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# Nonadditive Genetic Effects on Hostility in South Korean Adolescent and Young Adult Twins

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Hostility has been shown to be a vulnerability marker for various health problems. The present study examined genetic and environmental contributions to individual differences in hostility in South Korean adolescent and young adult twins. Seven hundred and nineteen same- and opposite-sex twin pairs aged from 13 to 23 years completed a hostility scale. The scalar sex-limitation model was applied to the data. The best fitting model indicated that 34% of the total variation of hostility was attributable to genetic factors operating in a nonadditive manner. The remaining 66% of the variance was associated with nonshared environmental influences and measurement error. These findings were largely consistent with results from previous twin studies of personality based on Caucasian twins, rendering support for the pervasive influence of genetic non-additivity on human personality traits and the generalization of the heritability of personality across human populations.

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Hostility has been shown to be a risk factor for cardiovascular disease, atrial fibrillation (Eaker et al., 2004; Everson-Rose & Lewis, 2005), posttraumatic stress disorder (Lasko et al., 1994), eating disorders (Truglia et al., 2006), and premature mortality due to accidents, suicide, and violence (Iribarren et al., 2005; Romanov et al., 1994). Thus, it is important to understand genetic and environmental origins of hostility for the development of optimal prevention and therapeutic intervention strategies for health problems associated with hostility.

Previous twin studies have yielded mixed results of genetic and environmental influences on hostility: reported heritability estimates ranged from 20% to 98% (Cates et al., 1993; Pedersen et al., 1989; Raynor et al., 2002; Smith et al., 1991). This inconsistency in the estimates of heritability in the literature of hostility appeared to be due in part to small samples that most twin studies of hostility employed and to the complexity of the construct of hostility. It has been suggested that hostility is a multidimensional construct that includes affective (anger and irritability), behavioral (aggressive behaviors), and attitudinal (cynicism and

suspiciousness) components (Dembroski & Costa, 1987). It is likely that the estimates of heritability in the literature of hostility vary substantially because various twin researchers have used different instruments that measure varying dimensions of hostility. For clear understanding of the genetic causes of the variation of hostility, future twin researchers should refine the hostility phenotype and focus on a specific aspect.

Recently, Rebollo and Boomsma (2006) studied the genetic etiology of anger, an emotional component of hostility, in a large sample of adolescent and young adult twins (age range 12 to 25 years) and their parents. The authors administered the Spielberger's Trait Anger Scale (TAS) to 2664 twin families. Interestingly, the authors found significant dominance genetic effects on anger. Furthermore, the authors found sex differences in dominance genetic effects, favoring males (26% vs. 0%). In the Rebollo and Boomsma (2006) study, additive genetic effects were higher for females than for males (34% vs. 23%). The authors speculated that these sex differences in additive and nonadditive genetic effects might be related to sex differences in the risk for cardiovascular diseases.

The major aim of the present study was to investigate genetic and environmental influences on individual difference in hostility in South Korean adolescent and young adult twins. Given the widespread evidence for genetic nonadditivity documented for personality traits (Keller et al., 2005), the present study attempted to detect nonadditive genetic influences on hostility. Using the hostility scale (Koskenvuo et al., 1988), the affective component of hostility was examined.

As the majority of twin research on hostility so far has involved Caucasian twins, little is known about genetic and environmental contributions to the variation of hostility in Asian populations. The magnitude

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and nature of genetic and environmental contributions to hostility can differ in various human populations for the variations in allelic frequencies and culture across populations. For instance, it has been shown that Asians tend to experience anger less intensely and for shorter periods than Americans do (Scherer et al., 1988), and that Asians tend to use more introspective strategies comprising of repression and rational coping self-statements to anger provocation than White people (Suchday & Larkin, 2004). It was important, therefore, to undertake genetic and environmental analyses of hostility utilizing a South Korean twin sample.

## Materials and Methods

### Sample

The sample was drawn from twin participants in the ongoing South Korean Twin Registry (SKTR). The SKTR is a volunteer registry of South Korean twins and their families. Twins in the SKTR are recruited from a variety of sources including the medical records of maternity hospitals, twin mothers' clubs, media advertisements, and kindergartens and schools throughout South Korea. A detailed description of the recruitment procedure of the SKTR was reported in Hur et al. (in press). Twins' zygosity in the SKTR was determined from the twins' parents' responses to a zygosity questionnaire that includes questions regarding physical similarities and frequency of confusion by family members and others of the twins.

