Body Size in Five-Year-Old Twins: Heritability and Comparison to Singleton Standards

G. Frederiek Estourgie-van Burk,[1,2] Meike Bartels,[2] Toos C. E. M. van Beijsterveldt,[2] Henriette A. Delemarre-van de Waal,[1] and Dorret I. Boomsma[2]

[1] Department of Pediatric Endocrinology, VU University Medical Center, Amsterdam, the Netherlands
[2] Department of Biological Psychology, Vrije Universiteit, Amsterdam, the Netherlands

The aim of this study is to examine causes of individual differences in height, weight and body mass index (BMI) in 5-year-old children registered with the Netherlands Twin Register. In addition, we examine whether the results of twin studies can be expanded to the singleton population by comparing the data from twins to Dutch reference growth data and by looking at the twins' target height, which was derived from parental height. For 2996 5-year-old twin pairs, information on height and weight and on parental height was available. Univariate and bivariate genetic analyses of height and weight and univariate analyses of BMI were conducted. In order to compare the twins to the singleton population, standard deviation scores (SDS) for height, BMI and target height were calculated based on Dutch reference growth charts for the general population from 1997. Genetic influences were an important source of variation in height, weight and BMI and the main source of covariation between height and weight. Additive genetic factors accounted for 69% and 66% of the individual differences in height in boys and girls, respectively. For weight, heritability estimates were 59% in boys and 78% in girls and for BMI 34% and 74%. The influence of common environment on height was 25% and 27%, on weight 24% and 10% and on BMI 44% and 12% in boys and girls. The bivariate model showed a large overlap between the genes influencing height and weight. Genes explain 78% (in boys) and 76% (in girls) of the covariance between weight and height. At the age of 5 years, female twins were as tall as singleton children, while male twins were shorter than singletons. For both boys and girls, however, mean height SDS was 0.6 standard deviation scores below the mean target height. All twins had lower BMI than singletons. Twins grow fairly well compared to singletons, but they grow below their target height. This may be due to the above average height of twin parents.

Growth during fetal life, childhood and adolescence is influenced by many factors. Size at birth depends, in addition to the length of gestation, on the intrauterine environment and on the small but significant influence of genetic factors (Tanner, 1978). During infancy, gestational age and fetal growth are still important factors of growth, but as their influence decreases, genetic factors become more important (Levine et al., 1987; Van Dommelen et al., 2004a). Genetic effects redirect growth towards the genetic target level in childhood. Height is a highly heritable trait. Van Dommelen et al. (2004a) studied the height and weight process in Dutch twins during the first 2.5 years of life. They found that the variance in length at birth is mostly explained by gestational age and common environmental factors. At birth genetic factors account for only 10% to 15% of the variance in length, increasing to 52 to 58% at the age of 2 years, when the influence of gestational age has almost disappeared. Evidence for the increasing importance of genetic factors also comes from a large longitudinal family study, which showed sibling correlations to increase from 1 (.4) to 4 years of age (.53; Byard et al., 1983a). The Louisville Twin Study showed that at the age of 6 years, genetic factors account for 94% of the variance in height. During puberty intrapair similarity in height decreases, but increases again in adulthood, explaining 67% to 94% of the variation in height (Akerman & Fischbein, 1992; Fischbein, 1977; Phillips & Matheny, Jr., 1990; Schousboe et al., 2004; Silventoinen, Sammalisto, et al., 2003).

