Associations of 18-Year-Old Daughters’ and Mothers’ Serum Leptin, Body Mass Index and DXA-Derived Fat Mass

Sanae Tanaka¹, Wu Bin², Mari Honda², Seiki Nanbu¹, Kazuhisa Suzuki¹,², Keisuke Fukuo¹,², and Tsutomu Kazumi¹,²

¹Department of Food Sciences and Nutrition, School of Human Environmental Sciences, Hyogo, Japan
²Open Research Center for Lifestyle-related Diseases, Mukogawa Women’s University, Hyogo, Japan

Aim and Methods: We assessed the relationship of the body mass index (BMI) of 187 college female students aged 18 years with the reported BMI of their middle-aged biological parents measured on 2 occasions: when the parents were 18–20 years old and at the time of the study. The relationships of fat mass measured using whole body dual energy X-ray absorptiometry (DXA) and serum leptin levels were also determined between 148 daughters and middle-aged parents (148 mothers and 59 fathers).

Results: The BMI of daughters was associated with their mothers’ BMI (r=0.30, p<0.0001) but not with their fathers’ BMI measured when they were 18 years old. Daughters’ BMI showed a stronger association with the current BMI of their mothers BMI (r=0.36, p<0.0001) than that of their fathers’ BMI (r=0.19, p=0.01). In addition, the serum leptin levels of daughters were correlated with their mothers’ leptin values (r=0.22, p=0.04). Further, not only total body fat mass (r=0.19, p<0.05) but also fat mass in the trunk (r=0.18, p<0.05) and legs (r=0.17, p<0.05) was associated between daughters and their mothers.

Conclusion: The significant correlation between daughters’ and mothers’ BMI measured when their mothers were 18 years old did not result from shared environmental factors, including the intrauterine environment. The results in the present study therefore suggest that adiposity in 18-year-old daughters may be influenced by the maternal effect. The associations of serum leptin and DXA-derived fat mass between daughters and their mothers may support our hypothesis.


Key words: BMI, Leptin, Fat mass, DXA, Offspring-parent relation

Introduction

The contributions of genetic and environmental factors to obesity in humans have been investigated in a variety of family studies covering a wide range of age¹. Genetic transmission as well as familial aggregation of obesity have been reported². Pedigree studies provide one means for disentangling the genetic and environmental sources of covariation among traits.

Inspection of significant familial patterns can lead to certain genetic and environmental inferences. For example, a pattern of significant correlations among siblings and between parents and offspring (who share about half their genes), but not between spouses (who share few genes, assuming random mating), suggests genetic heritability. Similarly, cross-trait familial correlations lead to the same type of genetic and environmental inferences. A pattern of significant cross-trait correlations between parents’ body size and offspring’s insulin level, and among siblings, but not spouses, would suggest that a common gene (or genes) influences both traits.

Because, as a general rule, mitochondrial DNA is exclusively maternally inherited³ and because mitochondria are fundamental in mediating effects on...
energy dissipation, we assessed the relationship between body mass index (BMI), a surrogate of body fat, in young women and their mothers, and compared with their fathers’ BMI. These comparisons used BMI measured on 2 occasions; when parents were 18 years old and at the time of the study. We also measured serum leptin and fat mass using dual energy X-ray absorptiometry (DXA), a well-established technique of measuring body composition that has been validated against most other reference measures. Since age is one important factor known to affect mitochondrial function, we studied healthy, young and slim people as described below.

**Methods**

The study population consisted of 2 groups of young women and their biological parents. The young women were students of the Department of Food Sciences and Nutrition, Faculty of Environmental Sciences, Mukogawa Women’s University (Nishinomiya, Japan). The study was approved by the MWU ethnic committee and written informed consent was obtained from all participants.

One group consisted of 208 female college students aged 18 years, who entered the Department of Food Sciences and Nutrition, School of Human Environmental Science, Mukogawa Women’s University, in 2001. Height and weight were measured to the nearest 0.1 cm and 0.1 kg, respectively. Current BMI (weight in kg/ height in m) was calculated from these measurements. Anthropometric measurements were also performed 12 months later.

We asked their parents to recall their body weight at the age of 18 years and at the time of the study. Self-reported heights and weights, which were shown to respond closely to measured heights and weights, were available in 187 out of 208 students (Table 1).

Fat mass and serum leptin were measured in another set of 148 daughter-mother pairs and 59 daughter-father pairs (Table 2). Daughters were students of the University and the characteristics of the daughters and their mothers are described in detail elsewhere. Body composition was determined using whole-body DXA (QDR-2000, software version 7.20D; Hologic, Bedford, MA, USA) as previously reported. The software provides estimates of lean tissue mass, fat mass, and bone mineral mass for the total body and for standard body regions. With the use of specific anatomic landmarks, regions of the head, trunk, arms, and legs were differentiated as shown in Fig. 1. Legs included both lower extremities and gluteal regions.

