Published in final edited form as:

Behav Genet. 2012 January ; 42(1): 86–95. doi:10.1007/s10519-011-9486-x.

Genetic Variance of Body Mass Index from Childhood to Early Adulthood

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Abstract

Research has been conducted to determine genetic and environmental components of body mass index (BMI). The portion of phenotypic correlation attributed to genetic, and environmental effects, the effects of puberty stage on BMI means and variances, and consistency of parent/twin report remain largely unknown. The current study seeks to address these questions using four waves of data from 1480 twin pairs in the Swedish Twin Registry: Swedish Twin Study of Child and Adolescent Development. Two Cholesky decomposition models were fit (parental and twin report). For wave 2, a univariate model was fit allowing puberty stage moderation. Parent/twin concordance of reported BMI is high. Genetic factors are largely responsible for phenotypic correlation: puberty stage has a significant effect on BMI variance, with higher genetic variance at more advanced puberty stages. Results provide additional information about this phenotype and suggest early adolescent and parental reports for BMI are roughly equivalent.

Keywords

Body mass index; Puberty; Heritability; Twin; Longitudinal; TCHAD

Introduction

Much attention is given to body mass index (BMI) and its relationship to health. BMI and mortality have a J-shaped relationship suggesting those with low and high BMIs are at increased risk for disease and death (Freedman et al. 2006). Over the last decades BMI has been increasing (Centers for Disease Control 2011; Da Veiga et al. 2004). These increases are occurring more rapidly than genes can evolve. This suggests that environmental factors have an important influence on BMI. However, despite the increasing prevalence of obesity, it is well established that there are strong genetic components to BMI (Maes et al. 1997; Silventoinen and Kaprio 2009). Heritability estimates of BMI range between 0.63 and 0.90 (Stunkard et al. 1986; Allison et al. 1996; Maes et al. 1997) with the remainder of the variance being attributable to environmental sources plus measurement error. Studies have begun to identify a relationship between BMI and single nucleotide polymorphisms (SNPs) and copy number variants in a number of genes (Wang et al. 2010; Glessner et al. 2010; Wardle et al. 2008; Scuteri et al. 2007; Saunders et al. 2007; Hunt et al. 2008; Frayling et al. 2007; Dina et al. 2007). These studies lend further credence to the importance genetic factors have on BMI.

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Several twin studies have examined the stability of genetic and environmental influences on BMI developmentally. These include studies on the stability of genetic and environmental influences on BMI from adolescence into adulthood and from early to late adolescence. Haberstick et al. (2010) conducted a longitudinal study examining the genetic and environmental influences on BMI at ages 16 years, 17 years, and 22 years (Haberstick et al. 2010). This study found that the magnitude of the genetic and environmental components of BMI are relatively stable across time, although the genetic cross-time correlation (>0.84) was much higher than the environmental correlation (<0.67) (Haberstick et al. 2010). In a longitudinal study of Finnish twins aged 11–12 years, 14 years, and 17 years, Lajunen and colleagues (2009) found the genetic component was higher in later adolescence than early adolescence for both males and females (Lajunen et al. 2009). There was also a higher correlation between the genetic components over time than the environmental components (Lajunen et al. 2009). Furthermore, results from this study suggest common environmental influences are important in early but not later adolescence.

The molecular genetic literature provides further evidence for stability in genes influencing BMI in childhood and adulthood. For example, SNPs in the *FTO* gene are associated with childhood and adult obesity (Frayling et al. 2007; Dina et al. 2007). Taken together these studies provide some indication that several of the same genetic influences contribute to the variability in BMI across time, but environmental influences change from early adolescent into adulthood. In fact, common environmental influences seem important in early adolescents, but not beyond that developmental stage (Haberstick et al. 2010; Lajunen et al. 2009).

