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Changes in genetic and environmental influences on disordered eating between early and late adolescence: A longitudinal twin study

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Abstract

Background: We investigated the genetic and environmental contributions to disordered eating (DE) between early and late adolescence in order to determine whether different sources of heritability and environmental risk contributed to these peak times of emergence of eating disorders.

Methods: Adolescent female twins from the Australian Twin Registry were interviewed over the telephone with the Eating Disorder Examination (EDE). Data were collected at 12-15 and 16-19 years (Wave 1: N=699, 351 pairs; Wave 3: N=499, 247 pairs). Assessments also involved self-report measures related to negative life events and weight-related peer teasing.

Results: Unstandardised estimates from the bivariate Cholesky decomposition model showed both genetic influences and non-shared environmental influences increased over adolescence, but shared environmental influences decreased. While non-shared environmental sources active at ages 12-15 continued to contribute at 16-19 years, new sources of both additive genetic and non-shared environmental risk were introduced at ages 16-19. Weight-related peer teasing in early-mid adolescence predicted increases of DE in later adolescence, while negative life events did not.

Conclusions: Two-thirds of the heritable influence contributing to DE in late adolescence was unique to this age group. During late adolescence independent sources of genetic risk, as well as environmental influences are likely to be related in part to peer teasing, appear key antecedents in growth of DE.

Key words: Global EDE, adolescents, twins, genetic, environmental, longitudinal

It has been known for some time that eating disorders “run in families”, with a 7- to 12-fold increase in the prevalence of anorexia nervosa (AN) or bulimia nervosa (BN) in relatives of eating disordered probands compared to families of controls (Klump *et al.* 2001). Historically, and in common with many other types of psychopathology, there has been a view that families cause eating disorders, a view reinforced by patients referred for treatment, who most commonly report the perceived cause of the eating problem to be a dysfunctional family (Tozzi *et al.* 2003). However, over the last 20 years a number of twin studies have shown that there is a clear and substantial genetic contribution to both BN and AN (Fairburn & Harrison, 2003), in conjunction with an influence of the non-shared environment. The role of the shared environment in disordered eating (DE) is less certain (Bulik *et al.* 2000).

A more nuanced understanding of how genes and the environment impact on the aetiology of DE is slowly emerging. Two longitudinal studies suggest shifts in genetic and environmental risk factors between childhood and adolescence. In a study of female twins at ages 11, 14 and 18 years (Klump *et al.* 2007), the shared environment was found to be a significant contributor to DE in the pre-pubertal age group, with negligible impact at age 14. Conversely, the impact of heritability was negligible at 11 years, but increased significantly by age 14. The source of genetic variance active at 11 years of age continued to be a major contributor to variance at ages 14 and 18 years, and a new source of genetic variance appeared at age 14 which made only small contributions to the variance at ages 14 (1%) and 18 (18%), with no new sources of genetic influence appearing at 18 years. In contrast, new sources of non-shared environmental variance appeared at each age. Authors of this longitudinal study (and other cross-sectional investigations) suggest that puberty and associated ovarian hormones may contribute to an environmental trigger “switching on” the main source of genetic risk for DE (Klump *et al.* 2003; Culbert *et al.* 2009; Klump *et al.* 2012; Klump, 2013). A second longitudinal study, this time assessing weight and shape concern in adolescent twins (Wade *et al.* 2013) over three occasions (12-13, 13-15 and 14-16 years), showed a similar pattern of results, with non-shared environmental influences largely specific to each age cohort, and genetic influences influential at 12-13 years continuing to contribute to subsequent age cohorts, with independent sources of genetic influence emerging at ages 13-15, but not in later adolescence. This latter study omitted to examine older adolescents, where the transition from adolescence to adulthood represents

an important risk period for the emergence of eating disorders, especially those related to binge and purge behaviours (Lewinsohn *et al.* 2000; Hudson *et al.* 2009).