Variance in weight at birth is, like birth length, mainly explained by gestational age and common environmental factors (Baker et al., 1992; Hur et al., 2005; Livshits et al., 2000; Van Dommelen et al., 2004a). During infancy genes become increasingly important in determining weight, explaining nearly 60% of the variance at 2 years of age, while the influence of gestational age is reduced to practically none. At the age of 4 to 5 years genetic factors explain 61% to 74% of the variance in weight (corrected for height;

Koeppen-Schomerus et al., 2001; Wilson, 1979). In adolescence and adulthood, genes account for about 64% to 90% of the variation in body mass index (BMI; Akerman & Fischbein, 1992; Pietilainen et al., 2002; Plomin et al., 2001; Stunkard et al., 1986). Most of these estimates come from twin studies. Family and adoption studies also show that weight and BMI are heritable traits (Annest et al., 1983; Biron et al., 1977; Burns et al., 1989; Byard et al., 1983b; Treuth et al., 2001). Estimates from these studies range from 17% to 52%. The lower estimates from family studies may be due to the different ages of family members who are included in a family design.

The correlation of length at birth with adult height is low. During the first years of life, the correlation rises steeply and by age 5 is of the order of .8 (Tanner, 1978). This would imply that 5-year-olds grow according to their genetic target level. Therefore, it is interesting to study whether genetic effects have become more evident at the age of 5 years and whether influences of environment have decreased compared to younger ages.

It may have become apparent that weight can be expressed in several ways: weight corrected for age; weight corrected for height; and BMI. In most studies, particularly in adolescents and adults, BMI is used as it provides more information on body composition than weight. In children aged 2 years and older, BMI has been recommended as a measure for overweight (Cole et al., 2000; Dietz & Robinson, 1998), and BMI reference charts have been published in several countries, among which the Netherlands (Fredriks et al., 2000).

We will study the individual differences in height, weight and BMI around the age of 5 years in a univariate genetic analysis. In addition, weight and height will be studied using bivariate analyses in order to investigate whether there is an overlap in genetic effects between height and weight. We will investigate whether the results from this twin study can be generalized to the general population.

It is well known that birth length and weight of twins is compromised due to a combination of intrauterine growth retardation and shorter gestational age (Alexander et al., 1998; Glinianaia et al., 2000; Kiely, 1990; Min et al., 2000). Several studies have shown that these differences in body size between twins and singletons disappear during childhood, but in a few studies differences remain until in adulthood (Buckler & Green, 2004; Luke et al., 1995; Moilanen & Rantakallio, 1989; Pietilainen et al., 1999l; Wilson, 1979). A previous study in 2-year-old Dutch twins demonstrated that height was below the median of the reference population, while the BMI was nearly at the level of the reference population (Van Dommelen et al., 2004b). We will investigate the persistence of twin-singleton differences through early childhood in relation to the Dutch reference growth charts (Fredriks et al., 2000).

Furthermore, we will study whether 5-year-old twins grow according to their target height, which will be calculated using parental height.

Methods

Procedure and Subjects

All data were obtained from the Netherlands Twin Register (NTR) at the Vrije Universiteit in Amsterdam, the Netherlands (Boomsma et al., 1992). For this study data from twins from the birth cohorts 1986 to 1998 were used. Near the twins' 5th birthday, questionnaires were mailed to the families. Mothers were asked to report up to five different measurements of height and weight of the twins, starting from their 3rd birthday onwards. Information on parental height and weight was collected by questionnaires at the time of registration of the twins. Of the Young NTR twins, 94% are registered by their parents within the first year of life. The initial sample was composed of 7051 twin pairs. For 3565 twin pairs at least one measurement between the age of 4.5 and 5.5 years was reported. Many mothers (n = 3486) reported measurements beyond the age of 4.5 to 5.5 years. If height or weight data deviated 2.5 standard deviations from the mean value corrected for age using Dutch reference growth charts from 1997, data were checked for data-entry errors in the questionnaire (Fredriks et al., 2000). After exclusion owing to extreme values (n = 137 twin pairs), the sample consisted of 3428 twin pairs. Four hundred and twenty-one twin pairs of non-Dutch parents (except if one parent was Dutch and the other West-European) were excluded. This is in line with the criteria used by the Dutch growth study (Fredriks et al., 2000). Zygosity was determined for 955 same-sex twin pairs by DNA typing or blood group polymorphisms and for all other same-sex twins by questionnaire items on similarity. The agreement between zygosity assigned by the replies to the questions and zygosity determined by DNA markers/blood typing is around 93% (Rietveld et al., 2000). After exclusion owing to missing information on zygosity (n = 11 twin pairs), there were 2984 twin pairs for height analysis and 2996 twin pairs for weight analysis. The final sample for height analysis was composed of 471 monozygotic males (MZM), 512 dizygotic males (DZM), 550 monozygotic females (MZF), 480 dizygotic females (DZF), 491 dizygotic opposite-sex male born first (DOSMF) and 480 dizygotic opposite-sex female born first (DOSFM) twin pairs. For weight analysis, the final sample was composed of 478 MZM, 517 DZM, 561 MZF, 478 DZF, 499 DOSMF, and 463 DOSFM twin pairs. One hundred and thirty twin pairs were incomplete for height data and 118 twin pairs for weight data. Information on height was available for 2690 fathers and 2748 mothers, and information on weight for 2687 fathers and 2744 mothers.