It has been established that BMI is relatively stable over time. For example, approximately 70% of obese adolescents will be obese adults (Parsons et al. 1999). Additional research is needed to help determine if the stability of BMI over time is primarily attributable to genetic or environmental sources. BMI is influenced by biologic factors such as puberty. Puberty is characterized by increased hormone levels and rapid growth and development. At the time of initial data collect, at least a portion of participants in both the Haberstick et al. and Lajunen et al. were likely to have begun puberty, but puberty measures were not included in either of these studies. In another longitudinal study of twins aged 12–16, the authors concluded that twins were likely in the midst of puberty and genetic factors were important in BMI during puberty; however a direct measure of puberty stage was not used (Cornes et al. 2007). Therefore, additional research is needed to fully understand the effect of puberty on BMI.

It is also unknown if parent and twin (child) report of BMI are similar. Elucidating the concordance between these two sources could 1) help determine the accuracy of reports; 2) provide information regarding the possibility of asking children as young as age 13 about their height and weight; 3) provide information regarding similarities and difference from longitudinal models using parent or child reports; and 4) help indicate if reports gathered by parent and child sources could potentially be used in conjunction with one another for samples in which data were collected from parents about a child in childhood and the child about them self in later waves of data collection.

The aim of the current study is to address these unanswered questions in the Swedish Twin Registry: Swedish Twin Study of Child and Adolescent Development (TCHAD).

Methods

TCHAD

The TCHAD sample has previously been described (Lichtenstein et al. 2007). Briefly, TCHAD is an ongoing longitudinal study of 1480 twin pairs born between May 1985 and December 1986. Data were collected when the twins were the following ages: 8–9 years; 13–14 years; 16–17 years; 19–20 years. Both twins' parent and twin reports are contained in these data (Lichtenstein et al. 2007). All data were collected in accord with the Declaration of Helsinki and all research were approved by an institutional review board.

Zygosity Determination

Zygosity determination was based on twin and parental reports of the similarity of the twins' appearances during child and how often the twins were confused for one another as children. To test accuracy of this algorithm; zygosity determination was made using 16 DNA polymorphisms on 106 same-sex twin pairs. The DNA analysis showed the algorithm was 95% accurate (Lichtenstein et al. 2007).

Measures and Current Sample

BMI—BMI was calculated as weight in kg/ height in m². Height and weight were reported by the twins' parent at waves 1–3 and by the twins at waves 2–4.

Puberty—At wave 2, The Pubertal Development Scale (Petersen 1988) was administered to twins' parents and twins. Participants were asked five questions regarding each twin's physical maturation including: if a height growth spurt had occurred; if growth of body hair was present on armpits and genitalia; if skin changes were noticed; if the voice had deepened (males only); if facial hair growth was present (males only); if breast development was present (females only); and if menstruation had begun (females only). An algorithm was created to determine puberty stage. If any question had a missing value, puberty status was coded as missing. Stages were grouped follows: prepubertal; beginning pubertal; mid-pubertal; advanced pubertal; and post pubertal. All five puberty stages were used for all models.

Statistical Methods

Means [standard deviations (sd)] were calculated for BMI at each wave for males and females and are given separately for each reporter. The data were checked for outliers and individuals with a BMI ± 3 standard deviations from the mean were considered to be outliers and removed for the analyses. Pearson's product-moment correlations were performed between parent and twin reported BMI for waves 2 and 3, and Spearman's correlations were performed for parent and twin report of puberty at wave 2. These correlations were performed separately for males and females.

This study was based on the classic twin design and its assumptions. Mainly, monozygotic twins (MZ) twins share all genes, and dizygotic (DZ) twins share on average half their genes. The basic principle behind twin modeling is the similarity of MZ twins to that of DZ twins can be compared. This allows for estimates of the role of genetic and environmental factors. These factors can be divided in additive genetic factors (A), common environmental factors (C), and unique environmental factors (E). Measurement error is included in E (Neale and Maes 1998; Neale and Cardon 1992).

