

Sources of Variability in John Henryism

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Objectives: To decompose sources of individual differences in coping as measured by John Henryism among African Americans.

Methods: Analyses described in this study are based on the pairwise responses from 180 pairs of same-sex, African-American twin pairs who participated in the Carolina African-American Twins Study of Aging (CAATSA).²¹ The sample consisted of 85 monozygotic (MZ) and 95 dizygotic (DZ) twin pairs.

Results: Environmental factors account for most of the variance (65%) in John Henryism scores, with the remaining variance attributable to additive genetic factors (35%). The test of the genetic component suggested that the 35% represented a statistically significant proportion of variance.

Conclusions: The vast majority of recent studies on African Americans and health outcomes have focused on the impact of psychosocial factors on diseases such as hypertension and diabetes, with relatively little attention to possible genetic contributors. Previous research on psychosocial indices and their relationship to cardiovascular health among African Americans has focused on assessment and epidemiological explorations rather than understanding the etiology of variability in such measures.

Key words: John Henryism ■ twins ■ genetics ■ environment ■ aging

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INTRODUCTION

Chronic exposure to adverse psychosocial stressors has been posited as a possible determinant of the disproportionate prevalence of cardiovascular disease experienced by African Americans.¹⁻⁴ The biological dysregulation promoted by psychosocial stressors may be moderated by any number of individual coping strategies. In particular, active or high-effort coping in response to everyday demands has been linked with positive health outcomes, among mostly Caucasian samples.⁵⁻⁹ However, recent research in the contexts of laboratory stressors and epidemiological studies on coping and hypertension suggests that this is not the case in all social contexts.^{10,11} Specifically, coping resources and information may act as proxies for control over outcomes and enhance the role of active coping efforts in successful outcomes.

The high-effort coping style John Henryism is conceptualized as “a strong behavioral predisposition to cope actively with psychosocial and environmental demands.”¹¹ The pattern is characterized by efficacious mental and physical vigor, a strong commitment to hard work and a single-minded determination to succeed.¹¹ John Henryism may be an adaptive strategy when sufficient financial, material and interpersonal resources are available.¹² By contrast, among those who have limited educational attainment, financial and other resources, and consequently increased susceptibility to the products of social inequity, high levels of John Henryism may lead to frustration and disappointment, rumination, negative social consequences as well as chronic physiological arousal.^{11,13-14} Indeed, mounting evidence supports John Henryism as a risk factor for cardiovascular dysregulation, primarily for African Americans, among those in low socioeconomic strata.¹¹⁻¹⁸ These dysregulated physiological responses may increase the risk for premature cardiovascular-related morbidity and mortality among African-Americans.

There is evidence that John Henryism levels among African Americans may vary by a range of

sociodemographic characteristics, including age and gender.¹⁹⁻²⁰ We have previously called for increased attention to determinants of the inter-individual variability in African Americans' adoption of the John Henryism style.¹⁴ Quantitative genetic designs that include twins are an important step in identifying determinants of individual differences by describing the environmental and genetic contributions to variance in coping among African Americans.²¹ These designs also allow the decomposition of environmental factors into shared and unique, nonshared environmental components of variance. There is evidence from studies of Caucasian twins that both environmental and genetic influences impact variability in psychosocial factors such as life satisfaction and social support.²²⁻²⁴ There are no studies of the genetic and environmental contribution to psychosocial factors among African Americans. The purpose of this paper is to examine the relative contribution of genetic and environmental factors to coping as indexed by John Henryism among adult African Americans. In addition to the influence of socialization (including verbal programming and behavioral modeling),^{25,26} we hypothesized that genetic factors would significantly contribute to inter-individual variability in coping among adult African Americans.

METHODS

Analyses described in this study are based on the pairwise responses from 180 pairs of same-sex, African-American twin pairs who participated in the Carolina African-American Twins Study of Aging (CAATSA).²⁷ The sample consisted of 85 monozygotic (MZ) and 95 dizygotic (DZ) twin pairs. Details on the registry and sample ascertainment can be found elsewhere.²⁷ Briefly, birth records from North Carolina Register of Deeds Offices from selected counties (those with significant African-American populations) were used to identify participants for the CAATSA study. Potential subjects were contacted by phone and asked to participate. Those who agreed (83%) participated in an in-person interview, which included measures of health status, cognition and psychosocial measures.

