Eur J Epidemiol (2012) 27:247–253
DOI 10.1007/s10654-012-9678-6

REVIEW

Heritability of body mass index in pre-adolescence, young adulthood and late adulthood

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Received: 17 December 2010 / Accepted: 7 March 2012 / Published online: 18 March 2012

Abstract Increased body mass index (BMI) is a worldwide health issue. Individual differences in the susceptibility to increased BMI could be related to genes or environment. We performed a systematic review of genetic studies on BMI in pre-adolescence, young adulthood and late adulthood. We searched PubMed and EMBASE with *heritability, body mass index, BMI, weight, height, anthropometry* and *twins* as search terms. Studies reporting intra-pair correlations of healthy twin pairs that were raised together were included. This resulted in the inclusion of 8,179 monozygotic (MZ) and 9,977 dizygotic (DZ) twin pairs from twelve published studies in addition to individual participant data for 629 MZ and 594 DZ pairs from four twin registries. Structural equation modelling with intra-pair twin correlations showed that the heritability of BMI remained high over all age categories ranging from 61 % (95 % CI 54–64 %) to 80 % (95 % CI 76–81 %) for male and female subjects combined, while unique

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environmental influences increased from 14 % (95 % CI 13–15 %) to 40 % (95 % CI 37–43 %) with increasing age. Heritability of BMI remains consistently high over different age categories. Environmental changes over time do not seem to have as big a relative impact on an individual's weight as previously reported, suggesting a mainly genetic influence on variation in BMI over the years.

Keywords Twins · Heritability · BMI · Body mass index · Anthropometry · Life span

Abbreviations

A	Additive genetic influences
BMI	Body mass index
C	Common environmental influences
CAATSA	Carolina African American Twin Study of Aging
DZ	Dizygotic
E	Unique environmental influences
EFPTS	East Flanders Prospective Twin Study
IPD	Individual participant data
LLTS	Leuven Longitudinal Twin Study
MTR	Murcia Twin Register
MZ	Monozygotic
NTR	Netherlands Twin Registry
TEDS	Twins Early Development Study
WHO	World Health Organisation

Introduction

Between 35 and 65 % of the population in Europe and nearly 80 % of the United States is overweight (body mass index; BMI $\geq$ 25 kg/m^2) or obese (BMI $\geq$ 30 kg/m^2) according to the latest estimates by the World Health Organisation (2010). Furthermore, nearly 43 million children under five were overweight in 2010 [1]. Increased body mass is related to many health problems, including cardiovascular disease and type 2 diabetes [2]. Causes of obesity and overweight have been linked to the imbalance between calorie intake and physical activity [3]. Over the past few decades, food has become more available, and cheaper. This, and the changes in marketing of food, has led to people feeling more inclined to go out for meals and buy more highly processed foods [4]. Jobs and modes of transport allow us to be sedentary for most of the day, causing more energy intake than expenditure [5].

In addition to these environmental explanations for excessive weight gain, there is evidence that BMI also has a strong genetic component. Results on the difference in the genetic influence at different developmental stages are inconclusive. It has been reported that the heritability may increase from childhood into adolescence [6, 7] and decrease [8] after that. This means that the relative importance of genetic and environmental effects on the variance of BMI change over time. Common environmental influences within a twin pair seem to have a larger influence in early life [9], than in adulthood where common environmental effects are negligible [7, 8]. Only a few studies have looked into gender differences in the heritability of BMI. There is room for debate whether the heritability of BMI in males and females is influenced by the same [10] or different [11] genes. Wisniewski et al. [12] concluded in their review that gender differences in relation to childhood obesity in particular have been understudied. They argue that body composition and hormonal influences are different for males and females, and thus gender differences should be investigated when looking at risk factors for obesity.

In this systematic review we studied the heritability of BMI, using previously reported intra-pair twin correlations from published studies and unpublished individual participant dataset (IPDs) from four additional twin registries. We used a cross-sectional method to investigate how the heritability of BMI changes in pre-adolescence, young adulthood and late adulthood, and as a function of sex.

Methods

Search methods

We conducted a structured literature search of MEDLINE and EMBASE databases at regular intervals until January 2012. Search terms that were used as free text and MeSH terms were *heritability, body mass index, BMI, weight, height, anthropometry* and *twins*.