Reliability of parent-reported height and weight was examined in a subsample of 94 twins, for which both maternal report as laboratory-measured height and weight were available (van Baal et al., 1996). Maximum time between measured and reported date was 3 months. The correlation between laboratory-

measured and parent-reported data was .96 for height and .92 for weight.

Hereafter, height, weight and BMI between 4.5 and 5.5 years of age will be referred to as just height, weight and BMI.

Genetic Analyses

The descriptive analyses were performed by using SPSS-12 (SPSS Inc). All analyses were conducted using raw data. Effects of birth order, zygosity and sex on means and variances of height, weight and BMI were tested in univariate (saturated) models using the statistical package Mx (Neale, 1999). Age at measurement was included as a covariate. Twin correlations and the 95% confidence intervals were calculated for the six zygosity groups to get a first impression of the genetic and environmental influences on the variance in height, weight and BMI. We compared dizygotic (DZ) same-sex correlations to DZ opposite-sex correlations to explore whether the same genes and environmental factors play a role in males as in females. For height and weight, monozygotic (MZ) and DZ cross-twin cross-trait correlations were calculated using a bivariate script.

Genetic analyses were performed using structural equation modeling (SEM) implemented in the statistical package Mx (Neale, 1999). The total variation in height, weight and BMI was decomposed into sources of additive genetic variance (A), common environmental variance (C) and unique environmental variance (E). A is due to additive genetic effects of different alleles, C is due to common environmental influences shared by members of a twin pair, and E is due to unique environmental influences not shared by members of a twin pair. E also includes measurement error and is therefore always included in the models. A full univariate ACE model was fitted to the height, weight and BMI data. The variance components A, C and E were estimated separately for males and females. We tested whether A, C and E for males and females could be constrained to be equal. Significance of the A and C component was tested by dropping the component from the model. Submodels were compared to the full ACE model using the likelihood ratio test. Genetic and environmental influences on height and weight were estimated using a full bivariate ACE model which decomposes the variance of each measured variable and the covariance between the measured variables into genetic and environmental sources. Significance of the individual path coefficients was tested by constraining paths to zero. Based on the twin correlations the full ACE model was tested against an AE model for height and weight and for males and females separately.

Twin–Singleton Comparison

Data from twins were compared to data from the general population using standard deviation scores (SDS). SDS for height and BMI were calculated with the software package Growth Analyser 3 (2004),

