Genetic influences on core self-evaluations, job satisfaction, and work stress: A behavioral genetics mediated model

Timothy A. Judge [a,*], Remus Ilies [b], Zhen Zhang [c]

[a] Mendoza College of Business, University of Notre Dame, Notre Dame, IN 46556, United States
[b] Department of Management and Organization, National University of Singapore, Singapore
[c] W. P. Carey School of Business, Arizona State University, Tempe, AZ 85287, United States

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ABSTRACT

In this study we investigated the mediated influence of core self-evaluations (CSE) on employee health problems via job satisfaction and work stress, and the degree to which genetic factors explain these mediated relationships. Based on data obtained from a sample of 594 Swedish twins (114 monozygotic twin pairs and 183 dizygotic twin pairs), conventional path analysis results supported the mediated effects of CSE on employee health via job satisfaction and work stress, after controlling for conscientiousness and extraversion. Behavioral genetic analyses showed significant heritability of all four variables. Moreover, we found that the mediated relationships via job satisfaction and work stress are explained by genetic factors, such that the genetic source of job satisfaction and work stress mediates the genetic influence of CSE on health problems. These results highlight the role played by genetic factors in better understanding the relationships between CSE, work attitudes, and health outcomes.

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Introduction

If general research areas can be evaluated based on the importance of their contributions, in the past quarter-century, arguably no area would rank ahead of behavioral genetics. An early finding from behavioral genetics research is now unsurprising: That enduring individual differences such as intelligence and personality are substantially (though not completely) heritable (Bouchard, 2004). From this base, researchers found that many presumably contextualized psychological variables are heritable to varying degrees, including outcomes both socially desirable [e.g., exercise participation (Bryan, Hutchison, Seals, & Allen, 2007), second language acquisition (Dale, Harlaar, Haworth, & Plomin, 2010), perceived social support (Bergeman, Neiderhiser, Pedersen, & Plomin, 2001), mental health (Keyes, Myers, & Kendler, 2010)] and undesirable [e.g., smoking (Boardman, Blalock, & Pampel, 2010), drug use (Haberstick et al., 2011), negative attitudes toward homosexuals (Verweij et al., 2008), psychiatric disorders (Khan, Jacobson, Gardner, Prescott, & Kendler, 2005)]. Indeed, genetic effects are so strong and pervasive that the proposition that *all human characteristics are heritable* has been labeled by Turkheimer (2000) as the First Law of Genetics. Taking account of the insights produced by behavioral genetics research, Johnson, Turkheimer, Gottesman, and Bouchard (2009) concluded, "By now we have a fundamental understanding that genetic influences are involved in all aspects of psychology and behavior."

Not surprisingly, organizational psychology and behavior has been affected by, and has contributed to, this body of research. Studies by Arvey, Bouchard, and colleagues identified genetic sources of central work criteria, including job satisfaction (Arvey, Bouchard, Segal, & Abraham, 1989; Arvey, McCall, Bouchard, Taubman, & Cavanaugh, 1994), work values (Keller, Bouchard, Arvey, Segal, & Dawis, 1992), job and occupational switching (McCall, Cavanaugh, Arvey & Taubman, 1997), entrepreneurship (Zhang et al., 2009), and leadership emergence (Arvey, Rotundo, Johnson, Zhang, & McGue, 2006). Other researchers have investigated the heritability of additional organizational concepts: perceptions of organizational climate (Hershberger, Lichtenstein, & Knox, 1994) and vocational interests (Lykken, Bouchard, McGue, & Tellegen, 1993). Cumulatively, these studies have dovetailed with the broader behavioral genetics literature in revealing that, to a substantial degree, organizational attitudes and behavior are heritable.

The contributions and significant impact of these studies notwithstanding, one important area for further development in the organizational behavior literature are models which may explain these genetic effects. As noted by Ilies, Arvey, and Bouchard (2006), "Progress in understanding the role of genetic differences has been rather slow-paced" (p. 126). Why is it so important to explain genetic sources of organizational behavior variables? Most

* Corresponding author. Fax: +1 352 392 6020.
E-mail address: tjudge@nd.edu (T.A. Judge).

fundamentally, such inquiry advances understanding of the real (vs. apparent) associations among organizational concepts. When a model among concepts is properly articulated and tested, it has the ability to test the degree to which apparently situational mediational relationships at the phenotypic level in fact result from underlying genetic and shared environmental influences.

Specifically, if we observe an association between a personality trait, a work attitude, and an outcome or criterion variable, it is possible that the interpretations we make about the nature and meaning of such a meditational relationship can only be properly understood once we consider the degree to which these relationships are due to genetic effects, environment effects, or both. If genetic effects predominantly explain the associations, it suggests a different causal association than typically assumed. In such a case, it is not that a work attitude or perception causes an outcome in the way most organizational behavior researchers assume, but, rather, that genetic differences lead individuals to hold the attitude and experience the outcome. As noted by O'Connor, Caspi, DeFries, and Plomin (2000), connections between individual differences in adjustment and many outcomes that were previously thought to be explained entirely by environmental differences are now thought to be substantially explained by genetic differences. Under these circumstances, as noted by Neiss, Rowe, and Rodgers (2002), "The apparent phenotypic mediation is spurious on other sources of individual differences" (p. 273).

The only such mediational efforts in organizational behavior research have been studies conducted by Ilies and colleagues. Relying on meta-analytic data, Ilies and Judge (2003) found that positive and negative affectivity better explained genetic sources of job satisfaction than the Big Five traits. Using a similar approach, Ilies, Gerhardt, and Le (2004) found that general mental ability and the Big Five traits explained some of the heritability of leadership emergence. While these studies contributed to our understanding of the degree to which genes underlie associations between personality and work outcomes, as the authors note, these studies were limited by the data on which they are based and in the assumptions they make. Ilies et al. (2006) note, "The limitations of the Ilies and Judge (2003) method can be avoided by using primary twin or familial data to investigate mediated genetic effects" (p. 133). They also note, "Specific operational models explaining the mechanisms through which genetics influence certain organizational outcomes can and should be developed and tested" (Ilies et al., 2006, p. 135).

Accordingly, the purpose of the present study is to develop and test a model linking a personality trait (core self-evaluations) to an important outcome (employee health problems) as mediated by two work variables—job satisfaction and work stress. While we test an overall model of the relationships among these variables, the heart of the intended contribution of this study is to shed light on the degree to which the relationships among the variables in the model are genetic in nature. Uncovering genetic bases for mediated relationships among perceptions of the work environment and outcomes provides support for the "nature of nurture" perspective (Butcher & Plomin, 2008; Plomin & Asbury, 2005), and suggests different theoretical and practical implications than those typically assumed. In the next section of the paper, we introduce the model, and then develop hypotheses for the core linkages within the model.