While no specific genes have been identified for eating disorders yet, weight teasing has been identified as a potentially important environmental influence, predicting frequent dieting in girls, as well as binge eating and unhealthy weight control in boys (Haines *et al.* 2006). Relatedly, perceived pressure to be thin predicts growth in importance of weight and shape in adolescents (Wilksch & Wade, 2010), a diagnostic criterion for eating disorders, and comments about amounts eaten or appearance by family members as children were growing up is a retrospective correlate for both AN and BN (Fairburn *et al.* 1999; Wade *et al.* 2007). In addition, an increase in negative life events predicted onset of eating disorders in a female adolescent sample (McKnight Investigators, 2003), with retrospective recall of negative life events predicting onset of BN (Welch *et al.* 1997).

Given the omission of an older adolescent group in a previous longitudinal examination of the cognitive component of DE (Wade *et al.* 2013), and lack of research addressing important environmental risk factors in the context of any shifts in genetic contribution to DE within this developmental period, the purpose of the current study was twofold. First, we wished to examine contributions of genetic and environmental variance contributing to DE in adolescents, measured using the global score of the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), used as the primary indicator of outcome in treatment trials of eating disorders, both AN and BN, in children and adults (le Grange *et al.* 2007; Fairburn *et al.* 2009; Lock *et al.* 2010; Wade *et al.* 2011). It incorporates *both* cognitive and behavioural indicators of DE. In particular, our aim was to examine any changes in genetic and environmental influences between early (i.e., before age 16) and late adolescence (i.e., from 16 years of age) in order to revisit the question of whether new sources of heritability emerge later in adolescence, given that the only previous longitudinal study of DE suggested that this is not the case (Klump *et al.* 2007). Second, we examined the predictive role of two risk factors for DE, namely peer teasing about weight and negative life events. While puberty has been considered as an environmental influence in early adolescence, we wished to investigate the nature of any emerging environmental risk factors over late adolescence.

METHOD

Participants

The current study used outcome measures derived from three waves of data from adolescent female-female twin pairs, described previously (Wade *et al.* 2008; Wilksch & Wade, 2009, 2010; Wade *et al.* 2013). Given that we wished to examine DE outcomes in non-overlapping age groups that typified the peak risk periods of emergence of eating disorders (early and late adolescence), only the first and third wave of data collection were used as the age at the middle wave of data (Wave 2; mean age 15.10 years, $SD=0.83$, range: 13.76-17.56) overlapped with age at the first and third waves. Data pertaining to environmental risk factors (i.e., weight-related peer teasing, negative life events) were derived from Waves 1 and 2.

At Wave 1, twin pairs who were between 12 and 15 years of age registered with the Australian Twin Registry (ATR) were approached, along with their parents, to participate in the present study by the ATR. Seven hundred and nineteen families were contacted and invited to participate, and of these 411 (57.2%) agreed, 237 (32.9%) declined, and 71 (9.9%) did not reply. Researchers approached those agreeing to take part and sent self-report questionnaires to both parents, including those families where the parents did not live together. After parents returned the questionnaires, the EDE (Fairburn & Cooper, 1993) was conducted by telephone, such that 699 twins were interviewed at separate times with a different interviewer for each child in the family. The sample was Caucasian and the socioeconomic indexes for areas (SEIFA) – a standardised 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 ($SD = 11.36$). In order to ensure no overlap in ages between Waves 1 and 3, 8 pairs of twins where one or more was aged 16 at the time of first assessment were removed from the data set, such that the mean age of the twins who participated was 13.90 years, $SD=0.80$ (range: 12.70-15.47)¹.

¹A representative study of Australian children (Longitudinal Study of Australian Children) has identified 74% of girls aged 10-11 years, and 92% aged 12-13 years showed signs of puberty onset (Gray & Sanson, 2005; Gray & Smart, 2008; Edwards, 2012; Edwards, 2014). This suggests that the vast majority of the younger cohort have entered puberty and is unlikely to be pre-pubertal.

All twins were contacted again at Wave 2 (including non-responders); the mean duration of time between Waves 1 and 2 was 1.15 years ($SD = 0.17$). A total of 514 parents completed questionnaires (86% of Wave 1) and 669 twins completed interviews (96% of Wave 1). Mean age was 15.10 years, ($SD=0.83$; range: 13.76-17.56).

