Regional body fat distribution in relation to pubertal stage: a dual-energy X-ray absorptiometry study of New Zealand girls and young women

Ailsa Goulding, Rachael W Taylor, Elspeth Gold, and Nicholas J Lewis-Barned

ABSTRACT  A cross-sectional study of 140 healthy, non-obese women and growing girls aged 8–27 y was undertaken to examine changes in total-body and regional fat and fat-free lean tissue mass by Tanner stage of pubertal development with dual-energy X-ray absorptiometry. Absolute fat mass and absolute fat-free lean tissue mass were higher at successive Tanner stages (1 through 5) but the proportional increase was greater for fat: total fat mass (kg) was about threefold higher in Tanner stage 5 than in stage 1 ($P < 0.001$), whereas lean tissue mass (kg) in Tanner stage 5 was about double that in stage 1 ($P < 0.001$). Furthermore, although the regional distribution of lean tissue mass in the trunk and legs remained fairly constant at different pubertal stages, the regional distribution of fat was altered significantly, becoming more central and less peripheral. Trunk fat (as a percentage of total body fat) was significantly higher at stage 5 than at stage 1 ($P < 0.001$). In the whole population, body mass index was positively correlated with trunk fat ($r = 0.662, P < 0.0001$) and negatively with leg fat ($r = -0.457, P < 0.0001$). We conclude that girls accumulate a higher proportion of their total adult fat mass than of their total adult lean tissue mass during puberty, and that regional fat patterns become more android and less gynoid with maturity. *Am J Clin Nutr* 1996;64:546–51.

KEY WORDS  Puberty, regional fat distribution, dual-energy X-ray absorptiometry, gynoid and android fat patterning, lean tissue mass

INTRODUCTION

Sex affects body composition and the distribution of body fat. Women have more fat and less lean tissue than do men and they typically store more fat in the hips and thighs (gynoid distribution) and less centrally in the trunk (android distribution) (1). These sex differences are not evident in early childhood, however, and are believed to originate during puberty (2, 3). To date, no quantitative dual-energy X-ray absorptiometry (DXA) measurements of regional fat and lean tissue distribution by Tanner stage of pubertal development have been reported.

DXA is considered to be ideal for examining body composition in children because it is quick, accurate, and involves a low radiation exposure ($< 2 \mu Sv$ per scan) (3). It is simple to use, sensitive to small changes (4–6), has good reproducibility, and is suitable for large population samples (1, 7–9). Body-composition results of DXA agree with those of direct chemical analysis (10). DXA accurately quantitates both total and regional lean, fat, and bone tissues, enabling fat in the hips and thighs to be distinguished from fat in the trunk (1).

In contrast, hydrodensitometry, which is considered to be the gold standard for body fat determination, does not provide any estimate of gynoid or android fat distribution and is unsuitable for use in young children. Skinfold thicknesses and waist-to-hip circumference ratios are not considered to provide good measurements of regional fat patterning in children (11–13). The high radiation level of computerized tomography (CT) precludes general use in children whereas both CT and magnetic resonance imaging are highly expensive and neither measures total body fat. The main critique of DXA for examining fat patterning is that, unlike magnetic resonance imaging and CT, DXA does not differentiate between subcutaneous and intraabdominal fat (11, 14). However, Treuth et al (15) recently published promising equations for estimating intraabdominal fat in adult women with DXA and simple anthropometric measures; estimations with these equations gave good agreement with direct CT estimations. This supports the view that DXA provides useful information on body composition and fat distribution.

Quantification of regional fat patterning is important for evaluating the health risks of an individual because of known associations of a central body fat distribution with risks of cardiovascular disease and diabetes in later life (16–18). Currently, there is a paucity of quantitative information regarding the development of central adiposity in children. The purpose of the present cross-sectional study was to examine the relation between pubertal stage and regional distribution of fat and lean tissue mass in nonobese girls and young women through use of DXA.

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SUBJECTS AND METHODS

Subjects

We measured body composition in 140 healthy girls, adolescents, and women living in Dunedin, a town in the South Island of New Zealand: 119 participants were aged from 8 to 16 y and 21 were mature adults (aged 18-27 y). No subjects were underweight or obese: all children had body mass index (BMI) values above the 5th and below the 90th percentile for age (19); adults had BMI values between 20 and 26. None of the subjects was taking any medication affecting body composition. Subjects were grouped by pubertal stage of development, determined by self-assessment according to Tanner stage (20). This method has been validated (21). SD scores for height- and weight-for-age were calculated with the data of Tanner and Whitehouse (22); SD scores for all Tanner stage 5 subjects were calculated on the basis of data for 18-y-olds. The mean age of menarche was 12.55 ± 1.19 y. All women in the Tanner stage 5 group had regular menstrual cycles. No blood samples were collected and energy intake was not assessed. The study was approved by the ethics committee of the Southern Regional Health Authority and written consent was obtained from participants or a parent or guardian.