Model and hypotheses

The hypothesized path model appears in Fig. 1. Some links in the model, and some of the underlying sources of variance in the variables, are assumed rather than formally hypothesized. Because the link between job satisfaction and work stress may be reciprocal (Judge, Boudreau, & Bretz, 1994), we do not specify a causal
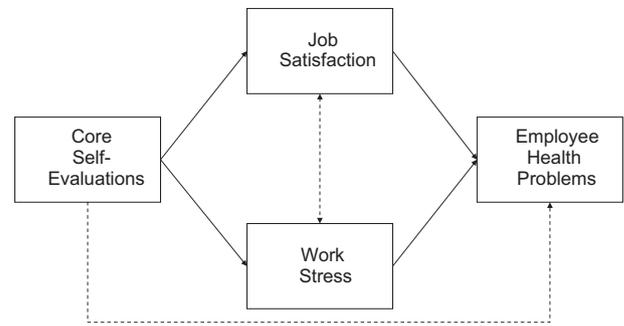

Fig. 1. Hypothesized path model.

direction, but instead assume and stipulate a non-causal link between the two variables. In addition, we do not formally hypothesize a genetic source of variance in the four variables in the path model, though we do test for heritability in subsequent behavioral genetics models given that it is a necessary condition for some hypotheses that follow. Concerning the heritability of CSE, though only one previous study showed heritability of a measure of CSE for a sample of female twins (Zhu & Arvey, 2006), several studies have supported the genetic basis of some of the individual core traits, namely neuroticism (Jang, Livesley, & Vernon, 1996) and self-esteem (Kendler, Gardner, & Prescott, 1998; Neiss, Sedikides, & Stevenson, 2006). As for job satisfaction, several studies by Arvey and colleagues (Arvey et al., 1989, 1994) have found measures of job satisfaction to be heritable. We are not aware of any evidence on the heritability of measures of job or work stress. However, there is ample reason to believe that work stress is heritable, too. Autonomic reactions to stressors – such as elevated heart rate, blood pressure, and galvanic skin response – are substantially heritable (Lensvelt-Mulders & Hettema, 2001), as are putative causes, such as stressful life events (Kendler & Baker, 2007). Moreover, Federenko et al. (2006) found significant heritability ($h^2 = .30$) for a measure of perceived stress. Thus, though direct evidence is lacking, evidence indirectly supports an expectation that work stress is heritable. Finally, it is of no surprise—given a voluminous body of research showing substantial heritabilities for nearly every health condition (Johnson & Krueger, 2005)—to expect that health problems are heritable.

Regular path modeling hypotheses

The hypothesized model, in its phenotypic (traditional path-analytic) form, is relatively straightforward. Each link in the model has been supported by past research. Judge and Bono's (2001) meta-analysis revealed that each of the core self-evaluations traits is positively related to job satisfaction, and that in all cases the confidence intervals overlapped – meaning that these positive relationships were indistinguishable, as predicted by the framework. Moreover, studies utilizing direct measures of core self-evaluations have shown equally consistent relationships with job satisfaction (Brown, Ferris, Heller, & Keeping, 2007; Judge, Erez, Bono, & Thoresen, 2003). Compared to job satisfaction, there is considerably less research on the relationship of core self-evaluations to work stress, but the literature suggests a significant, negative relationship. Brunborg (2008) and Kluemper (2008) found that core self-evaluations was negatively correlated with perceptions of job stress. Thus, the extant literature supports links of core self-evaluations with job satisfaction and with work stress.

Similarly, the associations of job satisfaction and work stress with health problems are well documented in the literature. In terms of work stress, numerous studies have found a positive

relationship between work or job stress and health problems (e.g., Glomb et al., 1997; Grzywacz et al., 2007). In their classic review, Beehr and Newman (1978) note that the very nature of stress itself produces physiological changes (e.g., levels of catecholamine, glucose, and cortisol in the blood; increases in heart rate and blood pressure, etc.) that precipitate health problems (e.g., gastro-intestinal disorders, coronary heart disease, asthmatic attacks, etc.). There is even evidence that stress is associated with susceptibility to infectious diseases (Cohen & Williamson, 1991). Psychologically, Christie and Barling (2009) argued that the positive relationship between work stress and health problems is explained by depletion of resources, such that when individuals perceive and respond to stress at work, they use up their bank of resources which, in turn, compromises their ability to cope with the stressors or other heath risk factors.

Comparatively less research has linked job dissatisfaction to health problems, though the available literature suggests that a link is there (Cass, Siu, Faragher, & Cooper, 2003). Utilizing a panel design, Fischer and Sousa-Poza (2009) found that job satisfaction was associated with later measures of objective health conditions. As noted by Rosse and Hulin (1985), job dissatisfaction may have negative health consequences because it reflects an inability to adapt to one's work role. More generally, the subjective well-being literature suggests that while health affects life satisfaction (Diener, 1984), satisfaction may stimulate engagement in positive health behaviors that later affect health outcomes (Grant, Wardle, & Steptoe, 2009), and positive emotional states have positive effects on health indicators (faster cardiovascular recovery, reduced inflammation, resilience to infection) and health outcomes (Pressman & Cohen, 2005).

The final link to be discussed in the model—between core self-evaluations and health—has been tested less often. Several studies have found that core self-evaluations is negatively associated with burnout (Best, Stapleton, & Downey, 2005; Laschinger & Finegan, 2008). Tsaousis, Nikolaou, Serdaris, and Judge (2007) found that core self-evaluations was significantly negatively related to both physical and psychological health problems. Even less studied are explanations for why core self-evaluations may be associated with better health, or fewer health problems. Given the links of core self-evaluations with job satisfaction and work stress previously noted, and of these latter constructs with health problems, it seems likely that at least part of the association between core self-evaluations and health problems is explained by job satisfaction and work stress.

H1. Core self-evaluations (H1a) and job satisfaction (H1b) are negatively related to employee health problems, and work stress (H1c) is positively related to employee health problems.

H2. Job satisfaction (H2a) and work stress (H2b) partially mediate the relationship between core self-evaluations and employee health problems.

Behavioral genetics hypotheses

In linking the one personality variable and the two work variables to employee health problems, the heart of this study concerns the underlying causes of these relationships. Our general thrust here is that to understand these relationships, or relationships among organizational behavior variables more broadly, one must investigate their genetic origins. As Bouchard (2004, p. 148) noted: "A simple answer to the question of why scientists study genetic influences on human behavior is that they want a better understanding of how things work, that is, better theories." Plomin and Asbury (2005, p. 90) comment: "Given that environmental measures as well as behavioral measures show genetic influence, it is reasonable to ask whether associations between environmental and behavioral measures are mediated genetically...Genetic factors can mediate the correlation to the extent that the environment represents a direct response to genetically influenced characteristics."

Olson, Vernon, Harris, and Jang (2001) commented that it was quite unlikely that there were direct, one-to-one causal links between genes and attitudes. Genetics provide for general predispositions or natural tendencies, which in turn shape environmental experiences in ways that increase the likelihood of the individual thinking, feeling, or acting in a certain way. Ilies et al. (2006, p. 131) agreed, elaborating with respect to organizational behavior, "Genes do not directly cause attitudes or behaviors, but they encode evolved neurophysiological systems that have adaptive value (e.g., the behavioral approach system promotes fitness by facilitating the acquisition of resources related to reproductive success)."

If genetic factors significantly affect measures of relevant environments, as evidence strongly suggests they do (Plomin, DeFries, McClearn, & McGuffin, 2008), how can this be the case? The answer, as noted by Plomin and Asbury (2005), is that environments may be considered extended phenotypes, "reflecting genetic differences between individuals as they select, modify, and construct their own experience of the world" (p. 90). There are various ways this extended phenotype may come about. One possibility is *active genotype-environment correlation*, which occurs when individuals' inherited traits influence their life choices (McGue & Bouchard, 1998). For example, individuals with genotypes that are expressed in core self-evaluations may chose jobs that are more likely to be intrinsically rewarding; CSE has been linked to the choice of intrinsically challenging work (Srivastava, Locke, Judge, & Adams, 2010). Another possibility is *evocative genotype-environment correlation*, which occurs because an individual's experiences are a function of the reactions his or her genetically influenced attitudes or behaviors elicit from others (McGue & Bouchard, 1998, p. 16). For example, individuals may have a "positivity" gene (as indicated by one or more of the genetic markers reviewed shortly) that causes others to treat the person more positively, in turn. Such treatment will then serve as a source of self-verification (Swann, Chang-Schneider, & McClarty, 2007), and may manifest itself in the mediated relationships hypothesized here. Alternatively, some have suggested that CSE is consistent with an approach-avoidance motivation framework (Ferris et al., 2011). As such, a gene that promotes self-positive thinking may be the same gene that fosters greater approach and lesser avoidance motivation.

