Genetic and environmental contributions to anxiety among Chinese children and adolescents – a multi-informant twin study

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Background: Child and adolescent anxiety has become a major public health concern in China, but little was known about the etiology of anxiety in Chinese children and adolescents. The present study aimed to investigate genetic and environmental influences on trait anxiety among Chinese children and adolescents. Rater, sex, and age differences on these estimates were also examined. Methods: Self-reported and parent-reported child’s trait anxiety was collected from 1,104 pairs of same-sex twins aged 9–18 years. Genetic models were fitted to data from each informant to determine the genetic (A), shared (C), and non-shared environmental (E) influences on trait anxiety. Results: The parameter estimates and 95% confidence intervals (CI) of A, C, and E on self-reported trait anxiety were 50% [30%, 60%], 13% [1%, 28%], and 24% [22%, 27%], respectively. The heritability of anxiety was higher in girls for self-reported data, but higher in boys for parent-reported data. There was no significant age difference in genetic and environmental contributions for self-reported data, but a significant increase of heritability with age for parent-reported data. Conclusions: The trait anxiety in Chinese children and adolescents was highly heritable. Non-shared environmental factors also played an important role. The estimates of genetic and environmental effects differed by rater, sex and age. Our findings largely suggest the cross-cultural generalizability of the etiological model of child and adolescent anxiety. Keywords: Anxiety, Chinese children and adolescents, genetic and environmental influence, heritability, twin study.

Introduction
Anxiety is a common mental health problem in childhood and adolescence (Rapee, Schniering, & Hudson, 2009). Both subthreshold anxiety symptoms and clinical anxiety disorder are associated with significant functional impairments, onset of adult anxiety disorders, and suicide risk (Balazs et al., 2013). With rapid economic growth and drastic social transformation in China during the past two decades, child and adolescent mental health deteriorated with increased amounts of anxiety and other problems (Xin, Zhang, & Liu, 2010). Although without nationally representative epidemiological data in China, existing community studies depicted alarming statistics. For example, the three-month and lifetime prevalence of anxiety disorder in Chinese youths is 3.1%–9.2% (Gau, Chong, Chen, & Cheng, 2005) and 30.2% (Leung et al., 2008), comparable to those in Western samples (Rapee et al., 2009). Several survey studies have reported similar or an even higher level of anxiety symptoms in Chinese youths than in their Western counterparts (Su, Wang, Fan, Su, & Gao, 2008; Zhao, Xing, & Wang, 2012). Child and adolescent anxiety has become a major public health concern in China. However, little research has examined the etiology of anxiety in Chinese children and adolescents, especially from the genetic perspective.

Etiological research in Western countries supported both genetic and environmental roles in the etiology of child and adolescent anxiety (Gregory & Eley, 2007; Rice & Thapar, 2009). A meta-analysis of twin and adoption studies showed that genetic factors accounted for 48%, while shared and non-shared environments accounted for 12% and 40% of variations in child and adolescent anxiety respectively (Burt, 2009). Further investigations concluded no qualitative sex difference in genetic and environmental etiologies of child and adolescent anxiety (Franić, Middeldorp, Dolan, Ligthart, & Boomsma, 2010). However, the quantitative sex differences findings varied across studies. Specifically, several studies with self-reported anxiety suggested higher heritability in girls than in boys (Bartels, van de Aa, van Beijsterveldt, Middeldorp, & Boomsma, 2011; Eaves et al., 1997; Topolski et al., 1997). In contrast, evidence from parent-reported data suggested higher heritability of anxiety in boys than in girls (Eaves et al., 1997; Topolski et al., 1999). Additionally, other studies suggested no quantitative sex differences in genetic and environmental influences (Burt, 2009; Lau, Eley, & Stevenson, 2006; Legrand, McGue, & Iacono, 1999; Rice, van den Bree, & Thapar, 2004; Zavos, Rijsdijk, & Eley, 2012). These discrepant findings are partly due to different measures and raters across studies. Thus, more studies,
especially those with multiple raters (i.e. child and parents) using parallel measures of anxiety, are needed to further delineate the quantitative sex differences issue.