For the current study, two Cholesky decomposition models were fit. Figure 1 depicts the model fit for parental report of twin 1. Here the direct paths represent the within wave estimates and the cross paths represent the inter-wave concordance. The same model was fit

for twin report, however waves 2, 3, and 4 were modeled. Puberty stage was used to regress main effect on the mean BMI at wave 2 in both the parent and twin model. Sex limitation and moderation on the variances was not fit in multivariate models because of known limitations (Neale et al. 2006).

The five-group (MZ males, MZ females, DZ males, DZ females, and opposite-sex twins) model was fit; means and variances were equated across twin order, zygosity group, male opposite-sex twins were equated with male same-sex twins and female opposite-sex twins were equated with female same-sex twin, but based on results from saturated model comparisons twins were not equated across sex. In the first model parental reported BMI at waves 1, 2, and 3 were entered in that order. In the second model, twin reported BMI at waves 2, 3, and 4, were entered in that order. The models allowed for the main effects of puberty stage to be regressed out of the mean for the respective BMI variable for wave 2 only; the parent reported puberty variable was used in the parent model and the twin reported puberty variable in the twin model. Submodels were then fit removing the effects of puberty status on the means in both the parent and twin model. To determine if genetic variance differed significantly among waves, the genetic variance between two waves at a time were constrained to equal one another. If a significantly worse fitting model resulted, the parameters differ significantly.

To further understand the relationship between puberty and BMI, an additional univariate model with puberty moderation on the means and variances was fit for the twin report since we believed these puberty data to be most accurate. This model included an additional parameter to model qualitative sex differences. All statistical analyses were conducted in the R software version 2.12.2 (R Development Core Team 2010); all twin modeling was conducted using OpenMx version 1.06 (Boker et al. 2010).

Results

For males, reported BMIs means and parent twin are contained in Table Ia and correlations are presented in Table Ib. In general, parent-twin correlations in their report of twin BMIs were high. As expected, BMI increases over time as reported by both parents and twins.

At wave 2, the majority of twins had begun puberty, but had not fully matured. As expected, overall females were at later stages of puberty than males. Male pubertal stages are as follows: prepubertal, 228 (parent), 101(twin); beginning pubertal, 345(parent), 443 (twin); mid-pubertal 381 (parent), 468 (twin); advanced pubertal 52 (parent), 46 (twin). There were no post pubertal males by either parent or twin report. The parent-twin correlation for pubertal status was moderate (0.70). Pubertal status reported for females are as follows: beginning pubertal 1 (parent), 5 (twin); mid-pubertal 716 (parent), 736 (twin); advanced pubertal 342 (parent), 356 (twin). There were no females reported as pre-pubertal or post pubertal. Parent twin correlation for female puberty stage was high (0.94).

Model fit statistics are contained in Table II. The full model allowing for the effects of puberty on the BMI means fit significantly better than the more parsimonious model without puberty moderation on BMI means; therefore all reported estimates are from the moderation model. Parameter estimates and 95% confidence intervals from the Cholesky decomposition models are contained in Tables III–VI. Tables III and IV show parent and twin parameter estimates for males. The within wave estimates are contained on the diagonal, the proportion of phenotypic correlation attributed to each parameter are contained on the lower off diagonal, and the genetic and environmental correlations are contained on the upper diagonal.

The heritability estimates increased across waves with the largest increase coming between waves 1 and 2; this resulted from the parent reported model. Table VII indicates genetic variance differed significantly between waves in the parent model, but in the twin model genetic variance was not significantly different across waves. Following childhood, the genetic parameter was responsible for over 60% of the variance in BMI. Looking at both the parent report and twin report models, genetic sources accounted for 67%–83% of the correlation in BMI across waves. The common environment became less important over time and accounted for under 30% of the phenotypic the correlation across time. The unique environmental parameter became more important over time, but did not contribute strongly to the cross-time correlation of BMI.