using the Dutch reference growth charts for the general population from 1997 (Fredriks et al., 2000). $SDS = (X–X_1)/Sx$, where X is the twin's measurement, X_1 is the mean value at the child's age in the general population, and Sx is the standard deviation at a given age in the general population. These scores indicate the standard deviation the relevant measurement differs from the mean of the Dutch reference growth charts from 1997. SDSs are used as they are a convenient way of comparing specific groups to the general population. Target height was calculated as the average of father's and mother's height plus 11 cm in boys and minus 2 cm in girls (Fredriks et al., 2000). Target heights were also converted into SDS. In addition, height was corrected for target height (HcTH = height SDS minus target height SDS). We also compared maternal and paternal height to reference standards, as twinning rates increase with increasing maternal height (Basso et al., 2004; Reddy et al., 2005). SDS of paternal and maternal height was calculated using the Dutch reference growth charts for the general population from 1980 (Roede & Van Wieringen, 1985). To assess whether twins differed from singletons in terms of height, target height, HcTH and BMI, mean SDSs were constrained to zero in Mx. A one-sample t test in SPSS was used to test whether paternal and maternal height SDS differed significantly from the mean of the general population, that is, zero.

Results

Genetic Analyses

Table 1 provides means for height, weight and BMI of first-born and second-born twins separately. The mean age of the sample was 5.14 years. First, we tested the effect of age, birth order, zygosity and sex on mean height, weight and BMI in the univariate saturated models. Age (years) affected height (cm) and weight (kg) significantly ($p < .05$; b = 7.91 and b = 2.45 respectively), while no effect of age was found on BMI ($p = .77$). Mean height, weight and BMI were significantly lower in second-born twins compared to first-born twins ($p < .05$). Therefore, means were estimated separately for first-born and second-born twins in the genetic models. Dizygotic same-sex twins (DZSS) and dizygotic opposite-sex (DOS) twins were comparable for mean height ($p = .29$). MZ twins were significantly shorter than DZ twins ($p < .05$). Small and significant, but inconsistent differences were shown for weight and BMI between MZ, DZ and DOS twins ($p < .05$). Boys were significantly taller than girls ($p < .05$). No sex effect was shown on mean weight and BMI ($p = .18$ and $p = .76$ respectively). The variances of height and BMI were not influenced by birth order, zygosity and sex. Regarding weight, we found an inconsistent pattern of effects of birth order, zygosity and sex on the variance, which resulted in a moderate fit of the saturated model compared to the univariate genetic model. The –2 log-likelihood of the

Table 1

Mean Observed Height (cm), Weight (kg) and BMI (kg/m²) of First-Born and Second-Born Twins Respectively

	Males			Females		
	N*	Mean*	SD*	N*	Mean*	SD*
Height†	1433/1440	113.6/113.1	5.4/5.3	1481/1484*	112.9/112.6	5.4 /5.4
Weight†	1456/1436	19.5/19.1	2.7/2.6	1475/1507	19.1/18.9	2.7/2.8
BMI†	1335/1322	15.1/14.9	1.4/1.5	1353/1377	15.0/14.8	1.5/1.6

Note: * first-born/second-born

† mean height, weight and BMI significantly different in first-born and second-born twins ($p < .05$)

N = number of individuals; SD = standard deviation

bivariate saturated model was 55,525.878 with 11,624 degrees of freedom. We constrained the cross-trait correlations to be equal in twin 1 and twin 2 and the cross-twin cross-trait correlations to be equal across twins. This did not worsen the statistical fit ($\Delta\chi^2 = 16.461$, $\Delta df = 12$, $p = .171$).

Table 2 shows the twin correlations for height, weight and BMI. The higher MZ twin correlations versus DZ twin correlations indicate a large influence of genetic factors on all variables. However, the fact that the MZ correlations are less than twice the DZ correlations demonstrates influences of shared environment as well. Similarity in same-sex and opposite-sex twin correlations indicate influences of the same underlying set of genes for boys and girls. The correlations between height and weight within an individual are very similar in all zygosity groups. The cross-twin cross-trait correlations were calculated to explore the genetic and environmental influences on the observed association between height and weight. As can be seen in Table 2 the MZ cross-twin cross-trait correlations are higher than the DZ cross correlations suggesting that the association between height and weight is at least partly due to genetic factors. However, the MZ cross correlations are not twice as high as the DZ cross correlations, which indicates influence of common environment as well.

