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Genetic and Environmental Etiologies of Adolescent Dysfunctional Attitudes: A Twin Study

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Despite the importance of dysfunctional attitudes in the development and maintenance of depression, little is known about the etiological origin of dysfunctional attitudes. The Dysfunctional Attitudes Scale for Children was administered to 674 adolescent twins derived from the Beijing Twin Study (BeTwiSt). Four hundred and thirty-nine monozygotic and 235 same-gender dizygotic twin pairs were included. Approximately 54% were females. The age range of the twins was 11–17 years. Model-fitting analyses were conducted. Biometric genetic model-fitting estimates indicated that additive genetic factors accounted for 31% (95% CI: 11%, 45%) of variance in adolescent dysfunctional attitude. The influence of shared environmental factors was small and negligible (9% [95% CI: 0%, 27%]). Non-shared environmental factors explained 60% (95% CI: 55%, 66%) of variance. Equating the estimate parameters across gender or age groups resulted in a non-significant difference of model fit, but there were trends suggesting higher heritability in females and older adolescents. Our results provide evidence for moderate heritability of dysfunctional attitudes in adolescents. Dysfunctional attitudes can be used as an endophenotype to identify risk genes for depression.

■ **Keywords:** dysfunctional attitudes, genetic and environmental influences, twin study

One of the most prominent etiological models of depression is Beck's cognitive theory (Beck, 1967, 1987), which posits that depressogenic schemas confer vulnerability to depression. Beck defined the latent schemas as stored bodies of knowledge (i.e., mental representations of the self and prior experiences) that affect how the individual perceives, encodes, and retrieves information. Beck posited that depressogenic schemas are typically represented as sets of dysfunctional attitudes such as 'If I fail at something, it means I'm a total failure', or 'I am nothing if a person I love doesn't like me'. Beck's cognitive theory proposes that dysfunctional schemas confer individual vulnerability to depression, especially in the occurrence of negative events. Moreover, dysfunctional schemas are presumed to endure beyond the depressive episode, representing stable vulnerability factors for depression onset and recurrence.

Beck's (1967, 1987) theory is supported by earlier research in adults that demonstrates dysfunctional attitudes mainly or interacting with stress prospectively predicting an episode of major depression (Alloy et al., 2006a; Otto et al., 2007) or increase in depressive symptoms (Brown et al., 1995; Dykman & Johll, 1998). Subsequently, studies have provided evidence for the applicability of Beck's theory to adolescents (Abela & Hankin, 2008). More specifically, several prospective studies of Western samples have shown that adolescents who possess a high level of dysfunctional attitudes are more likely than other adolescents to experience increase in depressive symptoms or onset of clinical depression, especially following the occurrence of negative events (Abela & Skitch, 2007; Hankin, 2008a; Hankin et al., 2008; Lewinsohn et al., 2001; Young et al., 2012). Moreover, two recent studies of Chinese adolescents also reported that higher levels of dysfunctional attitudes were associated with greater increase in depressive symptoms following the occurrence of negative events (Abela et al., 2011; Cui et al., 2013). Despite the importance of dysfunctional attitudes in the development and maintenance of depression, little is known about the etiological origin of dysfunctional attitudes. Identifying these causes is an important step toward understanding and preventing depression.

Beck (1987) proposed that adverse experiences during childhood might contribute to the development of dysfunctional attitudes. Extant empirical studies that have examined causes of dysfunctional attitudes have mainly focused

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on social and interpersonal factors (Hankin et al., 2009). More specifically, several studies have shown that emotional maltreatment (Gibb et al., 2003), verbal peer victimization (Gibb et al., 2004), negative feedback from parents (Alloy et al., 2006b), and negative life events (Young et al., 2012) are associated with a higher level of dysfunctional attitudes. Lee and Hankin (2009) reported that dysfunctional attitudes mediate the association between insecure attachment and an increase in symptoms of depression and anxiety during adolescence.