In early 2006, a mail survey, including the hostility scale, was sent to adolescent and young adult twins registered with the SKTR who were living in Seoul and three counties in South Korea. These areas were chosen for the mail survey in 2006 because, at the time of the mail survey, permissions for the participation in research had been obtained from the twins in these areas. The overall response rate of the mail survey in 2006 was approximately 32%. Among the twins who returned the mail survey, 41 twin pairs were excluded from the present analyses because their zygosity was ambiguous. The final sample included 719 pairs of twins, where 176 pairs were male monozygotic twins (MZM), 324 pairs female monozygotic twins (MZF), 60 pairs male dizygotic twins (DZM), 61 pairs female dizygotic twins (DZF), and 98 pairs opposite-sex twins (OSDZ).

The sample in the present study ranged from 13 to 23 years of age, with a mean of 16.8 years and a standard deviation of 2.4 years. There were almost twice as many monozygotic (MZ) twins in the present sample as dizygotic (DZ) twins. These rates of MZ and DZ twins were largely consistent with the twin birth rates in the South Korean population for the birth cohorts in the present study (Hur & Kwon, 2005). As in most volunteer twin samples (Lykken et al., 1978), however, the present sample has an overrepresentation of female twins.

### Measure

The hostility scale used in the present study comprises three items derived from factor analyses

(Koskenvuo et al., 1988). The scale includes self-ratings of anger-proneness, irritability, and argumentativeness. These three items of the scale are presented as a 5-point Likert scale. Responses to the three items are summed to yield a total score of hostility (range 3–15). This scale has been shown to be significantly correlated with Spielberger's TAS and to have satisfactory test-retest and internal consistency reliabilities in population-based samples (Romanov et al., 1994). The internal consistency reliability of the scale in the present sample was .71.

### Analytic Methods

To fulfill the goals of the present study, twin correlations for the five groups of twins (MZM, MZF, DZM, DZF, and OSDZ) were compared and the scalar sex-limitation model (Neale & Cardon, 1992) was applied to the twin data. Prior to correlational analyses and model-fitting analyses, assumptions regarding means and variances across zygosity and sex were tested, and the main effects of sex and age were examined.

The scalar sex-limitation model in the present study includes additive and nonadditive genetic and nonshared environmental factors, while allowing sex differences in these factors (Neale & Cardon, 1992). Sex and age were used as covariates in the model to control their main effects on the means. The raw data option in Mx (Neale et al., 2003) was used for the model-fitting analyses. Mx calculates twice the negative log-likelihood ( $-2LL$ ) of the data. Variations on the scalar sex-limitation model were made to determine the best fitting, most parsimonious model to explain genetic and environmental influences on hostility. Two criteria were used in deciding on the best fitting, most parsimonious model: the likelihood ratio test (LRT) and the Akaike information criterion ( $AIC = X^2 - 2df$ ). As the difference in  $-2LL$  is chi-square distributed with degrees of freedom equal to the difference in degrees of freedom, when two models were nested, LRT was applied to evaluate the significance of the eliminated parameters. AIC quantifies the information content of a model in terms of the joint criterion of fit and parsimony (Akaike, 1987). Thus, the smaller the AIC, the better the fit of the model to the data. When two models were not nested, the model that yielded lower AIC was chosen as more parsimonious model.

## Results

### Test of Assumptions and Twin Correlations

Table 1 provides tests for means, variances, and maximum likelihood correlations for the five groups of twins. Means were not significantly different across zygosity within each sex (Models 2 and 3), and differences in variance were not significant (Models 4 and 5). Model 6 equated variances to be equal between males and females, which yielded a nonsignificant difference in  $-2LL$ , suggesting that variances of the hostility score are similar in males and females. Models 7 and 8 tested the main effect of sex and age,

**Table 1**

Tests for Means, Variances, and Maximum Likelihood Twin Correlations for the Hostility Scale