Table 3 gives the results for the univariate genetic modeling. Univariate full ACE models with sex differences fitted the height, weight and BMI data best. Dropping additive genetic or common environmental factors to zero caused a significant worsening of fit ($p < .01$).

To gain insight into the overlap between height and weight, data were analyzed in a bivariate analysis, for which the full ACE model was used (Table 4). The variance components for males and females could not be constrained to be equal ($p < .01$). Removing the shared environmental factors from the full ACE model significantly worsened the statistical fit for both height and weight in boys and in girls ($p < .01$). Dropping A, C or E on the covariance between height and weight caused a significant loss of fit in both sexes ($p < .01$), which indicates an overlap in genetic and environmental factors for height and weight.

Variance and covariance component estimates of the genetic models are provided in Table 5, based on the bivariate model (estimates from the univariate and bivariate models for height and weight were similar). Genetic factors explained 74% of the variance in BMI females and only 34% of the variance in BMI in males. Common environmental influences were more important in males, explaining 44% of the variance, versus 12% in females. For weight, the estimates were more similar in boys and girls. Additive genetic factors accounted for 59% of the variance in boys and 78% in girls, while 24% of the variance in boys and 10% in girls was explained by the common environment. The estimates for height were nearly the same in boys and girls. Additive genetic effects explained 66% and 69% of the variance in height in boys and girls respectively, while the common environment accounted for

Table 2

Twin Correlations for Height, Weight and BMI, Cross-Trait Correlations (Between Height and Weight Within a Twin) and Cross-Twin Cross-Trait Correlations (Between Height of One Twin and Weight of the Co-Twin) with 95% Confidence Intervals by Zygosity Group (Age Used as a Covariate)

	Height	Weight	BMI	Height–Weight	Cross-twin cross-trait
MZM	.95 (.94–.96)	.83 (.80–.85)	.79 (.76–.82)	.66 (.62–.71)	.61 (.56–.66)
DZM	.57 (.51–.63)	.52 (.45–.58)	.62 (.56–.66)	.69 (.65–.72)	.34 (.27–.40)
MZF	.93 (.92–.94)	.90 (.88–.91)	.86 (.84–.87)	.69 (.65–.73)	.64 (.60–.69)
DZF	.62 (.56–.67)	.51 (.44–.58)	.53 (.47–.59)	.64 (.60–.69)	.36 (.29–.42)
DOSMF	.61 (.55–.67)	.52 (.45–.58)	.47 (.39–.53)	.69 (.64–.72)	.38 (.31–.44)
DOSFM	.57 (.51–.63)	.42 (.35–.50)	.47 (.39–.53)	.67 (.63–.71)	.34 (.27–.40)

Table 3

Univariate Saturated and Genetic Model-Fitting Results for Height, Weight and BMI

	−2LL	df	χ²	Δdf	c.t.m.	p	AIC
Height							
0. saturated	32,724.365	5807					
1. full ACE	32,728.554	5819	4.189	12	0	.980	−19.811
2. no sex differences*	32,740.721	5822	12.167	3	1	.007	6.167
3. AE males	32,791.666	5820	63.113	1	1	.000	61.113
4. AE females	32,784.753	5820	56.199	1	1	.000	54.199
5. CE males	33,193.924	5820	465.371	1	1	.000	465.371
6. CE females	32,738.316	5820	9.763	1	1	.002	7.763
Weight							
0. saturated	25,732.995	5765					
1. full ACE	25,766.001	5777	33.006	12	0	.001	9.006
2. no sex differences	25,783.856	5780	17.855	3	1	.000	11.855
3. AE males	25,771.422	5778	5.421	1	1	.020	3.421
4. AE females	25,770.061	5778	4.060	1	1	.044	2.060
5. CE males	25,891.498	5778	125.497	1	1	.000	123.497
6. CE females	25,996.740	5778	230.739	1	1	.000	228.739
BMI							
0. saturated	17,704.983	5294					
1. full ACE	17,725.814	5306	20.831	12	0	.053	−3.169
2. no sex differences*	17,758.222	5309	32.409	3	1	.000	26.409
3. AE males	17,772.438	5307	46.624	1	1	.000	44.624
4. AE females	17,757.995	5307	32.181	1	1	.000	30.181
5. CE males	17,765.377	5307	39.564	1	1	.000	37.564
6. CE females	17,892.344	5307	166.531	1	1	.000	164.531