One issue the foregoing analysis has left unanswered is: What is this mediating gene? Specifically, what gene leads us to believe that the mediating effects of job satisfaction and work stress on the relationship between CSE and health problems is partly genetic? This, of course, is a difficult question to answer, for three reasons. First, most broad traits and attitudes are not caused by only one genetic marker (or SNP, for single-nucleotide polymorphism). It is likely that several genetic markers are at work here. Second, given the complexity of the DNA strand—there are approximately 10 million SNPs (Sherry et al., 2001)—it is often a daunting task to identify candidate SNPs. Finally, to isolate the SNPs hypothetically responsible for the associations among the variables in this model would obviously require genetic testing.

These caveats notwithstanding, there are some plausible genetic markers. Perhaps the most likely candidates are genes known to be implicated in individual differences in well-being (serotonin), as well as perhaps pain (norepinephrine, GABA) and rewards (dopamine). Indeed, some theory and research does provide indirect support for the hypothesis that the proposed genetic effects can be traced to specific genetic markers. Perhaps the most logical candidate is a serotonin receptor gene—5-HTTLPR. Although the

link between this polymorphism and psychological variables has generated its share of inconsistent results (see Uher & McGuffin, 2010), a 2004 meta-analysis supported the importance of 5-HTTLPR for measures of stress, depression, and neuroticism (Sen, Burmeister, & Ghosh, 2004). Several more recent studies have found that 5-HTTLPR was associated with measures of anxiety, depression, and neuroticism (Takano et al., 2007; Wray et al., 2009). Another recent study linked 5-HTTLPR to pain responses (Palit et al., 2011). In reviewing the literature, Wankerl, Wüst, and Otte (2010) conclude that 5-HTTLPR is central to the stress regulation process. Though we are aware of no studies that have linked the other core traits to 5-HTTLPR, given the findings for neuroticism measures, it does suggest that this polymorphism may explain the hypothesized mediating effect of work stress in the relationship between CSE and employee health problems.

Though the most obvious, 5-HTTLPR is not the only candidate to explain the genetic basis for CSE—work stress—health problems relationship. Two specific dopamine genetic markers—D19S254 and D1S534—were identified in a recent study of 1157 Dutch twins (Bartels et al., 2010). Reiner and Spangler (2010) identified a broad dopamine marker—D4—as associated with emotional stability and negative life events. Another study isolated several markers that were commonly associated with neuroticism, psychological distress, and depression (Luciano et al., 2010).

As for job satisfaction, several studies have found that individuals with short 5-HTTLPR allele paid more attention to positive affective pictures while selectively avoiding negative affective pictures (Beevers, Ellis, Wells, et al., 2009; Beevers, Wells, Ellis, et al., 2009). Because job satisfaction results from an appraisal of one's job features (Locke, 1969), this polymorphism may explain the CSE—job satisfaction—employee health problems mediated relationship as well. Indeed, Song, Li, and Arvey (in press) found individual differences in 5-HTTLPR (as well as a dopamine neurotransmitter—DRD4) was associated with job satisfaction. As suggested by Song et al. (in press), there are other genetic pathways that may be particularly relevant to job satisfaction. Beevers, Wells, and McGeary (2009) found that Val66Met, a polymorphism of the neurotrophic factor BDNF gene which is associated with reward sensitivity, was associated with rumination. Wells, Beevers, and McGeary (2010) further found that Val66Met interacted with 5-HTTLPR to predict dysfunctional thinking. Because dysfunctional thought processes have been linked to both personality and job satisfaction (Judge & Locke, 1993), this polymorphism may be the linking mechanism which explains the mediating role of job satisfaction in the CSE—employee health problems relationship. As such, we hypothesize:

H3a. The degree to which job satisfaction mediates the relationship between CSE and health problems is explained, in part, by genetic effects, such that the genetic source of job satisfaction partly mediates the genetic influence of CSE on health problems.

H3b. The degree to which work stress mediates the relationship between CSE and health problems is explained, in part, by genetic effects, such that the genetic source of work stress partly mediates the genetic influence of CSE on health problems.

Method

Participants and procedure

Participants comprising the sample used in this study were enrolled in the Swedish Adoption/Twin Study on Aging (SATSA; Pedersen, 2005). Data collection for the SATSA was conducted in 1984, 1987, 1990, and 1993; in this study we used data that were collected in 1984 (core self-evaluations, job satisfaction, and work stress) and in 1993 (health problems). The SATSA data have been used to examine a variety of topics, such as the biological underpinnings of changes in memory (Reynolds, Jansson, Gatz, & Pedersen, 2006). To our knowledge, no study has been published using the SATSA to examine the relationships among core self-evaluations, job satisfaction, work stress, and health outcomes.

The sample that provided the data used in our study consists of twins, both monozygotic (MZ or "identical") and same-sex dizygotic (DZ, or "fraternal"), who were either raised together or were separated at an early age and raised apart. Specifically, the sample includes 94 (47 pairs of) MZ twins reared apart, 134 (67 pairs of) MZ twins reared together, 196 (98 pairs of) DZ twins reared apart, and 170 (85 pairs of) DZ twins reared together, for a total sample size of 594 individuals.

Measures

Core self-evaluations

Core self-evaluations was measured using the following 10 items (negatively-worded statements were reverse coded): "*My greatest expectations of myself are not filled,*" "*I get depressed more often than others,*" "*I often feel insecure,*" "*I'm not a cheerful optimist,*" "*When I make plans, I'm almost certain that I can follow them through,*" "*I often feel inadequate at work,*" "*I often feel as though I have no control over what happens to me,*" "*Most of my expectations have been filled,*" "*Sometimes I feel as though I don't have enough control over my own life,*" and "*I am worried in case I fail.*" The reliability of this scale was $\alpha = .76$.

Because the SATSA study was initiated well before the Core Self-Evaluations Scale (CSES; Judge et al., 2003) was developed, consistent with Judge and Hurst (2007), we formulated the measure used in this study based on items that would meet the criteria provided by Judge, Locke, and Durham (1997). Specifically, the items were: (a) evaluation-focused (vs. purely descriptive); (b) self-oriented (vs. other-oriented); and (c) fundamental or general in scope (as opposed to very specific self-evaluations). Because the degree to which each of the aforementioned items adequately measures the core self-evaluations concept is subjective, we sought to further investigate the construct validity of this measure by comparing it with existing measures.

Accordingly, we administered the measure used here (SATSA CSE), along with two other core self-evaluations (CSE) measures – the CSES (Judge et al., 2003) and Judge and Hurst's (2007) 12-item CSE scale (JH CSE) – to a sample of 909 undergraduates at a large, public university. All three measures had acceptable levels of reliability: SATSA CSE, $\alpha = .81$; CSES, $\alpha = .86$; JH CSE, $\alpha = .81$. The SATSA CSE measure correlated $r = .84$ with the CSES and $r = .80$ with the JH CSE scale. This is essentially the same as these two measures (the CSES and the JH CSE scale) correlated with each other ($r = .83$). Moreover, in correlating the three measures with measures of the Big Five traits, which we also collected, the correlations were quite similar. The average absolute difference in the correlations of the SATSA CSE measure and the CSES with the Big Five traits was $|\Delta r| = .02$. The average absolute difference in the correlations of the SATSA CSE measure and the JH CSE measure with the Big Five traits was $|\Delta r| = .03$. In sum, these pieces of evidence indicate that the SATSA CSE measure converges with other measures of core self-evaluations.

