Body Mass Index and Breast Size in Women: Same or Different Genes?

Tracey D. Wade,[1] Gu Zhu,[2] and Nicholas G. Martin[2]

[1] *School of Psychology, Flinders University, Australia*
[2] *Queensland Institute of Medical Research, University of Queensland, Australia*

The objective of the current study was to investigate the heritability of breast size and the degree to which this heritability is shared with BMI. In a sample of 1010 females twins (mean age 35 years; *SD* = 2.1; range 28–40), self-report data pertaining to bra cup size and body mass index (BMI) was collected in the context of self-report data and an interview relating to disordered eating respectively. In a sample of 348 complete twin pairs who completed data collection (226 MZ pairs and 122 DZ pairs and 360 incomplete pairs (170 MZ and 190 DZ)), we found that the heritability of bra cup size was 56%. Of this genetic variance, one third is in common with genes influencing body mass index, and two thirds (41% of total variance) is unique to breast size, with some directional evidence of non-additive genetic variation. The implications of these findings with respect to previous research linking breast size with reproductive potential are discussed.

Keywords: breast size, body mass index, heritability, bivariate Cholesky

Individual differences in breast size are a conspicuous feature of variation in human females and have been variously associated with breast cancer risk (Thurfjell et al., 1996), where a larger bra cup size is associated with increased risk of cancer (Hsieh & Trichopoulos, 1991), and higher reproductive potential (Møller et al., 1995). While more recent research shows that it is the percentage of total radiologically dense breast tissue area that is associated with a four- to six-fold increase in breast cancer risk rather than breast size (Boyd et al., 1998), the link with reproductive potential continues to be of interest.

There is a developing literature on the relationship between female body weight, breast size and waist-to-hip ratio (WHR) and male ratings of attractiveness. Findings to date vary across studies, with recent work indicating that British Caucasian males prefer a high WHR black figure with small breasts and a high WHR white figure with large breasts (Swami et al., 2009). Ratings of attractiveness of such figures has been significantly and positively associated with ratings of health (Furnham et al., 2006), and women with large breasts have been inferred to have higher fecundity as assessed by measures of daily levels of 17-β-oestradiol (E2) and progesterone (Jasieńka et al., 2004). Thus male preference for larger breast size might be adaptive as it might contribute to higher reproductive success.

While the heritability of WHR has been previously examined (Nelson et al., 1999), where 48% of the variance has been attributed to additive genetic effects, no such examination of breast size has been conducted. Earlier observations have attributed breast size to energy intake early in life (Hsieh & Trichopoulos, 1991; Trichopoulos & Lipman, 1992) but it is likely that genetic factors will have a major influence as it does for WHR. Additionally, if breast size signals fertility, whatever selective forces have been and are still at work on breast morphology, they can only work on genetic variation.

Hence the aim of the current investigation is to examine the heritability of breast size. An obvious covariate of breast size is overall size of the woman; here we index this using body mass index (BMI). In contrast to breast size, the heritability of BMI has been extensively investigated, and twin studies suggest between 50 to 90% of the variance in BMI is accounted for by genetic factors (Maes et al., 1997; Schousboe et al., 2003). We report results of a bivariate genetic analysis of data on bra cup size and BMI in middle aged MZ and DZ twins, collected in the context of a study on eating habits and disorders, in order to investigate the heritability of breast size and the degree to which this heritability is shared with BMI.

Method

Participants

Participating twins were drawn from a cohort of 8536 twins (4268 pairs) born 1964–1971, who were registered as children with the Australian Twin Registry (ATR) during 1980–1982, in response to media and systematic appeals through schools. Female-female twins who had participated in at least one of two waves of data collection (Heath et al., 2001), one during 1989–1992 when the twins were aged 18–25 years, and

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Address for correspondence: Professor Tracey Wade, School of Psychology, Flinders University, PO Box 2100, Adelaide, SA, 5001, Australia. E-mail: tracey.wade@flinders.edu.au

the other during 1996–2000 when the median age of the sample was 30 years, were approached during 2001–2003 to participate in a third wave of data collection; of 2,320, individual twins approached 1,083 (47%) consented to participate (Wade et al., 2006b). 1002 (43%) completed a semi-structured interview over the telephone relating to current and lifetime eating and 1016 (44%) completed a mailed self-report questionnaire assessing various aspects of personality (Wade et al., 2006a), with 962 women completing both (42%). In all, 1056 females (46%) participated in at least one of the data collection components.