MEASURES

John Henryism

John Henryism was measured using the 12-item John Henryism Scale of Active Coping (JHAC12), which uses a five-point Likert-type scale to derive a total John Henryism score that ranges from 12–60 (with high scores representing higher levels of John Henryism). Five response options for each item extend from completely true to completely false. The JHAC12 has demonstrated acceptable internal consistency in male ($\alpha=0.67$, $n=180$) and female ($\alpha=0.71$, $n=242$) African-American samples.²⁸ Adult samples tend to score near the high end of the JHAC12.^{11,13} The assessment of John Henryism can, at times, present analytic and interpretive difficulties. Scores on the JHAC12 are often quite high for both blacks and whites. These high scale scores, normally averaging 50–54 out of 60, have been found in many John Henryism investigations and can make meaningful differences between high and low John Henryism groups difficult to discern. James and colleagues¹⁶ attributed the high scores to the possibility of social desirability biases in the scale. High John Henryism levels may be found because the construct taps factors such as hard work and determination, which are core American values (i.e., the Protestant work ethic).

Sociodemographics

Data on twin's age, gender and educational attainment were provided via self-report. Age and educational attainment were reported in number of years, and gender as male or female.

Zygosity

Zygosity was established using self-report and a physical similarity questionnaire,²⁹ which has been shown to predict zygosity with 93% accuracy, compared to genetic markers from blood.

Procedures

Participants were contacted from the CAATSA twin registry. Once they agreed to participate, they were scheduled a time for an interview. Participants read and signed an informed consent and then completed a 2.5-hour interview, conducted by trained

Table 1. Demographic characteristics of individuals and pairs

Variable	Mean (SD)	MZ Pairs Mean (SD)	DZ Pairs Mean (SD)
Age	47.28 (13.69)	47.08 (14.67)	47.46 (12.81)
Sex (% male)	37.7	37.1	39.1
Education	13.47 (4.55)	13.50 (5.93)	13.44 (2.87)
John Henryism	41.31 (4.92)	41.81 (5.07)	41.08 (4.59)

study personnel, in their home. The John Henryism measure was administered as part of the larger in-person interview. The items were read aloud to subjects to eliminate differences in education and reading ability. Participants received \$40 for their participation.

RESULTS

Statistical Analyses

Analyses of the John Henryism measure included phenotypic and quantitative genetic analyses. The phenotypic analyses were conducted to confirm the associations with demographic variables typically found using the JHAC measure.^{13,15-17} After the phenotypic correlations, linear regression was used to examine the contribution of these factors to individual variability in John Henryism. The results of the regression were used to inform the quantitative genetic analyses. Quantitative genetic analyses included the examination of intraclass correlations and biometric modeling to estimate the proportion of genetic and environmental (shared and non-shared) influences.

Phenotypic Analyses

The means and standard deviations for the demographic variables of interest are presented in Table 1. We examined the relationship between the variables by conducting correlations on the data with individual members of twin pairs (Table 2). We found that the relationships between education and age, and education and gender were significant. Generally, women reported more education than men. Further, educational attainment decreased as a function of increasing age. This negative relationship is common among older cohorts of African Americans who may not have had access to educational opportunities.²⁴

Linear regression analysis was conducted on individual scores for the John Henryism measure as the dependent variable; and age, education and gender as independent variables. We found that only gender ($p < 0.04$) was a significant predictor of scores on the John Henryism scale, with women scoring higher on average than men, although the group mean scores differed by only 1 point [men = 40.73 (SD = 5.80), women = 41.74 (SD = 4.28)]. We

calculated the within-pair correlations for MZ ($r = 0.38$) and DZ ($r = 0.16$) twin pairs. We also calculated residualized (for age, gender and education) correlations for our MZ ($r = 0.35$) and DZ ($r = 0.15$) twin pairs. These correlations suggest there is a significant genetic component in our results. We used the within-pair variance for the two types of twin pairs in the quantitative genetic analysis discussed in the next section.

