The Intergenerational Transmission of Thinness

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Objectives: To examine intergenerational associations for thinness and to compare maternal and paternal effects.


Participants: Families with children and adolescents aged 2 to 15 years with anthropometric data available for children and adolescents and 2 parents (N=4423 families, N=7078 children and adolescents).

Main Exposure: Trained interviewers collected height and weight measurements.

Main Outcome Measure: Child/adolescent thinness, categorized using International Obesity Task Force criteria.

Results: Of 7078 children and adolescents, 402 (5.7%) were categorized as being thin. Thinness was more common in 2- to 5-year-olds (odds ratio, 1.61; 95% confidence interval, 1.22-2.13) than in 11- to 15-year-olds and in children and adolescents from ethnic minority (black: 2.28; 1.22-4.26; and Asian: 3.65; 2.76-4.83) than white backgrounds, but no differences were observed by sex or socioeconomic status. The strongest predictor of child/adolescent thinness was parental weight status. The prevalence of thinness was highest (16.2%) when both parents were thinner and progressively lower when both parents were in the upper half of the healthy-weight range (7.8%) or were overweight (5.3%) or obese (2.5%), with no differences in the magnitude of maternal and paternal influences.

Conclusions: These results are consistent with the idea that many cases of thinness are likely to represent the low end of the healthy distribution of weight and, as such, are likely to have a primarily genetic origin.

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PEDIATRIC THINNESS IS A MAJOR HEALTH PROBLEM IN COUNTRIES WHERE MALNUTRITION IS RIFE AND CHILD HEALTH CARE IS LIMITED.1,2 However, even in countries where food supplies are secure and maternal and pediatric services are strong, a proportion of apparently healthy children and adolescents can be categorized as being underweight. Parents often are concerned about their feeding strategies if their child or adolescent is thin, but in some cases, low weight may simply represent the lower end of the healthy distribution of weight. Evidence that pediatric thinness is associated with familial tendencies toward thinness might alleviate parental concerns.

The prevalence of pediatric thinness, defined using the new International Obesity Task Force (IOTF) criteria,3 is approximately 5% to 7% in adolescents from Europe, the United Kingdom, and Australia.4-6 Few studies have examined the demographic correlates. The one study7 that examined socioeconomic status (SES) found no association between underweight and parental income or educational level, but some evidence exists for slightly higher rates of underweight in female vs male adolescents.3-6

Children’s and adolescents’ weights consistently are correlated with the weights of their biological parents, but detailed investigations of family concordance have concentrated on obesity.5,10 The only study that examined familial concordance for thinness used data from siblings in a large sample of Swedish men conscripted into military service.11 Thinness (defined in this study as the lowest body mass index [BMI] decile) in first-born brothers was strongly predictive of thinness in later-born brothers.

The possibility that specific genes exist for thinness (or obesity resistance) has been proposed,12,13 but most of the emerging evidence points to the genetic determinants of adiposity being quantitative across the en-
tire weight range. This is supported by recent molecular genetic findings using a set of single-nucleotide polymorphisms identified in the obesity field, which demonstrated a continuous association between BMI and the number of high-risk alleles. A pediatric study using the same single-nucleotide polymorphism set found that infants and children with fewer high-risk alleles were more likely to be diagnosed as having failure to thrive and gained less weight until 9 years of age. Consistent with a quantitative model, appetitive traits also show a continuous association with adiposity, with overweight children having low satiety responsiveness and high food responsiveness and underweight children having high satiety responsiveness and low food responsiveness.

We are not aware of any familial analyses of pediatric thinness analogous to the studies of intergenerational transmission of obesity; no family studies have used the new IOTF criteria for thinness, and no comparisons have been made between paternal and maternal effects. Therefore, the objectives of the present study were to investigate the demographic and familial predictors of pediatric thinness and to compare maternal and paternal effects.

STUDY POPULATION AND DESIGN

The Health Survey for England is a nationally representative, annual survey of individuals living in private households in England that uses a multistratified probability sampling design, with the smaller-user Postcode Address File serving as a sampling frame. Data are collected from all adults in the household and as many as 2 children or adolescents aged 2 to 15 years. For households in which 3 or more children or adolescents resided, interviewers selected 2 of those individuals at random. We combined the results from the 6 surveys conducted from January 1, 2001, through December 31, 2006, using data from all families with 2 parents who self-identified as the biological parents of the index children and adolescents. Each annual survey is a new random sample of households in England. This study did not require ethical approval from the University College London Research Ethics Committee because it used anonymous data in the public domain (Health Survey for England, 2001-2006), for which appropriate permission already had been obtained.