In addition to published articles, we obtained IPDs from the Netherlands Twin Registry (NTR) [13], Carolina African American Twin Study of Aging (CAATSA) [14], Murcia Twin Register (MTR) [15] and the Leuven Longitudinal Twin Study (LLTS) [16], which used subjects from the East Flanders Prospective Twin Study (EFPTS) [17], comprising a total of 1,223 twin pairs. The height and weight data in the IPDs were used to calculate BMI and intra-twin pair correlations in order for it to be in a comparable format to the published data.

Inclusion/exclusion criteria

Articles were included if the classic twin method was used on population-based samples, where monozygotic (MZ) and dizygotic (DZ) intra-twin pair correlations on a trait are compared. Finally, articles were selected if they reported intra-pair twin correlations on BMI of healthy MZ and DZ

twins that were raised together or published sufficient information to calculate these correlations.

Articles were excluded from the review if birth weight of the subjects was less than 500 g or gestational age at birth was less than 22 weeks, based on the minimum criteria for a viable live birth as described in the International Classification of Disease (10th ed.) [18]. If several articles were published on data from the same cohort, and it was unclear whether these were the same subjects or not, the largest study was selected for inclusion. IPD entries with missing zygosity, gender, age or BMI were excluded.

Statistical analyses

We grouped the studies into three age categories: pre-adolescence (9–11 years), young adulthood (18–22 years) and late adulthood (49–65 years). There were two longitudinal registries. The first one was the LLTS [16], for which we had IPD data. Average BMI was calculated for those subjects in the LLTS who had several measurements within one age category. Average BMI was log transformed to reduce skewness of the data, before intra-twin pair correlations were calculated.

The second longitudinal study was the Twins Early Development Study (TEDS). Data from this registry were used in three papers [19–21] within the 9–11 years category. Average correlations were calculated by applying a Fisher's z transformation on the extracted intra-twin pair correlations.

Structural equation modelling, using correlations derived from published articles and the IPDs, was done in Mx [22, 23] to estimate genetic (A) and common (C) and unique (E) environmental influences on the variance of BMI in each age category. Two models were fitted sequentially, as proposed by Sullivan et al. [24]. The first model allowed for the factor loadings on A, C and E to be freely estimated for each primary study. These factor loadings were set to be equal across all studies in the second model. Heterogeneity was determined by calculating Chi-square differences between both models, and studies were considered heterogeneous if the Chi-square difference was statistically significant at $p < 0.05$ [25]. Estimates from the second model are reported as pooled variance components [24]. We performed further sex-specific analysis, which only included articles that reported intra-twin pair correlations separately for men and women.

Results

Sixteen studies were included in the final analysis, resulting in a total of 1,912 pre-adolescent MZ and 2,499 DZ twin pairs, 5,367 MZ and 4,444 DZ young adult twin pairs, and 1,248 MZ and 1,375 DZ twin pairs in late adulthood from population based cohorts. Intra-pair correlations were obtained from twelve studies, of which seven were carried out in Europe [19–21, 26–29] and five in the United States [8, 30–33]. We acquired IPDs for the remaining studies from three European twin cohorts [13, 15–17] and an American twin registry [14].

General study characteristics and intra-pair correlations for MZ and DZ twins of all included studies can be found in Tables 1 and 2 respectively. The sex-specific analyses of the IPDs suggested no significant difference in variance components for male and female subjects. Therefore, we reported results on the combined male–female meta-analyses only. Forest plots of heritability estimates from each study are provided in Fig. 1.

Figure 2 shows the variance components estimates for all age categories. Heritability of BMI remains high over the age categories, with estimates of 0.75 (95 % CI 0.70–0.80) in pre-adolescence, 0.80 (95 % CI 0.76–0.81) in young adulthood, and somewhat lower in late adulthood at 0.61 (95 % CI 0.54–0.64).

Shared and unique environmental factors seem to follow opposite patterns from each other. As age increases, the proportion of shared environmental influences decreased from small to negligible. Overall, the proportion of variance explained by unique environmental factors seemed to generally increase from 0.14 (95 % CI 0.13–0.15) in pre-adolescence to 0.39 (95 % CI 0.36–0.43) in late adulthood.

