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ORIGINAL RESEARCH

Genetic and Environmental Influences on Depressive Symptoms in Chinese Adolescents

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Abstract Adolescent depression is common and has become a major public health concern in China, yet little research has examined the etiology of depression in Chinese adolescents. In the present study, genetic and environmental influences on Chinese adolescent depressive symptoms were investigated in 1,181 twin pairs residing in Beijing, China (ages 11–19 years). Child- and parent-versions of the children's depression inventory were used to measure adolescents' depressive symptoms. For self-reports, genetic factors, shared environmental factors, and non-shared environmental factors accounted for 50, 5, and 45 % of the variation in depressive symptoms, respectively; for parent-reports, genetic factors, shared environmental factors, and non-shared environmental factors accounted for 51, 18, and 31 % of the variation, respectively. These estimates are generally consistent with previous findings in Western adolescents, supporting the cross-cultural generalizability of etiological model of adolescent depression. Neither qualitative nor quantitative sex differences were found in the etiological model. Future studies are needed to investigate how genes and environments work together (gene–environment interaction, gene–environment correlation) to influence depression in Chinese adolescents.

Keywords Adolescent · Depressive symptoms · Genetic and environmental influences · Chinese twins

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Introduction

Epidemiological studies suggest that the prevalence of child and adolescent depression in China is rising in recent years (Dennis 2004; Lee et al. 2009; Lu et al. 2008; Ma et al. 2009; Phillips et al. 2009). For example, in a community-based sample of 6–14 year-old children in Wuhan, a large metropolitan city in south-central China, the prevalence of current clinically-diagnosed depressive disorder was found to be 2.8 % (Zhong et al. 2013), which is comparable with the results of surveys in Western countries (Costello et al. 2003; Demir et al. 2011; Ford et al. 2003). Several questionnaire-based studies reported similar or even higher prevalence rates of depression in Chinese adolescents (Abela et al. 2011; Greenberger et al. 2000; Happonen et al. 2002; Liu et al. 1999; Sun et al. 2010) than in Western adolescents (Merikangas et al. 2011; Rushton et al. 2002; Thapar et al. 2012). Emerging evidence therefore suggests that adolescent depression has become a major public health concern in China, yet little research has examined the etiology of depression in Chinese adolescents, and in particular, how genetic and environmental factors influence adolescent depression.

Etiological research in Western populations suggests that both genetic and social factors contribute to adolescent depression (Rice et al. 2002a; Rice and Thapar 2009). One

meta-analysis of twin and adoption studies (including 17 samples with 21,027 sibling pairs) found that genetic factors accounted for 44 % of the variance, while shared and non-shared environments accounted for 14 and 42 % of the total variation in child and adolescent depression, respectively (Burt 2009). Albeit extensive, previous studies have almost entirely employed Western samples, thus, it is still unknown whether prior findings can be generalized to other ethnic groups, and specifically to a population with genetic and cultural differences compared to their Western counterparts, such as residents of China, as detailed below.

The first aim of the present study was to examine the genetic and environmental contributions to adolescent depressive symptoms in a large Chinese adolescent twin sample. There are good reasons to expect somewhat different genetic and environmental influences on depression in Chinese adolescents relative to Western populations. Genetically, evidence has shown that the frequencies of some depression-related genetic variants (e.g. BDNF val66met polymorphism and 5-HTTLPR) are different between Eastern and Western populations (Chiao and Blizinsky 2010; Verhagen et al. 2008). In addition, the same allele may serve as a risk allele in one population (Western or Eastern), but as a protective allele in the other (Eastern or Western) (Chen et al. 2012; Long et al. 2013; Zhang et al. 2009). The environmental factors associated with depression may also differ for youth living in different cultures (Western vs. Eastern). Chinese adolescents live in a very different social context compared to Western adolescents. For example, students in China bear heavier academic stress and higher levels of parental expectations, which may render them more depressed than their Western counterparts (Hou et al. 2012). The mental health service utilization rates, however, are lower in Chinese children and adolescents compared with those in Western countries (Ryder et al. 2012). According to the bioecological model (Bronfenbrenner and Ceci 1994) and some empirical studies (Hicks et al. 2009; Lau and Eley 2008; Rice et al. 2006), social factors, such as family, school, and the peer group, can influence the expression of genetic predispositions to depression. Because there are differences in proximal social contexts surrounding adolescents between Eastern and Western cultures (Greenberger et al. 2000; Zhou et al. 2008), the extent to which genetic and environmental factors contribute to Chinese adolescents' depression may differ from Western populations.