At Wave 3 all twins, responders and non-responders, were again approached, of which 499 (71% of Wave 1) completed interviews, with a mean duration of time between Waves 1 and 3 of 2.96 years ($SD=0.27$), ranging from 1.91 to 4.65. The mean age of the twins was 16.90 years, $SD=0.70$ (range: 15.49-19.84), and age was significantly different from Wave 1 ($t[df=488] 261.18, p < .001$). Attrition at Wave 3 was unrelated to EDE scores at Wave 1 ($OR = 1.15, 95\% CI = 0.88-1.51$); this was also the case for the environmental risk factors at both Wave 1 and 2 respectively for life events ($OR=1.04, 95\% CI = 0.89-1.21$; $OR=0.96, 95\% CI = 0.87-1.06$) and weight-related peer teasing ($OR=1.00, 95\% CI = 0.76-1.34$; $W2: OR=1.01, 95\% CI = 0.72-1.42$). Whilst not significant, DZ twins showed a tendency to be less likely to participate at Wave 3 than MZ twins ($OR=0.72, 95\% CI: 0.51-1.00$).

Zygoty assignment used parental responses to standard questions about physical similarity and confusion of twins by parents, teachers, and strangers; this method gives better than 95% agreement with genotyping (Eaves *et al.* 1989). Where there was uncertainty ($N=46$ pairs), DNA testing was used to assign zygoty for 39 pairs (DNA was not available for seven pairs and these pairs were therefore not included in the analyses). At Wave 1 there were 367 monozygotic (MZ) twins (182 complete pairs and 3 incomplete pairs), 302 dizygotic (DZ) twins (151 complete pairs), and 7 pairs where zygoty was unknown i.e., 340 complete pairs and three incomplete pairs. Wave 2 constituted 330 complete pairs (178 MZ; 146 DZ; and, 6 without zygoty) and 8 incomplete pairs (5 MZ and 3 DZ). At Wave 3, there were 243 complete pairs (of which 136 were MZ, 106 DZ, and 1 without zygoty) and 5 incomplete pairs (4 MZ and 1 DZ). The number of twin pairs completing the EDE at Wave 1 was 340, with 243 complete pairs (72% of Wave 1) available across both Waves. The Flinders University Clinical Ethics Committee approved the data collection process; parents gave written informed consent and twins gave written assent.

Disordered eating

The twin interview consisted of two parts for Waves 1 and 2; the EDE (Fairburn & Cooper, 1993) and questions from self-report questionnaires assessing a range of variables including weight-related peer teasing and life events (Wilksch & Wade, 2009; 2010). Parents also completed self-report questionnaires at Waves 1 and 2, including twin weight, height, and life events. The interview at Wave 3 comprised only the EDE interview. Sixteen postgraduate Clinical Psychology trainees competent in the use of the EDE conducted all interviews.

The EDE interview generates a global measure of eating psychopathology (22 items) that includes four subscales weight concern, shape concern, eating concern and dietary restraint. It was adapted for the younger age of the sample simply by encouraging participants to verbalise the list related to the following question: “*imagine the things that influence how you feel about (judge, think, evaluate) yourself as a person – such as how you are doing at school, what sort of friend you are, how you get along with other people – and put these things in order of importance, where does your weight/shape fit in?*” The EDE is generally considered the ‘preeminent’ eating disorder interview assessment tool with sound validity and reliability (Berg *et al.* 2011; Berg *et al.* 2012; Berg *et al.* 2012a).

The global EDE was found to perform validly in our sample. First, construct validity was demonstrated by global EDE scores among those meeting diagnostic thresholds for key DE symptoms (e.g., fear of weight gain, purging) being significantly higher than those who did not meet criteria (data available from authors on request). Second, convergent validity was confirmed with medium-large correlations with Eating Disorder Inventory (EDI: Garner *et al.* 1983) *interoceptive awareness* and *drive for thinness* measures. Third, factorial invariance suggested that the global EDE remained invariant across Waves 1 and 3. The chi-square difference test showed the *metric invariance* model was not a significantly poorer fit to these data (χ^2 test for difference = 30.997, df = 21, p = .07), nor the *full threshold invariance* model (χ^2 test for difference = 166.52, df = 140, p = .06). Medium-large cross-wave correlations (ranging 0.37-0.42,) suggest the global EDE score remains quite stable over time. Fourth, the internal reliability of the global EDE was 0.93 at both Wave 1 and 3.