OUTCOME MEASURES

Trained interviewers collected data during household visits. Height was measured with stadiometers and weight using electronic digital scales. Parents’ BMI was calculated as weight in kilograms divided by height in meters squared and categorized into underweight (<18.5), healthy weight (18.5 to <25.0), overweight (25.0 to <30.0), and obese (≥30.0). Few parents were underweight (only 47 mothers [1.1%] and 24 fathers [0.5%]), but we subdivided the healthy-weight group at the 50th BMI percentile (which was 23.4 for men and 22.6 for women), overweight (25.0 to <30.0), and obese (≥30.0). Few parents were underweight (only 47 mothers [1.1%] and 24 fathers [0.5%]), but we subdivided the healthy-weight group at the 50th BMI percentile (which was 23.4 for men and 22.6 for women), overweight (25.0 to <30.0), and obese (≥30.0). Few parents were underweight (only 47 mothers [1.1%] and 24 fathers [0.5%]), but we subdivided the healthy-weight group at the 50th BMI percentile (which was 23.4 for men and 22.6 for women), overweight (25.0 to <30.0), and obese (≥30.0).

Children’s and adolescents’ BMI was categorized into IOTF cutoffs for thinness, healthy weight, overweight, or obesity using the updated Microsoft Excel 2003 (Microsoft Corporation, Redmond, Washington) growth macro (http://homepage.Mac.com/ftc), which classifies BMI in children and adolescents ages 2 to 18 years as thin, healthy weight, overweight, or obese, associated with predicted BMIs of less than 18.5, 18.5 through 24.9, 25.0 through 29.9, or 30.0 or higher at 18 years of age. The IOTF criteria further categorize thinness into grade 1 (predicted BMI, 17.0 to <18.5), grade 2 (predicted BMI, 16.0 to <17.0), and grade 3 (predicted BMI, <16.0).

Socioeconomic status was classified based on the occupation of the household reference person, defined as the one with the highest income. Occupation was categorized as manual (skilled, semiskilled, and nonskilled) or nonmanual (professional, managerial-technical, and skilled). Child/adolescent ethnicity was categorized as white (British or white Irish descent), black (African or Caribbean descent), Asian (Indian, Pakistani, Indian-Caribbean, or Indian-African descent), or other (Chinese, Japanese, Filipino, Vietnamese, or mixed-ethnic-group descent) based on parental endorsement.

STATISTICAL ANALYSIS

Data were analyzed using SPSS statistical software, version 17.0 (SPSS Inc, Chicago, Illinois), and Stata statistical software, version 10.0 (StataCorp, LP, College Station, Texas), for Windows. Descriptive statistics were completed for age, sex, ethnicity, occupation, height, weight, BMI (BMI SD for children and adolescents), and weight status. Logistic regression was used to examine relationships among parental weight status; family SES; child/adolescent sex, age, and ethnicity; and child/adolescent thinness. Complex samples analysis was used to control for clustering by family. To take into account the sampling method (a maximum of 2 children or adolescents per family), a child-weighting (or adolescent-weighting) variable based on weightings provided with each annual survey was used. Boot-strapping with 1999 resamplings was used to test differences between regression coefficients in models for paternal and maternal weight; also, bias-corrected confidence intervals (CIs) are reported. Missing data were excluded in regression analyses.

RESULTS

Response rates for the surveys varied from 61% to 67% across years of the Health Survey for England. Only families with anthropometric data for 2 parents (n = 4423) were included in these analyses, but we have shown in a previous analysis that this subsample is comparable to the full sample (n = 13 762) in demographic and anthropometric characteristics. In the subsample of mothers (n = 4423), 1.1% were categorized as thin, which was the same in the full sample (n = 8319). In the subsample of fathers (n = 4423), 0.5% were categorized as thin, which was also the same in the full sample (n = 5443). The prevalence of pediatric thinness was the same in the full sample (5.7%) and in the subsample with anthropometric data from both parents (5.7%).