The second aim of the present study was to examine sex differences with regard to genetic and environmental influences on adolescent depression. Studies in Western samples have shown that girls start to exhibit more depressive symptoms than boys at approximately age 13, and that this sex difference continues into adulthood (Ge et al. 1994; Hankin et al. 1998; Nolen-Hoeksema and Girgus 1994). This robust finding, however, has not been well replicated in samples of Chinese adolescents. Some studies have find more depressive symptoms in girls than in boys (Abela et al. 2011; Greenberger et al. 2000), but some studies have found no significant sex differences in mean levels of depressive symptoms (Liu et al. 1999; Tepper et al. 2008) and other studies have found greater rates of depressive symptoms in boys than in girls (Hong et al. 2009; Sun et al. 2010). These inconsistent findings prompted our interest exploring whether the genetic and environmental etiology of depression in Chinese adolescents differs by sex.

Behavioral genetic research on sex differences in the etiology of depression has considered both qualitative (i.e., there are some sex-specific genetic and/or environmental contributors to adolescent depression) and quantitative (i.e., the same genetic and environmental factors contribute to boys' and girls' depression, but these contributors influence the two sexes unequally) sex differences. After reviewing 34 twin studies, Franic et al. (2010) concluded that there were no qualitative sex differences in genetic and environmental etiologies of childhood and adolescent depression. However, the results of quantitative sex differences varied across studies. For example, Rice et al. (2002b) and Eley and Stevenson (1999) found stronger genetic effects on adolescent boys' than on adolescent girls' self-reported depressive symptoms. Conversely, Scourfield et al. (2003) found a greater genetic influence for girls than for boys using parent-report data. Studies based on data from the National Longitudinal Study of Adolescent Health also reported larger genetic influences on self-reported depressive symptoms for female than for male adolescents (Cho et al. 2006; Jacobson and Rowe 1999; McCaffery et al. 2008). In addition, three studies reported no sex differences in the magnitude of genetic and environmental influences on adolescent depressive symptoms (Eaves et al. 1997; Happonen et al. 2002; Lau and Eley 2006).

To our knowledge, only two twin studies in Eastern cultures have examined genetic and environmental influences on depression and sex differences associated with these influences. Using 490 pairs of adolescent and young adult twins (aged 13–23 years) from the South Korean Twin Registry (SKTR), Hur (2008) reported significant genetic and non-shared environmental effects on depressive symptoms in females (explained 41 and 59 % of total variance, respectively), but no significant genetic influences in males (shared and non-shared environmental effects explained 34 and 66 % of total variance, respectively). In another study with using 602 pairs of adolescent twins (ages: 11–19 years) from the Qingdao Twin Registry (QTR) in China, Unger et al. (2011) found no significant genetic influences on depressive symptoms in either girls

or boys, while shared and non-shared environmental factors significantly explained 38 and 62 % of total variance.

These findings seemed to suggest higher shared environmental effects and lower genetic effects (both males and females in QTR, males in SKTR) on depression in Eastern cultures, compared with the findings in Western populations. However, several limitations should be kept in mind when interpreting the results of the two previous studies. First, both studies were based on relatively small samples, especially when considering the number of dizygotic (DZ) twin pairs (QTR: 81 pairs of female DZ twins, 81 pairs of male DZ twins, 119 pairs of opposite-sex DZ twins; SKTR: 56 pairs of female DZ twins, 41 pairs of male DZ twins, 51 pairs of opposite-sex DZ twins). The small sample size limited the statistical power to draw reliable conclusions. Second, both studies only used self-report method to assess adolescents' depressive symptoms. Previous research has suggested that, twin studies based on parent-report data can provide additional information regarding the etiology of adolescent depression (Happonen et al. 2002).

In order to get a more accurate estimation of genetic and environmental effects in the two sexes, the present study used the data from the Beijing Twin Study (BeTwiSt) (Chen et al. 2013). The BeTwiSt is a longitudinal twin study based on a large and representative twin sample in Beijing, China. Both self-reported and parent-reported adolescent depressive symptoms were collected to provide perspectives from multiple informants. Both qualitative and quantitative sex differences in the etiological models were examined by employing both same-sex and opposite-sex twins.