An alternative explanation that has not received much attention is the role of genes. Several lines of evidence have already indicated the trait-like characteristics and genetic bases for dysfunctional attitudes. First, in a short-term prospective multi-wave study, Hankin (2008b) showed that dysfunctional attitudes in early and middle adolescence exhibit moderate stability over time. Further study supported dysfunctional attitudes as a stable trait across late adolescence to early adulthood (Wang et al., 2010). Second, several high-risk studies demonstrated that a history of depression is associated with elevations in dysfunctional attitudes (Farmer et al., 2001; Wang et al., 2010), even when sub-clinical depressive symptoms are controlled (Otto et al., 2007). Finally, the main or interactive effects of some specific genetic loci (e.g., BDNF Val66Met and 5-HTTLPR) are involved in dysfunctional attitudes (Wells et al., 2010; Whisman et al., 2011). However, to date, no empirical studies have quantitatively assessed the magnitude of genetic influence on dysfunctional attitudes (i.e., heritability). Thus, the first aim of the present study was to quantify the genetic and environmental influences on adolescent dysfunctional attitudes using a classic twin design.

Previous studies examining the mean level of dysfunctional attitudes showed no significant gender differences (Abela & Skitch, 2007; Abela et al., 2011). However, several studies have indicated that the interacting effect between dysfunctional attitudes and life stress on depressive symptoms is stronger in females compared with males (Dykman & Johll, 1998; You et al., 2009). Several behavioral genetic studies have also suggested gender differences in genetic and environmental etiologies of depression (Bierut et al., 1999; Kendler et al., 2001; Rice et al., 2002). Thus, beyond estimating genetic and environmental influences on dysfunctional attitudes in the entire sample, we were also interested in whether or not there is a gender difference in the etiological model.

Another interesting issue pertains to age differences. From the developmental perspective, research has shown that cognitive vulnerabilities, such as dysfunctional attitudes, probably emerged in late childhood and stabilized during adolescence (Abela & Hankin, 2008). Furthermore, dysfunctional attitudes exhibit more trait-like components across time in middle adolescence compared with early adolescence (Hankin, 2008b). In addition, D'Alessandro and Burton (2006) reported that the dysfunctional attitudes–stress interaction only predicted an increase in depressive symptoms in older children (aged $\geq$ 11–12 years) who have formal operational cognitive skills, but not in younger children (aged 7–10 years). Finally, behavioral genetic research has shown that the heritability of anxious/depressive symptoms and general cognitive ability increase with age (Haworth et al., 2009; Lamb et al., 2010). These findings highlight the need to examine age differences with respect to the impact of genetic and environmental factors on dysfunctional attitudes.

In sum, the purpose of the present study was to estimate the contribution of genetic and environmental factors to variation in adolescent dysfunctional attitudes using a classic twin study. Then we examined gender differences in the genetic and environmental etiologies of adolescent dysfunctional attitudes. Lastly, we examined how the genetic and environmental contributions to adolescent dysfunctional attitudes differ by age.

Materials and Method

Participants

This study was part of the Beijing Twin Study (BeTwiSt), a longitudinal study examining the genetic and environmental influences on psychological development and mental health problems among Chinese children and adolescents. The BeTwiSt sample was recruited from 620 elementary and secondary schools randomly selected from 18 districts and counties in the Beijing municipality. Detailed information about the recruitment process was described in a previous study (Chen et al., 2013).

The sample for the current study included 674 same-gender twin pairs with complete measures, among which 439 pairs were monozygotic (MZ) twins and 235 pairs were dizygotic (DZ) twins. Females accounted for 53.7% of the sample. The age of the study sample ranged from 11 to 17 years, with a mean (*M*) of 14.03 years and a standard deviation (*SD*) of 1.9 years. Ninety-two per cent of the participants were Han Chinese, and the rest were minorities. Regarding the educational level of the fathers of the participants, 6.8% attended primary school, 32.8% attended junior high school, 31.8% attended senior high school, 26.1% attained a college degree, and 2.5% had a graduate degree. For the education level of the mothers of the participants, the proportions for the above five academic qualifications were respectively 5.4%, 35.3%, 29.5%, 25.2%, and 4.6%. The representativeness of the twin sample was described in the prior study (Chen et al., 2013).

