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# A Study of Changes in Genetic and Environmental Influences on Weight and Shape Concern Across Adolescence

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The goal of the current study was to examine whether genetic and environmental influences on an important risk factor for disordered eating, weight and shape concern, remained stable over adolescence. This stability was assessed in 2 ways: whether new sources of latent variance were introduced over development and whether the magnitude of variance contributing to the risk factor changed. We examined an 8-item WSC subscale derived from the Eating Disorder Examination (EDE) using telephone interviews with female adolescents. From 3 waves of data collected from female–female same-sex twin pairs from the Australian Twin Registry, a subset of the data (which included 351 pairs at Wave 1) was used to examine 3 age cohorts: 12 to 13, 13 to 15, and 14 to 16 years. The best-fitting model contained genetic and environmental influences, both shared and nonshared. Biometric model fitting indicated that nonshared environmental influences were largely specific to each age cohort, and results suggested that latent shared environmental and genetic influences that were influential at 12 to 13 years continued to contribute to subsequent age cohorts, with independent sources of both emerging at ages 13 to 15. The magnitude of all 3 latent influences could be constrained to be the same across adolescence. Ages 13 to 15 were indicated as a time of risk for the development of high levels of WSC, given that most specific environmental risk factors were significant at this time (e.g., peer teasing about weight, adverse life events), and indications of the emergence of new sources of latent genetic and environmental variance over this period.

*Keywords:* weight and shape concern, twins, genetic, environmental, longitudinal

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Weight and shape concern has been identified as one of the most potent and best-replicated risk factors for both bulimia nervosa and anorexia nervosa (Jacobi & Fittig, 2010). The construct has been assessed with various measures across different studies but consists predominantly of items that relate to body dissatisfaction (e.g., “How often have you worried about having fat on your body?”), in addition to an item that assesses the degree to which weight/shape influences feelings of self-worth (e.g., “How much has your weight/shape made a difference in how you feel about yourself?”). This latter item relates to the diagnostic criterion for eating disorders known as “undue influence of body shape or weight on self-evaluation” (American Psychiatric Association, 1994), which has been described as the “core psychopathology” of eating disorders (Cooper & Fairburn, 1993) and is included as one of the diagnostic criterion for both bulimia nervosa and anorexia nervosa. At 4-year follow-up, Killen and colleagues (1996) showed that females who reported the highest weight and shape concerns at baseline (average age of 14.9 years) also recorded the greatest incidence of partial syndrome eating disorders, whereas those who reported the least weight concerns at baseline recorded no incidence of partial syndrome eating disorders. A 7-year longitudinal study by Field and colleagues (2008) found that 12-year-old females reporting high weight concern were 2.7 times more likely to start binge eating and 2.3 times more likely to start purging at follow-up. The McKnight Investigators (2003) found that higher scores on a factor assessing concerns with weight and shape were a significant predictor of eating-disorder onset in Grade 6 to Grade 9 females over a 4-year time period. Wilksch and Wade (2010) found that undue influence of shape and weight in 14-year-old females was a significant predictor of disordered eating behaviors at 1-year follow-up. Furthermore, Haines, Kleinman, Rifas-Shiman, Field, and Austin (2010) conducted a prospective study that demonstrated a direct association between weight concern and purging, binge eating, and overweight status in females. The findings are consistent with those of Allen, Byrne, McLean, and Davis (2008), who found weight concern predicted the onset of binge eating in a sample of 8- to 13-year-old females.

Given the importance of this risk factor, many different behavioral genetic investigations of weight and shape concern, and related constructs, exist that try to identify to what degree latent genetic and environmental (shared and nonshared) sources of variance contribute to this phenotype. A study of weight concern and shape concern in adult women, using the separate scales of a semistructured interview from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), showed that only the environment (shared and nonshared) contributed to weight concern, whereas genetic variance substantially contributed to shape concern (62%), with the remainder of the variance being accounted for by the nonshared environment (Wade, Martin, & Tiggemann, 1998). Three investigations of the undue influence of body shape or weight on self-evaluation exist. The first, a Norwegian twin study, using a single self-report item for males and females aged 18 to 31 years (Reichborn-Kjennerud et al., 2004), found that only shared and nonshared environment contributed to the phenotype. In contrast, investigations of Australian adults (Wade & Bulik, 2007) and young adolescents (Wilksch & Wade, 2009a) found a small contribution of heritability to combined items assessing undue influence of weight and shape using the EDE—25% and 15%, respectively. An examination of a four-item measure of

disordered eating, which contained two items relating to weight and shape concern, found that genetic and nonshared environment contributed to individual variation in adolescents aged 14 to 18 years (Slof-Op 't, et al., 2008). Examination of body dissatisfaction in 11- and 18-year-olds showed that genetic variance contributed at both age groups (49% and 60%, respectively), as did the nonshared environment (48% and 40%, respectively), with a small contribution (3%) of the shared environment at age 11 but not at age 18 (Klump, McGue, & Iacono, 2000). In different studies, both genetic and nonshared environment impacted on body dissatisfaction in women aged 22 to 27 years (Keski-Rahkonen et al., 2005), and on salience of weight and shape of women aged 28 to 36 years (Wade, Wilkinson, & Ben-Tovim, 2003), with the genetic variance ranging from 39% to 59%.

Although the various investigations across different populations suggest that latent genetic and environmental risk factors contribute to weight and shape concern, both estimates of the variance and evidence pertaining to the presence of the shared environment is somewhat varied. This could be partly explained by developmental shifts in both the sources of genetic and environmental influences contributing to weight and shape concern over adolescence and the magnitude of variance of these latent influences. A better understanding of these mechanisms can be somewhat resolved by an examination of how these latent risk factors change over adolescence. One cross-sectional study contributed to clarification of developmental changes in weight and shape concern, assessed with the 12 items (combined) from the self-report version of the EDE (EDE-Q; Fairburn & Beglin, 1994), by examining the latent genetic and environmental risk factors contributing to the phenotype across six age groups: 10 to 12, 13 to 15, 16 to 19, 20 to 25, 26 to 30, and 31 to 41 years of age (Klump et al., 2010). Changes in genetic effects were observed only between the 10- to 12-year group and the remainder of the age groups. In the former group, only environmental influences contributed to weight and shape concern (58% due to shared environment and 42% due to the nonshared environment), whereas the estimates for the latter group could be constrained to be the same from ages 13 to 41, with genetic and nonshared environment contributing to the variance at 54% and 46%, respectively. Somewhat in contrast, but examining a different phenotype, a cross-sectional study of disordered eating in female twins showed a sizable genetic contribution to eating disorder symptoms at ages 8 to 13 years, followed by a decrease in this contribution at ages 14 to 17 years (Silberg & Bulik, 2005). However, a longitudinal study is required to more definitively examine developmental shifts in the genetic and environmental variance contributing to weight and shape concern.