Method

Sample

This study was based on data from the BeTwiSt, a longitudinal twin study of Chinese adolescents. The primary aim of the BeTwiSt was to examine how genes, environment, and their interplay influence psychological development and mental health problems among Chinese adolescents. Approximately 1,400 pairs of twins aged 10–19 years were recruited from elementary and secondary schools randomly selected from all 18 counties or districts in the Beijing municipality. Detailed information about recruitment and assessment procedures are described elsewhere (Chen et al. 2013).

The sample for the present study included 1,181 pairs of twins. The breakdown by zygosity is as follows: 338 pairs of monozygotic male (MZM) twins, 122 pairs of dizygotic male (DZM) twins, 359 pairs of monozygotic female MZF twins, 148 pairs of dizygotic female (DZF) twins, and 214 pairs of opposite-sex dizygotic (DZOS) twins. The age of the study sample ranged from 11 to 19 years, with a mean age of 14.17 years ($SD = 2.28$ years). Ninety-two percent of participants were of Han ethnicity. Regarding fathers' highest educational attainment, 6.8 % had a primary school degree, 32.8 % had a junior high school degree, 31.8 % had a senior high school degree, 26.1 % had a college degree, and 2.5 % had a graduate degree. For the mothers' highest educational attainment, 5.4 % had a primary school degree, 35.3 % had a junior high school degree, 29.5 % had a senior high school degree, 25.2 % had a college degree, and 4.6 % had a graduate degree. Self- and parent-reported adolescent depressive symptoms during wave 1 were used in the present study. We asked one parent from each family, the one who knew their twin children better, to complete the parent-report questionnaire. Sixty-five percent of parent respondents were mothers, 34 % were fathers, and 1 % were other caregivers. The representativeness of the twin sample is described in a previous study (Chen et al. 2013). Specifically, we compared the basic demographic characteristics of the BeTwiSt sample to a population-based representative youth population in Beijing (Chen et al. 2009). No significant differences were detected in terms of perceived family social economic status, fathers' educational attainment, or parents' marital status or marital quality. The mothers' educational levels were significantly higher in the twin sample than in the general youth sample. Overall, these comparisons suggest that the representativeness of the twin sample was acceptable.

Measures

Depressive symptoms

Self-reported depressive symptoms were assessed with the Children's Depression Inventory (CDI) (Kovacs and Staff 2003), one of the most widely used measures of depression in children and adolescents (Mash and Hunsley 2005). The youth were asked to assess their behaviors over the previous 2 weeks. Each of the CDI items consists of three choices, with higher scores indicating higher severity: "1" = the absence of symptoms; "2" = mild symptoms; "3" = definite symptoms. The total scores ranged from 27 to 81. Psychometric studies of the CDI have shown a high degree of internal consistency, test-retest reliability, and construct validity, especially in nonclinical populations (Cole and Martin 2005). The Chinese version of the CDI (Chen et al. 2000) was used in this study. The internal consistency of the CDI in our sample was acceptable, with Cronbach's alpha of 0.86.

Parents reported on children's depressive symptoms via the CDI parent form (CDI-PF) (Kovacs and Staff 2003).

The CDI-PF retains the original content of the self-report CDI items, but is reworded for parental use. For example, the item "I feel sad all the time" is rephrased into "My child feels sad all the time." Previous studies show satisfactory psychometric characteristics of the CDI-PF (Cole et al. 2002; Tram and Cole 2006). In the current study, cronbach's alpha was .87.

Zygosity determination

The twins' zygosity was determined by a validated method combining DNA analysis (89.5 % of twins) and questionnaire data (10.5 % of twins) (Chen et al. 2010). For the DNA analyses, nine short tandem repeat loci, which are highly heterogeneous in the Chinese population, were used. Same-sex twins with at least one different genetic marker were classified as dizygotic twins; otherwise, the twins were classified as monozygotic (MZ) twins. The posterior probability of being MZ for same-sex twins with the same genotype in all nine loci was estimated to be 99.99 %. The validity of the zygosity determination questionnaire was examined by comparing it with the results of the DNA analyses. The predictive accuracy of the questionnaire method used in this study reached 91 % (Chen et al. 2010).