Quantitative Genetic Analysis

Quantitative genetic modeling is premised on differences among individuals on a trait of interest, or phenotype being attributed to two primary sources of variation: 1) additive genetic variance (V_A), and 2) variance due to and individual's experiences (V_E) (e.g., work history in adulthood). More explicitly, the phenotypic variance (V_P) can be expressed as:

$$V_P = V_A + V_E$$

If each term in the above equation is divided by V_P , such that the phenotypic variance now equals unity, the following expression results:

$$1 = h^2 + e^2$$

where h^2 is heritability, or the proportion of the phenotypic variance attributable to additive genetic variance. Additive genetic variation is the sum of the effects from genes influencing a trait. Using this formula, environmental effects that make up the remaining proportion of variance are partitioned into those that are common (shared) or unique (nonshared).³¹ Common environmental variation can be defined as the phenotypic variation due to the subjects living in the same family (e.g., parental socioeconomic status), thus, sharing the same environment³¹ and denoted as c^2 . The proportion of variance attributable to unique environmental influences is represented as e^2 . These are the unique experiences that contribute to twin dissimilarity. The unique environmental component also contains measurement error. Figure 1 depicts a structural model that consists of additive genetic, common environmental and unique environmental influences for a pair of twins. P_1 and P_2 are the phenotypic scores

Table 2. Phenotypic correlations among the variables of interest

	Age	Sex	Education	John Henryism
Age	1.00			
Sex	0.026	1.00		
Education	-0.289**	0.133**	1.00	
John Henryism	0.024	0.040	-0.071	1.00

The correlations between sex and age, educations and John Henryism are Spearman correlations; * $p < 0.01$

for twin 1 and twin 2, respectively, and A_1 and A_2 are the latent additive genotypic values for the pair of twins. E_1 and E_2 represent environmental influences specific to each twin. Designation of twins as twin 1 or 2 is based on random assignment.

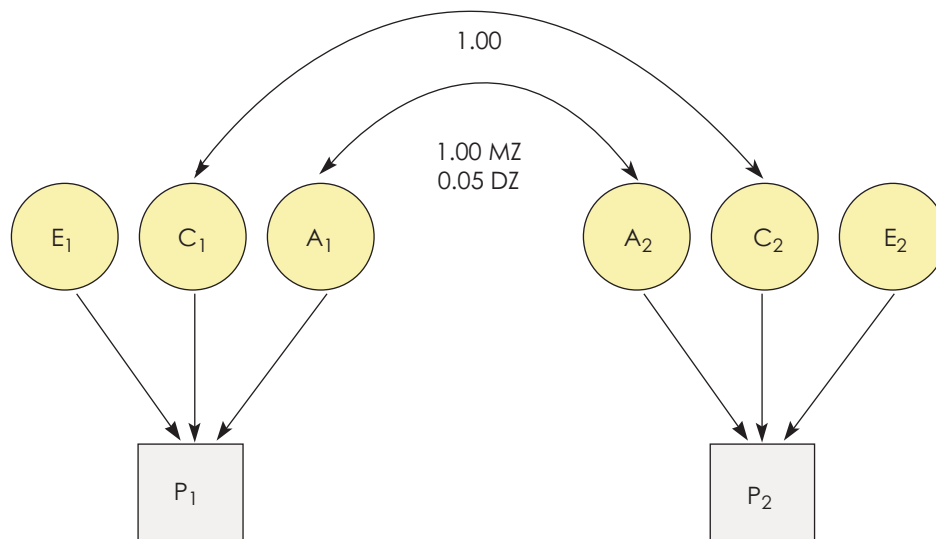
Although the components of variance are latent or unobserved variables in quantitative genetic analyses, they nonetheless can be estimated from MZ and DZ twin correlations and variances.³¹ The correlation between genotypic values in MZ twin pairs is constrained to equal 1.0 since they are genetically identical, while the correlation between genotypic values in DZ twins is constrained to equal 0.5 since they share, on average, half of the segregating alleles. The expected correlation between twin 1 and twin 2 on a single phenotype is then a function of the genes and environment that they share and can be derived by aid of the path diagram. Using a latent A and E model produces an upper heritability estimate that includes both the influence of genes and shared environment. This approach was used for the present analyses to maximize our ability to detect heritable influences. Examination of the intraclass correlations indicates quite small a correlation between twins for John Henryism. The correlations are almost double for MZs compared to DZs, indicating a relative change in genetic influence for the measure. Overall, the correlations remain low to moderate, suggesting a low-to-moderate influence of genetics on coping as is defined by the JHAC scale. Using these assumptions in structural equation models, observed data can be compared to expected values and the results tested via a Chi-squared statistic. Here, a nonsignificant Chi