Another interesting issue pertains to the developmental differences in genetic and environmental contributions to anxiety. A meta-analysis of studies with self-reported data indicated increasing heritability of anxiety from adolescence to young adulthood (Bergen, Gardner, & Kendler, 2007). Using a large sample of Dutch twins, Lamb et al. (2010) and Bartels et al. (2011) found an increasing heritability of self-reported anxious/depressed symptoms from late childhood through adolescence to young adulthood. A similar pattern of finding was demonstrated in a longitudinal twin study of adolescent trait anxiety in the U.S. (Garcia et al., 2013). Nevertheless, several contemporaneous studies with self-reported data demonstrated no significant age difference in genetic and environmental contributions to child and adolescent anxiety (Eley & Stevenson, 1999; Lau et al., 2006; Lau, Gregory, Goldwin, Fine, & Eley, 2007; Legrand et al., 1999; Ogliari et al., 2006). Thus, there is no consensus on whether the genetic and environmental influences change with age. Furthermore, most studies used the self-reported data whereas studies with parent-reported and multi-informant data are scarce.

These findings have greatly advanced our understanding of the etiologies of anxiety in child and adolescents. However, as these studies were almost entirely conducted in the Western societies, it remains unclear on the generalizability to other ethnic populations, especially those in different cultures, such as Chinese youths. To our knowledge, only two twin studies of anxiety were conducted in Chinese samples. Using 602 pairs of adolescent twins from Qingdao Twin Registry (QTR) in China, Unger et al. (2011) found small genetic influences (10%), larger shared (37%) and non-shared environmental influences (53%) on anxiety symptoms assessed by the Zung Anxiety Scale (Zung, 1971). Kuo, Lin, Yang, Soong, and Chen (2004) examined 279 pairs of twins and same-sex siblings from the Taipei Adolescent Twin/Sibling Family Study and found small genetic influences (19%), but larger shared environmental effects (58%) on anxious/depressed symptoms in boys. These preliminary findings seemed to suggest larger shared environmental effects and smaller genetic effects on anxiety in Chinese adolescents, which are different from findings in Western samples. However, the relatively small samples, especially the number of dizygotic twin pairs, in the two studies, may limit their power to draw solid conclusions about genetic and environmental influences, and sex differences in the etiological model. Moreover, both studies examined self-reported anxiety symptoms only, and thus may not have fully captured child’s problems in different contexts (Achenbach, 2006).

The present study aimed to investigate genetic and environmental influences on trait anxiety among children and adolescents using a large twin sample from China, a non-individualistic society. We collected both self-reported and parent-reported twin’s anxiety phenotypes to obtain multiple-informant data. Furthermore, we examined quantitative sex differences and explored age differences in the etiological model. This study provided a unique opportunity for investigating genetic and environmental etiologies of child and adolescent anxiety in a collectivistic culture. Potential cross-cultural similarities or differences in the etiological model of anxiety might be revealed or suggested by comparing our results to the findings in Western individualistic cultures.

**Method**

**Participants**

This study used data from the Beijing Twin Study (BeTwiSt), a longitudinal twin study of Chinese children and adolescents. Approximately 1,400 pairs of twins aged 8–19 years were recruited from 620 elementary and secondary schools that were randomly selected from 18 counties or districts in the Beijing municipality. Detailed information about recruitment procedures and assessment were described in Chen et al. (2013). The present study included 1104 pairs of same-sex twins: 795 pairs of monozygotic (MZ) twins (380 males) and 309 dizygotic (DZ) twins (144 males). The proportion of same-sex DZ twins was similar to other Asian twin samples (Ando et al., 2006; Hur, Shin, Jeong, & Han, 2006), but smaller than that of Western samples (Bulmer, 1970). The higher DZ twinning rates in Western samples has been suggested attributed to the older maternal age and higher proportion of mothers being treated with ovulation-inducing hormones and in vitro fertilization (IVF) in Western societies (Imaizumi, 2003).

Participants’ age ranged from 9 to 18 years (M = 13.52, SD = 2.57). Fifty-three percent of participants were females, and 92% were of Han ethnicity. With regard to highest educational attainment, 6.8% fathers had primary school degree, 32.8% had junior high school degree, 31.8% had senior high school degree, 26.1% had college degree, and 2.5% had graduate degree. The corresponding percentages for mothers’ highest educational attainment were 5.4%, 35.3%, 29.5%, 25.2%, and 4.6%. We asked one parent from each family who know their children best to complete the parent-report questionnaire. Respondents were 65% mothers, 34% fathers, and 1% other caregivers.