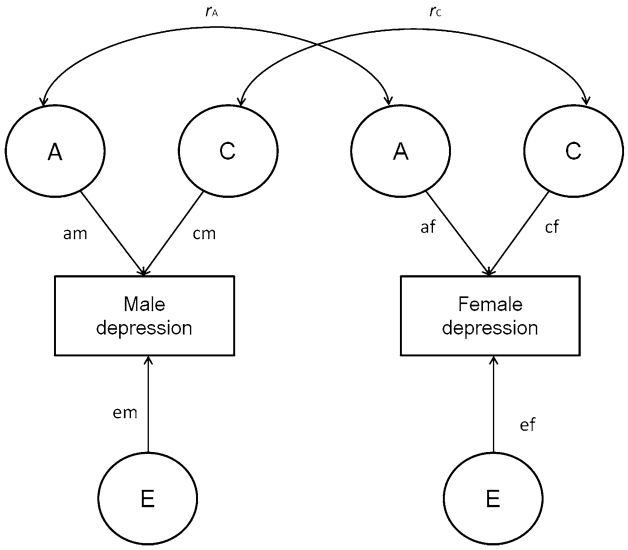

Fig. 1 *Sex-limitation model for adolescent depressive symptoms*. The magnitude of additive genetic (**a**), shared environmental (**c**), nonshared environmental (**e**) influences may differ for males and females (am ≠ af, cm ≠ cf, em ≠ ef), and/or the genetic (r_A) or shared environmental (r_C) correlation among opposite-sex twins may fall below the expected genetic (0.50) and shared environmental (1.00) correlations for same sex dizygotic twins

Assessment procedure

All twins and their parents signed informed consents before participation. Arrangements were made for the twins to stay in their classrooms after school. After describing the purpose and procedures of the study, trained research staff distributed the questionnaires to the twin participants and instructed them to complete the questionnaires independently. Research staff members were present to answer any questions that the students might have about the questionnaires. Participants were assured of the confidentiality of their responses and the voluntary nature of their participation. All procedures had been approved by the Institutional Review Board.

Data analysis

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 is used to estimate the contribution of genetic and environmental factors to the individual differences in the phenotype of interest. As the current study aimed to clarify both qualitative and quantitative sex differences in genetic and environmental influences on adolescent depressive symptoms, the Pearson correlations of five twin groups (i.e., MZM, MZF, DZM, DZF, and DZOS) were computed first. To obtain parameter estimates of genetic, shared, and non-shared environmental effects, and to test for sex difference in etiological model, maximum-likelihood model fitting to the variance-covariance matrices using the structural equation modeling package Mx (Neale et al. 2006) was undertaken.

To examine both qualitative and quantitative sex differences, it is necessary to first fit a full sex-limitation model and then two nested sub-models, which progressively model fewer parameters (Fig. 1). In *the full sex-limitation model*, additive genetic (A), shared (C) and nonshared environmental (E) parameters were allowed to differ between males and females, assuming that the magnitudes of influence of A, C, and E on depressive symptoms may vary in males and females. For DZOS twins, the correlations for the A factors (r_A) and C factors (r_C) were estimated freely. Because a model estimating both r_A and r_C simultaneously is not identifiable, the two correlations were estimated separately in two non-nested models. The fits of these two non-nested models were compared with the akaike information criterion (AIC) and the model with the smaller AIC was selected as the best fitting model. Then, data were fit into the first nested submodel, *the common effect model*, which constrained the DZOS twins' r_A to 0.5 or r_C to 1.0 but allowed the A, C, E parameters for males and females to differ. The significance of difference in fits between the common effect model and the full sex-limitation model was tested to examine qualitative sex differences. The second nested

Table 1 Means and standard deviations of depressive symptoms grouped by zygosity and sex

	Self-reports		Parent-reports	
	Mean	SD	Mean	SD
MZT Female	36.82	6.40	33.04	4.69
DZT Female	37.55	6.62	33.62	5.18
MZT Male	37.70	6.77	33.72	5.75
DZT Male	37.73	6.87	34.65	5.37
OST Female	37.52	6.38	33.85	5.90
OST Male	37.23	6.69	33.54	4.97
Total	**37.35**	**6.60**	**33.58**	**5.26**

MZT monozygotic twins, *DZT* dizygotic twins, *OST* opposite-sex twins, *SD* standard deviation

sub-model is *the scalar model*, which constrains the DZOS twins' r_A to 0.5 or r_C to 1.0 and constraint the A, C, E parameters for males and females to be equal. The scalar model also allows sex differences in the variance of the phenotype. The significance of difference in fits between the scalar model and the common effect model tested quantitative sex differences. The self- and parent-reported adolescent depressive symptoms were examined separately.