The sample included 348 complete twin pairs who completed Wave 3 data collection, comprising 226 MZ pairs and 122 DZ pairs, and 360 incomplete pairs (170 MZ and 190 DZ), where only one twin of a pair participated. Participation at the third wave of data collection has previously been shown not to be predicted by the number of eating problems at Wave 1 nor by any of the 16 individual eating problems making up this total, including ever suffered from or been treated for eating disorder, low body weight, binge eating, obesity, weight loss, anorexia nervosa, bulimia (Wade et al., 2006c). Neither did BMI at Wave 1 predict participation at Wave 3 (OR = 0.99, 95% CI: 0.94–1.03, p = .56). Zygosity was determined on the basis of 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). The Flinders University Clinical Research Ethics Committee approved the study and written informed consent was obtained.

Measures

Self-reported body mass index (BMI) was calculated using weight (kg)/height^2 (metres). Participants were asked what bra cup size they normally wore: A, B, C, D, E, F, G+, where A is the smallest size and G+ the largest. These answers were converted to a scale of 1 to 7.

Statistical Analyses

For the purpose of the following analyses, all data were treated as continuous. Using the statistical package Mx (Neale, 1997), a full information maximum likelihood (FIML) approach was employed with raw data, where complete and incomplete pairs of twins are included in the analyses. FIML can reduce the impact of any respondent bias when the data are missing at random (Little & Rubin, 1987). FIML estimation has been found to be superior to the three ad hoc techniques (listwise deletion, pairwise deletion, and mean imputation) in multiple regression models as FIML parameter estimates had less bias and sampling variability than the other three methods (Enders, 2001). Univariate and bivariate genetic models were fitted using standard methods (Neale & Cardon, 1992).

Results

The mean age of the women at the time of the data collection was 35 years (SD = 2.11; range 28–40). The mean BMI value was 24.09 (SD = 4.91) and ranged from 14.20 to 63.98, where 10 people were classified as morbidly obese with a 40 > BMI < 50, and 2 people (MZ cotwins) had a BMI > 50. The BMI of 63.98 is unlikely to be an error as the identical co-twin, assessed at a different interview and by a different interviewer, had a BMI of 54.56. For genetic analysis four extreme values were reduced to 45.00 (4.5 SD) before log transformation. Bra cup size ranged from 1 (A) to 7 (G+), with a mean of 2.67 (between B and C cup) and a SD of 1.05. The distribution of bra cup size in the total sample is shown in Figure 1.

BMI and bra size were significantly positively skewed (respective z values 20.69 and 4.61). Normality was best achieved by using a natural log (ln) transformation for BMI (Kolmogorov-Smirnov Z = 2.46, p < .001), the quadratic function ($X^{0.25}$) for bra size (Kolmogorov-Smirnov Z = 5.90, p < .001). Logistic regression revealed no significant differences between the monozygotic and dizygotic twins with respect to mean values for BMI (OR=1.01, 95% CI: 0.98-1.03) and bra cup size (OR = 1.03, 95% CI: 0.92-1.17). Neither was cooperation bias detected as there were no significant differences between complete and incomplete pairs for mean values of BMI (OR = 1.00, 95% CI: 0.97-1.03) and bra cup size (OR=1.11, 95% CI: 0.98-1.26).

The number of complete pairs with data for BMI was 204 (MZ) and 109 (DZ), the number of complete pairs with data for bra cup size was 226 (MZ) and 122 (DZ), and the number of pairs with complete data for both variables was 204 (MZ) and 109 (DZ). The FIML correlations for BMI and bra cup size with age as fixed effects are shown in Table 1. Given the narrow age range of this sample, regression on age (and age^2) was negligible but was nevertheless retained for univariate and bivariate model fitting.