Blood samples were obtained in the morning after a 12-hr overnight fast. Leptin concentrations were assessed by an RIA kit from LINCO research (St. Charles, MO, inter-assay CV = 4.9%).

Statistical analysis was performed with Stat View. Data are presented as the means ± SD. Spearman’s correlation coefficients were calculated to determine the

**Table 1.** Current BMI and BMI at age 18 in daughters and their parents

<table>
<thead>
<tr>
<th></th>
<th>Daughters (n = 186)</th>
<th>Mothers (n = 186)</th>
<th>Fathers (n = 179)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>19</td>
<td>48 ± 3</td>
<td>51 ± 4</td>
</tr>
<tr>
<td>BMI at age 18 (kg/m²)</td>
<td>20.2 ± 2.4</td>
<td>20.0 ± 2.0</td>
<td>20.5 ± 2.3</td>
</tr>
<tr>
<td>Current BMI (kg/m²)</td>
<td>20.1 ± 2.2</td>
<td>21.8 ± 2.7</td>
<td>23.7 ± 2.3</td>
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<tr>
<td>Mean ± SD</td>
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**Table 2.** Fat mass measured using DXA and serum leptin in daughters and their parents

<table>
<thead>
<tr>
<th></th>
<th>Daughters (n = 148)</th>
<th>Mothers (n = 148)</th>
<th>Fathers (n = 59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>20.0 ± 0.8</td>
<td>50 ± 4</td>
<td>52 ± 5</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>20.4 ± 2.2</td>
<td>22.0 ± 2.8</td>
<td>24.1 ± 2.3</td>
</tr>
<tr>
<td>Body fat mass (kg)</td>
<td>14.4 ± 4.4</td>
<td>16.1 ± 5.9</td>
<td>15.5 ± 6.2</td>
</tr>
<tr>
<td>Trunk fat mass (kg)</td>
<td>7.0 ± 2.5</td>
<td>8.7 ± 3.6</td>
<td>9.7 ± 4.1</td>
</tr>
<tr>
<td>Leg fat mass (kg)</td>
<td>5.6 ± 1.5</td>
<td>5.3 ± 1.8</td>
<td>4.0 ± 1.6</td>
</tr>
<tr>
<td>Leptin (ng/mL)</td>
<td>8.6 ± 3.9</td>
<td>7.6 ± 4.9</td>
<td>3.6 ± 2.3</td>
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<tr>
<td>Mean ± SD</td>
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**Fig. 1.** Standard regions of DXA scanning: 1, head; 2, trunk; 3, arms; 4, legs.
Correlation coefficients of serum leptin and fat mass relation coefficients with their fathers’ BMI were 0.06 current BMI (their mothers’ BMI at age 18). Daughters’ BMI at age 19 was strongly associated with BMI measured 12 months later were essentially same. They were 18 years old. The results using students’ 18-year-old daughters’ BMI was associated with their fathers; however, it is noted that associations were stronger between daughters’ and mothers’ BMI than between daughters’ and fathers’ BMI. In addition, 18-year-old daughters’ BMI was associated with their mothers’ BMI measured when they were 18 years old. Not only serum leptin levels but also total body fat mass measured using DXA were associated between daughters and their mothers, whereas there was no relation between daughters and their fathers (Table 3). In addition, trunk and leg fat mass of daughters were associated with those of their mothers but not with their fathers. Fat mass of the arms did not show a sig-

Results

As shown in Fig. 2, 18-year-old daughters’ BMI was associated with the current BMI of their mothers and fathers; however, it is noted that associations were stronger between daughters’ and mothers’ BMI than between daughters’ and fathers’ BMI. In addition, 18-year-old daughters’ BMI was associated with their mothers’ BMI measured when they were 18 years old. In contrast, there was no correlation between 18-year-old daughters’ BMI and fathers’ BMI measured when they were 18 years old. The results using students’ BMI measured 12 months later were essentially same. Daughters’ BMI at age 19 was strongly associated with their mothers’ BMI at age 18 (r = 0.35, p < 0.001) and current BMI (r = 0.34, p < 0.001). Corresponding correlation coefficients with their fathers’ BMI were 0.06 and 0.14 (not significant).