Results

The descriptive statistics for self- and parent-reported adolescent depressive symptoms in groups by zygosity and sex are shown in Table 1. Sex and age differences in the mean level of depressive symptoms were first examined. We found no significant sex differences in the means of either self- ($t = -1.26$, $p = 0.21$) or parent-reported adolescent depressive symptoms ($t = -1.24$, $p = 0.18$). Adolescent depressive symptoms increased significantly with age (self-report: $r = 0.12$, $p < 0.001$; parent-report: $r = 0.11$, $p < 0.01$). The correlation between self- and parent-report data was moderate and significant ($r = 0.50$, $p < 0.001$). The residuals after regressing out the effects of sex and age were used in following analyses (McGue and Bouchard 1984).

The Pearson correlations for the five groups are shown in Table 2. For both self- and parent-report data, the twin correlations of MZ twins were larger than those of the same-sex and opposite-sex DZ twins, suggesting substantial genetic influences. Furthermore, the twin correlations of DZOS twins were similar to those of same-sex DZ twins, suggesting that there may be trivial qualitative sex differences.

Table 3 provides the model-fitting results and parameter estimates for self- and parent-reported adolescent depressive symptoms. For the self-report data, the fits of the full sex-limitation models (full r_A and r_C) were very similar; we selected the full r_A model as the baseline model. When the freely estimated r_A was set to 0.5 (common effect model), there was no significant change in Chi square ($\Delta\chi^2$ (1) = 0, $p > 0.5$), suggesting no qualitative sex differences. Allowing the genetic and environmental estimates to be equal between the sexes (scalar model) resulted in a non-significant change in Chi square ($\Delta\chi^2$ (3) = 3.10, $p > 0.2$), indicating no quantitative sex differences. Thus, the best-fitting model for self-reported depressive symptoms was the scalar model with no qualitative and quantitative sex differences, in which genetic factors accounted for 50 %, while non-shared environmental factors accounted for 45 % of individual differences in adolescent depression. The shared environmental effect was negligible and not significant statistically.

For the parent-report data, the full r_A model was also selected as the baseline model, as the fits of the two full sex-limitation models (full r_A and r_C) were very similar. When the freely estimated r_A was set to 0.5 (common effect model), there was no significant change in Chi square ($\Delta\chi^2$ (1) = 0, $p > 0.5$). Additionally, allowing the genetic and environmental estimates to be equal across sexes (scalar model) resulted in a nonsignificant change in Chi square ($\Delta\chi^2$ (3) = 3.10, $p > 0.2$). The best-fitting model for parent-reported depressive symptoms was also the scalar model, in which genetic factors accounted for 51 %, while non-shared environmental factors accounted for 31 % of individual differences in adolescent depression. Furthermore, unlike the self-reports, the shared environmental effect for parent-reported adolescent

Table 2 Twin correlations for age-regressed Chinese adolescents' depressive symptoms

Depressive symptoms	Twin correlations				
	Monozygotic twins		Dizygotic twins		
	Male	Female	Male	Female	Opposite-sex
Child-report	0.52 (285)	0.56 (342)	0.27 (106)	0.38 (142)	0.27 (187)
Parent-report	0.74 (225)	0.60 (278)	0.41 (87)	0.47 (117)	0.48 (150)

All correlation coefficients were significant at $p < 0.01$ level. The number of twin pairs is shown in brackets