Measures

Dysfunctional attitudes. The Dysfunctional Attitudes Scale for Children (DAS-C; D'Alessandro & Burton, 2006) was used to measure the dysfunctional attitudes of the

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participants. For each item, children were asked to rate how much they agree by choosing from a 5-point Likert scale (1 = strongly disagree; 2 = mostly disagree; 3 = agree a bit; 4 = mostly agree; 5 = strongly agree). Past research has shown DAS-C to be reliable and valid; the 3-week test–retest reliability was 0.80 (D'Alessandro & Burton, 2006; Rawal et al., 2013). The Chinese version of the DAS-C was developed using the back-translation method. The internal consistency of the scale in our sample was satisfactory, with a coefficient alpha (α) of 0.82.

Zygosity determination. For the 674 pairs of same-gender twins, 89.5% of the zygosity of the pairs was determined by DNA analyses. The zygosity of the remaining pairs (10.5%) was determined by a valid questionnaire method (Chen et al., 2010) because the saliva samples yielded insufficient DNA. For DNA analyses, nine short tandem repeat (STR) loci, which are highly heterogeneous in the Chinese population, were used. Same-gender twins with at least one different genetic marker were classified as DZ twins, otherwise the twins were classified as MZ twins. The posterior probability of being MZ for same-gender twins with the same genotype in all nine loci was estimated to be 99.99%. The validity of the questionnaire method was examined through comparison with the results of DNA analyses. The predictive accuracy of the questionnaire method used in this study reached 91% (Chen et al., 2010).

Procedures

All twins and their parents signed informed consent before participation. During the survey of the second wave of BeTwiSt, two research staff members visited each family in their home to administer the assessments. Twins reported their dysfunctional attitudes and depressive symptoms by DAS-C and Children's Depression Inventory (CDI) independently. The participants were assured of the confidentiality of their responses and the voluntary nature of their participation. All procedures were approved by the Institutional Review Board.

Statistical Analyses

The twin design relies on different levels of genetic relatedness between MZ twin pairs who are genetically identical, and DZ twin pairs who share one-half of the additive genetic effects. This difference was used to estimate the contribution of additive genetic (A), shared environmental (C), and non-shared environmental (E) influences to the individual differences in the phenotype of interest. Shared environmental influence is defined as a non-genetic influence that results in similarity within the twin pairs, while non-shared environmental influence results in differences within the twin pairs, which also includes measurement error.

Structural equation modeling packages (Mx) utilize maximum likelihood estimation procedures on raw data to estimate variance components. The fit statistics provided by Mx for raw data modeling is minus twice the log likelihood (-2LL) of the observations. This is not an overall measure of fit, but provides a relative measure of fit because differences in -2LL between models are distributed as chi-square (χ^2). Therefore, to examine the overall fit of the genetic model, it is necessary to compare the -2LL with that of a saturated model. Consistent with the principle of parsimony, the fit of sub-models was assessed by χ^2 difference tests and the Akaike's information criterion (AIC = χ^2 - 2*df*), with lower χ^2 values and more negative AIC values suggesting a better fit. Confidence intervals of parameter estimates were obtained by maximum likelihood.

The statistical significance of genetic, shared, or non-shared environmental effects was tested by checking whether or not the confidence interval of parameters included zero. Quantitative gender and age differences were examined by comparing the saturated model with freely estimated A, C, and E parameters across gender or age group and the restricted model, where A, C, and E parameters were equated for males and females, or for younger (aged 11–13 years) and older groups (aged 14–18 years). The difference in -2LL between the saturated model and the restricted model is χ^2-distributed with degrees of freedom equal to the difference in degrees of freedom. The likelihood ratio test (LRT) was applied to evaluate the significance of the constraint. If the difference in -2LL was significant, the constraint was rejected; if the difference is non-significant, the constraint was accepted and the parsimonious model (with fewer estimated parameters) was approved.