Parent and twin results for females are contained in Tables V and VI. Similar to males, the heritability estimates increased over time, but the largest increases were seen between waves 2 and 3 in both the parent report and twin report models; Table VII indicates that genetic variance increased significantly across waves in the parent model, and increased between waves 2 and 4 in the twin model, with waves 2 and 3 bordering on differing significantly. In childhood, genetic factors accounted for over 50% of the variance in BMI and in late-adolescents and early adulthood, genetic factors accounted for over 65% of the variance in BMI. Genetic factors were responsible for 51% of the correlation in BMI between wave 1 and wave 2 in the parental report model and 78% of the correlation in BMI between wave 3 and wave 4 in the twin report model. Also similar to males, the common environment became less important over time, however this parameter contributed more strongly to the correlation in BMI across time in females. The unique environmental parameter followed the same pattern in females as males and increased in magnitude over time.

Puberty stage was used to regress out the mean effect of puberty on the BMI means at wave 2 in both the parent and twin models. The parent model yielded a β coefficient of pubertal stage on BMI of 0.17 for males and $-.53$ for females. For the child model, the β coefficient for males was -0.0004 and 0.00003 for females. Removal of puberty moderation for both the parent and twin models resulted in significantly worse fitting models (Table II).

In the univariate analysis, the correlation between the male and female genetic influences on BMI was 0.87 [95% confidence interval (CI): 0.11–1.00]. This suggests that overall at wave 2 males and females share 87% of genes responsible for variance in BMI. Total variance in males was lower with increasing maturity (Figure 2a). In males, the variance in BMI explained by the genetic component ranged from 0.32 (95% CI: 0.10–0.71) in prepuberty to 0.87 (95% CI: 0.73–0.93) in mid-puberty and the variance in BMI explained by the C parameter ranged from 0.63 (95% CI: 0.23–0.86) in prepuberty to 0.03 (95% CI: 0.00–0.17) in mid-puberty. The variance explained by the unique environmental component was relatively stable across puberty stages (Figure 2b). The mean value of BMI was 18.4 kg/m^2 in prepuberty and 19.1 kg/m^2 in mid-puberty. In females, the total variance became greater with increasing maturity (Figure 2c). As shown in figure 2d, the variance in BMI explained by genetic factors ranged from 0.82 (95% CI: 0.59–0.89) in mid-puberty to 0.91 (95% CI: 0.48–0.95) in advanced puberty; the variance explained by C ranged from 0.05 (95% CI: 0.00–0.27) to 0.02 (95% CI: 0.00–0.45) and that of E ranged from 0.14 (95% CI: 0.10–0.18) to 0.07 (95% CI: 0.04–0.12). All five puberty stages for males and females were entered into the this model, but given the small number of males in advanced puberty and the small number of females in beginning puberty we do not report these values.

Discussion

The current study provides additional information about the BMI phenotype. For waves 2 and 3, relatively similar patterns are seen in parental and twin reports in males and females

and correlation between BMI for parent and twin reports were quite high. The 95% CI in the parent and child models overlap and, with the exception of wave 3 parameter estimate for E in males, the point estimates of the parent model is contained in the 95% CI of the child model and the point estimates of the child model is contained in the 95% CI of the parent model. The Results of the current study expand results of previous investigations (Haberstick et al. 2010; Lajunen et al. 2009) by providing additional information regarding the proportion of phenotypic correlation across time which can be attributed to genetic and environmental sources, providing information regarding parent/twin correlation of BMI, and indicate the importance of puberty stage on BMI variance. This information can help further the understanding of developmental factors which influence BMI.

Inter-rater reliability and consistency is often of great concern. In the current study, the correlations between parent and twin report were moderate to high. In addition, the parameter estimates for parent and twin report were comparable as indicated by overlapping confidence intervals. This indicates that during the adolescent years, obtaining BMI information from parent or child is likely to yield similar results and are therefore roughly equivalent. This suggests that BMI collected from either reporter at this age is likely acceptable for use in research studies and is likely subject to the same pitfalls of other self-reported BMI data. Given the similarities between parent and twin report and the similarities in longitudinal analysis of parent and child report, it may be possible to combine these reports into a single model as twins age if additional data are collected. However, inter-rater consistency may not generalize beyond this phenotype. For example, the parent-male twin report of puberty status did not yield nearly as high correlation as any of the parent/twin BMI correlations.