Job satisfaction

Overall job satisfaction was measured with 20 items tapping satisfaction with various aspects of one's job (e.g., supervision, coworkers, work, working conditions). Using a 1–5 response scale, individuals evaluated items such as: "Supervisors really support

the workers," "I enjoy my work," "Some people at work cause trouble because they talk behind the backs of others" (reverse-scored), and "My work is stimulating." The reliability of this 20-item scale was α = .84.

Work stress

Work stress was measured with six items. Individuals responded to the items using a five-point (1 = *agree*, 3 = *neither agree nor disagree*, 5 = *do not agree*) response scale, which was subsequently reverse-scored so that high scores reflect high levels of work stress (i.e., 5 = *agree*, 3 = *neither agree nor disagree*, 1 = *do not agree*). Sample items included: "I have more to do than I have time for at work," "I think that my work is demanding," and "I feel tense when I work." The reliability of this six-item scale was α = .73.

Health problems

Participants' health problems were measured via participants' self-reports, in 1993, on whether they had experienced (1 = yes or 0 = no) a series of 25 health conditions. These conditions included angina, phelbitus, bronchitis, allergies, migraines, dizziness, anemia, arthritis, hip problems, ulcer, gall bladder problems, liver problems, circulatory problems, among others. A response of yes was coded 1 and a response of no was coded 0. The responses were summed across the 25 conditions for an individual. Coefficient alpha was .71 for this measure.

Control variables

We controlled for *education,* twins' *contact frequency* with their co-twins, *conscientiousness*, and *extraversion* in the analysis. Prior research has shown that education level is associated with one's core self-evaluations and perceptions of one's job (and potentially their work stress and satisfaction). Thus, controlling for education can rule out this potential confound. Education level was measured as a categorical variable (1 = high school or less, . . . 4 = doctoral degree). Twins' contact frequency was measured using a 1-6 scale reflecting the degree of contact by phone and in person, with higher scores indicating greater contact. We averaged both twins' responses to obtain the contact frequency measure for the pair. Conscientiousness and extraversion were measured with 14- and 17-item scales, respectively, obtained in 1984. Items were evaluated on a 5-point Likert scale (1 = *strongly disagree* and 5 = *strongly agree*). Sample conscientiousness items include "I am punctual" and "I strive to achieve as much as possible." The reliability of the conscientiousness scale was α = .73. Sample extraversion items include "I like being with people" and "I prefer to stay in background" (reverse-scored). The reliability of the extraversion scale was α = .94. Finally, we did not statistically control for gender (in the sense of having gender as a control variable in a multiple regression). Rather, we followed the more comprehensive approach of prior research (e.g., Zhang et al., 2009) by conducting separate analyses for both gender groups. In no case were the relationships among the hypothesized variables affected by gender.

Analyses

To test H1a, H1b, H1c, H2a and H2b, we conducted conventional path analyses that treat the twins as individuals. Because the two twins in a pair are not independent observations, we used robust standard errors (Huber, 1981) in the path analysis that can provide accurate statistical tests. Furthermore, we used 1000-replication bootstrapping to obtain the bias-corrected 95% confidence intervals for the path estimates and the mediated effects.

To test H3a and H3b, we first examined a series of univariate genetic models to separately estimate the heritability of CSE, job satisfaction, work stress, and health problems. These models were estimated in Mplus (Muthén & Muthén, 2010) by comparing the covariance among twins' scores on the variable of interest (i.e., CSE, job satisfaction, work stress, or health problems) across the four types of participant pairs (MZ reared apart, MZ reared together, DZ reared apart, DZ reared together). In these univariate models, the variance of each of the variables is decomposed into: additive genetic variance, shared environmental variance, and non-shared environmental variance (which includes measurement error). As shown in Fig. 2, additive genetic effects (latent variable A) reflect effects of the summation of genes across loci, whereas shared (latent variable C) and non-shared (latent variable E) environmental effects refer to environmental effects that contribute to twin similarities and differences, respectively. The four groups involved different patterns of constraints as follows: within a pair of twins, latent variables A_1 and A_2 are correlated 1.0 or 0.5 for MZ and DZ pairs, respectively (because MZ and DZ twins share, on average, 100% and 50% of their genes), whereas latent variables C_1 and C_2 are correlated 1.0 for twins reared together and uncorrelated for twins reared apart. The latent variables E_1 and E_2 are uncorrelated in all groups because E represents non-shared environmental effects.

Using the notations from Fig. 2, the total variance in CSE (V_{CSE}) can be decomposed into additive genetic variance (a^2), shared environmental variance (c^2), and non-shared environmental variance (e^2).

$$V_{CSE} = a^2 + c^2 + e^2 \qquad (1)$$

Heritability is then estimated as the proportion of the total variance that reflects additive genetic influences:

$$h^2 = a^2/V_{CSE} \qquad (2)$$

Following established procedures in conducting univariate genetic analyses (e.g., Arvey, Zhang, Avolio, & Krueger, 2007), the paths estimated by the full, or the ACE, model (containing all the three types of effects) were examined for significance and the
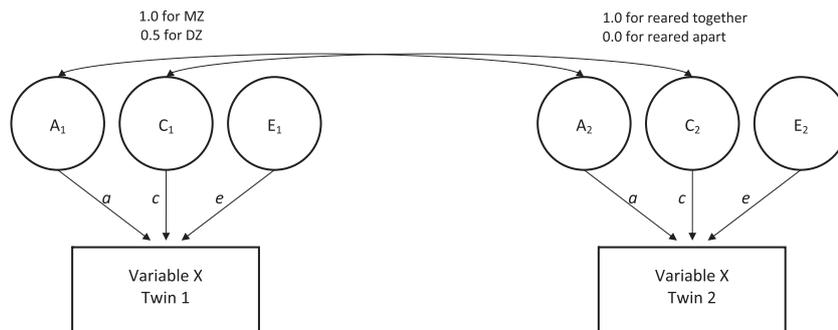

Fig. 2. Conceptual univariate ACE model. MZ = monozygotic twins. DZ = dizygotic twins. *a*, *c*, and *e* refers to genetic, shared environmental, and non-shared environmental influences, respectively. Subscripted numbers 1 and 2 refer to the first and second twin in a pair.

model was tested against alternative (simpler, nested) models (e.g., AE model, which eliminates shared environment effects). Models were compared using the chi-square (χ^2), as well as examining model fit indices including Comparative Fit Index (CFI) and the Root-Mean-Square Error of Approximation (RMSEA), as recommended by various authors (e.g., Bentler, 1990; Hu & Bentler, 1995; MacCallum, Browne, & Cai, 2006; MacCallum, Roznowski, Mar, & Reith, 1994). We also examined the Akaike Information Criterion (AIC; Akaike, 1987) because it is useful for model comparisons (Tanaka, 1993), and because it adjusts for the parsimony of a model.

Second, based on the results of univariate models, we conducted multivariate analyses to examine whether the genetic influences of CSE on health problems are, in part, mediated by the genetic components of job satisfaction and work stress. We used the established Cholesky decomposition approach to model the genetic and environmental influences among the variables. Fig. 3 shows a simplified version of the multivariate model in which only one mediator is shown and the shared environmental (C) factors were not shown for clarity purposes. Paths a_{21} and a_{31} represent the influences of the genetic factor of CSE on the mediator and health problems, respectively. Path a_{32} shows the genetic influence of the mediator on health problems. Paths a_{22} and a_{33} represent the remaining genetic influences after partialling out those from the genetic factors of CSE and the mediator. Analogically, paths a_{21}, a_{32}, and a_{31} correspond to the conventional mediation paths a, b, and c' using Barron and Kenny's (1986) terms. A partial mediation regarding genetic influence of CSE on health problems is supported when all three paths are significant whereas a full mediation exists when both a_{21} and a_{32} are significant but a_{31} is not significant.
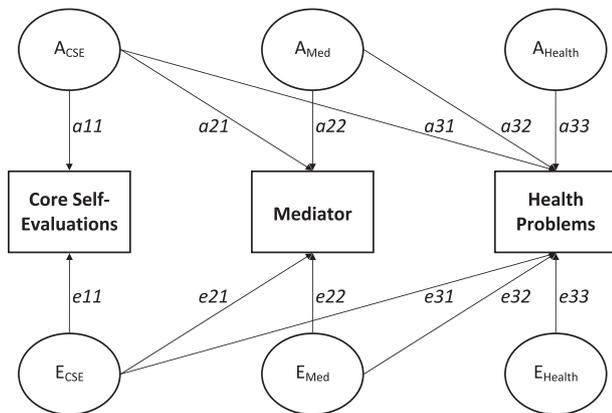

Fig. 3. Conceptual multivariate model (for one twin). Only one mediator is shown and the shared-environmental factors were omitted for clarity purposes. Actual analyses used two mediators simultaneously. CSE = core self-evaluations.