square indicates that the data fit the expectations set in the structural equation models. In addition to Chi square, indices of practical fit, which control for the effects of for sample size on Chi square, are also calculated by the statistical package. These include Root Mean Square (RMS) and Goodness of Fit Index (GFI).³² The genetic component was tested for significance by eliminating the parameters that correspond to each latent factor, producing reduced models. The goal was to establish the most parsimonious model, one with fewer parameters or latent variables, yet still fitting the data. Parameter significance is assessed by dropping individual parameter estimates and comparing the full model to the reduced models using a Chi square, where:

$$\chi^2_{\text{Reduced}} - \chi^2_{\text{Full}} = \chi^2$$

with the degrees of freedom equal to the difference in the degrees of freedom for the models ($df_{\text{Reduced}} - df_{\text{Full}} = df$).³² Models were fitted using Lisrel 8.5 statistical modeling package. The model-fitting results are presented in Table 3. As can be seen, environmental factors account for most of the variance (65%) in John Henryism scores, with the remaining variance attributable to additive genetic factors (35%). Dropping the parameters corresponding to the latent genetic factor produced a significant change in the Chi square. The Chi square for the reduced model was 38.72. Subtracting this from the Chi square for the full model resulted in a Chi-squared delta of 25.41 with a df delta of 1. This change in Chi-squared value is significant at $p < 0.001$.

Figure 1. Path diagram of the univariate model used to examine genetic and environmental influences on John Henryism



A: the additive genetic variance; C: the shared environmental variance; E: the unique environmental variance

DISCUSSION

To our knowledge, based on a review of the literature, no published studies have attempted to decompose the contribution of genetic and environmental influence on coping among African Americans. Since one body of research argues that the excess rates in cardiovascular disease (CVD) among African Americans is in large part a function of genetic factors³³—while another shows that John Henryism and low resources predict increased risk for CVD—then John Henryism may be one key mechanism for the gene and CVD link. The association between John Henryism and measures of cardiovascular functioning has been supported in several studies,^{11,13-14} but the etiology of individual differences in John Henryism has been less well understood. In this study, we used a quantitative genetics approach to elucidate sources of individual variability in coping among adult African Americans. We decomposed the high-effort coping style, John Henryism, into genetic and environmental components of variance. Not surprisingly, our results indicated that the vast majority of individual variability in John Henryism was attributable to environmental factors. Interestingly, however, we found that more than one-third of the variance in John Henryism can be attributed to genetic factors. Our analysis of the proportion of genetic and environmental variance in John Henryism is largely consistent with that of previous investigations conducted among predominately Caucasian populations. For example, Busjahn et al.³⁴ found evidence of significant genetic influence on a series of 19 distinct coping styles in a sample of 212 MZ and DZ twin pairs. Other work³⁵ has suggested a genetic influence for some active coping styles in comparison to other strategies. Our findings, taken in conjunction with previous research, suggest that: 1) genetic factors appear to be important in accounting for individual variability in coping for both African Americans and Caucasians, and that 2) the relative proportion of genetic influence is similar across ethnic groups. Many have suggested that the development of coping styles such as John Henryism develops via learning that occurs in social contexts.^{13,25,26} Our findings are consistent with these notions, which suggest that the preponderance of individual variability in John Henryism among African Americans in coping arises from environmental influences, likely to be primarily from learned strategies to manage perva-