The heritability estimates of the current study are similar to those in previous reports (Stunkard et al. 1986; Allison et al. 1996; Maes et al. 1997). However, the current study indicates that the genetic component in BMI is lower in childhood (prepuberty) and increases in early adolescents after which time it remains relatively stable. The largest change in factors associated with the variance in BMI comes between waves 1 and 2 for males and 2 and 3 for females suggesting both sex differences and the importance of puberty on the heritability of BMI. As expected, common environmental influences which were prominent in childhood approach having no influence on the variance of BMI in early adulthood and the unique environmental influences increase over time. The relative importance of genetic factors increases with age indicated by the relatively high and increasing proportion of BMI correlation attributed to genetic sources across time.

In univariate analyses of twin report at wave 2, we examined the influence of puberty stage on BMI means and variances. As puberty stage becomes more advanced, the genetic component contributes more strongly to the variance in BMI. This illustrates that puberty is plausibly involved in the increasing importance of genetic factors and decreasing importance of common environmental factors involved in BMI variance. In early adolescence, many of the same genes appear to be responsible for BMI variance in males and females. The actual estimation of this parameter indicates that 87% of the genes were the same; this was not significantly different from unity. Therefore, during puberty males and females share the majority of the genetic factors responsible for BMI variance.

Limitations

Results of this study should be evaluated with regard to study limitations. First, we only have parental report at wave 1 and twin report at wave 4. It is unknown how this influences results given some differences exist in estimates from parent and twin report. We are also unable to know which report was more accurate, however the general pattern was similar for

both parent and child report of BMI. Second, we only had puberty status taken at wave 2; therefore we could only evaluate puberty at a single time point. However, it is unlikely that many participants entered puberty prior to wave 1 and most participants had begun puberty by wave 2. In addition, regressing out the mean effects of puberty on BMI resulted in only a slightly better AIC which could be attributable to sample size. However, based on all fit indices, the moderation models are the better fit models. Further, the nature of the relationship between BMI and puberty may complicate puberty moderation of BMI (Eaves and Erkanli 2003). In the multivariate model this would be compounded and other difficulties would arise (e.g., moderation would moderate variances and covariances), therefore moderation was only applied to the univariate model. Third, individuals who are overweight are less likely to report themselves as such (Elgar et al. 2005) and this could influence results. Fourth, although the patterns of parameter estimates were consistent across parent and child report; we are unable to determine if the same influences underlie the two types of reports. Fifth, results may not generalize beyond twins born in Sweden between 1985 and 1986.

Conclusion

High parent-twin correlations in BMI suggest that data collection from either report is likely equally appropriate for use in research studies and likely to give the similar results. Genetic factors are largely responsible for BMI correlation across time and may help to explain the difficulty of altering weight and maintaining weight loss. The importance of puberty stage on factors that effect BMI variance is confirmed by univariate models; steep increases occur in the magnitude of the genetic component of BMI during puberty.

Acknowledgments

We would like to thank Swedish Council for Working Life and Social Research and the Swedish Research Council. Dr. Dellava was funded by NIMH T32MH20030 (PI: Michael C. Neale) and 5R37DA018673 (PI: Michael C. Neale). We would also like to thank all the participants who made this study possible.

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Figure 1.