Below, in the results section, we detail how the variance of health problems can be explained by the genetic (as well as non-shared environmental) factors associated with CSE and the mediators. The proportions of explained variances are computed from the path coefficients estimated in the multivariate model.

Results

Individual-level descriptive statistics and intercorrelations among the study variables as well as the control variables are provided in Table 1. This table shows significant relationships of core self-evaluations with job satisfaction ($r = .33$, $p < .001$), work stress ($r = -.28$, $p < .001$), and health problems ($r = -.31$, $p < .001$). Among the control variables, only conscientiousness was significantly, but only moderately, related to core self-evaluation ($r = .15$, $p < .001$). Job satisfaction and work stress were significantly correlated with employee health problems ($r = -.22$ [$p < .001$] and $r = .24$ [$p < .001$], respectively).

In Table 2, we report cross-variable cross-twin correlations for both monozygotic (upper panel) and dizygotic (lower panel) twins. To estimate these correlations, we restructured the dataset so that the number of records was equal to the number of twin pairs, and separate variables were included for each twin (e.g., core self-evaluations of Twin 1 was one variable, core self-evaluations of Twin 2 was another variable). In each case, and as expected, the Twin 1 – Twin 2 correlations for the same variables were higher for MZ twins than DZ twins.

We tested H1 and H2 using conventional path modeling with robust standard errors (which corrected for the non-independence of two twins in a pair). Fig. 4 depicts the estimated unstandardized coefficients. CSE is positively related to job satisfaction ($b = .24$, $p < .001$) and negatively related to work stress ($b = -.39$, $p < .001$) and these two mediators, in turn, predict health problems ($b = -.70$, $p < .05$, $b = .64$, $p < .01$, respectively). The mediated effect via job satisfaction was $-.17$ ($p < .05$, bootstrapped 95% CI = $-.35$, $-.02$), and the mediated effect via work stress was $-.25$ ($p < .01$, bootstrapped 95% CI = $-.51$, $-.07$). Moreover, CSE has a direct effect ($b = -1.05$, $p < .001$) on health problems. Overall, these path analysis results supported H1a, H1b, H1c, H2a, and H2b.

Before testing H3a and H3b, we first estimated univariate ACE models for each variable. When modeling the covariance structure of the twins' scores on CSE, the ACE model provided a reasonable fit to the data, as shown by the fit indices provided in Table 3. The estimated A, C, and E factors accounted for 40%, 7%, and 53% of the variance in CSE. The 95% confidence intervals for the A and E factors excluded zero but that for the C factor included zero ($-.12$, .27). Therefore, we estimated the AE model for CSE (see Table 3). The fits of the ACE and AE models for CSE were not substantially different, and thus we retained the AE model as the final model, because it is more parsimonious (i.e., it is a simpler model

Table 1
Means, standard deviations, and intercorrelations among study variables (individual-level).

	M	SD	1	2	3	4	5	6	7	8
Core self-evaluations	3.62	.63	(.76)							
Job satisfaction	3.59	.51	.33[***]	(.84)						
Work stress	2.74	.77	−.28[***]	−.22[***]	(.73)					
Health problems	5.33	2.97	−.31[***]	−.22[***]	.24[***]	(.71)				
Education	1.50	.82	.07	.17[***]	.09	−.04	–			
Contact frequency	3.67	1.00	.01	−.01	.02	−.07	.02	–		
Conscientiousness	3.71	.45	.15[***]	.27[***]	.05	−.01	.01	−.05	(.73)	
Extraversion	3.09	.41	.03	.11[*]	.10[*]	.04	.05	−.02	.26[***]	(.94)

Notes: N = 594 individuals. Coefficient alphas are reported on the diagonal.
[*] $p < .05$ (two-tailed).
[***] $p < .001$ (two-tailed).

Table 2
Cross-variable cross-twin correlations for MZ and DZ twin pairs.

Variable	1	2	3	4	5	6	7
MZ twin pairs							
1. CSE (twin 1)	–						
2. Job satisfaction (twin 1)	.37***	–					
3. Work stress (twin 1)	−.27**	−.22	–				
4. Health problems (twin 1)	−.25*	−.27**	.31**	–			
5. CSE (twin 2)	.45***	.23*	−.04	−.31**	–		
6. Job satisfaction (twin 2)	.01	.38***	−.16	.10	.24*	–	
7. Work stress (twin 2)	−.19	−.22	.38***	.51***	−.30**	−.08	–
8. Health problems (twin 2)	−.12	−.15	.27**	.48***	−.25*	−.16	.40***
DZ twin pairs							
1. CSE (twin 1)	–						
2. Job satisfaction (twin 1)	.40***	–					
3. Work stress (twin 1)	−.40***	−.28**	–				
4. Health problems (twin 1)	−.39***	−.25*	.36***	–			
5. CSE (twin 2)	.20	.09	−.17	−.19	–		
6. Job satisfaction (twin 2)	.15	.13	−.21*	−.25*	.29**	–	
7. Work stress (twin 2)	.08	.03	.03	.15	−.24*	−.28**	–
8. Health problems (twin 2)	−.16	−.01	−.02	.33**	−.34**	−.29**	.12

Notes: CSE = core self-evaluations. For MZ twins, N = 114 pairs. For DZ twins, N = 183 pairs.
 * $p < .05$ (two-tailed).
 ** $p < .01$ (two-tailed).
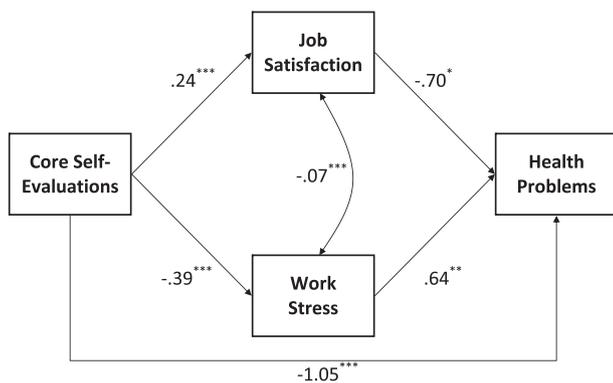 *** $p < .001$ (two-tailed).

Fig. 4. Path model with estimated unstandardized coefficients. Robust standard errors were used in analysis. Control variables (education, contact frequency, conscientiousness, and extraversion) were not shown for clarity purposes. The mediated effect via job satisfaction was −.17*, and the mediated effect via work stress was −.25**. * $p < .05$, ** $p < .01$, *** $p < .001$ (two tailed).

that fit the data equally well as the ACE model). In the AE model, the *A* and *E* factors explained 44% and 56% of the variance, respectively. In other words, the heritability of CSE is h^2 = .44, showing that 44% of the differences among individuals in measured CSE scores are associated with between-individual differences in genotype. The environment effects only model (CE model) for core self-evaluations did not fit the data well, and fit the data significantly worse than the ACE model ($\Delta\chi^2$ = 14.58, Δdf = 1, $p < .001$).

