefficient $c_u$ | 1351.41 (559) | 3.66 (8) | .89 | 233.41 | 174.91 |

−2LL = −2 log likelihood; fixing a coefficient means to constrain the term as zero. A particular model is nested within the one immediately above it.

**Table 6**
Fit indexes of the univariate moderation models on leadership role occupancy.

| Moderator variable | Sample size (MZ/DZ pairs) | Model | Fit indexes | | | | |
|---|---|---|---|---|---|---|---|
| | | | −2LL | Δ-2LL (Δ*df*) | *p*-Value | AIC | Sample-size adjusted BIC |
| Socioeconomic status | 103/67 | ACE | −995.08 | – | – | 2006.16 | 2005.91 |
| | | CE | −1059.41 | 64.33 (2) | .000 | 2130.82 | 2130.64 |
| | | AE [a] | −995.08 | .00 (2) | 1.00 | 2002.16 | 2001.97 |
| Perceived parental support | 89/54 | ACE | −729.19 | – | – | 1474.39 | 1472.78 |
| | | CE | −738.42 | 9.23 (2) | .01 | 1488.83 | 1487.62 |
| | | AE [a] | −729.19 | .00 (2) | 1.00 | 1470.39 | 1469.18 |
| Perceived conflict with parents | 89/54 | ACE | −671.36 | – | – | 1358.72 | 1357.11 |
| | | CE | −683.42 | 12.06 (2) | .001 | 1368.85 | 1367.64 |
| | | AE [a] | −671.36 | .00 (2) | 1.00 | 1354.72 | 1353.52 |

[a] Indicates the best-fitting models. The ACE model was based on Eq. (2). In the CE model, both $a$ and $\beta_X$ were fixed at zero. In the AE model, both $c$ and $\beta_Y$ were fixed at zero.

**Table 7**
Estimates based on the best-fitting univariate moderation models.

| Moderator variable | Sample size (MZ/DZ pairs) | Path coefficient estimates (95% CI) | | | | |
|---|---|---|---|---|---|---|
| | | $a$ | $\beta_X$ | $e$ | $\beta_Z$ | $\beta_M$ |
| Socioeconomic status | 103/67 | .35 (.21, .51) | −.39 (−.70, −.12) | .74 (.61, .90) | .49 (.02, .59) | −.04 (−.16, .08) |
| Perceived parental support | 89/54 | .30 (.22, .47) | −.29 (−.48, −.05) | .68 (.56, .81) | .35 (.05, .47) | .01 (−.12, .10) |
| Perceived conflict with parents | 89/54 | .43 (.21, .57) | .26 (.10, .46) | .58 (.46, .77) | −.03 (−.14, .12) | .09 (−.01, .20) |

The path coefficient estimates based on the AE moderation models are reported in Table 7. Significant values for $\beta_X$ reveal substantial moderating effects of the moderator on the genetic influences on leadership. As Table 7 shows, SES significantly and negatively moderates the genetic influence on leadership role occupancy ($\beta_X = -.39$; 95% bias-corrected bootstrapped confidence intervals: −.70 to −.12). To facilitate the interpretation of the results, we report the model-predicted genetic and non-shared environmental influences on leadership under three conditions of SES (see Table 8). In particular, when SES takes the value of −1.0 *SD*, the mean, and +1.0 *SD*, the corresponding genetic influence is .76, .18, and .01, respectively.

Fig. 3 shows the moderating effects of SES on the genetic and non-shared environmental variances of the leadership variable. To derive the genetic component of the leadership variable at different levels of the moderator, we generated random values of the moderator within ±1.0 *SD* from its mean, and entered these values in Eq. (3) along with the path coefficient estimates from Table 7 (i.e., $a$, $\beta_X$, $e$, and $\beta_Z$). The environmental component of the variance of leadership role occupancy was calculated in an equation with the environmental variance, rather than genetic variance, as the numerator. The genetic and environmental components of the variance of the leadership variable were then plotted against different levels of SES within ±1.0 *SD* from its mean. As Fig. 3 shows, with the increase of SES (which is a standardized variable), the genetic effects on leadership decrease and the non-shared-environmental effect increase.