Protocol

A short medical history was taken and subjects were weighed and measured without shoes and while wearing light clothing. All metal objects (buckles, watches, zips, and jewelry) were removed before body composition was measured. DXA was then performed in fast mode with a total-body scanner (model DPX-L, software version 1.3z; Lunar Corporation, Madison, WI) to directly measure bone mineral content (BMC), fat mass, and fat-free lean tissue mass. In our hands the CVs for these measurements in vivo match those obtained by others (1, 3, 7, 23) and were as follows: BMC, 1.35%; fat mass, 2.58%; and fat-free lean tissue mass, 0.88%. Standard Lunar software options were used to define regions of the body (head, arms, trunk, and legs) as described by others (1, 3). The upper border of the trunk region is delineated by a horizontal line below the chin, the vertical borders lie lateral to the ribs, and the lower borders are formed by oblique lines passing through the middle of the femoral necks. The leg region includes all tissue below the oblique lines through the femoral necks. The upper limbs external to the trunk region define the arm region and the composition of the head is the difference between total body composition and the tissue content of the trunk, leg, and arm regions. Total body percentage fat was calculated as fat mass divided by (fat mass + lean tissue mass + BMC) × 100/1. The fat and fat-free lean composition of the different body regions are expressed both in absolute values (kg) and as a percentage of total-body tissue mass.

Statistical analysis

Data were analyzed with the statistical package SPSS-X (SPSS, Inc, Chicago). Results are expressed as means ± SDs. When one-way analysis of variance indicated significant differences among group means, the differences were investigated with Duncan’s multiple-range test. The significance level was set a priori at P < 0.05. Pearson’s correlation coefficients (r) were determined to test linear relations among variables.

RESULTS

Height, weight, BMI, percentage fat, and absolute tissue contents of fat, fat-free lean tissue, and BMC were greater with each successive stage of puberty (Table 1). At Tanner stage 1, fat mass was only 36% of the adult fat content, whereas lean mass was already 54% of the adult lean mass. Fat mass was about threefold higher in stage 5 than in stage 1, whereas lean mass was only twofold greater, showing that a larger proportional gain in total-body fat stores than in total-body lean tissue mass takes place throughout puberty.

In every body region the proportional differences between Tanner stages 1 and 5 were greater for fat mass than for lean mass (Table 2). Figure 1 and Figure 2 confirm that considerable changes in regional fat distribution take place with advancing maturity; truncal fat increased steadily, accompanied by a slight decrease in leg fat; in contrast, the body distribution of lean tissue remained fairly constant at different Tanner stages. The proportion of lean tissue in the trunk was only slightly higher at stage 5 than at other stages, whereas for the legs, lean tissue was a little higher at stages 2 to 5 than at stage 1. The fraction of total body fat and total lean tissue present in the head and neck region became smaller with advancing maturity, in contrast with gains elsewhere. Thus, at Tanner stage 1, 10.1% of body fat and 6.7% of lean mass were

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Anthropometric data and total body composition by Tanner pubertal stage</th>
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<tbody>
<tr>
<td>Stage 1 (n = 45, 8–11 y)</td>
<td>Stage 2 (n = 33, 8–14 y)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>9.6 ± 0.8*</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>136.1 ± 6.7*</td>
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<tr>
<td>Height SDS</td>
<td>0.55 ± 0.80*</td>
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<tr>
<td>Weight (kg)</td>
<td>31.7 ± 4.5*</td>
</tr>
<tr>
<td>Weight SDS</td>
<td>0.52 ± 0.78*</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>17.06 ± 1.52*</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>21.6 ± 5.9*</td>
</tr>
<tr>
<td>Fat (kg)</td>
<td>6.78 ± 2.67*</td>
</tr>
<tr>
<td>Lean (kg)</td>
<td>22.83 ± 2.65*</td>
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<tr>
<td>BMC (kg)</td>
<td>1.104 ± 0.167*</td>
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</tbody>
</table>

1 ± SD. Means not sharing a common superscript are significantly different, P < 0.05. BMC, bone mineral content.

2 Standard deviation score for age (22).
in the head region compared with 2.1% of body fat and 6.7% of body lean mass at Tanner stage 5.