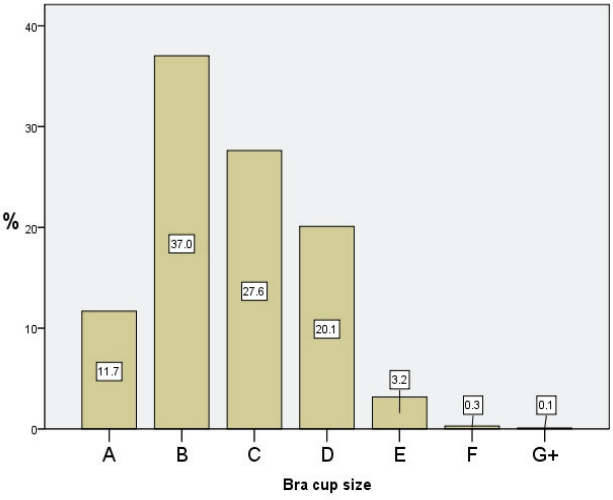

Figure 1
Histogram of Bra cup size for all subjects (N = 1010).

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Table 1

FIML Correlations (and 95% confidence intervals) for Transformed BMI and Bra Cup Size Between Twin 1 (t1) and Twin2 (t2) for Monozygotic (MZ) (upper diagonal) and Dizygotic (DZ) twins (lower diagonal) (corrected for age)

Variables	BMI t1	BRA t1	BMI t2	BRA t2
BMI t1	1	0.54	**0.73**	0.39
		(0.45–0.61)	(0.66–0.77)	(0.23–0.43)
BRA t1	0.43	1	0.34	**0.58**
	(0.31–0.54)		(0.28–0.49)	(0.48–0.65)
BMI t2	**0.43**	0.09	1	0.51
	(0.25–0.56)	(0.09–0.27)		(0.41–0.58)
BRA t2	0.22	**0.16**	0.45	1
	(0.05–0.37)	(0.02–0.32)	(0.32–0.55)	

Note: twin correlations are shown in **bold**; Natural log (ln) transformation for BMI and the quadratic function ($X^{0.25}$) for bra size.

Univariate twin model fitting results are shown in Table 2 and for both variables, the AE submodel was the most parsimonious as judged by the Akaike Information Criterion (AIC).

The results of bivariate model fitting are shown in Table 3. Once again, the AE model gave the best fit and the proportions of variance explained by the latent sources of A and E are shown in Figure 2. It can be seen that only 15% of the variance of bra cup size is due to the additive genetic sources contributing to BMI with a further 41% of genetic variance specific to BRA. The correlation between genetic sources contributing to BMI and bra cup was 0.52 and the correlation for non-shared environmental sources was 0.44.

Discussion

Our main aim was to conduct a bivariate genetic analysis of data on bra cup size and BMI in adult female MZ and DZ twins. Doing so has allowed us to answer two previously unanswered questions: (1) what is the heritability of breast size, and (2) to what degree are the genetic factors contributing to breast size overlapping with BMI. In answer to this first question, we found that the heritability of bra cup size was 56%. Therefore while earlier research has focused on environmental explanations for breast size such as energy intake early in life (Hsieh & Trichopoulos, 1991; Trichopoulos & Lipman, 1992), our results suggest that there is a substantial genetic contribution

Table 2

Results of Univariate Twin Model Fitting to Twin Data for Transformed of Body Mass Index (BMI) and Bra Cup Size (BRA) Corrected for Age

Variables	Model tested	Model #	-2LL	df	AIC	Compare	χ^2	df	Prob.
BMI	ADE	1	2653.3	994	665.3				
	ACE	2	2652.5	994	664.5	2:3	0.8	*1*	3.9E-01
	AE	**3**	**2653.3**	**995**	**663.3**	**3:1**	**0.0**	*1*	**1.0E+00**
	CE	4	2669.5	995	679.5	4:2	17.0	*1*	3.7E-05
BRA	ADE	1	2777.8	1006	765.8				
	ACE	2	2779.9	1006	767.9	2:3	0.0	*1*	1.0E+00
	AE	**3**	**2779.9**	**1007**	**765.9**	**3:1**	**2.2**	*1*	**1.4E-01**
	CE	4	2799.1	1007	785.1	4:2	19.2	*1*	1.2E-05

Note: Best-fitting model is **bolded**.