A model in which the proportion of variance explained by A, C and E were equated over all studies for pre-adolescence and late adulthood fitted as well as the model with free estimates for each individual study, suggesting there was no significant heterogeneity between the studies. However, this was not the case in the young adulthood category, where significant heterogeneity between the studies was present ($\chi^2 = 21.98$, df $= 9$, $p = 0.01$).

Discussion

Our findings show that heritability of BMI remains high over several age categories. Unique environmental influences on BMI increase with increasing age group, while common environmental factors decrease to being negligible at an older age.

Many studies, including ours, have shown that BMI is highly heritable [10, 21, 27]. So far, about 20 susceptibility genes have been associated with obesity [34, 35]. However, these identified candidate genes only account for about 1 % of the total genetic variation of obesity and leave much to be discovered [36]. More specifically, matters are complicated by the fact that different obesity related genes come into play at different stages in life; some play a role in

Table 1 Characteristics of studies included in systematic review

References	Country	Method BMI measurement	Total MZ pairs	Total DZ pairs
Wardle et al.[21]	United Kingdom	Self measurement and clinical examination	1,813	3,279
Haworth et al. [19]	United Kingdom	Self report	2,209	2,536
Haworth et al. [20]	United Kingdom	Self report	1,857	1,669
Faith et al. [31]	United States	Clinical examination	41	25
Silventoinen et al. [28]	Sweden	Military database	1,582	1,864
Harris et al. [26]	Norway	Self report	866	751
Korkeila et al. [27]	Finland	Self report	1,173	2,340
Carmichael and McGue [8]	United States	Self report	137	136
Cardon et al. [30]	United States	Clinical examination	134	134
Wade et al. [33]	United States	Clinical examination	527	248
Nelson et al. [29]	Sweden	Clinical examination	27	36
Stunkard et al. [32]	United States	Clinical examination	1,974	2,097
LLTS [16]	Belgium	Clinical examination	158	146
NTR [13]	Netherlands	Self report	341	277
CAATSA [14]	United States	Clinical examination	66	86
MTR [15]	Spain	Self report	64	85

MZ monozygotic, *DZ* dizygotic, *LLTS* Leuven Longitudinal Twin Study, *NTR* Netherlands Twin Registry, *CAATSA* Carolina African American Twin Study of Aging, *MTR* Murcia Twin Register

either childhood or adult obesity, whereas others are associated with both [34, 37], emphasizing the importance of studying heritability of BMI at difference ages. No studies, to our knowledge, have reported on the change in heritability over a lifespan. We investigated the heritability of BMI in several age categories in an attempt to study this change.

We found that the heritability of BMI remains high throughout the three age categories and generally increases from pre-adolescence into young adulthood, in accordance with previous studies [19, 38], while it decreases after young adulthood. However, a recent study by Ortega-Alonso et al. [39] found that the heritability of BMI does continue to increase up to old age. Ortega-Alonso et al. [39] suggest that narrower age ranges may be able to show this increase later in life, as the rate of change seems to slow down with age. Contrary to this study, which only included women in young to late adulthood, our analyses included both male and female subjects from pre-adolescence to late adulthood. The inclusion of male subjects in our analyses could be an alternative explanation for why we have found a decrease of heritability in late adulthood, where Ortega-Alonso et al. [39] did not. More data on BMI of older twins would be needed to verify or refute this.

We have shown that common environmental factors have less influence on individual difference in BMI in late adulthood, whereas unique environmental influences increase steadily from pre-adolescence to late adulthood. This pattern is not surprising as we expect their interests and lifestyles to diverge as twins separate from their shared household [40]. Our findings of high heritability, while common environmental factors become negligent after young adulthood add confidence to the growing evidence within the field [41].

To our knowledge, ours is the largest meta-analysis on the heritability of BMI at several ages that combines published correlations and raw data in the same analysis. A large review of published estimates by Maes et al. [42] showed similar results. Additionally, Maes et al. [42] performed a more comprehensive raw data analysis which included data on relative body weight of parents, siblings, spouses and offspring of twins. This method allows for the investigation of any kind of special twin environment that could influence heritability estimations, which is not possible with the classical twin design that we have used in the current meta-analysis. Not only do familiarity, genes and environment separately play a role in determining BMI, interactions between food intake, physical activity and genetic predispositions to excessive weight gain are also possible explanations for obesity [43, 44]. Our data did not allow us to investigate possible gene-environment interactions. Another limitation to our study is our choice of three age categories. The timing of pubertal growth spurts depend on genetics [45, 46], diet and physical activity [47]. Our conclusion might have been different if we had been able to include more categories in our analyses. However, due to lack of raw data for subjects in puberty, we decided to not include this in our analyses. More data would be needed to adequately investigate this. Due to the nature of