Study participant characteristics are given in Table 1. Data for 2 parents and at least 1 child or adolescent were available for 4423 families with 7078 children or adolescents, of whom 4423 (62.5%) were the older or only offspring in the family and 2635 (37.5%) the younger offspring. Combining data across all years, no families had both parents underweight. A total of 619 families (14.0%) had 2 healthy-weight parents, of whom 179 (28.9%) had 2 lower-health-weight parents. In the total sample of children and adolescents, 402 (5.7%) met the IOTF criteria for thinness, of whom 74 (1.8%) were categorized as thinness grade 0 or 1.
We examined the association of demographic and parental variables with risk of pediatric thinness using a multivariable analysis that controlled for all the predictor variables, clustering by family, and weighting by sampling method. Childhood/adolescent thinness did not show a significant trend during the years included in this analysis, and no significant sex differences were observed. Also, no difference was observed in prevalence between households with lower (manual occupation) and higher (nonmanual occupation) SES. Thinness was more common in younger children (8.1% in 2- to 5-year-olds vs 4.9% in 6- to 10-year-olds and 4.7% in the 11- to 15-year-olds; Table 2). Thinness also was more common in children and adolescents from ethnic minority backgrounds (11.4% of those from nonwhite ethnic groups combined vs 4.2% in whites).

**CHILD/ADOLESCENT THINNESS AND PARENTAL WEIGHT STATUS**

With 2 thinner parents, the prevalence of offspring thinness was 16.2%. With 2 upper-healthy-weight parents, 7.8% of children and adolescents were thin. This decreased to 5.3% with 2 overweight parents and 2.5% when both parents were obese. **Figure 1** illustrates the frequency of child/adolescent thinness by joint parental weight status. Because of the low numbers of children and adolescents with grades 2 and 3 thinness, we have not repeated the analyses with this group.

We used logistic regression to assess the association between parental weight combinations and child/adolescent thinness controlling for survey year, SES, child/adolescent age, sex, and ethnicity; clustering by family; and weighting for sampling method. The upper-healthy-weight group was used as the reference category. Having 2 thinner parents was associated with an increased risk of child/adolescent thinness (odds ratio [OR], 1.86; 95% CI, 0.97-3.55) compared with having 2 upper-healthy-weight parents. Having 2 overweight parents conferred lower risk (OR, 0.54; 95% CI, 0.30-0.99) and having 2 obese parents was associated with even lower risk (0.27; 0.12-0.60).

**CHILD/ADOLESCENT THINNESS AND MATERNAL OR PATERNAL INFLUENCE**

We used logistic regression to compare paternal and maternal influences controlling for year of survey; SES, child/adolescent age, sex, and ethnicity; clustering by family; and weighting for sampling method (Table 2). Although the odds of child/adolescent thinness with a lower-healthy-weight parent (vs an upper-healthy-weight parent) were slightly higher for mothers (OR, 1.56; 95% CI, 1.16-2.11) than fathers (1.43; 0.98-2.09), boot-strapping analysis showed that the difference in ORs had a standard score of 0.13, corresponding to statistical nonsignificance (bias-corrected 95% CI, −0.67 to 0.76).

**USING MIDPARENTAL BMI AS A SIMPLE MARKER OF FAMILIAL RISK**

A simple way to quantify combined parental risk is to calculate midparental BMI (the mean of the BMIs of the 2
parents), which we then categorized into quintiles. A linear trend of child/adolescent thinness was observed across quintiles of midparental BMI (Figure 2). Figure 2 also indicates that the distribution of thinness grades 2 and 3 has a similar pattern, although the slope (measured as change in percentage of child/adolescent thinness per midparental BMI quintile; \( m = y_2 - y_1 / x_2 - x_1 \)) is less pronounced (\( m = -0.39 \) for grades 2 and 3 and \( m = -1.89 \) for all grades of thinness combined).

Figure 3 illustrates differences between age groups. The slope is steepest for 11- to 15-year-olds (\( m = -2.14 \)), but the 2 younger age categories show similar slopes (2-5 years: \( m = -1.65 \); 6-10 years: \( m = -1.62 \)), although the age \( \times \) midparental BMI quintile interaction was not significant (\( P = .23 \)).