Model	-2LL	df	$\Delta$ -2LL	$\Delta$ df	p
1. Saturated	6580.9	1439			
2. Means: MZM1 = MZM2 = DZM1 = DZM2 = OSDZM	6583.2	1443	2.27	4	.69
3. Means: MZF1 = MZF2 = DZF1 = DZF2 = OSDZF	6585.3	1443	4.41	4	.35
4. Variances: MZM1 = MZM2 = DZM1 = DZM2 = OSDZM	6583.0	1443	2.10	4	.73
5. Variances: MZF1 = MZF2 = DZF1 = DZF2 = OSDZF	6584.1	1443	3.22	4	.52
6. Variances: males = females	6587.1	1448	6.22	9	.72
7. Sex covariate = 0	6586.6	1440	5.73	1	.02
8. Age covariate = 0	6593.8	1440	12.86	1	.00
9. Correlations: MZM = MZF, DZM = DZF	6581.3	1441	0.42	2	.81
10. Correlations: DZM = DZF = OSDZ	6581.1	1441	0.25	2	.88

Note: MZM1, MZM2 = The first- and the second-born twins of male MZ twins; MZF1, MZF2 = The first- and the second-born twins of female MZ twins; DZM1, DZM2 = The first- and the second-born twins of male DZ twins; DZF1, DZF2 = The first- and the second-born twins of female DZ twins; OSDZ = opposite-sex DZ twins; OSDZM = male twins from the OSDZ group; OSDZF = female twins from the OSDZ group.

respectively. Both models produced significant differences in -2LL: males were higher on the score of hostility than females, and older twins were more hostile than younger twins.

Maximum likelihood twin correlations for hostility were .29 for MZM, .35 for MZF, .05 for DZM, .13 for DZF, and .03 for OSDZ. In both males and females, MZ correlations were more than double the DZ correlations, suggesting the existence of a nonadditive genetic contribution to hostility. Model 9 tested whether MZ and DZ twin correlations were similar in males and females. This test resulted in a nonsignificant change in -2LL. Model 10 tested whether OSDZ twin correlation was significantly different from the same-sex DZ twin correlations. Again, the difference in -2LL did not attain statistical significance in Model

10, suggesting that the same genes might be operating in the variation of hostility for males and females.

#### Analyses of Fitting the Scalar Sex-Limitation Model

Table 2 presents the results of fitting the scalar sex-limitation model. The full scalar sex-limitation model (Model 1) yielded -2LL of 6591.4 for 1453 df. The estimates for this model suggested some sign of sex differences in additive (A) and nonadditive (D) genetic factors in hostility: nonadditive genetic effects were higher for males than for females (31% vs. 5%), whereas additive genetic effects were higher for females than for males (30% vs. 0%). Model 2 set the magnitude of additive and nonadditive genetic and nonshared environmental factors to be equal across sex. The difference in -2LL in Model 2, however, was not statistically significant.

In Model 3, the A parameter was eliminated from Model 2, whereas in Model 4, the D parameter was

**Table 2**

Results of Fitting the Scalar Sex-Limitation Model

Model	Goodness-of-Fit Statistics						Parameter Estimates			
	-2LL	df	$\Delta$ -2LL	$\Delta$ df	p	AIC	Sex	A	D	E
1. Scalar sex-limitation	6591.4	1453	—	—	—	—	M	.00	.31	.69
							F	.30	.05	.65
2. A, C, & E equated across sex	6593.8	1456	2.45	3	.49	-3.56	M	.00	.34	.66
							F	.00	.34	.66
<b>3. Drop A from Model 2</b>	<b>6593.8</b>	<b>1457</b>	<b>2.45</b>	<b>4</b>	<b>.67</b>	<b>-5.56</b>	<b>M</b>	<b>.00</b>	<b>.34</b>	<b>.66</b>
							<b>F</b>	<b>.00</b>	<b>.34</b>	<b>.66</b>
4. Drop D from Model 2	6596.7	1457	5.39	4	.25	-2.61	M	.32	.00	.68
							F	.32	.00	.68
5. Drop A & D from Model 2	6652.5	1458	61.1	5	.00	51.10	M	—	—	1.0
							F	—	—	1.0

Note: The best fitting, most parsimonious model is indicated in bold. M = Males, F = Females.

A = additive genetic factors, D = nonadditive genetic factors, E = nonshared environmental factors.

dropped. No significant change in  $-2LL$  occurred in either of these two models. When the A and D parameters were simultaneously dropped from Model 2, however, a significant change in  $-2LL$  occurred (Model 5), suggesting that the variation of hostility cannot be explained without genetic effects. When the goodness-of-fit indices were compared between Models 3 and 4, AIC was much lower in Model 3 than in Model 4 ( $-5.56$  vs.  $-2.61$ ), indicating that Model 3 is better than Model 4. These results indicate that nonadditive genetic effects are more important than additive genetic effects in individual differences in hostility in South Korean adolescents and young adults. In Model 3, nonadditive genetic effects and nonshared environmental effect including measurement error were 34% (95% confidence interval [CI]: 30%–36%) and 66% (95% CI: 64%–70%), respectively.