Note: −2LL = −2 log-likelihood, df = degrees of freedom, χ² = chi-square statistic, Δdf = difference in degrees of freedom, c.t.m. = compared to model, p = probability value, AIC = Akaike's information criteria, A = additive genetic influences, C = common environmental influences, E = unique environmental influences.

 * no sex differences in variance component estimates (sex differences in means allowed)

25% and 27%. The influence of the unique environment on the variance in height, weight and BMI varied from 5% to 22%. The bivariate model showed that the covariance between height and weight could be mainly explained by additive genetic factors (78% and 76%), while 14% and 16% of the covariance was explained by the common environment. The genetic correlation (r_g) was .84 in males and .70 in females, indicating a large overlap between the two sets of genes. The common environmental correlation (r_c) was .38 and .63 respectively and the unique environmental correlation (r_e) was .58 and .61 respectively.

Twin–Singleton Comparison

Table 6 presents the mean SDS for height, target height, HcTH and BMI. Comparing twins to children from the general population at the age of 5, male twins were significantly shorter ($p < .05$), while female twins were as tall as singletons ($p = .072$). All twins had a significantly lower BMI than singletons at the age of 5 years. Mean SDS for maternal and paternal height were 0.26 and 0.10 respectively, implying that twin parents were significantly taller compared to the general population. This result leads to an above average mean target height SDS for twins with values

of 0.45 SDS for male twins and 0.58 SDS for female twins. No effect of zygosity was shown on mean target height SDS ($p = .86$). Studying height SDS, twin girls were comparable to children from the general population, but when controlling for target height (HcTH) female twins were, like male twins, significantly shorter than singletons ($p < .05$). Mean HcTH SDS did not differ between boys and girls ($p = .071$). No zygosity effect was demonstrated on mean HcTH SDS ($p = .29$).

Discussion

The purpose of this study was to estimate the contribution of genetic and environmental influences to the variation in height, weight and BMI at age 5 years and to compare body size of twins to that of singletons. The genetic analyses showed that height, weight and BMI are highly genetic traits. When we compare the results at age 5 years to the results at age 2 years of a previous study in Dutch twins (Van Dommelen et al., 2004a), genetic influences have become more evident. This is in line with our expectation that genetic effects become more important as children grow older. For height, heritability increased from 58% to 69% in

Table 4

Bivariate Saturated and Genetic Model-Fitting Results for Height and Weight

	−2LL	df	χ²	Δdf	c.t.m.	p	AIC
Height–Weight							
0. saturated	55,542.339	11,636					
1. full ACE	55,590.747	11,666	48.408	30	0	.018	−11.592
2. Drop A on covariance males	55,847.200	11,667	256.452	1	1	.000	254.452
3. Drop A on covariance females	55,771.887	11,667	181.139	1	1	.000	179.139
4. Drop C on covariance males	55,599.455	11,667	8.707	1	1	.000	6.707
5. Drop C on covariance females	55,599.007	11,667	8.260	1	1	.000	6.260
6. Drop E on covariance males	55,760.317	11,667	169.570	1	1	.000	169.570
7. Drop E on covariance females	55,822.996	11,667	232.248	1	1	.000	230.248

Note: −2LL = −2 log-likelihood, df = degrees of freedom, χ² = chi-square statistic, Δdf = difference in degrees of freedom, c.t.m. = compared to model, p = probability value, AIC = Akaike's information criteria, A = additive genetic influences, C = common environmental influences, E = unique environmental influences.