Results

Descriptive Statistics and Pearson Correlations

The mean values and *SD*s of dysfunctional attitudes are shown in Table 1. There were no significant differences in the mean level of dysfunctional attitudes between males and females, $t(629) = -0.33$, $p = .74$, and between the younger (aged 11–13 years) and older (aged 14–17 year) groups, $t(629) = -0.56$, $p = .58$. The interaction between gender and age was also non-significant, $F(1, 627) = 0.15$, $p = .93$. No significant mean difference was found between MZ ($M = 55.66$, $SD = 12.44$) and DZ ($M = 56.57$, $SD = 12.00$) twins, $t(629) = -0.88$, $p = .38$. The phenotypic variance was homogeneous across zygosity groups ($F = 0.81$, $p = .37$), gender ($F = 3.00$, $p = .08$), and age group ($F = 3.22$, $p = .07$). We also compared the mean levels of dysfunctional attitude between our sample (aged 11–17 years, $N = 674$, $M = 55.97$, $SD = 12.29$) and the sample of Canadian children (aged 7–14 years, $N = 241$, $M = 55.01$, $SD = 16.02$), in which the DAS-C scale was originally developed and validated (D'Alessandro & Burton, 2006). Results showed that there was no significant mean difference ($t = -0.85$, $p = .20$).

Before estimating the parameters of genetic and environmental effects in model-fitting analyses, Pearson correlation coefficients of DAS-C scores within twin pairs were

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TABLE 1

Descriptive Statistics and Pearson Correlation Within Twin Pairs

	Female			Male			Younger (11–13 years)			Older (14–17 years)		
	MZ	DZ	Total	MZ	DZ	Total	MZ	DZ	Total	MZ	DZ	Total
Mean	55.15	56.96	55.83	56.22	56.01	56.15	56.00	54.95	55.66	55.39	57.65	56.21
SD	12.33	10.83	11.81	12.57	13.51	12.86	12.89	13.12	12.95	12.11	11.10	11.79
Pearson correlations	0.42	0.26		0.37	0.24		0.37	0.24		0.40	0.25	
N	226	136	362	213	99	312	187	92	279	252	143	395

Note: All Pearson correlation coefficients were significant at the 0.05 level.

TABLE 2

Parameter Estimates With 95% Confidence Intervals and Fit Indices Across Genders

						Female			Male		
Model	-2LL	$\Delta\chi^2$	Δdf	p	AIC	A	C	E	A	C	E
Model 1	7969.39			-	2241.39	0.32 (0.05, 0.49)	0.10 (0.00, 0.34)	0.58 (0.51, 0.65)	0.28 (0.00, 0.44)	0.09 (0.00, 0.36)	0.63 (0.56, 0.72)
Model 2	7969.44	0.05	7	1.0	2227.44	0.32 (0.05, 0.49)	0.10 (0.00, 0.34)	0.58 (0.51, 0.65)	0.28 (0.00, 0.44)	0.09 (0.00, 0.36)	0.63 (0.56, 0.72)
Model 3	7971.09	1.65	3	.65	2223.09	0.31 (0.11, 0.45)	0.09 (0.00, 0.27)	0.60 (0.55, 0.66)	0.31 (0.11, 0.45)	0.09 (0.00, 0.27)	0.60 (0.55, 0.66)

Note: Model 1: A, C, and E parameters and mean values were freely estimated. Model 2: A, C, and E parameters were freely estimated but mean values were equated. Model 3: A, C, and E parameters and mean values were equated across genders.