## Discussion

Hostility has received increasing attention since it was identified as a vulnerability marker for various mental and physical illnesses. The significant nonadditive genetic effects on hostility found in the present South Korean twins are generally congruent with a growing body of evidence based on Caucasian twins for the importance of nonadditive genetic influences on personality traits (Keller et al., 2005). Nonadditive genetic effects refer to interactive effects where the effects of alleles differ in the presence of other alleles. Studies have demonstrated that an interaction between the 5-HTT gene and DRD4 alleles influences the trait of negative emotionality in infants in such a way that infants homozygous for the 5-HTTLPR s allele who also lack long DRD4 alleles showed higher negative emotionality than those who have long DRD4 alleles (e.g., Auerbach et al., 1999).

Although shared environmental influences on hostility were not considered in the model-fitting analyses in the present study, the DZ twin correlations of less than half the MZ twin correlations clearly suggest that environmental factors shared by family members like family socialization are not important in determining individual difference in hostility in South Korean adolescents and young adults.

The estimate of genetic influence (34%) on hostility found in the present South Korean twin sample is broadly in the range of those reported in previous studies of personality based on Caucasian twins, supporting the generalization of the heritability estimate of personality to various human populations. Sex differences in the magnitude of additive and nonadditive genetic influences found in the Dutch twin family study by Rebollo and Boomsma (2006) were not observed in the present sample, however. Several factors might contribute to the discrepancy between the results from the Dutch twin family study and the present findings. First, hostility is likely to be a polygenic trait. Possible differences in the frequency of genes for hostility between Dutch and South Korean

population may have produced somewhat different results in the two studies. It is well documented that the allele frequencies of 5-HTTLPR known to be involved in emotional behaviors are different in different human populations (Kunugi et al., 1997). Second, detecting nonadditive genetic effects and sex differences require a large sample (Martin et al., 1978). In the present study, a lack of statistical power associated with small twin samples, especially the small DZ twin samples, may be responsible for a failure to detect sex differences in additive and nonadditive genetic factors. However, one should note that additive and nonadditive genetic estimates for the full scalar sex-limitation model (Model 1) differed in males and females, and the pattern of sex difference was consistent with that found in the study by Rebollo and Boomsma (2006). The SKTR is planning to collect data from siblings of twins to complement DZ twins. Future studies using the SKTR sample may be able to identify sex differences in additive and nonadditive genetic effects on hostility.

Although the present results of nonadditive genetic factors in hostility largely support the behavioral genetic literature of personality, the estimate of nonadditive genetic factors found in the present study should be interpreted with caution. Martin et al. (1978) argued that classical twin studies typically do not have sufficient power to detect the nonadditive genetic factor due to its high correlation with the additive genetic factor. The sample size in the present study may have been too small to reliably discriminate between additive and nonadditive genetic effects. Thus, 34% of genetic nonadditivity found in the present study is likely to be a sum of both additive and nonadditive genetic effects.

Another limitation of the present study is that the response rate of the mail survey in the present study is relatively small (32%). Thus, the finding of the present study may not generalize to South Korean adolescents and young adults at large.

## References

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## References

- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332.
- Auerbach, J., Geller, V., Lezer, S., Shinwell, E., Belmaker, R. H., Levine, J., & Ebstein, R. (1999). Dopamine D4 receptor (D4DR) and serotonin transporter promoter (5-HTTLPR) polymorphisms in the determination of temperament in 2-month-old infants. *Molecular Psychiatry*, *4*, 369–373.
- Cates, D. S., Houston, B. K., Varak, C. R., Crawford, M. H., & Uttley, M. (1993). Heritability of hostility-