Although no longitudinal study of weight and shape concern exists, one longitudinal study examined changes during adolescence in latent genetic and environmental influences on disordered eating (Klump, Burt, McGue, & Iacono, 2007), a phenotype which overlapped with the weight and shape concern construct, as it included body dissatisfaction and weight preoccupation in addition to binge eating and the use of compensatory behaviors. In a study of female twins at ages 11, 14, and 18 years, the shared environment was found to be a significant contributor to disordered eating and attitudes in the youngest, prepubertal age group, but its impact was negligible at ages 14 and 18 years. Conversely, the impact of heritability was negligible at 11 years, but increased at ages 14 and 18, where the magnitude of the estimates for the three sources of

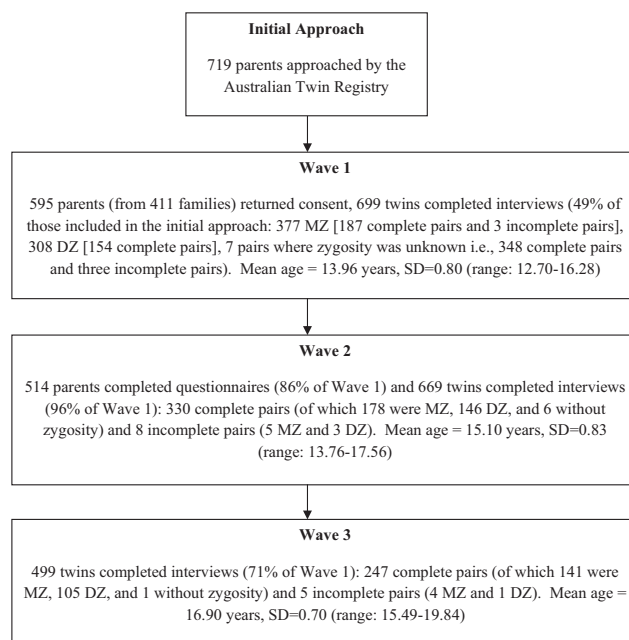
variance could be constrained to be the same within each source of variance. Although the source of genetic variance that was active at 11 years of age continued to be a major contributor to the variance at ages 14 and 18 years, a new source of genetic variance appeared at age 14 years but made only small contributions to the variance at ages 14 (1%) and 18 (18%), and there were no new sources of genetic influence at age 18 years. A similar pattern was observed for shared environmental influences, but, in contrast, new sources of nonshared environmental variance appeared at each age, indicating new environmental experiences occurring over development that influenced the development of disordered eating. This study and cross-sectional investigations support the suggestion that puberty and associated ovarian hormones activated at puberty “switch on” the main source of genetic risk for disordered eating (Klump, McGue, & Iacono, 2003; Culbert, Burt, McGue, Iacono, & Klump, 2009).

The aim of the current study is to therefore examine developmental shifts in the genetic and environmental factors contributing to weight and shape concern over three age cohorts during adolescence (e.g., 12 to 13, 13 to 15, and 14 to 16 years) in female twins, using frequent assessments over this time period, that is, three over a 3-year period. We seek to address two related questions: First, are new sources of latent variance introduced over development, and second, does the magnitude of variance contributed by these latent risk factors change over development? Data were modeled to determine whether genetic and environmental sources independent of those influencing weight and shape concern at ages 12 to 13 years become influential at 13 to 15 and 14 to 16 years, or, conversely, whether variation across the three periods was influenced by single generic and/or environmental sources. This modeling also allows us to test if the magnitude of latent variance changes across the age cohorts. Given previous research, we hypothesized that the shared environment would have a negligible impact on our phenotype, given the postpubertal status of our population and that independent sources of nonshared environmental variance would emerge at each age cohort. In order to further examine this latter hypothesis, we also examined specific sources of environment and their patterns of association with weight and shape concern across the age cohorts.

## Method

### Participants

The three waves of data from the current study are from female twin adolescents, as shown in Figure 1, where the participants and methodology of ascertainment for this sample were previously described (Wade, Byrne, & Bryant-Waugh, 2008; Wilksch & Wade, 2009a, 2010). Female–female twin pairs who were registered with the Australian Twin Registry (ATR) and were between 12 and 15 years of age, and their parents, were approached to participate in the present study by the ATR. Of the 719 families approached, 411 (57.2%) agreed to participate, 237 (32.9%) declined, and 71 (9.9%) did not reply. Families were then approached by the researchers with self-report questionnaires sent to both parents, including those families where the parents did not live together. When questionnaires were returned from the parents, the EDE (Fairburn & Cooper, 1993) was conducted over the telephone with the twins at separate times and with a different interviewer for



Note: there was no significant difference in age between MZ and DZ twins at Wave 1 ( $t[336]=0.25$ ,  $p=0.81$ ), Wave 2 ( $t[336]=0.54$ ,  $p=0.59$ ) or Wave 3 ( $t[335.57]=0.72$ ,  $p=0.48$ ).

Figure 1. Flow diagram depicting the three waves of the data collection process.

each child in the family. The sample was Caucasian and the socioeconomic indexes for areas (SEIFA)—a standardized measure of socioeconomic status with a mean of 100 ( $SD = 15$ ), using an amalgam of parental occupation, education (years of school), and income from 2006 census data related to the postcode of primary residence (Farish, 2004)—was 101.14, with a standard deviation of 11.36.

At Waves 2 and 3, all twins—responders and nonresponders—were approached. Different interviewers at each wave were used for each individual. The mean duration of time between Waves 1 and 2 was 1.15 years ( $SD = 0.17$ ), and the mean duration of time between Waves 1 and 3 was 2.96 years ( $SD = 0.27$ ), ranging from 1.91 to 4.65 years. The ages were significantly different between Waves 1 and 2 (13.96 vs. 15.10 years,  $t[df = 667] = -42.09$ ,  $p < .001$ ) and Waves 2 and 3 (15.10 vs. 16.90 years,  $t[df = 496] = -51.59$ ,  $p < .001$ ). Blood samples were obtained from the twins involved in the third wave of data collection. Zygosity assignment was based on parental responses to standard questions about physical similarity and confusion of twins by parents, teachers, and strangers, methods that give better than 95% agreement with genotyping (Eaves et al., 1989). Where there was uncertainty ( $N = 46$  pairs), DNA testing was used to assign zygosity for 39 pairs (DNA was not available for seven pairs and these pairs were therefore not included in the analyses).