TABLE 3

Parameter Estimates With 95% Confidence Intervals and Fit Indices Across Age Groups

						Younger (11–13 years)			Older (14–17 years)		
Model	-2LL	$\Delta\chi^2$	Δdf	p	AIC	A	C	E	A	C	E
Model 1	7972.27				2244.28	0.30 (0.00, 0.45)	0.06 (0.00, 0.36)	0.64 (0.55, 0.72)	0.32 (0.06, 0.47)	0.09 (0.00, 0.33)	0.59 (0.52, 0.66)
Model 2	7972.28	0.01	7	1.0	2230.28	0.30 (0.00, 0.45)	0.06 (0.00, 0.36)	0.64 (0.55, 0.72)	0.32 (0.06, 0.47)	0.09 (0.00, 0.33)	0.59 (0.52, 0.66)
Model 3	7972.98	0.70	3	.87	2224.98	0.31 (0.11, 0.45)	0.09 (0.00, 0.27)	0.60 (0.55, 0.66)	0.31 (0.11, 0.45)	0.09 (0.00, 0.27)	0.60 (0.55, 0.66)

Note: Model 1: A, C, and E parameters and mean values were freely estimated. Model 2: A, C, and E parameters were freely estimated but mean values were equated. Model 3: A, C, and E parameters and mean values were equated across age groups.

computed in MZ and DZ twins. For the entire sample, the magnitude of the correlation coefficient in MZ twins ($r = 0.39$) was greater than the magnitude of the correlation coefficient in DZ twins ($r = 0.25$), but less than twice the correlation coefficient in DZ twins. Similar patterns were noted when the Pearson correlations were computed separately by gender or age groups (aged 11–13 years vs. 14–17 years; see the third row in Table 1). These results suggest additive genetic, and shared and non-shared environmental influences on adolescent dysfunctional attitudes (i.e., ACE model).

Model-Fitting Analyses

The results of model-fitting across genders are shown in Table 2. The difference in fit between models 1 and 2 was not significant, $\Delta\chi^2$ (7) = 0.01, $p = 1.0$, as well as between models 2 and 3, $\Delta\chi^2$ (3) = 0.70, $p = .87$, suggesting that both mean level and magnitudes of genetic and environmental influences on dysfunctional attitudes did not differ between the younger (aged 11–13 years) and older (aged 14–17 years) groups. Thus, we found that across gender and age, the magnitudes of genetic, shared, and non-shared environmental influences on adolescent dysfunctional attitudes were 31% (95% CI: 11%, 45%), 9% (95% CI: 0%, 27%), and 60% (95% CI: 55%, 66%), respectively.

The results of model-fitting across age groups, as shown in Table 3, demonstrated similar patterns. The difference in fit was not significant between models 1 and 2, $\Delta\chi^2$ (7) = 0.01, $p = 1.0$, as well as between models 2 and 3, $\Delta\chi^2$ (3) = 0.70, $p = .87$, suggesting that both mean level and magnitudes of genetic and environmental influences on dysfunctional attitudes did not differ between the younger (aged 11–13 years) and older (aged 14–17 years) groups. Thus, we found that across gender and age, the magnitudes of genetic, shared, and non-shared environmental influences on adolescent dysfunctional attitudes were 31% (95% CI: 11%, 45%), 9% (95% CI: 0%, 27%), and 60% (95% CI: 55%, 66%), respectively.

Discussion

Even though dysfunctional attitude has been demonstrated as a vulnerability to adolescent and adult depression, little is known about the genetic and environmental origins of dysfunctional attitudes. The present study, for the first time, quantitatively investigated genetic and environmental influences on individual differences in dysfunctional attitudes using a twin design.