Body Mass Index (BMI) centile

As data relating to parental report of the twins' weight and height were highly correlated, the mothers' report was used, aside from instances where these data were missing, when the fathers' report was used. At Wave 3, twins reported their own weight and height. We adopted the Center for Disease Control recommendation to use BMI-for-age (or BMI centile) in this sample. Mean (standard deviation) BMI centile and range for both twin 1 and twin 2 with analogous details for the global EDE score are reported **Table 1**.

Measures of specific risk²

At Waves 1 and 2, a measure of weight-related peer teasing (McKnight Investigators, 2003) was undertaken by twins. The scale consisted of eight items (e.g., In the past year, how often have girls - including sisters - made fun of you because of your weight?) where responses were made on a 3-point scale (1=never, 2=sometimes, 3=a lot) from which a mean score was calculated. Cronbach's alpha was 0.87 and 0.89, respectively. Parents were asked to report if the twin had encountered negative life events in the preceding 12 months (yes/no) to Waves 1 and 2, listing 13 items: including death of a family member, moving home, starting at a new school, parental separation, or a breakdown of a close friendship. A count variable of those life events was created which summed the aforementioned items.

Statistical Analysis

Twin correlations. All subsequent analyses treated data as continuous, and a full information maximum likelihood (FIML) approach was used with the statistical package Mx (Neale, 1994) designed to employ structural equation modelling approaches to twin data with complete and incomplete pairs of twins across the waves automatically creating respective mean vector and covariance matrix for each data point (Neale, 1994). The twin pair correlations for monozygotic (MZ) and dizygotic (DZ) twins for each phenotype were calculated. As MZ twins share 100% of their genes, but DZ twins share only 50%

² ACE bivariate Cholesky modelling of life events at Wave 1 and Wave 2 showed heritable influences conveyed respectively 15.15% and 13.82% of total variance, in addition to shared environmental influences (78.69% and 78.64%) and non-shared environmental influence (6.16% and 7.53%). Analogous modelling for weight-related peer teasing identified genetic variance of 13.65% and 3.69%; with shared environmental variance of 22.63% and 43.93%, and non-shared environmental variance of 63.72% and 52.38%.

(on average), additive genetic effects on a phenotype can be deduced from MZ twin correlations being approximately double DZ twin correlations (Plomin *et al.* 1990).

Multivariate model fitting. A bivariate Cholesky decomposition model was used to analyse DE at Wave 1 and 3; models were fitted with and without adjustment for BMI-centile. The structure of the model can be seen in **Figure 1**. As bivariate models use both variances of individual variables and covariances between the different variables to estimate parameter they are more powerful than their univariate counterparts (Neale *et al.* 2003). Further, use of repeated measures enables correction of any ascertainment bias due to differential attrition (Little & Rubin, 1987), also diminishing proportion of measurement error attributed to the non-shared environment (Bulik *et al.* 1998).

Initially, models were fitted in which the magnitude of the parameter estimates was allowed to vary across assessment periods (i.e., Wave 1 and 3), starting with a full model (i.e., containing the additive genetic variance [A], shared environment [C] and non-shared environment [E] sources of variance). Subsequently, a series of nested models examined whether all sources of variance were necessary, where 95 % confidence intervals (CI) for all estimates assisted identifying the significance of the models. Twice the difference in the log likelihood (-2lnL) between a higher order and sub-model 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 can be used to determine if the sub-model is significantly worse fitting than the full model. Typically, where models do not differ significantly, the Akaike's Information Criterion (AIC) determines the selection of the sub-model as the best fitting model, where the lowest value represents the best combination between explanatory power and parsimony.

Relationship between specific risk variables and Wave 3 disordered eating. To investigate putative influences on DE at 16-19 years, LMM analyses were conducted with five models: (a) weight-related peer teasing at Wave 1; (b) weight-related peer teasing at Wave 2; (c) change in level of peer teasing between Wave 1 and Wave 2; (d) total life events at Wave 1; and, (e) total life events at Wave 2. All models adjusted for baseline (Wave 1) EDE global scores and BMI centile by including as covariates. Effect sizes (ES) were computed using $[2\sqrt{F}]/\sqrt{df(\text{error})}$.