males and from 52% to 66% in females, from age 2 to 5. The heritability estimates for weight showed an increase from 58% to 78% in females, while the heritability in males remained the same (59%). Shared environmental influences on the individual differences in weight are larger in males than females, which is even more evident in the BMI analysis. The 95% confidence intervals for the weight estimates of boys and girls overlap, while there is no overlap between the intervals of BMI. We did not expect to find these sex differences, based on the hypothesis that infancy and

childhood growth is similar in boys and girls (Karlberg, 1989). A large twin study in 4-year-olds found comparable heritability estimates (60%) for weight corrected for height in boys and girls (Koeppen-Schomerus et al., 2001).

The covariance between height and weight was mostly explained by genetic factors. The high genetic correlation between height and weight demonstrates that these two traits are mainly under the control of the same additive genetic factors at the age of 5. Focusing on the nature of common environmental

Table 5

Standardized and Unstandardized Estimates of Variance and Covariance Components for Height and Weight (Bivariate Model) and of Variance Components for BMI

	Standardized			Unstandardized		
	V_A	V_C	V_E	V_A	V_C	V_E
Height						
M	.69 (.62–.77)	.25 (.18–.33)	.05 (.05–.06)	18.34	6.53	1.40
F	.66 (.56–.74)	.27 (.19–.36)	.07 (.06–.08)	17.35	7.24	1.88
Weight						
M	.59 (.49–.69)	.24 (.15–.33)	.17 (.15–.20)	3.94	1.63	1.16
F	.78 (.67–.85)	.10 (.04–.22)	.12 (.10–.13)	5.63	0.76	0.84
BMI						
M	.34 (.23–.46)	.44 (.33–.54)	.22 (.19–.25)	0.74	0.96	0.47
F	.74 (.67–.80)	.12 (.06–.19)	.14 (.12–.16)	1.68	0.28	0.32
Height–Weight	Cov_A	Cov_C	Cov_E	Cov_A	Cov_C	Cov_E
M	.78 (.69–.87)	.14 (.05–.23)	.08 (.07–.10)	7.14	1.25	0.73
F	.76 (.61–.87)	.16 (.05–.30)	.08 (.07–.10)	6.92	1.47	0.77
Height–Weight	r_g	r_c	r_e			
M	.84	.38	.58			
F	.70	.63	.61			

Note: V_A = % of variance explained by additive genetic factors (heritability), V_C = % of variance explained by common environment, V_E = % of variance explained by unique environment, Cov_A = % of covariance between height and weight explained by additive genetic factors (heritability), Cov_C = % of covariance explained by common environment, Cov_E = % of covariance explained by unique environment, M = males, F = females, r_g = genetic correlation, r_c = common environmental correlation, r_e = unique environmental correlation.

95% confidence intervals in brackets.

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Table 6

Mean Standard Deviation Scores (SDS) for Height, Height Corrected for Target Height (HcTH) and BMI of First-Born and Second-Born Twins Respectively; Mean SDS for Target Height

	Males		Females	
	Mean*	SD*	Mean*	SD*
SDS height†	−0.12/−0.21‡	1.14 / 1.13	0.03/−0.03	1.13/1.12
SDS HcTH†	−0.59/−0.69‡	1.03/1.04	−0.57/−0.62‡	1.05/1.04
SDS BMI†	−0.41/−0.53‡	1.03/1.06	−0.44/−0.56‡	1.11 / 1.22
SDS target height	0.45‡	0.73	0.58‡	0.79

Note: * First-born/second-born

   † mean SDS significantly different in first-born and second-born twins ($p < .05$)

   ‡ mean SDS significantly different from 0 ($p < .05$)

   SD = standard deviation.

influences as another overlapping factor for the association between height and weight, nutrition, family environment (e.g., socioeconomic status) and assortative mating are known to influence variance in height and weight (Silventoinen, 2003).