However in order to examine discrete age ranges over adolescence, the focus of the current analyses was the data contained in the bolded lines in Table 1, accompanied by the descriptives of the three different age cohorts examined in the analyses: Cohort A (12 to 13 years), Cohort B (13 to 15 years), and Cohort C (14 to 16 years). The Flinders University Clinical Research Ethics Commit-

Table 1

*Descriptives for the Three Cohorts Over the Three Waves of Data Collection: Number of Pairs, Means (Standard Deviations) and Ranges for Age, BMI, and WSC*

	Age group				
	12–13 years	13–15 years	14–16 years	15–18 years	17–19 years
Wave 1	<i>Cohort A, Wave 1</i>	<i>Cohort B, Wave 1</i>	<i>Cohort C, Wave 1</i>		
<i>N</i> pairs	<b>133</b>	<b>122</b>	<b>96</b>		
Age	<b>13.15 (.22)</b> <b>12.74–13.49</b>	<b>13.97 (.30)</b> <b>13.50–14.49</b>	<b>15.01 (.33)</b> <b>14.50–16.28</b>		
BMI T1	<b>19.02 (2.92)</b> <b>13.74–31.20</b>	<b>19.98 (3.26)</b> <b>13.17–33.07</b>	<b>21.33 (3.88)</b> <b>14.53–34.81</b>		
BMI T2	<b>19.11 (3.00)</b> <b>14.10–31.89</b>	<b>19.69 (3.22)</b> <b>12.99–30.11</b>	<b>20.96 (3.94)</b> <b>15.70–34.48</b>		
WSC T1	<b>0.58 (0.84)</b> <b>0–4.50</b>	<b>0.88 (1.14)</b> <b>0–5.75</b>	<b>1.05 (1.15)</b> <b>0–4.25</b>		
WSC T2	<b>0.74 (1.04)</b> <b>0–5.63</b>	<b>0.67 (0.88)</b> <b>0–4.75</b>	<b>1.48 (1.51)</b> <b>0–6.00</b>		
Wave 2		<i>Cohort A, Wave 2</i>	<i>Cohort B, Wave 2</i>	<i>Cohort C, Wave 2</i>	
<i>N</i> pairs		<b>124</b>	<b>116</b>	<b>94</b>	
Age		<b>14.26 (.26)</b> <b>13.76–15.23</b>	<b>15.14 (.41)</b> <b>14.15–16.71</b>	<b>16.15 (.35)</b> <b>15.57–17.56</b>	
BMI T1		<b>20.13 (3.00)</b> <b>14.98–29.38</b>	<b>20.58 (3.16)</b> <b>14.52–30.78</b>	<b>21.52 (3.21)</b> <b>11.90–34.71</b>	
BMI T2		<b>19.96 (3.14)</b> <b>14.84–34.72</b>	<b>20.36 (3.16)</b> <b>14.34–30.47</b>	<b>21.29 (3.31)</b> <b>13.73–34.14</b>	
WSC T1		<b>0.64 (0.93)</b> <b>0–5.63</b>	<b>0.82 (1.19)</b> <b>0–5.13</b>	<b>0.89 (1.05)</b> <b>0–4.75</b>	
WSC T2		<b>0.68 (0.88)</b> <b>0–5.00</b>	<b>0.79 (0.96)</b> <b>0–5.00</b>	<b>1.09 (1.21)</b> <b>0–4.88</b>	
Wave 3			<i>Cohort A, Wave 3</i>	<i>Cohort B, Wave 3</i>	<i>Cohort C, Wave 3</i>
<i>N</i> pairs			<b>93</b>	<b>89</b>	<b>69</b>
Age			<b>16.09 (.29)</b> <b>15.50–16.98</b>	<b>16.99 (.42)</b> <b>16.24–18.30</b>	<b>17.91 (.39)</b> <b>17.34–19.83</b>
BMI T1			<b>21.71 (3.98)</b> <b>14.55–32.87</b>	<b>21.40 (3.39)</b> <b>13.87–35.88</b>	<b>22.88 (3.61)</b> <b>16.41–39.06</b>
BMI T2			<b>21.09 (3.28)</b> <b>14.78–33.50</b>	<b>20.65 (2.58)</b> <b>13.87–26.82</b>	<b>22.81 (4.03)</b> <b>16.58–40.74</b>
WSC T1			<b>0.54 (0.74)</b> <b>0–4.25</b>	<b>0.92 (1.24)</b> <b>0–4.75</b>	<b>0.93 (0.94)</b> <b>0–3.63</b>
WSC T2			<b>0.70 (1.00)</b> <b>0–6.00</b>	<b>0.89 (1.06)</b> <b>0–5.13</b>	<b>1.15 (1.36)</b> <b>0–5.75</b>

*Note.* The data utilized in the current study are in bold. BMI = body mass index; T1 = Twin 1; T2 = Twin 2; WSC = weight and shape concern.

tee approved the data collection process, and written informed consent from parents and written assent from the twins was obtained after the procedures had been fully explained.

### Weight and Shape Concern

The telephone interview consisted of two parts. The first part utilized the EDE (Fairburn & Cooper, 1993), and the second part consisted of questions from various self-report questionnaires that assessed a range of variables, including life events, temperament, and family functioning (Wilksch & Wade, 2009a, 2010). All interviewers were postgraduate clinical psychology trainees ( $n = 16$ ) who had been trained in use of the EDE. All interviews were taped, and corrective feedback was provided until the interviewer had acquired the skills required to complete the interview independently, considered to be attained when there was no disagreement more than 1 point on the Likert scales on all items. Through-

out the interviewing process, monthly group meetings were held to discuss the interview process in order to ensure interview fidelity.

The EDE was modified slightly for use with a younger population, as previously described in detail (Wade et al., 2008; Wilksch & Wade, 2009a, 2010), and included questions that form four subscales: dietary restraint, and eating, shape, and weight concern over the last 28 days. Each item in these subscales is assessed by the interviewer on a 7-point Likert scale ranging from 0 to 6. We chose to focus on the factor previously identified as having the greatest stability in this population at Wave 1 (Wade et al., 2008), an 8-item score that utilized items from the weight concern subscale (dissatisfaction with weight, reaction to prescribed weighing, importance of weight) and the shape concern subscale (dissatisfaction with shape, importance of shape, discomfort seeing body, avoidance of body exposure, feelings of fatness).

The EDE is among the most widely used assessments of eating disorder pathology (Berg et al., *in press*). The EDE has been found to have good convergence with the subscale scores of the self-report version of the same instrument, the EDE-Q (Berg, Peterson, Frazier, & Crow, 2011). Test-retest reliability in clinical populations over 2 to 7 days and over 6 to 14 days has ranged from 0.50 to 0.88 for the subscale scores, including 0.50 to 0.76 for shape concern and 0.52 to 0.71 for weight concern (Berg, Peterson, Frazier, & Crow, 2012). Internal consistency for the shape and weight concern subscales has ranged from 0.68 to 0.85 and 0.51 to 0.76, respectively, and interrater reliability for these two subscales has ranged from 0.84 to 0.99 and 0.65 to 0.99 (Berg et al., 2012). Although temporal stability of the EDE over long periods has not been reported in community samples, the temporal stability of the EDE-Q in an Australian adult community sample, aged 18 to 45 years, over a median period of 315 days was 0.75 for shape concern and 0.73 for weight concern (Mond, Hay, Rodgers, Owen, & Beumont, 2004). The EDE has also been shown to satisfactorily distinguish between people with an eating disorder and controls (Berg et al., 2012).