RESULTS

Twin correlations

Table 2 reports the cross-twin, cross-trait correlations of the global EDE score at Wave 1 and 3 for twin 1 and 2. Bolded correlations highlight correlations within each twin pair, and show MZ correlations ($r_{\text{Age } 12-15}: .43$; $r_{\text{Age } 16-19}: .36$) are observed to be greater than DZ ($r_{\text{Age } 12-15}: 0.20$, $r_{\text{Age } 16-19}: 0.01$), such that it is probable additive genetic factors contribute to the association between EDE levels in both early and late adolescence.

Multivariate model fitting

The results of the Cholesky model fitting are shown in **Table 3**. The ACE model fit was retained as recommended (Sullivan & Eaves, 2002) although the AE sub-model achieved greater parsimony, without decrement in model fit (as determined by the lowest AIC value). The standardised and unstandardised parameters (and 95% confidence intervals), percentage variance, and standardised estimates adjusting for the influence of BMI centile for the ACE model are shown in **Figure 1**. The unstandardised parameter estimates of genetic influence more than doubled over time (from 0.08 to 0.18) accompanied by a 30% increase in non-shared environmental influence (0.27 to 0.38). Importantly, late adolescence includes new sources of both genetic and non-shared environmental influences that predominate the risk period in late adolescence, with two-thirds of the heritable influence contributing to DE in late adolescence being unique to this age group. There is a moderate correlation between the latent genetic risk variables ($r_A = 0.63$; 95% CI = -1.0-1.0), shared environment ($r_C = 1.0$ (95% CI 1.0-1.0), and between the two latent non-shared environment variables ($r_E = 0.26$; 95% CI = 0.19-0.45) contributing to DE.

Standardised estimates indicated that, in early adolescence, latent sources of genetic (19.0%), shared (18.7%) and non-shared environmental (62.3%) variance impact DE, with 29.6% of the sources acting on late adolescence being genetic, contrasting with shared (7.6%) and non-shared sources (62.7%) that decrease. When adjusting for the influence of BMI centile these estimates were similar despite confidence intervals being marginally broader, a probable consequence of reduced power as inferred by one further path becoming non-significant.

Relationship between sources of specific risk variables and disordered eating at Wave 3

Table 4 shows two of the five independent specific risk variables predicted growth in DE. This included higher levels of weight related peer teasing at Wave 2 and an increase in peer teasing between Waves 1 and 2. There was no relationship with life events in the previous 12-month period.

DISCUSSION

The role of genetic and specific risk factors on the development of DE remains unclear, given few existing longitudinal studies (Klump *et al.* 2007, Wade *et al.* 2013), especially those that examine a critical risk period for the emergence of DE: the transition from adolescence to adulthood. We therefore used longitudinal adolescent twin data to determine whether significant change in heritable variance of the DE phenotype occurred between early (before 16 years) and late adolescence (after 16 years). While non-shared environmental influence was observed to occur early and to persist across adolescence, we also found evidence for a new source of genetic influence contributing to expression of DE symptomatology in late adolescence. Importantly, this is the first study to report the presence of an independent heritable source - putatively, a late adolescence genetic diathesis. Notably, two-thirds of the heritable influence contributing to DE in late adolescence is unique to this age group. This timeframe accords with the later emergence of binge and purge symptomatology, well documented to have peak age of onset in late adolescence and early adulthood (Favaro *et al.* 2009; Kessler *et al.* 2013). A new source of non-shared environmental risk was also identified in late adolescence, accounting for 80% of the environmental contribution at this time. In accordance with the recommendation by Sullivan and Eaves (2002) we modelled influence derived from shared environmental sources, but as with other studies utilising Cholesky decomposition modelling with such data, we found non-significant influence - a likely consequence of insufficient power. Certainly, by late adolescence the variance accounted for by the shared environment is very small, estimated at 7.6%.