We also examined differences by SES and ethnicity. Slopes were similar for manual (\( m = -1.95 \)) and nonmanual (\( m = -1.85 \)) groups with no significant SES \( \times \) midparental BMI quintile interaction (\( P = .88 \)). The slope was steeper for families from nonwhite ethnic groups (\( m = -3.27 \)) than for whites (\( m = -1.49 \)), but the interaction with midparental BMI quintile was not significant (\( P = .14 \)).

In this population-based sample of 2- to 15-year-old children and adolescents, slightly more than 1 in 20 (ie, 5.7%) met the new IOTF criteria for thinness, of whom just more than 1 in 100 (ie, 1.1%) met criteria for the more extreme thinness grades (2 and 3). These rates are comparable to those reported from other European countries. In multivariate analyses adjusting for all other predictors and sampling methods, no differences were observed in prevalence by sex or SES, although thinness was more common in children and adolescents who were younger and from nonwhite ethnic groups.

As predicted, children and adolescents with thinner parents (eg, both parents below the 50th percentile of the healthy-weight group) were twice as likely to be thin (16.2%) as those with both parents in the upper half of the healthy-weight range (7.8%), and those with overweight or obese parents were progressively less likely to be thin (5.3% and 2.5%, respectively). This finding indicates not only that parents and their children and adolescents tend to be concordant for thinness but also that

Table 2. Multivariable Logistic Regression With Parent Weight Group, Survey Year, and Demographics for All 7078 Children and Adolescents

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Frequency of Child/Adolescent Thinness, No. (%)</th>
<th>Child/Adolescent Thinness, OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower healthy weight (n=970)</td>
<td>99 (10.2)</td>
<td>1.43 (0.98-2.09)</td>
</tr>
<tr>
<td>Upper healthy weight (n=973)</td>
<td>66 (6.8)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Overweight (n=3487)</td>
<td>185 (5.3)</td>
<td>0.84 (0.60-1.17)</td>
</tr>
<tr>
<td>Obese (n=1648)</td>
<td>52 (3.2)</td>
<td>0.59 (0.37-0.90)</td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lower healthy weight (n=1576)</td>
<td>149 (9.5)</td>
<td>1.56 (1.16-2.11)</td>
</tr>
<tr>
<td>Upper healthy weight (n=1572)</td>
<td>93 (5.9)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Overweight (n=2339)</td>
<td>109 (4.7)</td>
<td>0.67 (0.49-0.93)</td>
</tr>
<tr>
<td>Obese (n=1591)</td>
<td>51 (3.2)</td>
<td>0.51 (0.34-0.75)</td>
</tr>
<tr>
<td>Year of survey</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001 (n=1576)</td>
<td>70 (4.4)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>2002 (n=734)</td>
<td>50 (6.8)</td>
<td>1.65 (1.09-2.49)</td>
</tr>
<tr>
<td>2003 (n=1444)</td>
<td>64 (4.4)</td>
<td>1.02 (0.69-1.49)</td>
</tr>
<tr>
<td>2004 (n=1376)</td>
<td>119 (8.6)</td>
<td>1.10 (0.75-1.59)</td>
</tr>
<tr>
<td>2005 (n=674)</td>
<td>31 (4.6)</td>
<td>1.04 (0.64-1.69)</td>
</tr>
<tr>
<td>2006 (n=1274)</td>
<td>68 (5.3)</td>
<td>1.23 (0.84-1.81)</td>
</tr>
<tr>
<td>Child/adolescent age, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-5 (n=1872)</td>
<td>152 (8.1)</td>
<td>1.61 (1.22-2.13)</td>
</tr>
<tr>
<td>6-10 (n=2587)</td>
<td>128 (4.9)</td>
<td>1.04 (0.79-1.36)</td>
</tr>
<tr>
<td>11-15 (n=2619)</td>
<td>122 (4.7)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Child/adolescent sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n=3583)</td>
<td>202 (5.6)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Female (n=3495)</td>
<td>202 (5.8)</td>
<td>1.05 (0.85-1.31)</td>
</tr>
<tr>
<td>SES</td>
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<td></td>
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<tr>
<td>Nonmanual (n=4032)</td>
<td>230 (5.7)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Manual (n=2899)</td>
<td>165 (5.7)</td>
<td>0.99 (0.79-1.27)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
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<tr>
<td>White (n=5624)</td>
<td>235 (4.2)</td>
<td>1.00 [Reference]</td>
</tr>
<tr>
<td>Black (n=183)</td>
<td>15 (8.2)</td>
<td>2.28 (1.22-4.26)</td>
</tr>
<tr>
<td>Asian (n=1014)</td>
<td>130 (12.8)</td>
<td>3.65 (2.76-4.83)</td>
</tr>
<tr>
<td>Other (n=251)</td>
<td>20 (8.0)</td>
<td>1.66 (0.99-2.78)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; OR, odds ratio; SES, socioeconomic status.