Weight and height of the twins were reported by both the mother and father separately at Waves 1 and 2. These reports were highly correlated and so the mother's report was used in the current report, or the father's report was used if the mother's was missing. At Wave 3, twins reported their own weight and height. The correlations between the weight ratios across the different waves were 0.71 (Waves 1 and 2), 0.82 (Waves 2 and 3), and 0.58 (Waves 1 and 3). Self-reported weight and height in adolescent populations has been shown to correlate strongly with actual weight (0.93 to 0.95) and height (0.89 to 0.94; Brener, Mcmanus, Galuska, Lowry, & Wechsler, 2003; Goodman, Hinden, & Khandelwal, 2000; Strauss, 1999). Although there is no information about the accuracy of parent-reported weight and height for adolescents, parents have been found to be more accurate than teens in identifying obesity in the adolescent (Goodman et al., 2000).

### Measures of Specific Environmental Constructs

The self-report questionnaires assessed specific environmental constructs at Wave 1 that have been previously implicated in the development of disordered eating. These measures have been previously described (Wilksch & Wade, 2010) and included parental expectations and parental criticism from the Frost Multidimensional Perfectionism Scale (Frost, Marten, Lahart, & Rosenblate, 1990); the Internalization subscale of the Multidimensional Media Influence Scale (Cusumano & Thompson, 2001), which assessed the degree to which respondents wished to look like images in the media; the Perceived Sociocultural Pressure Scale (Stice, Ziemba, Margolis, & Flick, 1996), which assessed perceived pressure to be thin from friends, family, media, and dating partners; conflict between the parents, measured using the conflict subscale from the Family Environment Scale (Moos & Moos, 1986); and parental care was assessed separately for mother and father using the Parental Bonding Inventory (Parker, Tupling, & Brown, 1979). Cronbach's alphas ranged from 0.72 to 0.91. In addition, parents were asked whether the twin had experienced a major life event in the previous 12-month period (yes or no), including death of a family member, a move to a new home, a

move to a new school, separation from a parent, loss of a close friendship, or any other traumatic event.

### Statistical Analysis

**Reliability and validity of the weight and shape concern construct.** This was assessed within each of the three age cohorts in a number of ways. First, reliability was assessed by examining interrater reliability, internal reliability, and the intra-class coefficients. The Tucker congruence coefficient (Tucker, 1951) was used to assess similarity across factors. The factor invariance of the EDE weight and shape concern measure across cohorts and waves was examined using Mplus version 6.11 (Muthén & Muthén, 1998–2010). Weighted least squares estimation with mean- and variance-adjusted chi-square statistics (WLSMV) and theta parameterization was used to estimate all models. EDE items were specified as ordinal variables and the dependency among twin pairs was accounted for by clustering siblings within family units. Three models were tested. The first model was the configural invariance model, which estimates separate factor loadings and item threshold values (cutpoints between the ordinal responses) for each item at each wave. This is the "baseline" model against which the subsequent two models are compared. The second model was the metric invariance model, which fixes the factor loadings for each item to be equivalent across the three waves but allows the item thresholds to differ. The third model was the full invariance model, which fixes both the factor loadings and item threshold values across the three waves. If the chi-square difference value between the models is significant, it indicates that constraining the parameters of the nested model significantly worsens the fit of the model, which indicates measurement noninvariance. If the chi-square difference value is not significant, this indicates that constraining the parameters of the nested model did not significantly worsen the fit of the model, indicating measurement invariance of the parameters constrained to be equal in the nested model.

**Twin correlations.** For the purpose of the remaining analyses, data were treated as being continuous, and a full-information maximum likelihood (FIML) approach was used with the statistical package Mx (M. C. Neale, 1994), designed to apply structural equation modeling approaches to twin data. In the current study, raw data were analyzed in Mx, which incorporates complete and incomplete pairs of twins and those with missing data across the waves of data collection by automatically creating the appropriate mean vector and covariance matrix for each observation (M. C. Neale, 1994). The weight and shape concern phenotypes was transformed at each wave using the  $\lg_{10}(x + 1)$  function to improve normality, and were then standardized ( $Z$ -scores:  $M = 0 \pm 1$ ).

The correlations among the weight and shape EDE scores at each age group and the monozygotic (MZ) and dizygotic (DZ) correlations for each phenotype were examined, and the difference between the correlations was tested by comparing submodels constraining MZ and DZ correlations to be the same. Given that MZ twins share 100% of their genes, whereas DZ twins share, on average, only 50%, additive genetic effects on a phenotype are inferred when MZ-twin correlations are roughly double DZ-twin correlations (Plomin, DeFries, & McClearn, 1990). Shared environmental influences include environmental influences common to co-twins growing up in the same family and therefore contribute to

their behavioral similarity to an equal degree in both MZ and DZ pairs. Nonshared environmental influences (which include measurement error) are those unique to each co-twin and are inferred when MZ twin correlations are less than 1.00. Nonadditive genetic influences (known as dominance) are implied if MZ correlations are more than twice that of the DZ correlations.

**Multivariate model fitting.** We used a multivariate Cholesky decomposition model that included the weight and shape concern score for each of the three age cohorts. The structure of this model is shown in Figure 2. Multivariate models are more powerful than univariate models, as they use both variances of individual variables and covariances between the different variables to estimate parameters (B. M. Neale, Mazzeo, & Bulik, 2003). Our use of repeated measures can correct for any ascertainment bias resulting from differential attrition (Little & Rubin, 1987) and also reduces the contribution of measurement error to the nonshared environment (Bulik, Sullivan, & Kendler, 1998).

We first examined the models where the magnitude of the parameter estimates was allowed to differ across the three age cohorts, starting with a full model (i.e., containing the additive genetic variance [A], shared environment [C], and nonshared environment [E] sources of variance). We then fit a series of nested models in order to examine whether all sources of variance were required, reporting 95% confidence intervals (CIs) for all estimates, which helps when examining the significance of models. We then fit a further submodel, comparing it with the most parsimonious model, where we constrained the magnitude of the parameter estimates within each latent source of variance to be equal across the three age cohorts.