Table 2 Sample sizes and intra-pair correlations for monozygotic and dizygotic twins in each age category and gender

	Male				Female				Combined			
	Monozygotic		Dizygotic		Monozygotic		Dizygotic		Monozygotic		Dizygotic	
Article	N	r	N	r	N	r	N	r	N	r	N	r
Pre-adolescence												
Wardle et al. [21]*	845	0.84	818	0.45	968	0.87	840	0.55	1,813	0.86	3,279	0.49
Haworth et al. [19]*	679	0.38	547	0.44	804	0.88	593	0.52	1,483	0.86	2,090	0.47
Haworth et al. [20]*	–	–	–	–	–	–	–	–	1,857	0.86	1,669	0.50
Faith et al. [31]	–	–	–	–	–	–	–	–	41	0.85	25	0.24
LLTS [16]	24	0.88	16	0.36	18	0.95	21	0.57	42	0.93	37	0.55
NTR [13]	49	0.87	49	0.61	59	0.88	39	0.72	111	0.88	91	0.65
Young adulthood												
Silventoinen et al. [28]	1,582	0.84	1,864	0.42	–	–	–	–	–	–	–	–
Harris et al. [26]	380	0.70	342	0.36	486	0.79	409	0.36	–	–	–	–
Korkeila et al. [27]	379	0.77	817	0.41	468	0.72	830	0.33	–	–	–	–
Stunkard et al. [32]	1,974	0.81	2,097	0.42	–	–	–	–	–	–	–	–
LLTS [16]	18	0.86	19	0.28	19	0.90	17	0.39	37	0.88	36	0.36
NTR [13]	28	0.81	19	0.61	33	0.84	30	0.31	61	0.82	49	0.47
Late adulthood												
Carmichael and McGue [8]	–	–	–	–	–	–	–	–	137	0.69	136	0.33
Cardon et al. [30]	134	0.67	134	0.24	–	–	–	–	–	–	–	–
Wade et al. [33]	131	0.53	68	0.35	396	0.59	180	0.37	–	–	–	–
Nelson et al. [29]	27	0.67	36	0.33	–	–	–	–	–	–	–	–
CAATSA [14]	11	0.89	19	0.38	22	0.67	24	0.38	33	0.75	43	0.37
MTR [15]	–	–	–	–	64	0.73	85	0.31	–	–	–	–

* Average sample size and intra-pair correlations of these studies used in systematic review [$r_{monozygotic} = 0.86$ (N = 1,718), $r_{dizygotic} = 0.48$ (N = 2,346)]

LLTS Leuven Longitudinal Twin Study, *NTR* Netherlands Twin Registry, *CAATSA* Carolina African American Twin Study of Aging, *MTR* Murcia Twin Register

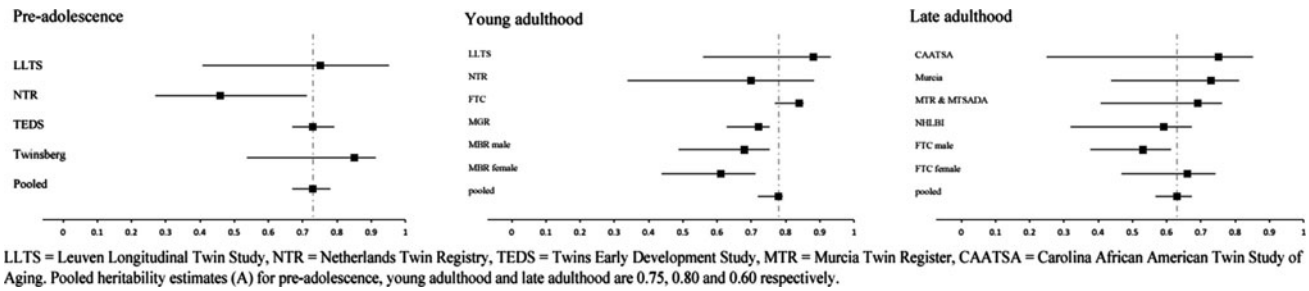

Fig. 1 Forest plots of individual and pooled heritability estimates for male/female subjects combined in pre-adolescence, young adulthood and late adulthood

our data we were also not able to adequately investigate possible sex differences in heritability of BMI. Some studies have shown that there is indeed a sex difference, such that heritability estimates for women are generally higher than for men [10, 48]. Information on dizygotic opposite sex twin pairs would be needed in order to perform these analyses.