In this study a birth-order effect was found on mean height, weight and BMI. First-born twins are slightly taller and heavier than second-born twins at the age of 5 years. Some studies reported a similar result, first-born twins being heavier at birth and at the age of 16 years, but in all these studies no explanations are given (Buckler & Green, 1994; Glinianaia et al., 2000; Pietilainen et al., 2002). It might be that the first-born twin is heavier and taller at birth due to better maternal-fetal nutrition, as its position is lower in the uterus. The differences in size at birth could be an explanation for the differences we noted at the age of 5 years.

Another goal of this study was to compare the growth of twins with the growth of children from the general population. Compared to singletons, twins are born substantially smaller, with a mean weight deficit of 30% and height deficit of 17% (Wilson, 1979). During infancy differences in body size between twins and infants from the general population decrease, but do not completely disappear, despite correcting for gestational age. At the age of 2 years, twin height is 0.3 SD below the reference population, while the BMI is nearly at the level of the reference population (Van Dommelen et al., 2004b). Some studies have shown that differences in body size between twins and singletons disappear at different ages in childhood, but in other studies differences remain until in adulthood (Andrew et al., 2001; Ljung et al., 1977; Moilanen & Rantakallio, 1989; Pietilainen et al., 1999; Wilson, 1979). Wilson (1979) concluded that the prenatal growth suppression on weight and height had fully disappeared by the age of 8 years compared to singleton standards. Two other studies showed that adolescent and adult twins are leaner than singletons, while height seemed to be comparable (Pietilainen et al., 1999). Unfortunately, most of these twin studies have not included siblings

or midparental or target height. One study included siblings and found that twins born appropriate for gestational age (birthweight greater than 10th percentile for singletons) are as tall as their siblings but lighter in childhood, while twins born small for gestational age (birthweight < 10th percentile for singletons) grow below their target height and singleton standards (Buckler & Buckler, 1987). They also showed midparental height to be 0.3 SD above average, but the study consisted of small numbers and compared children of different ages. This study, comparing twins to singletons, showed that male twins are significantly shorter, while female twins caught up in height (Table 6). We find more marked male twin–singleton differences and also greater shared environmental effects for boys than girls which may suggest perhaps lower generalizability of the findings to the general population for boys than for girls. When looking at height corrected for target height (HcTH), sex differences disappeared and both boys and girls grew below their target height. The target height of twins is above average, which can be partially explained by the fact that twin mothers are taller than women from the general population. This is in accordance with previous literature describing that twinning rates increase with increasing maternal height (Basso et al., 2004; Blickstein & Keith, 2005; Reddy et al., 2005). We also found paternal height to be above average, though to a lesser extent. One explanation for this finding may be assortative mating (Silventoinen, Kaprio, et al., 2003). The above average target height of twins implies that, although twin growth may be considered (nearly) normal compared to singleton standards, twin growth is restricted in respect to their target height. The growth restriction is unlikely to be of clinical importance, but it is an interesting finding, which needs more study in the future. To explore whether the differences in BMI and HcTH between twins and singletons are of genetic or environmental origin, a longitudinal design including siblings is needed.

Consistent with other studies, twins were significantly lighter than singletons (Buckler & Buckler,

1987; Ljung et al., 1977; Moilanen & Rantakallio, 1989; Pietilainen et al., 1999; Wilson, 1979). Some studies reported these differences to disappear in childhood, while others showed these differences to remain in adulthood. Our results showed a decrease in BMI compared to the age of 2 (Van Dommelen et al., 2004b). In the light of the increase in BMI and obesity in young children (Hirasing et al., 2001), this is an interesting finding. It may be that the finding is specific to twins, who grow up under environmental conditions in which they always have someone to play with and thus may show increased activity levels, as compared to other children.

Acknowledgments

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