As shown in Table 3, the univariate results for job satisfaction, work stress, and health problems were similar to those discussed above. In fact, the ACE models for job satisfaction and work stress estimated the effect of the shared environment to be zero, which is functionally equivalent to the AE model. The C factor for health problems was estimated at .22 with 95% confidence intervals including zero. The fit indices for AE models for the three variables suggest that the AE model fit the data reasonably well for these variables. Alternative models including only environmental components (CE model) did not fit the data well either in an absolute sense (e.g., CFI = .39 for job satisfaction) or relative to the ACE model (see Table 3; the χ^2 differences between ACE and CE models were statistically significant for all three variables).

Based on the best fitting univariate models, the heritability of job satisfaction was h^2 = .35, that of work stress was h^2 = .32, and that of health problems was h^2 = .47. Therefore, 35%, 32%, and 47% of the between-individual variance in job satisfaction, work stress, and health problems, respectively, were due to additive genetic effects. The heritability estimate for job satisfaction is consistent with previous estimates obtained from different samples (e.g., Arvey et al., 1989; see Ilies & Judge, 2003). We also conducted analysis on a sub-set of the sample (i.e., twins reared apart) and obtained nearly identical results. Particularly, for all four variables, the AE models were the best fitting models and the heritability estimates based on twins reared apart are nearly identical to those based on the full sample. The similarity in univariate analysis results for the whole sample and for twins reared apart confirmed the general finding in behavioral genetics literature that "the behavioral similarity of MZ twins is largely independent of whether they were reared together or apart" (McGue, Elkins, Walden, & Lacono, 2005, p. 995).

Furthermore, we conducted univariate analysis on male and female twins and compared the heritability estimates across gender groups. The results showed no gender effects on the heritability of the four variables. Specifically, after constraining the heritability estimates to be equal across gender groups, the chi-square changes ranged from .32 to 3.19 (Δdf = 1) and none of them was significant.

To test H3a and H3b, we estimated multivariate Cholesky decomposition models testing whether job satisfaction and work stress mediate, in part, the genetic influences of CSE on health problems. Because the covariances among twins' scores on each of the four variables were best explained by univariate AE models, the multivariate models only specified additive genetic effects and non-shared environmental effects for CSE, the mediators (job satisfaction and work stress) and the outcome (health problems). The fit indices for this model and a reduced model (in which non-significant paths were fixed to zero) are presented in Table 3, and the unstandardized path coefficients for the reduced model are presented in Fig. 5 (job satisfaction) and Fig. 6 (work stress). For clarity of presentation, control variables are not shown and we present one mediator per figure whereas in the analysis, both mediators were included simultaneously. As shown in Fig. 5, the path from the genetic factor of CSE to job satisfaction is a_{21} = .14 ($p < .05$), and that from the genetic factor of job satisfaction to

Table 3
Fit statistics for the univariate and multivariate models.

	A	C	E	$\chi^2(df)$	p	$\Delta\chi^2(\Delta df)$	CFI	AIC	RMSEA
Univariate models									
Core self-evaluations									
ACE model	0.40	0.07	0.53	16.76 (16)	.40	–	0.97	1149.51	0.025
	(.21, .58)	(−.12, .27)	(.38, .67)						
@AE model	0.44	–	0.56	17.32 (17)	.43	.56(1)	0.99	1148.07	0.016
	(.31, .57)		(.43, .69)						
CE model	–	0.37	0.63	31.34 (17)	.02	14.58***(1)	0.50	1162.10	0.107
		(.23, .51)	(.49, .77)						
Job satisfaction									
ACE model	0.35	0	0.65	15.19 (16)	.51	–	1.00	716.42	0.000
	(.15, .52)	(.00, .00)	(.46, .85)						
@AE model	0.35	–	0.65	15.19 (17)	.58	.00(1)	1.00	714.42	0.000
	(.15, .54)		(.46, .85)						
CE model	–	0.21	0.79	21.27 (17)	.21	6.08*(1)	0.39	720.51	0.061
		(.00, .41)	(.59, 1.00)						
Work stress									
ACE model	0.32	0	0.68	7.91 (16)	.95	–	1.00	985.95	0.000
	(.12, .52)	(.00, .00)	(.48, .88)						
@AE model	0.32	–	0.68	7.91 (17)	.97	.00(1)	1.00	983.95	0.000
	(.12, .52)		(.48, .88)						
CE model	–	0.15	0.85	14.37 (17)	.64	6.46*(1)	0.90	990.41	0.000
		(−.07, .36)	(.64, 1.00)						
Health problems									
ACE model	0.31	0.22	0.47	21.23 (16)	.17	–	0.96	2364.10	0.070
	(.08, .55)	(−.02, .46)	(.33, .60)						
@AE model	0.47	–	0.53	24.60 (17)	.10	3.37(1)	0.95	2365.47	0.080
	(.34, .60)		(.40, .66)						
CE model	–	0.48	0.52	27.04 (17)	.06	5.81*(1)	0.93	2367.91	0.094
		(.35, .61)	(.39, .66)						
Multivariate models for testing mediation									
Full 4-variable AE model									
(all paths shown in Fig. 3 were estimated)				180.40 (160)	0.13	–	0.93	8437.78	0.029
@Reduced AE model									
(non-significant paths were fixed to zero, see Figs. 5 and 6 for path estimates)				183.26 (166)	0.17	2.86 (6)	0.94	8428.64	0.026

Notes: For MZ twins, N = 114 pairs. For DZ twins, N = 183 pairs. @ indicates best-fitting model. 95% confidence intervals are reported in parentheses beneath the point estimates for A, C, and E factors. χ^2 = chi-square. df = degrees of freedom. CFI = comparative fit index. RMSEA = root-mean-square error of approximation. AIC = Akaike Information Criterion. Because univariate analyses showed that results based on the full sample remained the same when examining only twins reared apart, we combined the twins reared apart and reared together into MZ group and DZ group for multivariate analyses. We controlled for education, contact frequency, conscientiousness, and extraversion in multivariate analysis.

health problems is a_{32} = −1.17 (p < .001). Similarly, Fig. 6 shows that the path from the genetic factor of CSE to work stress is a_{21} = −.32 (p < .001), and that from the genetic factor of work stress to health problems is a_{32} = 1.65 (p < .001). The genetic influence of CSE does not directly impact health problems (path a_{31} is zero in both figures). Moreover, there was no residual genetic influence

on health problems after partialling out the genetic influences of CSE and the two mediators. Overall, the Cholesky model shows that job satisfaction and work stress, altogether, fully mediate the genetic influence of CSE on health problems. Given that CSE also directly influences health problems through non-shared environmental paths, we conclude that the mediated effects via
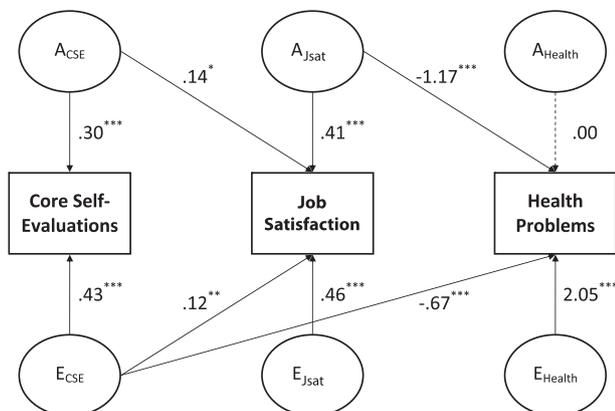