**Measures**

**Trait anxiety.** Anxiety was assessed using the Trait subscale of the Form Y of State-Trait Anxiety Inventory (STAI-T, Form Y) (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The STAI-T measures an individual’s stable susceptibility or proneness to experiences anxious mood or thoughts frequently. Example items included ‘I feel nervous and restless,’ and ‘I worry too much.’ On a 4-point Likert scale ranging from 1 = almost never to 4 = almost always, participants were asked to choose the statement that most closely describes how they generally feel. The Chinese version of STAI has been demonstrated reliable and valid (Li & Lopez, 2004). The Cronbach’s alpha of the STAI-T in our study was .89.
Parent-reported youth anxiety symptoms were assessed by the STAI Parent Form (STAI-PF). The STAI-PF consisted of the original STAI items but was reworded for parents to rate their children. For example, item ‘I feel nervous and restless’ was rephrased to ‘My child feels nervous and restless.’ The psychometric properties of the TAI-PF have been supported (Southam-Gerow, Flannery-Schroeder, & Kendall, 2003). In the present study, the Cronbach’s alpha of STAI-PF was .88.

Zygosity determination. About 90% of pairs of twins’ zygosity were determined by DNA analysis (Chen et al., 2010) wherein nine highly heterogeneous short tandem repeat loci 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 zygosity of the rest 10% of twin pairs were determined by a questionnaire. The validity of the questionnaire was tested by comparing to the DNA analyses results in previous pilot twin sample with predictive accuracy of 91% (Chen et al., 2010).

Procedure
All twins and their parents gave informed consent 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 assistants distributed the questionnaires to the twin participants and instructed them to complete the questionnaires independently. Research assistants 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. The questionnaires for parents were taken home by twins and mailed to our laboratory upon completion. All procedures had been approved by the Institutional Review Board.

Twin analysis
The twin design relies on different levels of genetic relatedness between MZ twins who are genetically identical and DZ twins who share one half of the additive genetic effects. This difference is used to estimate genetic and environmental contributions to individual differences in the phenotype. The total variance of a trait can be decomposed into the proportion due to additive genetic effects (A), to shared environmental (C), and to non-shared environmental (E) effects. Additive genetic effects represent the additive effects of genes at different loci affecting a trait. Shared environmental influences are non-genetic factors that serve to make twins more similar to one another, whereas non-shared environmental factors are those that impinge uniquely on individuals within a twin pair and tend to make them dissimilar from one another.

Structural equation modeling packages (Mx) that utilize maximum likelihood estimation method on raw data were used to estimate different variance components. The statistical significance of genetic, shared or non-shared environmental effects was tested by examining whether or not the confidence intervals (CI) of parameters included 0. Quantitative sex and age differences were examined by comparing the baseline model with freely estimated means, and A, C, and E parameters were constrained to be equal for males and females, or for younger (9–12 years, M = 10.85, SD = 1.05) and older groups (13–18 years, M = 15.12, SD = 1.75). The age group was created based on the operationalization of childhood versus adolescence in China. Generally speaking, children who are 12 years old or younger are in primary school, whereas children who are 13 years old or older are in middle or secondary school. The difference in the −2 log likelihood (−2LL) between the baseline model and the restricted model follows a χ² distribution. The likelihood ratio test (LRT) was used to evaluate the significance of the constraint. If the χ² difference test was significant, the constraint was rejected; if the χ² difference test was non-significant, the constraint was accepted and the parsimonious model (with fewer estimated parameters) was retained.

Results
Descriptive statistics and Pearson correlations
The means and standard deviations of trait anxiety in the whole sample and subgroups are shown in Table 1. Girls and boys had similar levels of self-reported trait anxiety, t(1102) = −0.15, p = .88, and parent-reported trait anxiety, t(1102) = −1.43, p = .16. Adolescents had significant higher level of self-reported, t(1102) = −3.38, p = .001, and parent-reported anxiety, t(1102) = −3.35, p = .001, than children. The Levene’s test of equality of variance showed that the phenotypic variances were homogeneous across zygosity, sex, and age groups, ps > .05. There was a moderate correlation between self- and parent-reported trait anxiety, r = .40, p < .001. The distributions of zygosity groups did not differ across sexes, χ² (1) = 0.13, p = .72, or age groups, χ² (1) = 0.33, p = .57. The distributions of trait anxiety scores approached normality in all groups. The residuals after regressing out the effects of sex and age were used in the following analyses (McGue & Bouchard, 1984).

Before estimating the parameters of genetic and environmental effects using model-fitting analyses, the Pearson correlation coefficients of trait anxiety within twin pairs (see Table 1) were computed in MZ and DZ twin pairs. For the whole sample, the correlation coefficients in MZ twins were greater than those in DZ twins, but less than twice the correlation coefficients in DZ twins. Similar patterns were found when the Pearson correlations were computed separately by sex and age groups (9–12 years vs. 13–18 years) (see Table 1). These results suggested additive genetic, shared, and non-shared environmental influences on trait anxiety in this sample (i.e. ACE model).