Table 3. Correlation coefficients of serum leptin and fat mass measured using DXA between daughters and their parents

<table>
<thead>
<tr>
<th></th>
<th>Daughter-Mother (n=148)</th>
<th>Daughter-Father (n=59)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>0.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.02</td>
</tr>
<tr>
<td>Serum leptin (ng/mL)</td>
<td>0.17&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.06</td>
</tr>
<tr>
<td>Fat mass (kg):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>body</td>
<td>0.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.12</td>
</tr>
<tr>
<td>trunk</td>
<td>0.18&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.11</td>
</tr>
<tr>
<td>legs</td>
<td>0.17&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.12</td>
</tr>
<tr>
<td>arm</td>
<td>0.06</td>
<td>−0.01</td>
</tr>
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<sup>a</sup>: p < 0.05, <sup>b</sup>: p < 0.01

Fig. 2. Correlations between daughters and their biological parents of BMI at age 18 (upper panel) and current BMI (lower panel).
significant association between daughters and parents.

Discussion

In the present study, we found a significant association of 18-year-old daughters' BMI, a surrogate of total body fat, with their mothers' BMI measured when they were 18 years old. In contrast, there was no association between daughters' BMI and fathers' BMI measured when they were 18 years old. In addition, not only serum leptin levels but also fat mass in the body, trunk and legs measured using DXA were associated in daughters and their mothers. There was no relation between daughters and fathers in serum leptin and fat mass in any regions measured in the present study. This discrepancy might be in part due to the smaller number of daughter-father pairs.

The underlying mechanisms of the associations of 18-year-old daughters' BMI with mothers' BMI measured when the mothers were 18 years old remain to be elucidated. We cannot rule out the possibility of genetic imprinting and sex-linked genetic transmission; however, significant correlations between 18-year-old daughters' BMI and mothers' BMI measured when mothers were 18 years old did not result from shared environmental factors, including intrauterine environment, although parent-offspring correlations in general do not allow the separation of genetic and environmental transmission. These results therefore suggest that their mothers' but not their fathers' mitochondrial function may influence adiposity in 18-year-old daughters because, as a general rule, mitochondrial DNA is exclusively maternally inherited and because mitochondria are fundamental in mediating effects on energy dissipation. Age and exercise are major factors known to affect the size and/or function of mitochondria. We therefore examined a homogeneous cohort of young healthy Japanese people. The mothers studied were only, on average, 30 years old when they passed their mitochondria on to their daughters, who were only 18 years old; therefore, the characteristics of these mitochondria could not be attributed to aging. In addition, 18–20-year-old college students are not sedentary, as compared to middle-aged individuals in the general population. Further, it was noted that the majority of the population (parents and daughters) did not suffer from metabolic syndrome, type 2 diabetes, or insulin resistance, but rather were slim, young and healthy people. We have recently reported that middle-aged mothers' BMI was associated with 18-year-old sons' BMI, aspartate and alanine aminotransferase and gamma glutamyl transpeptidase, all of which are mitochondrial enzymes, whereas middle-aged fathers' BMI was not, suggesting that 18 year-old sons' adiposity may be influenced substantially by a maternal effect. The significant association between 18 year-old daughters' BMI and mothers' but not fathers' BMI measured when the parents were 18 years old found in the present study may imply that mothers' mitochondrial function may influence adiposity in 18-year-old daughters, as described above.

Significant associations between 18-year-old daughters' BMI and the current BMI of their parents, although correlation coefficients to mothers' BMI were consistently greater than those to fathers' BMI, might be related to the dominant influence of maternal genes. Mitochondrial-specific genes could be potential candidates, but imprinted genes, in which only the maternal allele is expressed, might be also implicated. In addition to a genetic effect, the potential contribution of environmental and behavioral components also needs to be considered. The effects of the intrauterine environment might contribute to the stronger association of BMI between daughters and mothers. Further, a greater postnatal sharing of environmental factors between mothers and daughters than between fathers and daughters might also explain the stronger maternal effect.

In addition to BMI, serum leptin concentrations, another surrogate of total body fat, were correlated between daughters in early adulthood and middle-aged mothers. Furthermore, not only body fat mass but also fat mass in the trunk and legs measured using DXA, the gold standard to assess regional fat mass, were associated between daughters in early adulthood and middle-aged mothers. A strong genetic influence on BMI, leptin and fat mass has previously been reported. In contrast to the daughter-mother relationship, no significant association was found between daughters in early adulthood and middle-aged fathers, although these findings deserve further investigation, because daughter-father pairs were small in number.

We used recalled body weight and self-reported current weight. Self-reported weights at 50 years were reported to be accurate for both men and women. In addition, recalling past weight was not significantly influenced by the passage of time, the numbers of years of education, or the accuracy of current weight reports. In that report, correlations between recalling past weights and measured weights ranged from $r = 0.87$ at 18 years to 0.95 at 40 years. In a validation study in the Nurses' Health Study II, the difference between measured and self-reported body weight at age 18 was, on average, only 1.4 kg. The correlation
coefficient between recalled weight at age 18 and measured weight in physical examination records at age 18 has been reported to be 0.87.

**Acknowledgements**

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