Perceived parental support also negatively moderates the genetic influence on leadership role occupancy ($\beta_X = -.29$; 95% bootstrapped CI: −.48 to −.05, see Table 7); this result indicates that the heritability of leadership role occupancy is greater in magnitude for individuals who perceived having lower perceived parental support, as shown in Table 8. Fig. 4 illustrates the moderating effect of perceived parental support on the genetic and environmental influences on leadership role occupancy. Similar to the graph for SES, the genetic and environmental components of the

**Table 8**
Model-predicted estimates of genetic and environmental influences, as a function of the moderator variable.

| | Proportion of variance in leadership | |
|---|---|---|
| | Genetic | Non-shared environment |
| No-moderation model (population-average estimates) | .32 | .68 |
| *SES as the moderator* | | |
| −1 *SD* | .76 | .24 |
| Mean | .18 | .82 |
| 1 *SD* | .01 | .99 |
| *Perceived parental support as the moderator* | | |
| −1 *SD* | .76 | .24 |
| Mean | .16 | .84 |
| 1 *SD* | .01 | .99 |
| *Perceived conflict with parents as the moderator* | | |
| −1 *SD* | .07 | .93 |
| Mean | .35 | .65 |
| 1 *SD* | .61 | .39 |

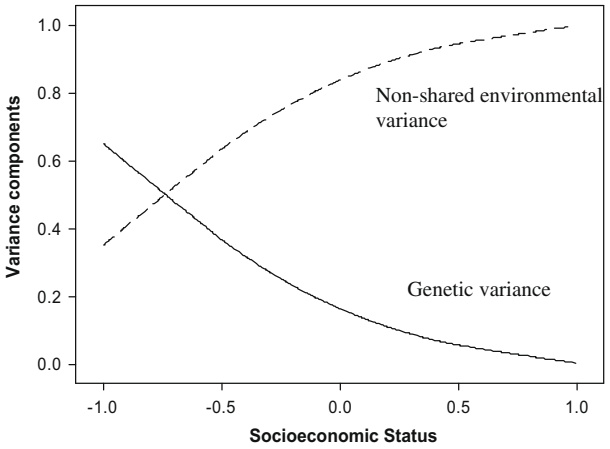


**Fig. 3.** Variance in leadership role occupancy as a function of family socioeconomic status, by source of variance. Socioeconomic status is a standardized variable with mean = 0 and *SD* = 1.0.

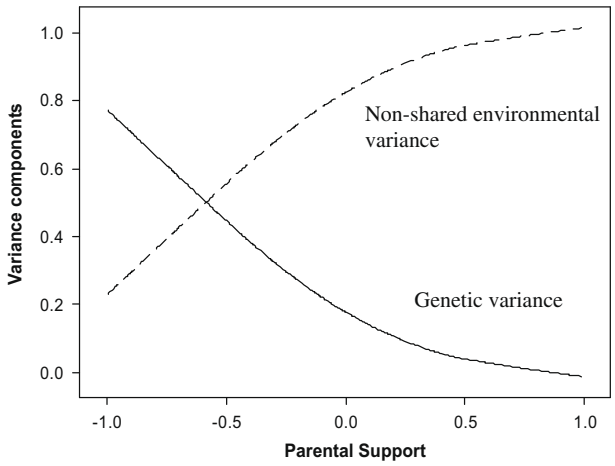


**Fig. 4.** Variance in leadership role occupancy as a function of perceived parental support, by source of variance. Perceived parental support is a standardized variable with mean = 0 and *SD* = 1.0.

variance of leadership role occupancy were then plotted against different levels of perceived parental support within ±1.0 *SD* from its mean. As Fig. 4 shows, with the increase of perceived parental support (a standardized variable), the genetic effects decrease and the non-shared-environmental effects increase.

Perceived conflict with parents positively moderated the genetic effect on leadership role occupancy ($\beta_X$ = .26; 95% CI: .10 to .46). Table 8 and Fig. 5 show the moderating effect of the conflict variable within the range of one standard deviation from the mean. With the increase of perceived conflict with parents, the genetic effects on the leadership variable increase and the non-shared-environmental effects decrease.