sive and unremitting psychosocial stressors (e.g., financial difficulties, familial instability, discriminatory acts, exposure to violence and limited access to quality healthcare).^{11,14} Notably, however, our findings suggest that a little more than one-third of the variance in John Henryism is accounted for by genetic factors, further supporting the notion that heritable biological processes underpin this active coping disposition.¹⁴ Genetic factors may work in an additive fashion to affect coping in a manner similar to the impact genetic factors have on other behaviors (e.g., through the action of neurotransmitters).³⁶ While our research does not identify specific candidate genes or environmental factors, they nevertheless highlight an important contributor to the strategies used by African Americans to manage the demands of their social environment. Caution should also be used in interpreting our findings due to the size of the sample. While CAATSA represents a very unique twin data set, it is rather small relative to conventional sample sizes for this type of data. It is, however, one of the largest in-person twin data sets and the only one that allows us to examine the variable of interest, John Henryism. We have given considerable thought to possible misinterpretations of our current findings. It is not our intent to suggest that racism and other social inequities are less problematic than the reactions of African Americans to these circumstances. We do not suggest that there is no need for societal change in attitudes and behaviors that fuel social inequity, nor should our results pathologize adaptive reactions to these profound stimuli. Rather, our study clearly illustrates that genetic and environmental influences interact dynamically and both play a relative role determining variability in coping. The large proportion of unique environmental variance supports the idea that environmental forces encountered by African Americans necessitate an active coping response.¹³ It is important to emphasize, however, that John Henryism should not be construed as a maladaptive coping strategy per se; rather, it is the combination of John Henryism within the context of socioeconomic adversity in which the adverse effects of John Henryism are expressed.¹⁴⁻¹⁵ Thus, we reject the view that our findings provide evidence of heritability in an adverse “trait.” These findings are as likely to indicate heritability in a very adaptive behavior pattern, given adequate socioeconomic circumstances. Taken

Table 3. Results of biometric model fitting

Genetic		Shared Environment		Nonshared Environment	
Parameter Estimate	Variance	Parameter Estimate	Variance	Parameter Estimate	Variance
0.588	0.35%	0.00	0.00	0.809	65%

π^2 (df=3)=0.06; p=0.998; root mean square=0.013; Goodness of Fit Index=0.999

together, however, we recognize the novelty of our approach and current results and the significant need to replicate our findings in additional populations of African Americans. One methodological point to note is that, throughout the John Henryism literature, use of the JHAC typically results in a negatively skewed distribution of scores.³⁷ This unique pattern of scores has provided some novel and important findings related to criterion validity. There are efforts by members of our research team to develop an enhanced psychometric version of this interesting and important test. While we observed a similar skewness to the scores in our sample, there was a significant range of scores that allowed the biometrical genetic analysis to be conducted. In addition, most important was the degree of similarity of scores between twins rather than actual individual scores.

CONCLUSION

There is increasing interest in understanding how psychosocial variables may be causally and otherwise related to variability in health outcomes, particularly among minorities. The vast majority of recent studies on African Americans and health outcomes have focused on the impact of psychosocial factors on diseases such as hypertension and diabetes, with relatively little attention to possible genetic contributors. Previous research on psychosocial indices and their relationship to cardiovascular health among African Americans has focused on assessment and epidemiological explorations rather than understanding the etiology of variability in such measures. This trend is likely due, in part, to the impact that the historical and negative connotation associated with those researchers who used studies of genetics in pseudoscientific paradigms to “prove” that African Americans were somehow biologically inferior or incapable of functioning at the level of their Caucasian counterparts. As a result, researchers are more likely to pursue topics that are less politically charged and more easily disseminated without controversy. The trend to exclude genetic factors may also be due to the tremendous difficulties recruiting African Americans into clinical and/or research trials that explore genetic factors. For example, identifying twins, establishing a twin research database and then maintaining rapport with African-American twins presents unique challenges and difficulties. Cohorts of African-American subjects who lived during times of segregation and more overt racism may also have world views, language and mannerisms that require special attention and experience to interpret in an ecologically valid and reliable manner. Although these trends exist, a small and growing minority of researchers, behavioral geneticists, psychologists and others remain com-

mitted to accurately portraying the influence of genetics on disease and behavior with sensitivities to the history and current context of their methods and dissemination. The current study is based on one such attempt to balance scientific exploration with historical sensitivities.

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