Fig. 5. Estimated multivariate model for job satisfaction as the mediator (for one twin). Actual analyses used two mediators simultaneously. CSE = core self-evaluations. Control variables (education, contact frequency, conscientiousness, and extraversion) were not shown for clarity purposes.
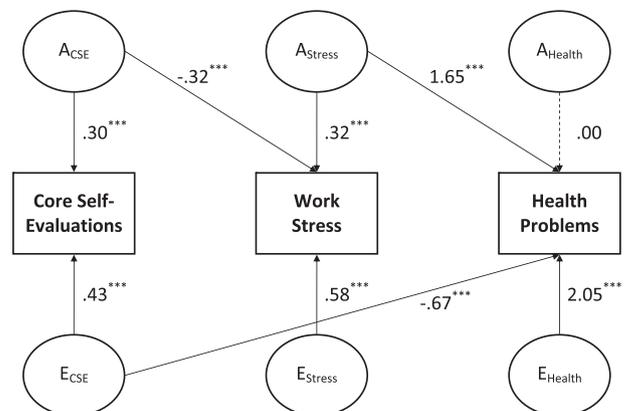

Fig. 6. Estimated multivariate model for work stress as the mediator (for one twin). Actual analyses used two mediators simultaneously. CSE = core self-evaluations. Control variables (education, contact frequency, conscientiousness, and extraversion) were not shown for clarity purposes.

Table 4
Proportions of health problems' genetic, non-shared environmental, and total variance that are explained by CSE, job satisfaction, and work stress.

	% Of the genetic or non-shared environmental variance	Corresponding % of the total variance
Health problems' genetic variance that is explained by:		
CSE	.00	.00
Job satisfaction	33.29 (17.69, 54.46)	15.55 (7.97, 25.06)
Work stress	66.71 (45.26, 82.24)	31.16 (17.75, 44.48)
Health problems' non-shared environmental variance that is explained by:		
CSE	9.60 (1.81, 21.97)	5.12 (1.01, 11.45)
Job satisfaction	.00	.00
Work stress	.00	.00

Notes: CSE = core self-evaluations. Reported values are percentages. 95% bias-corrected bootstrapped (with 1000 replications) confidence intervals are reported in parentheses.

job satisfaction and work stress are explained, in part, by genetic effects. Thus, H3a and H3b were supported.

These unstandardized path coefficients can be used to compute the proportions of the genetic and environmental influences on health problems that are due to the direct effect of CSE and mediated genetic effects via job satisfaction and work stress. As shown in Table 4, job satisfaction explained 33.29% of the genetic influence on health problems, i.e., $(-1.17)^2/[(-1.17)^2 + .00^2 + 1.65^2] = 33.29\%$. This corresponds to 15.55% of the total variance of health problems, i.e., $(-1.17)^2/[(-1.17)^2 + .00^2 + 1.65^2 + (-.67)^2 + 2.05^2] = 15.55\%$. Work stress explained 66.71% of the genetic influence on health problems, i.e., $(1.65)^2/[(-1.17)^2 + .00^2 + 1.65^2] = 66.71\%$. This corresponds to 31.16% of the total variance of health problems, i.e., $(1.65)^2/[(-1.17)^2 + .00^2 + 1.65^2 + (-.67)^2 + 2.05^2] = 31.16\%$. Regarding the non-shared environmental influence on health problems, none was due to job satisfaction or work stress (i.e., the path e_{32} was zero in Figs. 5 and 6). Rather, the environmental factor of CSE directly predicts health problems, and explained 9.60% $(= (-.67)^2/[(-.67)^2 + 2.05^2])$ of the non-shared environmental influence on health problems, which is 5.12% of the total variance of health problems, i.e., $(-.67)^2/[(-1.17)^2 + .00^2 + 1.65^2 + (-.67)^2 + 2.05^2] = 5.12\%$. Although not shown in Table 4, we found that CSE explained 10.38% $(= .14^2/[.14^2 + .41^2])$ and 49.06% $(= (-.32)^2/[(-.32)^2 + .32^2])$ of the genetic influence on job satisfaction and work stress, respectively. These correspond to 4.66% $(= .14^2/[.14^2 + .41^2 + .12^2 + .46^2])$ and 17.12% $(= (-.32)^2/[(-.32)^2 + .32^2 + .58^2])$ of the total variances of job satisfaction and work stress, respectively. The 95% confidence intervals of all the above percentages exclude zero.

Discussion

Our results show that job satisfaction and work stress – two constructs thought to be substantially influenced by the work environment – mediate, in part, the influence of CSE on health problems. Whereas this finding does not completely explain the processes by which having low CSE leads to increased health problems, it does provide an important step in elucidating why individual differences in broad personality traits such as CSE affect important and concrete outcomes such as health problems. But perhaps more importantly, our results shed light on the underlying nature of the mechanisms that link CSE to health problems. That is, we found that the mediation paths from CSE through job satisfaction and work stress to health problems are, in part, explained by genetics. This finding shows that broad self-evaluations influence satisfaction, stress and health problems not only by way of different perceptions or reactions to the work environment (e.g., those with low CSE would perceive more stressors in their work environment which would decrease satisfaction and increase stress and health problems). Rather, genetic factors associated with these constructs are also responsible for the mediated paths.

We believe this finding, that the mediated effects of CSE on health problems through job satisfaction and work stress are, in part, genetic, is important because it addresses the nature vs. nurture debate beyond partitioning between-individual variation in construct scores into genetic and environmental variation: We examined and showed that the mediated processes themselves are, in part, genetic in nature. On this topic, Plomin and Asbury (2005, p. 92) note that "Another important example in which genetic research is going beyond heritability is multivariate genetic analysis, which, as mentioned earlier, focuses on the covariance (correlation) between traits rather than the variance of each trait considered separately. It estimates the extent to which genetic factors that affect one trait also affect another trait." Kandler, Riemann, and Kämpfe (2009, p. 25) go a step further and suggest that "Genetically influenced personality traits are promising characteristics of individuals to explain the genetic influence on environmental measures, because personality affects how people create, interpret or perceive their environments or evoke reactions from other people."

Following Plomin and Asbury (2005) and Kandler, Riemann, and Kämpfe (2009), in this study, we conducted such multivariate genetic analyses that went beyond explaining the covariation among traits by (a) examining the covariation among a trait (CSE) and a health outcome, (b) explaining this covariation with job satisfaction and work stress (in traditional path analysis), and (c) examining the extent to which the genetic (environmental) effects of CSE on health problems are mediated by the genetic (environmental) components of job satisfaction and work stress. The nature of these analyses and their results are closely related to the areas of contribution of this research. Given our results, future research examining gene-environments correlations should attempt to uncover what are the decisions and activities influenced by the genotype which perhaps create work environments that lead to (dis)satisfaction or stress.

In order to extend these findings further, future research may well investigate finer-grained explanations of our genetic mediation effects (in essence, the mediation of mediation). Specifically, while our results suggest genetics is a partial explanation for the links of core self-evaluations, work stress, and job satisfaction with health problems, we do not know the mechanism of these genetic effects. Is it that a common gene causes individuals to be less positive in their reporting (about themselves, their work environment, and their health)—a genetic psychosomatic explanation? Or is it that a common gene causes individuals to be more functionally motivated (for example, to select work environments that cause less stress, foster more satisfying work, and promote better health)? It is important for future research to study the actual perceptions and behaviors associated with the genetic component of CSE that lead to more satisfying and less stressful jobs, and better perceived or actual health.