## Discussion

Our investigation into the role of social environmental factors in explaining the magnitude of genetic influences on leadership role occupancy led to consistent findings supporting the view that the presence of adversity and conflict facilitates the greater influence of genetic leadership potential. First, family SES, as an indirect measure of the quality of the environment during adolescence (indicating availability of resources), negatively moderated the genetic effects on leadership role occupancy, such that the genetic effects were higher for those from low-SES families. Second, perceptions of parental support and conflict with parents, as subjective indicators of the social/family environment in adolescence, also had significant moderating effects on the heritability of leadership role occupancy (negative for perceived parental support and positive for perceived conflict with parents), such that the genetic effects were higher for those reporting lower levels of perceived parental support and higher levels of perceived conflict with parents.

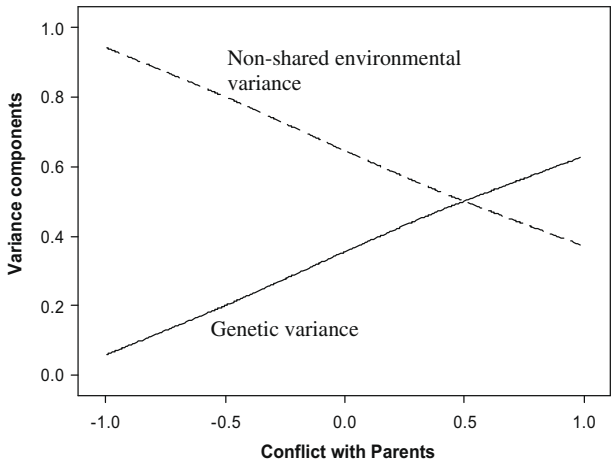


**Fig. 5.** Variance in leadership role occupancy as a function of perceived conflict with parents, by source of variance. Perceived conflict with parents is a standardized variable with mean = 0 and *SD* = 1.0.

Although much remains to be understood, our study shows that the economic and social characteristics of the environments that individuals experience in adolescence have important influences on the magnitude of genetic influences on leadership manifested later in life. The relative importance of genetic versus environmental factors in causing differences in observed leadership role occupancy appears to vary with SES, perceived parental support, and perceived conflict with parents. More enriched parental environments—characterized by higher SES, higher levels of perceived parental support, and lower perceived conflict with parents—were associated with a lower heritability of leadership role occupancy.

As noted, these findings are consistent with the conceptual argument proposing that the presence of adversity and conflict allows for a greater influence of genetic differences in capabilities related to leadership, and it is inconsistent with previous research on the moderating effect of characteristics of the early environment on intelligence (e.g., Turkheimer et al., 2003), which found that enriched family environments led to higher heritability on intelligence. We believe these contrasting results indicate the substantial differences between these two constructs. Based on 65 studies, Judge, Colbert, and Ilies (2004, p. 546) only found a low true correlation between paper-and-pencil intelligence and leadership emergence ($\rho$ = .19, uncorrected for range restriction, and $\rho$ = .25 corrected for range restriction). Whereas leadership skills can be taught and leadership potential can be developed, intelligence is believed to be much less malleable than leadership (e.g., Sternberg & Kaufman, 1998). Moreover, leadership skills are by nature interpersonal; thus, learning and developmental interventions are important for individuals to develop these skills. Intelligence, on the other hand, is an intrapersonal ability construct, and exhibits a high degree of between-individual stability across the life span (e.g., Cardon, Fulker, DeFries, & Plomin, 1992). For example, Deary, Whalley, Lemmon, Crawford, and Starr (2000) and Larsen, Hartmann, and Nyborg (2008) found that the between-individual differences in intelligence have substantial stability from childhood to late life. The correlation between two test scores at age 11 and age 77 was .63 (Deary et al., 2000), and the correlations between intelligence measures accessed 18 years apart ranged from .79 to .85 (Larsen et al., 2008).