Twice the difference in the log likelihood ( $-2\ln L$ ) between a higher order and submodel yields a statistic that is asymptotically distributed as chi square, with the degrees of freedom ( $df$ ) equal to the difference in their number of parameters, and this can be used to determine if the submodel is significantly worse fitting than the

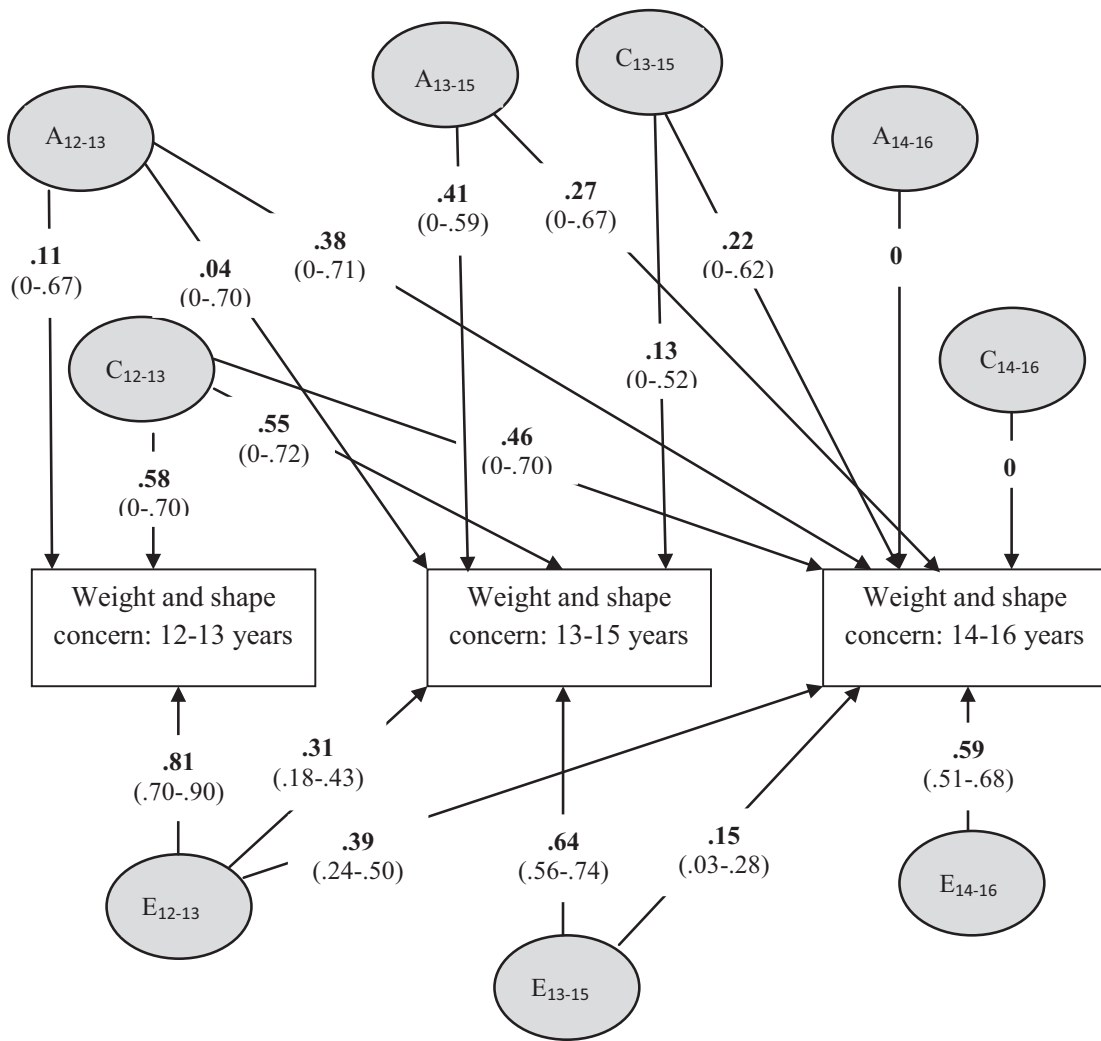


Figure 2. Path diagram showing the unstandardized parameter estimates (and 95% confidence intervals) for the Cholesky decomposition model for the observed weight and shape concern variables across the three age cohorts (A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences).

full model. In this case, the higher order model was the unconstrained ACE Cholesky. Typically, where models do not differ significantly, the Akaike's Information Criterion (AIC) is used to support the choice of a submodel as the best-fitting model, where the lower the value, the better the balance between explanatory power and parsimony.

**Association between specific environmental constructs and weight and shape concern.** Linear mixed models were used to examine cross-sectional associations between environmental variables and weight concern assessed at Wave 1 in each of the three age cohorts. The nonindependent data structure was accounted for by including the twin pair as a repeated measure within the family unit. Effect sizes were calculated using  $2t/\sqrt{df}$ .

## Results

### Reliability and Validity of the Weight and Shape Concern Construct

At Waves 1 and 3, this 8-item scale had high interrater reliability (0.980 and 0.997), where 20 different people across the age groups were randomly sampled from each wave. The internal reliability of the scale was generally high. The Cronbach's alphas for Cohort A were between 0.88 and 0.90, and between 0.90 and 0.91 for Cohorts B and C. Intraclass coefficients for the weight and shape concern measure across the waves of data were 0.46 (95% CI [0.37, 0.54]) for Cohort A, 0.58 (95% CI [0.50, 0.66]) for Cohort B, and 0.52 (95% CI [0.50, 0.61]) for Cohort C. The Tucker congruence coefficient assessed similarity across factors against Lorenzo-Seva and ten Berge's (2006) thresholds for meaningful similarity, where "fair" = .85 to .94 and "good"  $\leq$  .95, with results generally indicating good similarity. Values for Cohort A were .90 between Waves 1 and 2, and 0.97 for between Waves 1 and 3 and between Waves 2 and 3. Values for Cohort B were 0.97 between Waves 1 and 2 and between Waves 1 and 3, and 0.98 between Waves 2 and 3. Values for Cohort C were 0.98 between Waves 1 and 3 and 0.97 between Waves 2 and 3, and 0.98 between Waves 1 and 3.

When testing factorial invariance, a two-factor *configural invariance* model was initially specified with factor means fixed at 0, and factor variances and residual variances fixed at 1. All item factor loadings and item thresholds were then estimated separately.

This model was then compared with a *metric invariance* model in which factor loadings were constrained to be equal across assessment waves and cohorts, but item thresholds were free. The chi-square difference test based on the derivatives of the two models (DIFFTEST; Muthén & Muthén, 1998–2010) indicated that constraining the factor loadings did not significantly worsen the fit of the model ( $\chi^2$  test for difference = 11.88,  $df = 14$ ,  $p = .616$ ). The configural invariance model was then compared with a *full threshold invariance* model in which both item factor loadings and item thresholds were constrained to be equal across assessment waves and cohorts. Again, constraining both item factor loadings and item thresholds did not significantly worsen the fit of the model ( $\chi^2$  test for difference = 123.78,  $df = 107$ ,  $p = .128$ ).