Multivariate Cholesky for Twin 1 Parent Model

note: A=heritability component; C=common environment component; E=unique environment component; BMI=body mass index

Figure 2a.
Variance components for males by puberty stage at wave 2

Figure 2b.
Parameter estimates for males by puberty stage at wave 2

Figure 2c.
Variance components for females by puberty stage at wave 2

Figure 2d.
Parameter estimates for females by puberty stage at wave 2

Table 1aMean (standard deviation) of Body Mass Index (kg/m²) by Wave and Reporter

	Wave 1	Wave 2	Wave 3	Wave 4
Males Parent Report	16.2 (1.7)	19.0 (2.4)	20.8 (2.6)	n/a
Males Twin Report	n/a	18.9 (2.3)	20.8 (2.6)	22.4 (2.5)
Females Parent Report	16.1 (1.9)	19.0 (2.5)	20.4 (2.5)	n/a
Females Twin Report	n/a	18.9 (2.4)	20.5 (2.6)	21.3 (2.6)

Note: n/a=not available

Table 1b

Twin Correlations (95% Confidence Interval)

	MZM	MZF	DZM	DZF	OS
Parent Report BMI Wave 1	0.83 (0.78–0.87)	0.89 (0.86–0.92)	0.59 (0.46–0.69)	0.64 (0.52–0.73)	0.48 (0.39–0.57)
Parent Report BMI Wave 2	0.87 (0.83–0.90)	0.86 (0.82–0.89)	0.45 (0.30–0.58)	0.59 (0.47–0.69)	0.39 (0.28–0.49)
Twin Report BMI Wave 2	0.85 (0.80–0.89)	0.87 (0.83–0.90)	0.43 (0.26–0.57)	0.44 (0.30–0.57)	0.29 (0.17–0.40)
Parent/Twin BMI Correlation Wave 2	0.95 (0.94–0.96)	0.95 (0.94–0.96)	0.95 (0.94–0.96)	0.92 (0.90–0.94)	0.90 (0.88–0.92)
Parent Report BMI Wave 3	0.81 (0.75–0.86)	0.88 (0.83–0.91)	0.38 (0.20–0.53)	0.42 (0.25–0.56)	0.22 (0.08–0.34)
Twin Report BMI Wave 3	0.70 (0.62–0.77)	0.83 (0.78–0.87)	0.37 (0.22–0.51)	0.43 (0.29–0.55)	0.25 (0.14–0.36)
Parent/Twin BMI Correlation Wave 3	0.84 (0.80–0.87)	0.92 (0.90–0.94)	0.87 (0.83–0.90)	0.90 (0.87–0.92)	0.88 (0.86–0.90)
Twin Report BMI Wave 4	0.73 (0.63–0.81)	0.75 (0.68–0.81)	0.41 (0.18–0.59)	0.36 (0.20–0.52)	0.16 (0.01–0.32)
Parent Puberty Stage Report Wave 2	0.72*	0.95*	0.67*	0.94*	0.86*
Twin Puberty Stage Report Wave 2	0.91*	0.76*	0.51*	0.38*	–0.39*
Parent/Twin Puberty Stage Correlation	0.68*	0.72*	0.30*	0.26*	–0.31*

note: MZM=monozygotic twin male; MZF=monozygotic twin female; DZM=dizygotic twin male; DZF=dizygotic twin female; OS=opposite sex twin; BMI=body mass index; Spearman's correlations do not yield 95% confidence intervals

Table II**Cholesky Decomposition Model Fit Statistics**

	Parameters	-2LL	df	AIC	Δ -2LL	Δ df	p-value
Full Parent Moderation Model	44	16631.99	4361	7909.99	-	-	-
Parent Model no Moderation	42	16655.85	4363	7929.85	23.86	2	0
Full Twin Moderation Model	44	19076.86	4679	9718.86	-	-	-
Full Twin Model no Moderation	42	19084.55	4681	9722.55	7.69	2	0.02

Note: -2ll= -2 log-likelihood; df=degrees of freedom; AIC= Akaike information criteria

Table III
Parental Report Male Parameter Estimate (95% Confidence Interval) for Body Mass Index Across Waves