Many studies are investigating possible causes and remedies for obesity and its health consequences, with physical activity and inactivity [49–51] amongst the most

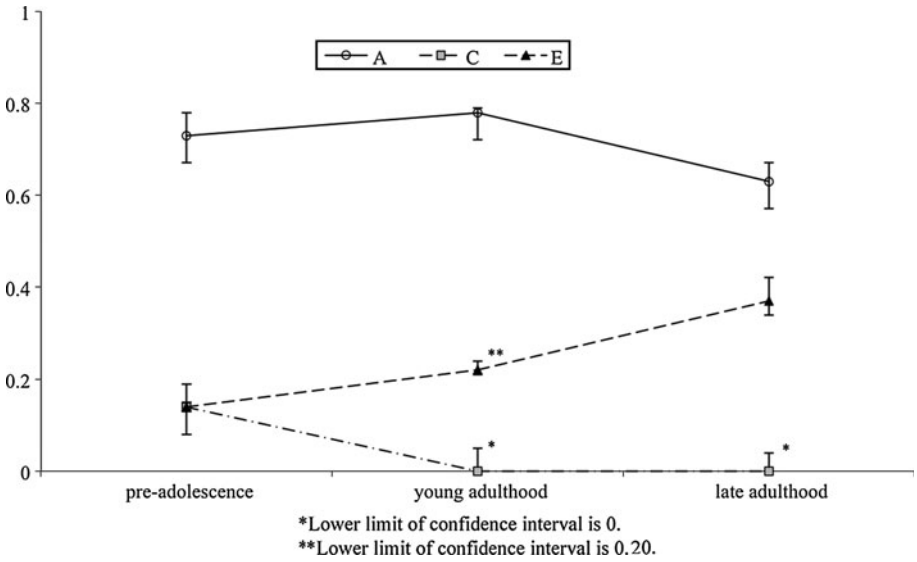

Fig. 2 Variance components estimates for male/female combined in the systematic review including 95 % CI, where *A* genetic influence, *C* common environmental influence and *E* unique environmental influence

common environmental factors that are studied. However, only few studies have looked into the genetic influences on obesity and how these changes over a lifetime [52]. Our findings suggest that there are differences related to either gene expression or gene-environment interaction at different ages across the age categories. The lack of significant heterogeneity in our pre-adolescent and late adulthood data from several countries and over a number of years suggests that genetic influences are still strong, despite the increased relative environmental influence on individual differences in BMI. This in turn suggests that the effectiveness of any environmental intervention would be largely dependent on an individual's genetic predisposition to weight gain and weight loss. Therefore, interventions would probably be most effective if aimed at certain risk groups as opposed to the entire population. This would, however, not be the most efficient method of treating and preventing obesity. Our findings also suggest that findings from longitudinal studies would be plausible, as the relative influences of the variance components seem to stay stable over time. In any of these instances, variance components estimates in young adulthood should be verified first. As longitudinal studies over an entire lifespan are not the most time-efficient when looking at changes in heritability of BMI, a cross-sectional study would be the best approximation. A cross-sectional or a combination of cross-sectional and longitudinal studies with raw data could shed more light on the relationship between genes, environment, age and BMI.

Acknowledgments The authors would like to thank Raymond Timmins of the Department of Public Health at the University of Birmingham (UK) for gathering a large part of the papers. This research was funded by the Birmingham Children's Hospital Research Foundation (BCHRF102f). The MTR is supported by the Seneca Foundation (08633/PHCS/08) and MICINN (PSI11560-2009). LLTS is supported by the Research Fund Katholieke Universiteit Leuven (OT/86/80), National Bank of Belgium, Fund for Medical Research Belgium (3.0038.82, 3.0008.90, 3.0098.91), and North Atlantic Treaty Organisation (860823). This study was previously published in abstract form in Twin Research and Human Genetics (2010): **13**(3), p279.

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