Table 3 Parameter estimates and fit indices for Chinese adolescents' twin data

Model	*df*	χ^2	AIC	Male				Female				Opposite-sex twins	
				A	C	E		A	C	E		r_A	r_C
Self-report													
Full r_A	8	22.18	6.18	0.49(0.22, 0.59)	0.03(0.00, 0.27)	0.48(0.40, 0.56)		0.50(0.25, 0.64)	0.07(0.00, 0.30)	0.42(0.36, 0.50)		0.50(0.12, 0.50)	1.0
Full r_C	8	22.18	6.18	0.49(0.22, 0.59)	0.03(0.00, 0.27)	0.48(0.40, 0.56)		0.50(0.25, 0.64)	0.07(0.00, 0.30)	0.42(0.36, 0.50)		0.50	1.0(0.00, 1.0)
Common	9	22.18	4.18	0.49(0.22, 0.59)	0.03(0.00, 0.27)	0.48(0.40, 0.56)		0.50(0.25, 0.64)	0.07(0.00, 0.30)	0.42(0.36, 0.50)		0.50	1.0
Scalar	**12**	**25.28**	**1.28**	**0.50(0.32, 0.60)**	**0.05(0.00, 0.21)**	**0.45(0.40, 0.51)**		**0.50(0.32, 0.60)**	**0.05(0.00, 0.21)**	**0.45(0.40, 0.51)**		**0.50**	**1.0**
Parent-report													
Full r_A	8	25.62	9.62	0.54(0.29, 0.74)	0.19(0.06, 0.43)	0.27(0.23, 0.34)		0.48(0.24, 0.67)	0.17(0.01, 0.38)	0.35(0.29, 0.42)		0.50(0.25, 0.50)	1.0
Full r_C	8	25.62	9.62	0.54(0.29, 0.74)	0.19(0.06, 0.43)	0.27(0.23, 0.34)		0.48(0.24, 0.67)	0.17(0.01, 0.38)	0.35(0.29, 0.42)		0.50	1.0(0.40, 1.0)
Common	9	25.62	7.62	0.54(0.29, 0.74)	0.19(0.06, 0.43)	0.27(0.23, 0.34)		0.48(0.24, 0.67)	0.17(0.01, 0.38)	0.35(0.29, 0.42)		0.50	1.0
Scalar	**12**	**28.72**	**4.72**	**0.51(0.35, 0.69)**	**0.18(0.01, 0.32)**	**0.31(0.27, 0.36)**		**0.51(0.35, 0.69)**	**0.18(0.01, 0.32)**	**0.31(0.27, 0.36)**		**0.50**	**1.0**

95 % Confidence intervals are shown in brackets.

Full r_A = full sex-limitation model allowing for quantitative sex differences, qualitative genetic sex differences, *full* r_C = full sex-limitation model allowing for quantitative sex differences, qualitative shared environmental sex differences, *Common* = common effects model allowing for quantitative sex differences, *Scalar* = scalar model, *AIC* = akaike's information criterion, *A, C, and E* = the proportion of variance in depressive symptoms explained by additive genetic, shared environmental, and nonshared environmental influences, respectively, r_A = the genetic correlation among opposite-sex twin pairs, r_C = the shared environmental correlation among opposite-sex twin pairs

depression was modest and significant, explaining 18 % of variation.

Discussion

Given the differences in genetic makeup and social contexts between Asian and Western countries, it is necessary to re-examine the genetic and environmental influences on adolescent depression and sex differences in Asian samples. Adolescent depression is common and has become a major public health concern in China, the country with the largest youth population. However, little research has examined the etiology of depression in Chinese adolescents. Thus, investigating the genetic and environmental etiology of depression has potential implications, especially for prevention and intervention of Chinese adolescent depression.

Behavioral genetic research in Western populations has demonstrated that adolescent depression was moderately heritable and non-shared environmental factors were an important contributor. We obtained similar findings in the present study. Our results showed that the heritability of depressive symptoms in Chinese adolescents was moderate (genetic factors explained about 50 % of variation); non-shared environmental factors accounted for a large amount of individual differences (45 % for self-reports, and 31 % for parent-reports). The consistency between findings of this study and the previous studies that had used Western population samples supports a cross-cultural generalizability of etiological models of adolescent depression.

Shared environmental effects were found to be modest in parent-reports, but negligible in self-reports. The higher estimates of shared environmental effects in parent-reported data might be due to shared rater effects (Bartels et al. 2004). We found similar moderate estimate of genetic influences on parent-report compared to self-report, which was consistent with previous studies with multiple informants (Happonen et al. 2002). As a whole, the genetic and environmental estimates in the current study were similar to findings in samples of Western adolescents (Burt 2009; Rice and Thapar 2009; Rice 2010).