Model-fitting analyses
Model-fitting analyses were firstly applied to the data of whole sample. The parameter estimates and their 95% CI of genetic (A), shared (C), and non-shared environmental (E) influences on self-reported trait anxiety were 50% [30%, 60%], 5% [0%, 24%], and 45% [40%, 49%]. For parent-reported data, the parameter estimates with 95% CI were 63% [47%, 78%], 13% [1%, 28%], and 24% [22%, 27%], respectively. To further test whether the child and their parents provided valid differential information on
child’s anxiety, we conducted bivariate genetic analyses and found that both self-reported and parent-reported child’s trait anxiety had significant unique genetic, shared, and non-shared environmental origins (see details in Appendix S1).

Model-fitting analyses were then conducted separately for males and females. Results are presented in Table 2. For self-reported trait anxiety, the heritability was higher and non-shared environmental effect was lower in females than in males. The difference in model fit between the baseline model and restricted model (A, C, E parameters were equated in males and females) was significant, \( \Delta \chi^2 (3) = 26.30, p < 0.01 \). However, for parent-reported data, the heritability was higher and shared environmental effect was lower in males than in females. Equating parameters across sexes also significantly worsened the model fit, \( \Delta \chi^2 (3) = 8.08, p < .05 \).

The results of model-fitting across age groups are shown in Table 3. For self-reported data, the difference in model fit between the baseline model and restricted model was non-significant, \( \Delta \chi^2 (3) = 3.00, p = .27 \), suggesting that the genetic and environmental influences on self-reported trait anxiety did not differ by age groups. However, for parent-reported data, there was an increase in heritability and a decrease in shared environmental effects from younger to older group. Equating parameters between age groups significantly worsened the model fit, \( \Delta \chi^2 (3) = 22.00, p < .01 \).

**Discussion**

Previous etiological research was primarily conducted in Western individualistic populations, thus much less is known about the etiologies of anxiety in children and adolescents from collectivistic cultural contexts, including China. The current study investigated the genetic and environmental etiologies of anxiety among Chinese children and adolescents, and tested the cross-cultural generalizability of the etiological model of anxiety.

**Heritability**

We found that trait anxiety in Chinese children and adolescents was highly heritable. The heritability of self-reported trait anxiety was 50% [30%, 60%], which was largely consistent with previous findings in Western samples (Franci et al., 2010; Zavos et al., 2012), including two prior studies using the same instrument (STAI): 31% [2%, 54%] in Lau et al. (2006), and 40% [11%, 51%] in Legrand et al. (1999). Moreover, we found large heritability of parent-reported trait anxiety: 63% [47%, 78%], which was comparable to previous studies of parent-reported anxiety symptoms (Eaves et al., 1997; Rice et al., 2004) and anxiety-related temperament trait (i.e. shyness/behavioral inhibition) (Eley et al., 2003). Taken together, these findings suggest...
### Table 2 Model fit indices and parameter estimates with 95% confidence intervals and fit indices by rater and sex groups

<table>
<thead>
<tr>
<th>Model</th>
<th>2LL</th>
<th>( \Delta \chi^2 )</th>
<th>( \Delta df )</th>
<th>( p )</th>
<th>AIC</th>
<th>95% CI Parameter Estimates</th>
<th>95% CI Fit Indices</th>
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<td><strong>Female</strong></td>
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<td>Self-reports</td>
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<td>A</td>
<td>C</td>
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<tr>
<td>M1</td>
<td>31,124.80</td>
<td>22,320.80</td>
<td>.56 [0.39, .67]</td>
<td>.07 [0.00, .23]</td>
<td>.37 [0.33, .41]</td>
<td>.47 [0.23, .52]</td>
<td>.00 [0.00, .21]</td>
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<tr>
<td>M2</td>
<td>31,151.07</td>
<td>26.3 &lt; .01</td>
<td>22,341.07</td>
<td>.50 [0.36, .59]</td>
<td>.06 [0.00, .19]</td>
<td>.44 [0.41, .48]</td>
<td>.50 [0.36, .59]</td>
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<tr>
<td>M1</td>
<td>28,282.42</td>
<td>19,478.42</td>
<td>.55 [0.40, .72]</td>
<td>.18 [0.02, .33]</td>
<td>.27 [0.24, .30]</td>
<td>.72 [0.55, .80]</td>
<td>.06 [0.00, .23]</td>
</tr>
<tr>
<td>M2</td>
<td>28,290.50</td>
<td>8.08 &lt; .05</td>
<td>19,480.50</td>
<td>.63 [0.52, .76]</td>
<td>.12 [0.00, .24]</td>
<td>.25 [0.23, .27]</td>
<td>.63 [0.52, .76]</td>
</tr>
</tbody>
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M1: Baseline Model with free A, C, E parameters and free means; M2: Restricted Model with equated A, C, E parameters and free means.