Our second aim explored the nature of specific risk factors for DE that occurred in early- to mid-adolescence, preceding the increase in genetic influence in later adolescence for DE. Despite negative life events having been shown to associate with DE in adolescence (e.g., McKnight Investigators, 2003), in the current sample it was not predictive of DE in late adolescence. There are a number of reasons for this: 12-month life events preceding the first two waves of data collection may not have provided

sufficient numbers of traumatic experiences to adequately produce a detectable effect; earlier sources of trauma may be more influential in the later development of DE e.g., parental loss in the form of separation prior to age 17 years was associated with increased risk for major depression as an adult (Kendler *et al.* 1992); the number of life events may be a blunt instrument in this context as opposed to a weighting based on self-report of impact; as the traumatic life events were reported by parents, it may be that the most traumatic events were kept secret from the parents by the twin, such as sexual assault or bullying at school, and thus were not assessed in the current study. This latter suggestion is somewhat supported by the finding that the life events measure was heavily influenced by the shared environment.

Contrastingly, both increased levels of weight-related peer teasing from early- to mid-adolescence, as well as greater weight-related peer teasing measured during mid-adolescence, were related to development of DE in late adolescence. These results underscore experience of weight-related peer teasing as being exceptionally challenging during adolescence, consistent with previous research that has highlighted the role of factors related to peer group acceptance in the emergence of DE. This is a developmental period combining significant hormonal changes usually functioning to redistribute or increase body adiposity (e.g., Garn *et al.* 1986) and transitions from relationships predominantly family-focused to greater independence and peer-oriented connectivity associated with increased investment in seeking acceptance by peers (Brown *et al.* 1986; Larsen *et al.* 1996).

Taken together, our two main study outcomes are suggestive of the possibility of an interaction between the emergence of a period of genetic risk for DE and weight-related peer teasing in mid to late adolescence. In other words, this period in adolescence conveys heritable risk for DE at a time when non-shared environmental influences increase in importance as greater emphasis is placed on independence and tighter peer-focused relationships. Our results suggest approaches that strategically targeting peer-relationships and skills for standing up to teasing and other weight related pressures within preventive interventions are likely to present key opportunities to protect older adolescents from the emergence of heritable risk factors for the development of DE, especially binge and purge behaviours. This is consistent with the success of approaches for young adolescents that prevent the growth of weight and shape concern by targeting internalisation of cultural appearance ideals promoted by media and peers (Richardson & Paxton, 2010; Bird *et al.* 2013; Wilksch *et al.* in press).

It is of interest to note in the current study that the profile of risk factors for DE change even while the mean levels of DE have remained stable. One recent study using the self-report version of the EDE showed mean scores were elevated slightly at 17 years, while symptoms prior (14 years old) and post (20 years old) were of similar magnitudes (Allen *et al.* 2013). It is possible that our groups have bisected Allen *et al.*'s peak symptom group, resulting in an "averaging" of their profile. Studies using other measures to track DE symptomatology over time show no coherent common trajectory across adolescence (e.g., Kotler *et al.* 2001; Klump *et al.* 2007; Neumark-Sztainer *et al.* 2011; Abebe *et al.* 2012).

The current study needs to be considered within a number of limitations, the first of which relates to no formal measure of puberty being available in the current study. However, the population-based age ranges suggests our sample is highly likely *not* to be pre-pubertal; rather, participants are already on a trajectory to sexual maturity. Further work is required to assess how stages within puberty progression impact on the results. Second, the low mean of the EDE global score, although the range was considerable, which may have limited error variance estimations. Third, while the impact of life events and weight-related peer teasing were found to be predominantly environmental origin, the small heritable influences contributed by these variables may conflate the additive genetic and non-shared environmental variance in our analysis. Fourth, the initial response rate was 49%, akin to equivalently large epidemiological studies of twins incorporating multiple time points (Stice *et al.* 2013; Wade *et al.* 2006). Fifth, while our primary analyses did not control for BMI centile, we did run additional models adjusting for BMI. Minor differences were observable, but confidence intervals were broadened, indicating a lack of power which argues for the use of larger samples in the investigation of the complexity of genetic and environmental risk factors on DE over adolescent development. Finally, participants reporting DE at Wave 1 were offered referrals, which may have influenced DE at Wave 3. Unfortunately no treatment data are available to test this suggestion.