	Wave 1 a^2	Wave 2 a^2	Wave 3 a^2	Wave 1 c^2	Wave 2 c^2	Wave 3 c^2	Wave 1 e^2	Wave 2 e^2	Wave 3 e^2
Wave 1	0.42 (0.26–0.62)	0.82 (0.61–0.98)	0.74 (0.52–0.91)	0.43 (0.23–0.58)	0.52 (0.19–0.81)	0.73 (0.26–1.00)	0.15 (0.12–0.20)	0.24 (0.06–0.40)	0.14 (–0.07–0.33)
Wave 2	0.67 (0.48–0.87)	0.62 (0.48–0.77)	0.78 (0.68–0.89)	0.28 (0.08–0.46)	0.25 (0.11–0.40)	0.96 (0.56–1.00)	0.05 (0.01–0.10)	0.12 (0.10–0.16)	0.37 (0.17–0.53)
Wave 3	0.68 (0.43–0.91)	0.69 (0.51–0.88)	0.7 (0.54–0.82)	0.29 (0.07–0.51)	0.24 (0.07–0.42)	0.13 (0.02–0.29)	0.04 (–0.02–0.10)	0.07 (0.03–0.12)	0.17 (0.13–0.22)

note: a^2 =heritability component; c^2 =common environment component; e^2 =unique environment component; within wave estimates are on the diagonal and proportion of BMI correlation across time attributed to each parameter are on the lower off diagonal and parameter correlation on the upper off diagonal.

Table IV
Twin Report Male Parameter Estimate (95% Confidence Interval) for Body Mass Index Across Waves

	Wave 2 a^2	Wave 3 a^2	Wave 4 a^2	Wave 2 c^2	Wave 3 c^2	Wave 4 c^2	Wave 2 e^2	Wave 3 e^2	Wave 4 e^2
Wave 2	0.72 (0.55–0.86)	0.94 (0.84–1.00)	0.78 (0.28–0.92)	0.15 (0.02–0.32)	0.79 (–1.00–1.00)	1.00 (–1.00–1.00)	0.13 (0.10–0.17)	0.19 (0.02–0.35)	0.15 (–0.08–0.36)
Wave 3	0.83 (0.63–0.99)	0.64 (0.45–0.77)	0.92 (0.83–1.00)	0.13 (–0.02–0.31)	0.10 (0.00–0.28)	0.75 (–1.00–1.00)	0.05 (0.00–0.09)	0.26 (0.21–0.32)	0.37 (0.23–0.49)
Wave 4	0.81 (0.58–0.98)	0.79 (0.58–0.92)	0.68 (0.47–0.79)	0.16 (0.00–0.37)	0.09 (–0.03–0.29)	0.08 (0.00–0.27)	0.04 (–0.02–0.10)	0.12 (0.07–0.18)	0.24 (0.19–0.32)

note: a^2 =heritability component; c^2 =common environment component; e^2 =unique environment component; within wave estimates are on the diagonal and proportion of BMI correlation across time attributed to each parameter are on the lower off diagonal and parameter correlation on the upper off diagonal

Table V

Parental Report Female Parameter Estimate (95% Confidence Interval) for Body Mass Index Across Waves

	Wave 1 a^2	Wave 2 a^2	Wave 3 a^2	Wave 1 c^2	Wave 2 c^2	Wave 3 c^2	Wave 1 e^2	Wave 2 e^2	Wave 3 e^2
Wave 1	0.56 (0.40–0.71)	0.76 (0.62–0.88)	0.48 (0.30–0.63)	0.33 (0.18–0.49)	0.61 (0.36–0.83)	0.68 (0.23–0.95)	0.11 (0.09–0.14)	0.34 (0.19–0.48)	0.26 (0.06–0.43)
Wave 2	0.51 (0.27–0.74)	0.42 (0.26–0.61)	0.61 (0.23–0.95)	0.41 (0.19–0.64)	0.42 (0.24–0.57)	1.00 (0.82–1.00)	0.08 (0.04–0.13)	0.16 (0.13–0.21)	0.47 (0.32–0.59)
Wave 3	0.63 (0.36–0.87)	0.57 (0.38–0.76)	0.72 (0.57–0.84)	0.31 (0.08–0.57)	0.34 (0.15–0.53)	0.15 (0.04–0.30)	0.06 (0.02–0.12)	0.09 (0.06–0.13)	0.13 (0.10–0.16)

note; a^2 =heritability component; c^2 =common environment component; e^2 =unique environment component; within wave estimates are on the diagonal and proportion of BMI correlation across time attributed to each parameter are on the off diagonal and parameter correlation on the upper off diagonal.