We found a strong, positive linear correlation between BMI and trunk fat ($r = 0.662, P < 0.0001$) and a negative correlation between BMI and leg fat ($r = -0.457, P < 0.0001$) (Figure 3), indicating that girls with higher BMI values carried more of their body fat in the trunk and less in the legs than did girls with lower BMIs. The trunk-to-leg ratios for fat mass increased in Tanner stages 1 to 5 from 0.743 ± 0.168 to 0.808 ± 0.218 to 0.911 ± 0.214 to 0.875 ± 0.200 to 1.000 ± 0.243 kg, respectively ($P < 0.05$, stage 1 versus stages 3 and 4; $P < 0.05$, stage 5 versus all other stages). In contrast, trunk-to-leg ratios for lean tissue mass remained steady; values were 1.39 ± 0.12 kg in stage 1 and 1.39 ± 0.11 in stage 5. Moreover, the proportions of total-body lean tissue in the legs ($r = 0.167, P < 0.048$) and trunk ($r = 0.226, P < 0.007$) were only weakly correlated with BMI (Figure 4).

**DISCUSSION**

This cross-sectional study extends previous work describing differences in body composition with advancing pubertal stage in women. In agreement with Rico et al (2) we found that progressive stages of puberty were associated with increases in both total fat mass and total lean body mass. We showed quantitatively that sexual maturation is also accompanied by considerable changes in the regional distribution of body fat, such that fat patterning changes from a pronounced gynoid distribution in young girls to a more android distribution in adult women. Our results support earlier studies in which skinfold thickness measurements were used (24). Furthermore, although none of our subjects was obese, we observed a positive correlation between BMI and truncal fat and a negative correlation between BMI and leg fat. These findings have been observed in adult women and children with increased adiposity (25, 26). Preferential central fat deposition was not accompanied by a more central distribution of lean tissue mass, indicating that differences were specific for fat and were not a general growth effect. Precocious accumulation of an android fat distribution increases the risks of metabolic complications later in life as opposed to a gynoid pattern of fat accumulation (27).

Previous cross-sectional studies in which DXA was used to measure changes in total body fat and lean tissue mass in growing girls all noted greater fat and lean tissue mass in older compared with younger girls (2, 3, 28). However, only one study assessed body composition in relation to pubertal state

![FIGURE 1](https://example.com/figure1.png)

**FIGURE 1.** Trunk fat as a percentage of total body fat and trunk lean tissue as a percentage of total lean tissue in groups of different pubertal status. a, significantly different from stages 1 and 2, $P < 0.05$; b, significantly different from stage 4, $P < 0.05$; and c, significantly different from all other stages, $P < 0.05$.

![FIGURE 2](https://example.com/figure2.png)

**FIGURE 2.** Leg fat as a percentage of total body fat and leg lean tissue as a percentage of total lean tissue in groups of different pubertal status. a, significantly different from stage 1, $P < 0.05$; b, significantly different from all other stages, $P < 0.05$.  

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**TABLE 2**

Regional content of fat and lean tissue by Tanner pubertal stage

<table>
<thead>
<tr>
<th></th>
<th>Stage 1 (n = 45)</th>
<th>Stage 2 (n = 33)</th>
<th>Stage 3 (n = 18)</th>
<th>Stage 4 (n = 23)</th>
<th>Stage 5 (n = 21)</th>
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<tbody>
<tr>
<td>Head</td>
<td></td>
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<tr>
<td>Fat (kg)</td>
<td>0.68 ± 0.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.73 ± 0.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.72 ± 0.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.79 ± 0.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.91 ± 0.32&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Lean (kg)</td>
<td>2.46 ± 0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.59 ± 0.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.70 ± 0.33&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>2.85 ± 0.33&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.83 ± 0.41&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>Arms</td>
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<tr>
<td>Fat (kg)</td>
<td>0.51 ± 0.24&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.82 ± 0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.89 ± 0.35&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.25 ± 0.67&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.56 ± 0.50&lt;sup&gt;b&lt;/sup&gt;</td>
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<td>Lean (kg)</td>
<td>2.03 ± 0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.64 ± 0.39&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.91 ± 0.38&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.66 ± 0.60&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.59 ± 0.88&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Trunk</td>
<td></td>
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<tr>
<td>Fat (kg)</td>
<td>2.40 ± 1.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.74 ± 1.68&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.75 ± 2.24&lt;sup&gt;b&lt;/sup&gt;</td>
<td>5.96 ± 2.79&lt;sup&gt;b&lt;/sup&gt;</td>
<td>8.15 ± 2.35&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Lean (kg)</td>
<td>10.53 ± 1.28&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.59 ± 1.46&lt;sup&gt;b&lt;/sup&gt;</td>
<td>14.22 ± 1.34&lt;sup&gt;b&lt;/sup&gt;</td>
<td>16.57 ± 1.82&lt;sup&gt;b&lt;/sup&gt;</td>
<td>20.36 ± 2.28&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Legs</td>
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<tr>
<td>Fat (kg)</td>
<td>3.17 ± 1.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.48 ± 1.27&lt;sup&gt;b&lt;/sup&gt;</td>
<td>5.02 ± 1.53&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.65 ± 2.40&lt;sup&gt;b&lt;/sup&gt;</td>
<td>8.20 ± 1.73&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Lean (kg)</td>
<td>7.64 ± 1.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.76 ± 1.30&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.01 ± 1.61&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.88 ± 1.61&lt;sup&gt;b&lt;/sup&gt;</td>
<td>14.69 ± 1.55&lt;sup&gt;b&lt;/sup&gt;</td>
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<sup>a</sup>, <sup>b</sup>, <sup>c</sup> ± SD. Means not sharing a common superscript are significantly different, $P < 0.05$.
assessed in this study, however, so few conclusions can be drawn about the effects of puberty on regional fat distribution.