Table 3

Results of Bivariate Cholesky Decomposition for Transformed Body Mass Index and Bra Cup Size Corrected for Age

Model tested	Model #	-2LL	df	AIC	Compare	χ^2	df	Probability
ADE	1	5178.2	1997	1184.2				
ACE	2	5180.7	1997	1186.7	2:3	1.1	*3*	0.77
AE	**3**	**5181.8**	**2000**	**1181.8**	**3:1**	**3.6**	*3*	**0.30**
CE	4	5217.6	2000	1217.6	4:2	36.9	*3*	< .001
E	5	5434.6	2003	1428.6	5:2	254.0	*6*	< .001

Note: Best-fitting model is **bolded**.

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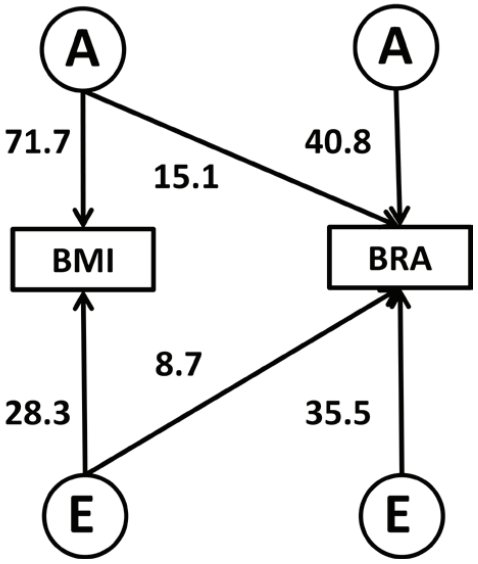

Figure 2

Path diagram showing the percentages of variance explained by the latent additive genetic (A) and unique environmental (E) influences (in circles) on transformed age-corrected residuals of body mass index (BMI) and bra cup size (BRA).

explaining the phenotypic diversity of breast size. In answer to our second question, of the genetic variance contributing to breast size, one third was in common with genes influencing BMI, and two thirds (41% of total variance) was unique to breast size.

Given that larger breast size has been postulated to be associated with greater reproductive success (Cant, 1981; Jasieńka et al., 2004), it is a puzzle that considerable phenotypic, and as shown here, genetic variance remains in the population. It seems more likely that breast size has been subject to stabilizing selection influences, perhaps affected by genetically programmed differences between males in their breast size preference. However, the picture is much more complex than this. Other variables affecting the survival of young offspring must be considered. For example, the ability to breastfeed must play a role, and larger breasts are not necessarily the most functional at this stage, given that babies of large breasted women have some difficulty in latching on to the nipple because they have such a tiny mouth in comparison to the areola. Thus the larger picture that informs what the selective forces are for breast size, and the direction in which they have acted, remains unclear.

To our knowledge, there are few other studies of bra cup size in an anthropometric setting and no others from a quantitative genetic perspective. In a classic study of clothing sizes in Dutch women, Vandenberg (1968) included 'chest girth' among 15 body measurements and found it loaded on the first factor of general size, with little indication of a specific loading. This is not surprising since chest girth confounds both breast size and trunk girth, with variance specific to the breast size being swamped by variance in overall size. While we believe that our focus on bra cup size enables us to ensure that these two factors are not confounded, the imperfect nature of our measured phenotype needs to be recognized, given that a 36DD bra may be the same volume as a 34F or 32G. Further examination of a better measure of the phenotype is required in order to confirm the results of the current research.

There are a number of other limitations of the current research in the context of which the results should be interpreted. First, the data for all variables are based on self-report. This may be particularly less than satisfactory for BMI where has been estimated at the kappa between self-reported and measured BMI is 0.705 for women (Craig & Adams, 2009). Second, we had no information pertaining to child bearing or surgical interventions that may impact on breast size. Third, we have a moderate response rate (47%), commensurate with another large Australian population study where an initial response rate for mid-age women was 54% (Brown et al., 1998). Previously no response bias due to a past history of disordered eating has been detected for this sample (Wade et al., 2006c), or other samples of Australian twins (Wade et al., 1997). We doubt, however, that any of these protective limitations would substantially alter our conclusion that there is a substantial genetic contribution to breast size of which two-thirds of the variance is relatively unique to this phenotype and not shared with BMI.

Disclosure Statement

The authors declared no conflict of interest.

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