### Twin Correlations

Shown in Table 2 is the cross-twin, cross-trait correlations for each wave of global EDE measurement for each twin. The correlations in bold indicate the correlations within each twin pair, and it can be observed that the MZ correlation is always higher than the DZ correlation for all the three age cohorts but not more than double. Although the MZ and DZ correlation could be constrained to be the same across the three age cohorts, the confidence intervals were broad. The presence of additive genetic variance is therefore indicated, along with both shared and nonshared environmental variance, and therefore the ACE model was chosen as the full model for testing.

### Multivariate Model Fitting

The results of the Cholesky model fitting are shown in Table 3, where all the submodels were significantly worse fitting than the full ACE model. The unstandardized parameters for the unconstrained ACE model are shown in Figure 2. Although we have limited power to make conclusions about the genetic pathways, an independent genetic source emerged at ages 13 to 15 years that also contributed to the phenotype at ages 14 to 16, and there was no evidence of an independent genetic source of variance at this age. Nonshared environmental influences were largely specific to each age cohort, although the nonshared environment contributing to the phenotype at ages 12 to 13 years continues to contribute to the weight and shape for subsequent age cohorts, as did the source

Table 2  
Cross-Twin and Cross-Wave FIML Correlations ( $\times 100$  With 95% Confidence Intervals)

	Twin 1			Twin 2		
	12–13 years	13–15 years	14–16 years	12–13 years	13–15 years	14–16 years
<b>Twin 1</b>						
12–13 years		65 (49, 77)	55 (30, 72)	<b>39 (16, 58)</b>	37 (17, 54)	34 (8, 54)
13–15 years	62 (42, 75)		66 (54, 75)	31 (8, 51)	<b>51 (37, 63)</b>	37 (19, 52)
14–16 years	58 (34, 75)	60 (45, 71)		17 (0, 44)	48 (33, 61)	<b>50 (38, 61)</b>
<b>Twin 2</b>						
12–13 years	<b>24 (0, 47)</b>	23 (0, 46)	12 (0, 38)		54 (35, 61)	59 (34, 75)
13–15 years	36 (14, 55)	<b>39 (22, 53)</b>	31 (13, 47)	51 (31, 67)		61 (48, 72)
14–16 years	18 (0, 41)	35 (17, 50)	<b>34 (18, 48)</b>	51 (26, 68)	62 (48, 73)	

Note. MZ twins are in the top diagonal; DZ twins are in the bottom diagonal. Twin pair correlations are in bold. MZ and DZ correlations could be constrained to be the same at ages 12–13 years,  $\chi^2(1) = 1.83$ ,  $p = .18$ ; 13–15 years,  $\chi^2(1) = 0.81$ ,  $p = .37$ ; and 15–17 years,  $\chi^2(1) = 1.38$ ,  $p = .24$ . DZ = dizygotic; FIML = full-information maximum likelihood; MZ = monozygotic.

Table 3  
 Test Statistics for Model Fitting on the Weight and Shape Concern Variable Using Cholesky Decomposition

Model #	Type of model	-2lnL	df	-2lnL <sub>Δ</sub> (df)	p	AIC
<b>1</b>	<b>ACE<sup>a</sup></b>	<b>3270.42</b>	<b>1297</b>			<b>676.42</b>
2	AE	3354.34	1303	83.92 (6)	<0.001	748.34
3	CE	3335.25	1303	64.83 (6)	<0.001	729.25
4	E	3453.99	1309	183.57 (12)	<0.001	835.99

Note. A = additive genetic influences; AIC = Akaike's Information Criterion; best fitting model in bold = nonshared environmental influences; -2lnL = 2 times the log likelihood using Model 1a as the comparison; -2lnL<sub>Δ</sub> (df) = differences in -2lnL values between each model.

<sup>a</sup> When the fully unconstrained ACE model presented in the table was compared with the fully constrained ACE model, there was no significant difference between the models,  $\chi^2$  (df = 6) = 6.85,  $p$  = .34 (i.e., the estimates for A, C, and E across the three age cohorts were not significantly different).

of variance emerging at ages 13 to 15 years. The shared environmental influence that was present at ages 12 to 13 remained influential over subsequent ages, with a new source of shared environment emerging at ages 13 to 15 that also contributed to ages 14 to 16.

The direction and magnitude of change over time are represented by the relative proportion of the total of the unstandardized values of the latent factors over time, displayed in Figure 3. The shared environment is relatively stable over increasing age, the nonshared environment increases slightly, and the genetic variance increases sixfold, catching up to the contribution than the shared environment at ages 14 to 16 years. When the fully unconstrained model was compared with the fully constrained model, there was no significant difference between the models,  $\chi^2$ (df = 6) = 6.85,  $p$  = .34, that is, the magnitude of genetic variance contributing to each age cohort could be constrained to be the same across the three age cohorts, as could the magnitude of the sources of the nonshared environment. The standardized estimates for A across the three age cohorts were 1% (95% CI [0, 45]), 17% (95% CI [0, 50]), and 22% (95% CI [0, 51]), respectively. The commensurate estimates for C were 34% (95% CI [0, 49]), 32% (95% CI [3, 52]), and 26% (95% CI [4, 81]), and for E were 65% (95% CI [49, 81]), 51% (95% CI [40, 64]), and 52% (95% CI [43, 64]).

### Association Between Specific Environmental Constructs and Weight and Shape Concern

The results of the cross-sectional linear mixed models are shown in Table 4, where large effect sizes for the association with weight

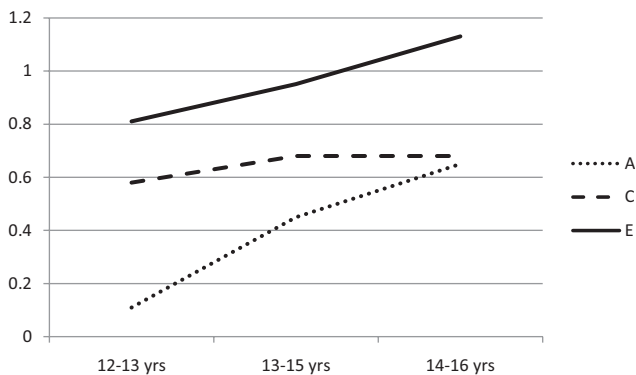


Figure 3. Combined unstandardized variance estimates for weight and shape concern over time for the best fitting model (A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences).

and shape concern and environmental variables were obtained for peer teasing about weight, pressure to be thin, and media internalization across all the age groups. Of note, adverse life events and parental expectations were significant only at ages 13 to 15 years, with a medium effect size. Also emerging more strongly at this age compared with the other two age cohorts were parental expectations. Maternal and paternal care emerged as stronger influences after age 12 to 13 years.