Future research should more explicitly aim to test the presence of gene and environment interactions occurring at later adolescence in terms of increasing risk for later onset DE in early adulthood. This is not a trivial problem, given previous research has found that 23% of women aged between 22 and 27 years of age experience DE in the 12-month period before assessment, which results

in a sustained negative impact on quality of life (Wade *et al.* 2012). A better understanding of how to protect young women from the onset of DE can be expected to result in a greater participation of young women in society, given the impact of DE on social, vocational and educational functioning, and resultant reduced productivity (Simon *et al.* 2005).

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Title for figure*Figure 1*

Bivariate Cholesky decomposition path diagram for disordered eating at 12-15 and 16-19 years of age showing the standardised [unstandardised] parameter estimates for A, C and E, proportion of total heritability (%), and BMI adjusted standardised estimates (*italics*) with respective 95% CI; (A=additive genetic influences; C= shared-environment; and E=non-shared environmental influences). Non-significant pathways are dashed, significant pathways are solid.

Figure 1

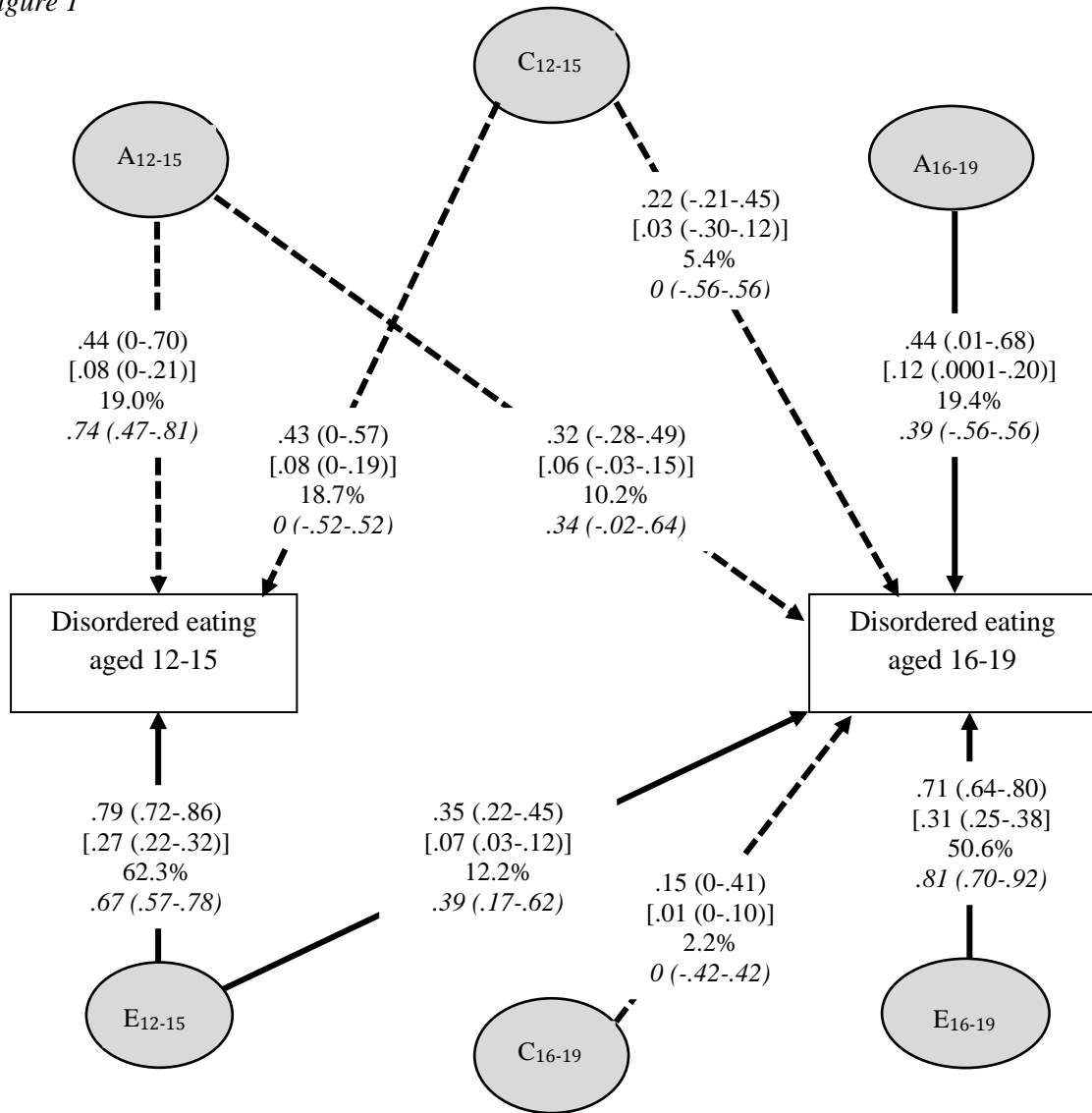


Table 1

BMI centile and global EDE means (SD) and range at Wave 1 (12-15 years) and 3 (16-19 years)