Our results showed that genetic factors play an important role in the etiology of adolescent dysfunctional attitudes, which accounted for approximately 31% of variation. The moderate heritability of dysfunctional attitude was slightly

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lower than that found for depressive symptoms in Western (Rice & Thapar, 2009) and East Asian youth (Hur, 2008). However, the genetic effect in dysfunctional attitude is very similar to the findings from twin studies of other cognitive vulnerabilities in depression. Specifically, Lau et al. (2006) demonstrated that the heritability of adolescents' attributional style (i.e., attributing negative events to internal, stable, and global causes) was 35%. Eley et al. (2008) showed that genetic factors accounted for 30% of variation in children's interpretation bias (i.e., interpretating ambiguous information as threatening). Moderate genetic effects were also reported for cognitive bias associated with anxiety and depressive symptoms in adolescents, which are referred to as sensitivity to symptoms of anxiety (attentional bias) and the belief that these are harmful (Zavos et al., 2010, 2012). Together with our findings, accumulating evidence suggests that cognitive vulnerabilities to depression have genetic roots and may function as an endophenotype to identify risk genes for depression. Indeed, several prior studies have indicated that several specific genetic loci (e.g., BDNF Val66Met and 5-HTTLPR) are associated with dysfunctional attitudes (Wells et al., 2010; Whisman et al., 2011).

The results of the present study also indicated differential effects of shared and non-shared environmental factors in the etiology of dysfunctional attitudes. Shared environmental influence was small, only accounting for 9% (0%, 27%) of variation, whereas non-shared environmental influence was large, accounting for 60% (55%, 66%) of variation. These findings are consistent with estimates in studies of depression and anxiety (Rice & Thapar, 2009). These findings also indicated that the environmental factors associated with dysfunctional attitudes, such as emotional maltreatment, verbal peer victimization, and negative feedback from parents, may exert influences through a non-shared environmental mechanism. Future studies using an MZ twin difference design may test this hypothesis empirically.

We found no significant difference in the mean level of dysfunctional attitudes across gender or age groups, which is consistent with prior findings (Abela & Skitch, 2007; Abela et al., 2011). Moreover, the magnitude of genetic and environmental influences on adolescent dysfunctional attitudes did not differ significantly between females and males, and between the younger (aged 11–13 years) and older (aged 14–17 years) groups. However, there were some interesting trends. Specifically, the genetic effects were stronger in females (32%) than in males (28%), and stronger in older adolescents (32%) than in younger adolescents (30%). These differences were not statistically significant and might be due to the relatively smaller sample size of this study. Thus, future research with a larger sample size may provide more definitive conclusions.

The present study had several limitations. First, we used the classic twin design to partition genetic, shared, and non-shared environmental influences on dysfunctional attitudes. Twin studies are susceptible to a violation of the equal environments assumption, which overestimates the importance of genetic effects. Fortunately, several studies have provided evidence of the validity of the equal environments assumption (Derks et al., 2006; Eriksson et al., 2006; Klump et al., 2000). Second, this study was based on participants from the general population. It remains to be seen whether the results can be generalized to clinical populations. It is noteworthy that the general population, rather than clinical samples, may be more appropriate for quantitative genetic studies, as the latter may restrict the variance of measures, and therefore reduce statistical power (Waldman, 2005). Third, as this finding was based on data collected in Chinese Han adolescents, the results may not be generalized to other ethnic groups. Nevertheless, we found no significant mean difference of dysfunctional attitudes between our sample and a European sample. Future studies are needed to investigate whether there are some culture-specific negative attitudes in the Chinese population.

Despite these limitations, the present study has potential implications. Previous studies have demonstrated dysfunctional attitudes as cognitive vulnerability to depression. The finding of moderate heritability in this study provides additional evidence indicating dysfunctional attitudes to be an endophenotype for depression. Future studies can use dysfunctional attitudes to identify risk genes for depression. Moreover, evidence of genetic influence on dysfunctional attitude and other cognitive vulnerabilities has reframed the traditional view of cognitive model of depression. The traditional view proposes that the formation of dysfunctional schemas is due to early adverse experiences. The dysfunctional schemas can be activated by later stressful events, and schema activation then influences individual's perceiving, encoding, and retrieving information, thus generate other cognitive vulnerabilities such as attention bias, interpretation bias, and negative attribution styles. The current study and previous findings of genetic influences on other cognitive vulnerabilities suggest that genes do play an important etiological role in the development of cognitive vulnerability to depression.

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