*Analyses adjusted for clustering by family and weighted for sampling method.

*Regression analyses for 6925 patients because some SES (n=147) and ethnicity (n=6) data were missing.
the risk of pediatric thinness is continuous across parental BMI groups. The same pattern was observed in the sample of Swedish siblings,11 but this is the first time, to our knowledge, that intergenerational transmission has been examined.

In the obesity literature, there has been particular interest in the stronger maternal than paternal influence on offspring obesity risk. Our earlier analysis of this same data set confirmed that mother-child associations were significantly higher than father-child associations.24 However, in the present analysis, we found no evidence that maternal transmission of thinness was any higher than paternal transmission. This finding indicates that thinness is unlikely to be a direct consequence of intrauterine or early feeding characteristics of thinner mothers but rather is influenced equally by both parents.

The fact that childhood/adolescent thinness was extremely rare in families with obese parents is consistent with the high heritability of weight15 and gives evidence that children and adolescents with fewer obesity-risk alleles are more likely to be underweight.15 Research into heritable appetitive traits finds a similar pattern in that children and adolescents with larger appetites (eg, high food responsiveness or low satiety responsiveness) are more likely to be overweight, but those with smaller appetites are more likely to be underweight.16,17 One implication of this apparent cross-protection between thinness and obesity is that a better understanding of thinness also may be informative regarding obesity.

Thinness was more common in younger children, but we found no sex differences, confirming results from Australia7; however, other European studies4-6 have reported slightly higher rates of thinness in adolescent girls. Black and Asian children and adolescents had higher rates of thinness than their white counterparts, and in the future, it will be important to compare ethnic subgroups with different ancestry, cultural heritage, and immigration history. We found no SES differences in the prevalence of thinness, which militates against some environmental explanations for low body weight. However, we cannot rule out those influences, and it would be interesting in future analyses to consider factors such as parental and child/adolescent physical activity levels.

There were limitations to the present study. In modern England, adult thinness is uncommon; no families in our cohort had 2 thin parents. We had to combine data from families in which the parents were underweight or in the lower half of the healthy-weight range to define a relatively thinner group; this still constituted only 4.0% of all families. Few children or adolescents had grade 2 or 3 thinness, making analyses of more severe thinness underpowered, although interestingly, the association with midparental weight looked similar to the association for all grades of thinness combined. We were not able to exclude children, adolescents, or adults who were thin because of illness, although to do so might have strengthened the observed parent-child and parent-adolescent associations. To compare maternal and paternal effects, we had to select 2-parent families, which excluded a sig-
significant number of families. However, we have shown previously that no differences existed in age or obesity prevalence in children or parents in the subsample and the full sample,

To our knowledge, this is the first study to investigate intergenerational transmission of thinness. We found evidence of a strong family association, with most thin children and adolescents coming from families in which both parents were thinner than average. These results are consistent with the idea that at least some cases of pediatric thinness represent the lower end of the healthy population weight distribution. As long as thinner infants stay on track within their (low) BMI centile, and especially if their parents are thin, there may be no cause for concern regarding the weight of those children and adolescents and no need for parents to change their feeding practices.

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Author Contributions: Drs Jarvis and Wardle had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Wardle. Acquisition of data: Jarvis. Analysis and interpretation of data: Whitaker, Jarvis, Boniface, and Wardle. Drafting of the manuscript: Whitaker and Wardle. Critical revision of the manuscript for important intellectual content: Whitaker, Jarvis, Boniface, and Wardle. Administrative, technical, and material support: Jarvis. Study supervision: Wardle.

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