	Data collection point	
	12-15 yrs M (SD)	16-19 yrs M (SD)
BMI centile Twin 1 12-15 years	52.92 (29.94)	54.04 (28.62)
<i>Range</i>	0.01-99.91	0.00-99.97
BMI centile Twin 2 16-19 years	50.77 (30.97)	49.97 (28.75)
<i>Range</i>	0.00-99.91	0.00-99.98
EDE global Twin 1 12-15 years	0.40 (0.61)	0.40 (0.65)
<i>Range</i>	0.00-3.99	0.00-4.46
EDE global Twin 2 16-19 years	0.45 (0.70)	0.43 (0.68)
<i>Range</i>	0.00-4.10	0.00-4.71

Note. BMI = body mass index; EDE = eating disorder examination.

Table 2

Cross-twin and cross-wave FIML correlations (95% Confidence Intervals) for twin 1 and twin 2; within-twin correlations are shown in bold

	Twin 1		Twin 2	
	12-15 years	16-19 years	12-15 years	16-19 years
Twin 1				
12-15 years	0.73 (0.62-0.80)	0.37 (0.23-0.49)	0.43 (0.30-0.54)	0.22 (0.06-0.38)
16-19 years	0.73 (0.62-0.80)	0.37 (0.23-0.49)	0.23 (0.06-0.38)	0.36 (0.20-0.49)
Twin 2				
12-15 years	0.20 (0.05-0.35)	0.21 (0.05-0.36)	0.23 (0.06-0.39)	0.35 (0.19-0.50)
16-19 years	0.09 (-0.11-0.28)	0.01 (-0.18-0.20)	0.23 (0.06-0.39)	0.35 (0.19-0.50)

Note. MZ above diagonal and DZ below diagonal

Table 3

Model comparisons of the bivariate Cholesky model of disordered eating between 12-15 years and disordered eating between 16-19 years of age.

Model	-2LL	df	AIC	χ^2 (df) <i>p</i>
ACE	2207.56	1161	-114.44	
AE	2208.40	1164	-119.61	0.84 (3) 0.84
CE	2212.70	1164	-115.31	5.13 (3) 0.16
E	2261.70	1167	-72.35	54.09 (6) <0.001

Note. A = additive genetic influences, C = shared environmental influences, E = non-shared environmental influences; AIC = Akaike's Information Criterion; preferred model (using AIC) in bold; $-2\ln L = 2$ times the log likelihood using ACE Model as the comparison; χ^2 (df) *p* indicates AE and CE have no significant decrement in fit, but ACE model is preferred based on recommendations by Sullivan & Eaves, (2002).

Table 4

Associations (unstandardised estimate and standard error) between disordered eating at Wave 3 and specified risk factors at Wave 1 (W1) and 2 (W2)

Independent variable ^a	EDE global 16-19 years (N=499)				
	Estimate (SE)	95% CI	<i>p</i>	F	ES
W1 peer teasing	-0.02 (0.07)	-0.15-0.11	0.751	0.10	0.03
W2 peer teasing	0.22 (0.06)	0.09-0.34	0.001	12.06	0.32
W1-W2 change in peer teasing	0.18 (0.05)	0.07-0.29	0.001	11.09	0.30
W1 life events	0.004 (0.03)	-0.05-0.06	0.86	0.03	0.02
W2 life events	0.02 (0.02)	-0.01-0.06	0.19	1.71	0.15

Note. ^aall models adjust for EDE global and BMI centile at Wave 1. Effect sizes (ES) were computed using $[2\sqrt{F}]/\sqrt{df(\text{error})}$.