Models with better model fit were bold.

### Table 3 Model fit indices and parameter estimates with 95% confidence intervals and fit indices by rater and age groups

<table>
<thead>
<tr>
<th>Model</th>
<th>2LL</th>
<th>( \Delta \chi^2 )</th>
<th>( \Delta df )</th>
<th>( p )</th>
<th>AIC</th>
<th>95% CI Parameter Estimates</th>
<th>95% CI Fit Indices</th>
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<td><strong>Younger group (9–12 years)</strong></td>
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<td>A</td>
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<td>Self-reports</td>
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<td>A</td>
<td>C</td>
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<tr>
<td>M1</td>
<td>31,106.57</td>
<td>22,302.57</td>
<td>.53 [0.30, .63]</td>
<td>.05 [0.00, .27]</td>
<td>.42 [0.37, .47]</td>
<td>.48 [0.30, .57]</td>
<td>.04 [0.00, .21]</td>
</tr>
<tr>
<td>M2</td>
<td>31,109.57</td>
<td>3.0 &lt; .27</td>
<td>22,299.57</td>
<td>.50 [0.36, .58]</td>
<td>.05 [0.00, .18]</td>
<td>.45 [0.42, .49]</td>
<td>.50 [0.36, .58]</td>
</tr>
<tr>
<td>M1</td>
<td>28,239.09</td>
<td>19,435.09</td>
<td>.46 [0.30, .65]</td>
<td>.30 [0.12, .46]</td>
<td>.24 [0.21, .27]</td>
<td>.74 [0.58, .77]</td>
<td>.01 [0.00, .16]</td>
</tr>
<tr>
<td>M2</td>
<td>28,261.11</td>
<td>22.0 &lt; .01</td>
<td>19,451.11</td>
<td>.64 [0.53, .76]</td>
<td>.11 [0.00, .22]</td>
<td>.25 [0.23, .27]</td>
<td>.64 [0.53, .76]</td>
</tr>
</tbody>
</table>

M1: Baseline Model with free A, C, E parameters and free means; M2: Restricted Model with equated A, C, E parameters and free means.

Models with better model fit were bold.
substantial genetic roots of trait anxiety in Chinese children and adolescents. Future studies can further identify the specific genetic variants accounting for the high heritability.

Rater difference in etiologies
We further examined the rater effect on the genetic and environmental influences. Consistent with previous research (Eaves et al., 1997; Thapar & McGuffin, 1995), there were lower genetic influences but higher non-shared environmental effects on self-reported anxiety, compared with parent-reports. One explanation is that parents may be rating enduring traits, whereas children may be reporting their current state (Gregory & Eley, 2007). Lau et al. (2006) provided some supportive evidence for this explanation by demonstrating a large genetic overlap underlying self-reported trait anxiety and state anxiety. Because the heritability of state anxiety was small, its overlap with genetic influences of trait anxiety may lower the heritability estimate of trait anxiety. Therefore, parent-reported trait anxiety may be a better phenotype for searching specific anxiety-related genetic variants.

Sex differences in etiologies
Sex differences in the genetic and environmental contributions were found and differed by raters (self vs. parents). Specifically, for self-reported data, there was greater heritability of trait anxiety in girls than in boys, consistent with previous studies (Bartels et al., 2011; Eaves et al., 1997). In contrast, we found greater heritability of parent-reported anxiety in boys than in girls, consistent with prior studies of parent-rated anxiety symptoms (Topolski et al., 1999) and shyness/inhibition trait (Eley et al., 2003). These findings seem to suggest higher genetic influences in child-reported girls’ anxiety and parent-reported boys’ anxiety. However, given that there were also null findings on sex differences (Burt, 2009; Lau et al., 2006; Legrand et al., 1999; Rice et al., 2004; Zavos et al., 2012), more empirical studies with both child-reported and parent-reported data are needed to better understand sex differences in the etiology of child and adolescent anxiety.