### Discussion

In the current study, we examined whether the genetic and environmental influences on an important risk factor for disordered eating—weight and shape concern—remain stable over adolescence or whether significant developmental shifts during this time could be observed. We first address the reliability and validity of our measure. The EDE was originally designed for use in clinical adult populations, and in the current study, the EDE was slightly modified for use in an adolescent population that was drawn from a community sample. The internal reliability of the weight and shape scale was high (.88 to .91), superior to previous studies showing internal reliabilities of the separate weight concern and shape concern scales ranging from .51 to .85. Our interrater reliability was also high, .980 to .997 at Waves 1 and 3, comparing well with previous examinations of the weight and shape concern subscales (ranging from 0.84 to 0.99 and 0.65 to 0.99, respectively). Interpretation of temporal stability is slightly more complex, in part because we expect weight and shape concern to increase over adolescence (Cooper & Goodyer, 1997), and there are no previous investigations of stability of the EDE over a long period of time in adolescent or adult populations to compare the current results. Test-retest of the EDE with clinical samples over a 2- to 14-day period has ranged from .50 to .76 for the weight and shape concern subscales, and thus our correlations across waves of .46 to .58 seems commensurate with what can be expected, but is lower than that obtained with an adult community sample over a median of 315 days using the EDE-Q (.73 to .75) and lower than 0.60, which has been used to indicate good stability with adolescents (McKnight Investigators, 2003; Klump et al., 2007). The equivalence or similarity of our weight and shape concern factor across the three waves of data collection was generally good. Finally, factorial invariance was indicated for the weight and shape concern measure across the three waves and three cohorts, suggesting that the factor is stable over increasing age, that is, the same construct is measured over time. Therefore, overall, we conclude that the measure was successfully adapted for use with this sample and appears to be both reliable and valid.

Table 4

*Associations (Estimate and Standard Error) Between Weight and Shape Concern and Sources of the Environment at Wave 1 Across the Three Age Cohorts, With Effect Sizes Bolded*

Variable	12–13 years			13–15 years			14–16 years		
	Estimate ( <i>SE</i> )	<i>p</i>	ES	Estimate ( <i>SE</i> )	<i>p</i>	ES	Estimate ( <i>SE</i> )	<i>p</i>	ES
Parental criticism	0.11 (0.03)	<0.001	0.48	0.13 (0.03)	<0.001	0.63	0.08 (0.03)	0.001	0.48
Parental expectations	0.06 (0.03)	0.06	0.23	0.11 (0.03)	0.001	0.45	0.01 (0.03)	0.69	0.06
Parental conflict	0.05 (0.03)	0.02	0.15	0.06 (0.02)	0.009	0.35	0.05 (0.02)	0.005	0.42
Maternal care	−0.03 (0.03)	0.18	0.17	−0.09 (0.02)	<0.001	0.48	−0.10 (0.02)	<0.001	0.60
Paternal care	−0.07 (0.03)	0.02	0.31	−0.14 (0.03)	<0.001	0.65	−0.11 (0.03)	<0.001	0.63
Peer teasing about weight	0.33 (0.03)	<0.001	1.52	0.33 (0.03)	<0.001	1.60	0.32 (0.03)	<0.001	1.84
Media internalization	0.42 (0.03)	<0.001	1.58	0.41 (0.03)	<0.001	1.64	0.27 (0.02)	<0.001	0.97
Pressure to be thin	0.25 (0.02)	<0.001	1.54	0.26 (0.02)	<.0001	1.60	0.23 (0.03)	<0.001	1.33
Adverse life events	0.03 (0.03)	0.42	0.07	0.13 (0.04)	<0.001	0.63	0 (0.04)	0.99	0.002

Note. ES = effect size, Cohen's *d*.

The next issue relating to the validity of our measure pertains to ongoing discussion in the literature as to whether interview or self-report measures are more accurate in assessing eating pathology in the context of a very reliable finding across 16 studies that higher scores of eating pathology are derived from the EDE-Q compared with the EDE (Berg et al., 2011). There is no definitive answer for the question relating to relative validity of the interview versus the self-report questionnaire, and arguments exist for both positions. The EDE is typically used as the gold standard when assessing the validity of the EDE-Q (e.g., Black & Wilson, 1996; Berg et al., in press), correlates more highly with daily food records when assessing binge episodes than the EDE-Q (0.56 to 0.93 compared with 0.31 to 0.63; Berg et al., 2012), and is a better indicator than the EDE-Q of severity of functional impairment and distress (Mond, Hay, Rodgers, & Owen, 2007). However, eating disorder symptoms are more likely to be endorsed under conditions of anonymity, perhaps due to the shame attached to such behaviors (Lavender & Anderson, 2009), and questionnaire and interview scores have been found to be more similar when interviews are conducted over the telephone rather than in person (Keel, Crow, Davis, & Mitchell, 2002). Given that the EDE interview was administered over the telephone in the current study, we can speculate that we have the best of both worlds: a degree of anonymity and the ability to probe replies and clarify answers.

A further issue of relevance to the interpretation of the results of the current study is that we would typically expect a lower estimate of the genetic variance when using diagnostic interviews as opposed to self-report questionnaires (Burt, 2009). The results support this trend, where we had estimates of genetic variance ranging from 1% to 22% across the three age cohorts for our eight-item measure compared with 54% for commensurate age groups using the 12-item EDE-Q measure of weight and shape concern (Klump et al., 2010). Contrary to our hypothesis, as well as indicating the contribution of both genetic and nonshared environmental variance to weight and shape concern, the results strongly supported the presence of the shared environment. The shared environment has previously been indicated in interview assessments of weight concern in middle-aged women (Wade et al., 1998), undue influence of weight in young women (Reichborn-Kjennerud et al., 2004), and undue influence of weight and shape in adolescents (Wilksch & Wade, 2009a). However, interview assessment of adults shows little support for the presence of the shared environ-

ment for either the two EDE items assessing undue influence or the remaining 10 items in the weight and shape concern subscale (Wade, Zhu, & Martin, 2011). Apart from preadolescent girls (Klump et al., 2000, 2007), the shared environment has not been indicated in self-report measures of body dissatisfaction or disordered eating. This may indicate that the shared environment may be most important for the undue influence aspect of weight and shape concern in younger women or girls and of little importance for the body dissatisfaction component of weight and shape concern. Alternatively, it may indicate that the way in which this construct is measured (interview vs. self-report) may affect similarity of twin-pair reports. However, given that different interviewers were used for each twin in the pair in the current study, this seems an implausible explanation.