Interestingly, even though when examining traditional covariation among construct scores, job satisfaction and work stress only partially mediated the effect of CSE on health problems (this was to be expected, given that CSE can influence other work and nonwork attitudes, behaviors and reactions that might be relevant to health), the genetic influence of CSE on health problems was fully mediated by the genetic components of job satisfaction and work stress. This finding suggests that genes, perhaps such as the serotonin receptor gene (5-HTTLPR), that are manifested in broad individual differences like CSE also influence a broad array of work- and nonwork-related constructs and thus explain, at least in part as our data suggest, the effects of traits on attitudes, reactions, health and perhaps behavior.

Another contribution of this study relates to the estimation of the heritabilities of the constructs considered in this research. Although, as noted in the introduction, perhaps it is not surprising,

we found that all construct were heritable to a substantial degree (heritabilities ranged between .32 and .47). One new finding concerns the heritability of work stress (.32) which was slightly lower than that of job satisfaction (.35). One interesting fact is that although these two heritabilities were very similar, CSE explained only 10.38% of the genetic influence on job satisfaction, compared to 49.06% of the genetic influence on work stress. Whereas the result with respect to job satisfaction is comparable with previous findings (Ilies & Judge, 2003) and thus not surprising, why would CSE explain such a large proportion of the genetic influence on work stress? Stress represents a response to stimuli in the work environment and is largely psychological in nature (Ilies, Dimotakis, & De Pater, 2010). It follows, to the extent to which the individual genotype influences the probability of exposure to stressors (e.g., Kandler et al., 2009), genetics would be more relevant to work stress than to attitudes (e.g., job satisfaction) because stress represents a more reactive process. Also, given these proportions of the genetic influence explained by CSE, future research should explore other possible mediators, which could be other traits, interactions among traits, or interactions between traits and environmental attributes. If self-concept evaluations are as tied to experience (social learning) as some argue (Bandura, 1999), and if experience itself is strongly hereditary (McGue & Bouchard, 1998; Plomin, 1994), then the degree to which the genetic source of core self-evaluations leads individuals to place themselves into environments that reinforce their core self-evaluations is an interesting avenue for future research.

Implications for research and practice

Given that core self-evaluations is generally treated as a trait variable in organizational behavior research, a reader of this article might conclude that while supporting the genetic basis of core self-evaluations and the outcome variables is interesting, it confirms implicitly-made assumptions rather than challenges current research or practice. Put another way: If CSE is partly genetic, and its associations with other variables are explained by genes, how might this inform current research or practice?

The key implication of these findings, we think, lies less in establishing the heritability of the variables than in demonstrating that the links among these variables are *themselves* substantially genetic. At a broad level, our results suggest that associations among organizational behavior concepts (in this study, job satisfaction, work stress, and employee health problems) are partly genetic, and genetic differences in core self-evaluations explain the source of some of these associations. Mindful of the possibility of overstating our research, we believe these findings challenge the very nature of these associations (and thus, possibly, among other organizational behavior variables). It is true that employees with dissatisfying jobs and stressful work report more health problems, but what is the cause of these associations? Typically, we assume they are contextually caused, such that if we undertake interventions to increase satisfaction or decrease stress, we will observe improved employee health. To a partial but important degree, our findings challenge that interpretation. Much of the reason why dissatisfied or stressed employees report health problems is because employees are genetically predisposed toward these attitudinal states (which itself is explained by the genetic source of core self-evaluations). Changing the work environment is not going to alter this genetic calculus.

This certainly does not mean that all interventions to improve the workplace are fruitless. After all, genes did not explain the entirety of the relations among the study variables. However, the results do suggest that, to a significant degree, the reason the work environment is associated with health is not due to the context, but rather due to the fact that individuals genetically predisposed

to have a positive self-concept select themselves into their work environments, or that genetic differences interact with the environment.

Dovetailing with the "nature of nurture" perspective in behavioral genetics research (Plomin et al., 2008), these findings question the foundation and efficacy of interventions that treat all individuals the same. As noted by Haworth, Asbury, Dale, and Plomin (2011) with respect to educational interventions, instead of thinking of environments or interventions counteracting genetic differences, we should instead accept that people differ in *how* and *how much* they respond to particular work environment features of interventions. One way to do that, of course, is to personalize work environments. To be sure, such personalization poses challenges to the benefits of standardized and formalized policies and procedures. We do not suggest that equal treatment of employees be abandoned altogether. Rather, behavioral genetics research in general, and our findings in particular, suggest that a more enlightened approach is to appreciate that organizations design management systems that foster a more active employee, who has a greater ability to select, modify, and control his or her work environment so as to optimize maximum performance. Fortunately, this is an area where technological changes also point to the need for organizations to be more flexible in the future. Recently, Morgeson, Dierdorff, and Hmurovic (2010) persuasively argued for the relevance of context to work design. We would add to that the relevance of individual differences that are genetically rooted.

Limitations

A limitation of the study is generalizability: Our results are based on a study of twins in a relatively small country (Sweden). It is possible that Swedes are different with respect to these variables than individuals in other countries (e.g., perhaps there is a higher range restriction in job satisfaction). However, there is no reason to believe that Swedes would have different genetic make-ups than people from other countries and therefore the generalizability of the results of our genetic analyses is less of an issue.

Another limitation concerns the measure (developed for this study) and nature of core self-evaluations. Some might therefore argue that the estimation of the heritability of CSE is not a new contribution, since the traits that indicate CSE, such as neuroticism and self-esteem, have been shown as heritable (Jang et al., 1996; Kendler et al., 1998). The advantage of CSE is that it broadens the measurement of the underlying trait and considers what these individual traits have in common. Put another way, if a researcher prefers to use measures of neuroticism or self-esteem in organizational behavior research, attention needs to be paid to the breadth of the measure as measures of neuroticism or self-esteem may not have the same predictive validity as broader measures. That being said, these findings, or other findings in CSE research, are not meant to preclude research on narrower traits. There are situations in which narrower measures yield equivalent or even better results.

More generally, we believe our main contribution rests on the findings about the mediated genetic effects on job satisfaction and work stress, and such mediated effects have not been proposed or found before with any of the CSE component traits. Indeed, these results might build upon work by Neiss and colleagues which has sought to explain, using nonwork mediators and outcomes, the genetic source of self-esteem's association with these variables (Neiss, Stevenson, Legrand, Iacono, & Sedikides, 2009; Neiss et al., 2005).

Even though our study focused on one antecedent of core self-evaluations – genes – our results also suggest that the environment plays a role (though, in fairness, much of the nonshared environmental variance is undoubtedly idiosyncratic). Future research

should consider environmental antecedents of core self-evaluations. Though less a feature of the organizational psychology literature, research in personality psychology has shown that personality can be influenced by work experiences. Clausen and Gilens (1990) found that women who had higher levels of labor force participation later reported higher levels of self-confidence. Roberts, Caspi, and Moffitt (2003) reported that occupational attainment was associated with decreases in negative emotionality. What remains to be seen is the relative influence of familial environment, educational attainment, and early work histories on the formation of core self-evaluations. Does early work success and attainment lead to subsequent increases in core self-evaluations, and are there potential interactions with the genetic source of core self-evaluations?

Conclusion

Results from the present study showed that core self-evaluations explained substantial proportions of the genetic source of job satisfaction and work stress which, in turn, explained the genetic source of health problems. Future research should build on these results by exploring environmental sources of core self-evaluations, job satisfaction, work stress and health, and possible genotype × environment interactions. Though a complete picture of the genomic structure of core self-evaluations is a long way off, these results nonetheless suggest that individuals are born with core evaluations that make them far from the "blank slate" that has been the assumption of some self-concept research, where self-concept is "seen as being acquired through interaction with other people rather than being inborn" (Robson, 1988, p. 7). As Pinker (2002) notes, "Everyone has a theory of human nature" (p. 1). It is our hope that these results inform our theories of the nature of self-concept generally, and core self-evaluations specifically.

Acknowledgment

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