### *Theoretical and practical implications*

The findings in this study suggest that a model in which leadership variance is merely partitioned into components attributable to genes and environmental effects lacks the capability to examine the dynamic interaction of genetic effects and developmental environments. As this study shows, examining G × E interactions can pinpoint the moderating relationships and show how the social environment interacts with genetic predispositions. When an individual lived in a family with higher parental support or lower conflict with parents, his/her opportunities of becoming a leader in the workplace were determined more by environmental factors rather than by genetic factors. In this sense, it is possible that creating more enriched environments for adolescents "levels the playing field", at least to some extent, such that whether one was born

with the "right" attributes for leadership matters less in the enriched environments.

In the current study, we focus on the interaction between genes and the environment. We neither predict nor find any main effects of the environmental variables on leadership role occupancy at work, and the correlations between the environmental variables and leadership were not significant. The absence of these main effects can mitigate the concerns on the confounding effects of G–E correlations.

These findings on G × E interaction on leadership have significant implications for leadership training and development in organizations. This study suggests that leadership development efforts in earlier life can matter even though leadership is influenced by genetic factors. It is known, for example, that transformational leaders can be developed to some extent (Barling, Weber, & Kelloway, 1996). Our findings suggest that early development interventions are likely to be more successful when they are accompanied by a resource-rich, supportive social environment. Nevertheless, it remains to be seen if development programs implemented later in life (at work) are more successful for those who experienced more enriched environments in adolescence, which would indicate that providing increased support and opportunities for adolescents may lead to more successful leadership development interventions later at work.

This study has several limitations. First, the self-reported measures of leadership role occupancy and perceived parental environment in adolescence may share common method variance. However, this concern is somewhat alleviated by the time lag among the measures (i.e., perceived parental environment measures were collected three years before the leadership data) and the method of computing the heritability of leadership role occupancy (based on correlations between independent reports of twins). In addition, the fact that the main objective of the study was to uncover the moderating effects on heritability estimates should further relieve concerns about this potential common rater problem.

Second, the subjective measures on family environments in adolescence may be subject to retrospective bias. Because holding more or less leadership roles is not likely to affect a person's memory retrieval with regard to their parental environments during adolescence, we believe any bias or inaccuracy in the retrospective measures of family environment would actually reduce the power of finding G × E interactions. Thus, the potential inaccuracies in memory retrieval may make our results conservative. Third, the study utilized white male twins born between 1961 and 1964. While narrowing down the age range and focusing on males could help to partial out the confounding effect of age and gender, the degree to which these findings generalize to females or other racial groups is unknown. These results need replication to affirm the stability of the relationships observed before we can generalize the results to a broader population.

Fourth, while we utilized ratings of objective characteristics of the family SES (i.e., father's occupation and mother's education), our measures of parental support and conflict with parents for the twins are inherently perceptual. Rater effects are probably responsible, in part, for the presence of genetic effects on these two "environmental" variables. Given the fact that our results suggest that G–E correlation did not substantially influence the G × E results, and that our analyses controlled for the main effects of "E" variables (and thus for their genetic component), we believe our results are informative in terms of demonstrating differential genetic influences under different environments. Similar measures on perceived environment and the same analytical approach have been used in prior studies on G × E interactions (e.g., perceived marital quality, South & Krueger, 2008). In addition, although the bootstrapping approach we used provided confidence interval estimates that are free of distribution assumptions, our dependent variable was not measured on an interval scale, which may reduce the variability of the measure. Future research needs to use interval-scale measures that fit with analytical methods based on normal distribution assumptions. Moreover, in replicating the results, future research needs to utilize larger samples of twins that provide increased statistical power to detect G × E interactions in the presence of G–E correlations.

This study's limitations should be considered vis-à-vis its innovativeness and contributions. This study attests to the importance of the adolescent social environment in the development of leadership potential later in life and is the first study to consider the role of the earlier developmental environment in influencing the genetic effect on leadership role occupancy at work. The results showed that workplace leadership role occupancy is less affected by genetic factors, and more affected by the environment, for those who had enriched and supportive environments. Early developmental factors, leading to favorable, opportunity-rich environments, may be especially important to the success of those who are not equipped with natural leadership endowments, by allowing them to more effectively capitalize on opportunities later in life.

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