Sex-specific differences in lean and fat mass patterning are generally attributed to differences in sex steroids and the hormonal milieu associated with puberty (27, 29, 30), although precise mechanisms are unclear. These hormonal factors include the net effects of estrogen, androgens, cortisol, growth hormone, insulin-like growth factor-I (IGF-I), and insulin. Ambient sex-hormone-binding globulin (SHBG) concentrations may also have an effect mediated through modifications in concentrations of unbound sex hormone. The increased proportion of android fat seen in our subjects with advancing pubertal stage suggests that estrogens do not play a dominant role because gluteal (hip-thigh) fat stores account for a high proportion of fat before puberty when estrogen concentrations are low (31). Androgen concentrations increase in girls during puberty and this may affect fat distribution as has been proposed for boys (3, 24). Testosterone concentrations and unbound androgenic activity have been shown to be correlated with body fat mass in girls during early puberty (30, 32). Androgens are largely bound to SHBG, which is higher in childhood than in early puberty (33) and is inversely related to central fat patterning in women (5, 25).

Because higher androgen concentrations themselves are known to lower SHBG concentrations, pubertal rises in androgens in girls may result in lower SHBG concentrations, higher concentrations of unbound androgen, and amplification of androgenic effects on fat distribution. Although high cortisol concentrations favor more central fat distribution and may have androgenic effects of their own (27, 34), there is little information about changes during puberty. Studies of growth hormone deficiency and excess, however, have indicated that growth hormone decreases android fat and increases lean mass (35–37). The sharp rise in growth hormone and IGF-I that accompanies early puberty would therefore be expected to limit accrual of abdominal fat in adolescent girls (29, 38). These rises may, however, also contribute to insulin insensitivity and hyperinsulinemia (39–41), and this appears to be more pronounced in girls than in boys (42). High circulating insulin concentrations suppress SHBG concentrations (43) and are correlated with BMI, growth hormone, IGF-I, and dehydroepiandrosterone sulfate concentrations, but are not closely related to gonadal hormone concentrations. Our findings suggest, in keeping with other researchers (29), that these effects may be important not only in determining the degree of fatness but also in determining the presence of increased abdominal fatness in young girls.

In conclusion, our study has shown that DXA has the sensitivity to detect rises in truncal fat associated with pubertal development in girls. The principal shortcomings of our study are that we have no information concerning genetics, plasma hormones, insulin sensitivity, lipid metabolism, energy expenditure, or past history of early growth and development for our subjects. Differences in these factors are likely to have influenced subject-to-subject variations in body fat and lean content and in fat patterning (44, 45). In addition, the cross-sectional design of our study makes it difficult to draw firm conclusions about progressive stage changes of body composition through puberty. Much further
work is required to achieve understanding of the underlying causes for the accentuated accumulation of fat in the upper body at puberty in growing girls. However, the subject is worthy of future investigation because fat patterns acquired in puberty and early adulthood may prove to be sensitive early predictors of risk for cardiovascular and metabolic disorders in adult life (24). Vague (27) pointed out that the presence of an abnormally pronounced android fat pattern may foretell the onset of metabolic complications up to 30 y later.

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REFERENCES

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