Age differences in etiologies
The age differences in genetic and environmental contributions also varied across raters. No significant age difference was found in child-reported anxiety, same with some studies (Eley & Stevenson, 1999; Lau et al., 2006, 2007; Legrand et al., 1999; Ogliari et al., 2006) but different from others (Bergen et al., 2007; Garcia et al., 2013; Lamb et al., 2010). For parent-reported data, we found a significant increase of heritability and decrease of shared environmental effect from childhood to adolescence, consistent with previous studies (e.g. Spatola, Rende, & Battaglia, 2010). One potential explanation for age-related increasing heritability is the gene–environment correlation (rGE) process (Knafo & Jaffee, 2013). For example, Eley, Napolitano, Lau, and Gregory (2010) found that highly heritable child anxiety can evoke maternal control, which may in turn exacerbate child’s anxiety (Affrunti & Ginsburg, 2012). Another explanation is the possible genetic innovation with development demonstrated by longitudinal research on anxiety (Kendler, Gardner, & Lichtenstein, 2008a) and phobias (Kendler et al., 2008b).

Cultural similarities or differences
Although two previous studies with smaller sample found low genetic influences (Kuo et al., 2004; Unger et al., 2011), our findings based on a large twin cohort demonstrated high heritability of anxiety in Chinese children and adolescents, consistent with previous findings in Western cultures (e.g. Franic et al., 2010; Rice & Thapar, 2009). In addition, sex differences in heritability of anxiety in our sample were also largely compatible with Western studies, that is, higher heritability in girls than in boys for self-report data (e.g. Bartels et al., 2011) and higher heritability in boys than in girls for parent-report data (Eaves et al., 1997; Topolski et al., 1999). These findings seem to support the cross-cultural generalizability of the etiological model of anxiety. However, potential cultural differences were also revealed. Different from previous findings in Western cultures (i.e. higher level of anxiety in adolescent girls than boys) (Beesdo, Knappe, & Pine, 2009), we found no significant difference in anxiety symptoms between Chinese girls and boys that is consistent with prior studies in Asian samples (Liu, Ma, Kurita, & Tang, 1999; Tepper et al., 2008). In sum, given the lack of twin studies in non-Western cultures, cross-cultural generalizability should be drawn with caution. More empirical studies with larger sample sizes are needed to examine the genetic and environmental influences on children and adolescents’ anxiety in China and other collectivistic societies.

Strengths and limitations
The strengths of the present study include a large sample size and using multiple informants. One major limitation is using the STAI-T to measure participants’ enduring tendency to experience anxiety symptoms. Thus, our findings may not be generalized to specific anxiety disorders or symptoms. However, previous multivariate twin studies supported a model with latent anxiety liability factor composing of genetically overlapped multiple anxiety disorders (Eley et al., 2003; Ogliari et al.,

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2010). Given the similar estimates of genetic and environmental effects between trait anxiety and anxiety disorders in prior studies (Francic et al., 2010; Rice & Thapar, 2009), we speculate that anxiety disorders in Chinese children and adolescents are also likely to be largely heritable and have substantial non-shared environmental origins. Future studies examining specific anxiety disorders in Chinese children and adolescents can empirically test this speculation.

Conclusion
To summarize, we found substantial genetic bases and important non-shared environmental etiology of trait anxiety among Chinese children and adolescents. The estimates of genetic and environmental effects were different across raters, sexes, and age groups. Importantly, the sex and age differences were further moderated by rater. Empirical cross-cultural studies are substantially needed to further understand cultural similarities and differences in the etiologies of anxiety.

Supporting information
Additional Supporting Information may be found in the online version of this article:
Appendix S1. Bivariate genetic analyses.

Acknowledgements
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Key points
- Child and adolescent anxiety has become a major public health concern in China. Unfortunately, little research has examined the etiology of anxiety in Chinese children and adolescents, especially from the genetic perspective.
- The trait anxiety in Chinese children and adolescents were highly heritable for both self-reported and parent-reported data. The heritability estimates were similar and even higher compared to findings in Western samples.
- The heritability of anxiety was higher in girls for self-reported data, but higher in boys for parent-reported data. There was no significant age difference in genetic and environmental contributions for self-reported data, but a significant age-related increase of heritability for parent-reported data.
- Our findings largely indicate the cross-cultural generalizability of the etiological model of childhood and adolescent anxiety. Future research can explore how the gene–environment interplay (i.e. gene–environment correlation and gene-by-environment interaction) influence the development of anxiety in Chinese children and adolescents.

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