We had two main aims of the current research: to explore whether new sources of latent variance were introduced over development and whether the magnitude of variance contributed by these latent risk factors changed over age. We can conclude, consistent with our hypothesis, that nonshared environmental influences were largely specific to each age cohort but each source continued to contribute significantly to subsequent age cohorts. We have somewhat less power with respect to the results pertaining to the shared environmental and genetic influences, given the presence of zero in the 95% confidence interval estimates. Sources of shared environment that were influential at 12 to 13 years continued to make a major contribution at ages 13 to 15 and 14 to 16, and an independent source of shared environment emerged at ages 13 to 15, which continued to influence weight and shape concern at ages 14 to 16. There were no new sources of shared environment or genetic influence emerging after ages 13 to 15. In addition to the genetic variance present at ages 12 to 13, our results indicate that a new (and indeed the singly most powerful) source of genetic variance emerged when the girls were postpubertal (ages 13 to 15). This suggests that weight and shape concern may be influenced differently from disordered eating, where it has been suggested that ovarian hormones associated with puberty are the main source of genetic risk for disordered eating (Klump et al., 2003, 2007; Culbert et al., 2009). We were also able to constrain the magnitude of all three latent influences to be the same across adolescence, though it should be noted that the genetic contribution to weight and shape concern did increase sharply in comparison with changes in environmental variance over age.

A novel contribution of the current research is our ability to link our behavioral genetic results with an examination of specific environmental variables in our population and how their association with weight and shape concern changes over the age cohorts. In terms of factors that are present at ages 12 to 13 and remain influential over subsequent age groups, large effect sizes were obtained for the association between weight and shape concern and the following variables: peer teasing about weight, pressure to be thin, and media internalization. Three variables emerged as becoming more important after ages 12 to 13, namely, adverse life events and parental expectations (both of which were significant only at ages 13 to 15 years), and maternal care was significant only at ages 13 to 15 and 14 to 16 years. These results suggest that sociocultural norms of thinness and attendant pressures remain insidious and powerful influences on weight and shape concern over adolescence. It is of interest that maternal care only emerges as significant after adolescence has commenced—it may be that this time of change and increased independence places duress on what would typically be seen as one of the most important relationships during childhood. The emergence of adverse life events and parental expectations at ages 13 to 15 only would indicate that this is a time of special risk for the development of weight and shape concern, as this is the time where most environmental risk factors appear active, as well as being a time when a new source of genetic risk is possibly introduced. This would accord with research that shows that weight and shape concern emerges before disordered eating (Cooper & Goodyer, 1997), and that the peak age of the onset for bulimia nervosa and related disorders is between ages 15 and 17 (Stice, Marti, Shaw, & Jaconis, 2009).

Although we should exercise caution in interpreting the results relating to genetic variance over time, the explanation for the strongest single genetic risk emerging at ages 13 to 15 is likely to indicate complex interactions between genes and the environment. Our results suggest that this is a period for exposure to more environmental events that may interact with genetic risk, thereby increasing that genetic risk, that is, a genotype–environment interaction (GxE). An example of this from an unrelated field is tobacco use in two historical cohorts in Sweden (Kendler, Thornton, & Pedersen, 2000), one where smoking was rare for women and one in which smoking had become widespread, showing respective heritability estimates in women of 0% and 63%, whereas heritability for men stayed relatively stable (63%). Environmental candidates for GxE, which have previously been found to increase heritability for disordered eating, are parental divorce (Suisman, Burt, McGue, Iacono, & Klump, 2011) and an increased exposure to dietary restraint (Racine, Burt, Iacono, McGue, & Klump, 2011). The challenges of entering adolescence may also represent a significant stressor for some individuals, which could implicate epigenetic action, where the environment can activate a specific gene. This could suggest a role for the serotonin transporter gene promoter polymorphism, which, although it is not directly associated with bulimic behaviors, is associated with a variety of potential endophenotypes for bulimic disorders, including affective instability and behavioral impulsivity in the face of adverse environmental challenges (Steiger et al., 2007). However, a range of other genes has been indicated but not definitively shown to be associated with disordered eating, including those related to the activity of gonadal hormones and brain-derived neurotrophic factor (Klump & Gobrogge, 2005).

The results of the current research should be viewed in the context of six important limitations. First, we had no measure of puberty in the current study, and therefore could not definitively ascertain that our adolescents in Cohort A had actually attained puberty. Therefore, we may have included twins with prepubertal status, which could increase shared environmental estimates (Klump et al., 2007). Second, although the range of weight and shape concern is large, the mean is relatively low and may limit our error variance estimations. However, in the current study, we observe relatively strong support for the reliability and validity of our measure. Third, we had a 49% response rate across the families of the twin population approached, which is commensurate with other large, Australian twin studies (Wade, Crosby, & Martin, 2006). Although previous research with Australian twin adult cohorts who have been asked to participate in studies on disordered eating have not shown a relationship between response rates and BMI or level of eating pathology, suggesting those with disordered eating did not avoid participating (Wade et al., 2006; Wade, Tiggemann, Martin, & Heath, 1997), the activity of such biases cannot be ruled out with respect to the current population. However, it should be noted that the use of multiple waves of data from the same respondents in the current study prevents any ascertainment bias that may appear over the course of the data collection in the current study (Wade, Neale, Lake, & Martin, 1999). Therefore, although, across the whole data set, we note that attrition differed between MZ- and DZ-twin pairs, with 75% of MZ-twin pairs completing Wave 3 assessments compared with 68% of DZ twins, the potential impact of imputing missing values in a larger proportion of DZ compared with MZ twins is minimized. Fourth and consistent with previous research (e.g., Klump et al., 2007), we did not control for body mass index (BMI). Previous research suggests that the genetic influences contributing to BMI and weight and shape concern are largely independent (Slof-Op't et al., 2008; Wade et al., 2011; Klump et al., 2000), and when we ran analyses covarying for BMI, there was not much difference in the pattern of results. Fifth, although we can draw robust conclusions about the estimates for the pathways associated with the nonshared environment, we have limited power to draw robust conclusions about the parameters associated with the shared environment and the genetic variance, given that zero is included in the lower bound of the confidence intervals. However, the results clearly indicate that both the shared environment and additive genetic action contribute to weight and shape concern over adolescence. Finally, analyses confounded cross-sectional comparisons of age cohorts with longitudinal changes and thus do not represent a purely developmental examination of changes over time.

Taken together, these results support the existence of developmental shifts in nonshared environmental effects for weight and shape concern over adolescence, with possible shifts also present for the nonshared environment and genetic effects. Our results suggest that the most vulnerable age for an increase in weight and shape concern, which is an important risk factor for disordered eating, is 13 to 15 years. This may be a peak developmental stage around which prevention programs should be aimed and tested. In particular, skills involving standing up to pressures around the thin ideal and stress management may be of special importance in such programs. To date, one such program that has targeted these risk factors in this age group has shown a long-term prevention effect

for weight and shape concern (Wilksch & Wade, 2009b), but the impact on the growth of disordered eating is as yet unknown.

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