However, our results were inconsistent with the previous two twin studies utilizing Asian samples. The study of the QTR in China suggested no genetic influences on depressive symptoms, but significant shared and non-shared environmental influences (Unger et al. 2011). Hur (2008) did find significant genetic influences on depression among South Korean females, but no genetic influences on depression for South Korean males. Different measurements might account for the discrepancy between the current study and two previous Asian studies (CDI vs. CES-D). Studies including multi-wave surveys showed

that CDI scores are more stable and represent more stable trait component, compared with CES-D scores (Cole and Martin 2005; Kenny and Zautra 2001; Tram and Cole 2006; Windle and Dumenci 1998). We speculate that the different time span referred to in the two measures may explain this phenomenon, i.e., the CES-D assesses individual's feelings or behaviors during the past 1 week, while the CDI measures individual's mood or behaviors during the past 2 weeks. Thus, it is possible that the depressive symptoms measured by the CDI have more genetic origins than symptoms assessed by the CES-D because they reflect a more stable and persistent set of symptoms. Similarly, previous empirical twin studies using Western populations have also found higher heritability of CDI scores (Eley 1997; Eley and Stevenson 1999; Happonen et al. 2002; Lau and Eley 2008; O'Connor et al.1998) than CES-D scores (Byers et al. 2009; Jacobson and Rowe 1999; Jansson et al. 2004; McCaffery et al. 2003, 2008). This finding reminds us that the specific measurements selected might affect heritability estimates of adolescent depression.

The mean level difference of adolescent depressive symptoms between sexes is non-significant in our sample. This finding is consistent with several previous studies with Chinese adolescent samples (Liu et al. 1999; Tepper et al. 2008), but is inconsistent with the robust finding in Western adolescents—higher level of depression in girls than in boys (Hankin et al. 1998; Nolen-Hoeksema and Girgus 1994). So far, to the best of our knowledge, few theories or hypotheses have been proposed to explain this interesting difference between Western and Chinese studies. We speculate that the cultural differences in the sex-differentiated impact of cognitive vulnerabilities to depression may partially account for this finding. As studies in Western samples demonstrate that the cognitive vulnerabilities (e.g. rumination) mediate the sex difference in adolescent depression (Nolen-Hoeksema 2012), but studies in Chinese adolescents have not replicated these findings (Hong et al. 2010). More empirical studies are needed to delineate the underlying mechanisms.

Regarding the sex differences in the etiologies of adolescent depression, we found no evidence of qualitative sex differences in the genetic and environmental influences on depressive symptoms in Chinese adolescents, which is consistent with findings in Western adolescents (Rice and Thapar 2009). We also found no sex differences in the quantity of genetic and environmental effects on Chinese adolescents' depression, which is consistent with several studies of Western adolescents (Eaves et al. 1997; Happonen et al. 2002; Lau and Eley 2006; Burt 2009). This finding, however, is inconsistent with the findings of Hur (2008). She reported a significant sex difference, with moderate genetic effects (41 % of variation) in females, no genetic effects but modest shared environmental effects (34 % of variation) in males. The discrepancy of evidence regarding sex differences in magnitude of genetic and environmental influences on adolescent depression has also been demonstrated in Western twin studies. Some studies have found greater genetic influences on depression in girls than in boys (Cho et al. 2006; Jacobson and Rowe 1999; McCaffery et al. 2008; Scourfield et al. 2003), whereas other studies have demonstrated opposite findings (Eley and Stevenson 1999; Rice et al. 2002b). Additional studies are needed to clarify the sex difference in genetic and environmental etiologies of adolescent depression, especially for Asians.

Conclusion

The heritability of Chinese adolescents' depressive symptoms was moderate, and the remainder of variance was mainly explained by nonshared environmental factors. Modest shared environmental influences were found in parent-reported adolescent depressive symptoms. The same genetic and environmental factors influence depressive symptoms in Chinese adolescent males and females, with similar quantity. These findings generally support the cross-cultural generalizability of the etiological model of adolescent depression. Future studies can be conducted to investigate the underlying mechanisms of how genes and environments act together to influence depression in Chinese adolescents.

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