Table VI
Twin Report Female Parameter Estimate (95% Confidence Interval) for Body Mass Index Across Waves

	Wave 2 a^2	Wave 3 a^2	Wave 4 a^2	Wave 2 c^2	Wave 3 c^2	Wave 4 c^2	Wave 2 e^2	Wave 3 e^2	Wave 4 e^2
Wave 2	0.55 (0.37–0.75)	0.86 (0.77–0.95)	0.85 (0.74–0.99)	0.30 (0.10–0.47)	1.00 (0.80–1.00)	1.00 (0.56–1.00)	0.15 (0.12–0.19)	0.44 (0.31–0.56)	0.17 (0.03–0.32)
Wave 3	0.65 (0.45–0.86)	0.68 (0.51–0.81)	0.87 (0.81–0.94)	0.26 (0.06–0.46)	0.14 (0.02–0.31)	1.00 (0.63–1.00)	0.09 (0.06–0.13)	0.18 (0.14–0.22)	0.26 (0.12–0.38)
Wave 4	0.72 (0.50–0.92)	0.78 (0.59–0.92)	0.69 (0.52–0.80)	0.24 (0.05–0.45)	0.16 (0.02–0.34)	0.11 (0.01–0.27)	0.04 (0.00–0.08)	0.06 (0.03–0.10)	0.21 (0.16–0.26)

note: a^2 =heritability component; c^2 =common environment component; e^2 =unique environment component; within wave estimates are on the diagonal and proportion of BMI correlation across time attributed to each parameter are on the lower off diagonal parameter correlation on the upper off diagonal.

Table VII

Comparison of genetic variance across waves

	-2LL	df	AIC	$\Delta -2LL$	Δdf	p-value
Full Parent Model	16631.99	4361	7909.99	n/a	n/a	n/a
Parent Model Genetic Parameter waves 1 and 2 Constrained-Males	16652.71	4362	7928.71	20.71	1	0.00
Parent Model Genetic Parameter waves 1 and 3 Constrained-Males	16664.14	4362	7940.14	32.15	1	0.00
Parent Model Genetic Parameter waves 2 and 3 Constrained-Males	16639.03	4362	7915.03	7.04	1	0.01
Parent Model Genetic Parameter waves 1 and 2 Constrained-Females	16633.33	4362	7909.33	1.34	1	0.25
Parent Model Genetic Parameter waves 1 and 3 Constrained-Females	16660.81	4362	7936.81	28.82	1	0.00
Parent Model Genetic Parameter waves 2 and 3 Constrained-Females	16648.41	4362	7924.41	16.42	1	0.00
Full Twin Model	19706.86	4679	9718.86	n/a	n/a	n/a
Twin Model Genetic Parameter waves 2 and 3 Constrained-Males	19077.18	4680	9717.18	0.33	1	0.57
Twin Model Genetic Parameter waves 2 and 4 Constrained-Males	19078.16	4680	9718.18	1.3	1	0.25
Twin Model Genetic Parameter waves 3 and 4 Constrained-Males	19077.17	4680	9717.17	0.31	1	0.58
Twin Model Genetic Parameter waves 2 and 3 Constrained-Females	19080.34	4680	9720.34	3.49	1	0.06
Twin Model Genetic Parameter waves 2 and 4 Constrained-Females	19083.48	4680	9723.48	6.62	1	0.01
Twin Model Genetic Parameter waves 3 and 4 Constrained-Females	19078.56	4680	9718.56	1.7	1	0.19

Note: -2ll= -2 log-likelihood; df=degrees of freedom; AIC= Akaike information criteria; n/a=not applicable