- related emotions, attitudes, and behaviors. *Journal of Behavioral Medicine*, 16, 237–256.
- Dembroski, T. M., & Costa, P. T. (1987). Coronary prone behavior: Components of the Type A pattern and hostility. *Journal of Personality*, 55, 211–235.
- Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., & Benjamin, E. J. (2004). Anger and hostility predict the development of atrial fibrillation in men in the Framingham offspring study. *Circulation*, 109, 1267–1271.
- Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health*, 26, 469–500.
- Hur, Y.-M., & Kwon, J. S. (2005). Changes in twinning rates in South Korea; 1981–2002. *Twin Research and Human Genetics*, 8, 76–79.
- Hur, Y.-M., Shin, J. S., Jeong, H.-U., & Han, J. Y. (in press). The South Korean Twin Registry. *Twin Research and Human Genetics*.
- Iribarren, C., Jacobs, D. R., Kiefe, C. I., Lewis, C. E., Matthews, K. A., Roseman, J. M., & Hulley, S. B. (2005). Causes and demographic, medical, lifestyle and psychosocial predictors of premature mortality: The CARDIA study. *Social Science and Medicine*, 60, 471–482.
- Keller, M. C., Coventry, W. L., Heath, A. C., & Martin, N. G. (2005). Widespread evidence for non-additive genetic variation in Cloninger's and Eysenck's personality dimensions using a twin plus sibling design. *Behavior Genetics*, 35, 707–721.
- Koskenvuo M., Kaprio, J., Rose, R. I., Kesaniemi, A., Sarna, S., & Heikkila, K. (1988). Hostility as a risk factor for mortality and ischemic heart disease in men. *Psychosomatic Medicine*, 50, 330–340.
- Kunugi, H., Hattori, M., Kato, T., Tatsumi, M., Sakai, T., Sasaki, T., Hirose, T., & Nanko, S. (1997). Serotonin transporter gene polymorphisms: Ethnic difference and possible association with bipolar affective disorder. *Molecular Psychiatry*, 2, 457–462.
- Lasko, N. B., Gurvits, T. V., Kuhne, A. A., Orr, S. P., & Pitman, R. K. (1994). Aggression and its correlates in Vietnam veterans with and without chronic posttraumatic stress disorder. *Comprehensive Psychiatry*, 35, 373–381.
- Lykken, D. T., Tellegen, A., & DeRubeis, R. (1978). Volunteer bias in twin research. *Behavior Genetics*, 17, 343–362.
- Martin, N. G., Eaves, L. J., Kearsy, M. J., & Davies, P. (1978). The power of the classical twin study. *Heredity*, 40, 97–116.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. London: Kluwer.
- Neale, M., Boker, S. M., Xie, G., & Maes, H. (2003). *Mx: Statistical modeling*. Richmond, VA: Department of Psychiatry.
- Pedersen, N. L., Lichtenstein, P., Plomin, R., DeFaire, U., McClearn, G. E., & Matthews, K. A. (1989). *Psychosomatic Medicine*, 51, 428–440.
- Raynor, D. A., Pogue-Geile, M. F., Kamarck, T. W., McCaffery, J. M., & Manuck, S. B. (2002). Covariation of psychosocial characteristics associated with cardiovascular disease: Genetic and environmental influences. *Psychosomatic Medicine*, 64, 191–203.
- Rebollo, I., & Boomsma, D. I. (2006). Genetic analysis of anger: Genetic dominance or competitive sibling interaction. *Behavior Genetics*, 36, 216–228.
- Romanov, K., Hatakka, M., Keskinen, E., Laaksonen, H., Kaprio, J., Rose, R. I., & Koskenvuo, M. (1994). Self-reported hostility and suicidal acts, accidents, and accidental deaths: A prospective study of 21,443 adults aged 25 to 59. *Psychosomatic Medicine*, 56, 328–336.
- Scherer, K. R., Wallbott, H. G., Matsumoto, D., & Kudoh, D. (1988). Emotional experience in cultural context: A comparison between Europe, Japan, and the United States. In K. R. Scherer (Ed.), *Facets of emotion: Recent research* (pp. 5–30). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Smith, T. W., McGonigle, M., Turner, C. W., Ford, M. H., & Slattery, M. L. (1991). Cynical hostility in adult male twins. *Psychosomatic medicine*, 53, 684–692.
- Suchday, S., & Larkin, K. T. (2004). Psychophysiological responses to anger provocation among Asian Indian and White men. *International Journal of Behavioral Medicine*, 11, 71–80.
- Truglia, E., Mannucci, E., Lassi, S., Rotella, C. M., Faravelli, C., & Ricca, V. (2006). Aggressiveness, anger and eating disorders: A review. *Psychopathology*, 39, 55–68.