

Infant growth is associated with fat accumulation and distribution measured by DXA in 982 healthy children aged 8 to 15 years

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Background

Early growth trajectories are associated with childhood BMI and fat distribution as well as adulthood type 2 diabetes^{1,2}.

Mechanisms by which early growth determines later adiposity and fat distribution remain unclear, but the effect may be mediated through adipokines.

Objective and hypothesis

We aim to describe the association between infant growth, adolescent fat distribution and serum adipokines. We hypothesize that poor and rapid early growth is associated with less gluteofemoral fat and more abdominal fat, respectively, accompanied by altered circulating adiponectin and leptin.

Methods

A prospective population-based birth cohort study was performed with measurement of weight, length/height, abdominal circumference and skinfold thickness at 0, 3, 18 and 36 months of age. Follow-up at 8-15 years (mean age 11 years) included whole-body DXA.

Change in each growth parameter (Δ SDS) from 0-3, 3-18 and 18-36 months was calculated. Catch-down and catch-up was defined as Δ SDS < -0.67 and > 0.67 , respectively. Total and regional fat percentage by DXA (figure 1) was measured in 982 children (426 girls) and fat% SDS was calculated. Serum leptin and adiponectin were analyzed. The effect of infant growth on fat% SDS was assessed in multivariable linear regression models adjusting for previous growth interval(s), birth weight SDS, maternal pre-pregnancy BMI and Tanner Stage. The effect of infant growth on circulating adiponectin and leptin was stratified by sex and further adjusted for age and fasting. Analyses on regional fat% SDS were also performed adjusting for total fat% SDS.

Results

Change in weight (Δ SDS) 0-3, 3-18 and 18-36 months showed the strongest association to body fat in early adolescence compared to change in abdominal circumference, height, BMI and skinfold thickness (Table 1). Birth weight SDS and change in weight were positively and independently associated with total fat accumulation, whereas the association with the android-to-gynoid ratio was not linear (figure 2). Adjusting the effect of infant growth on android fat% SDS for total fat% SDS reversed the association between BW SDS quartiles and android fat% (negative trend, $p < 0.05$).

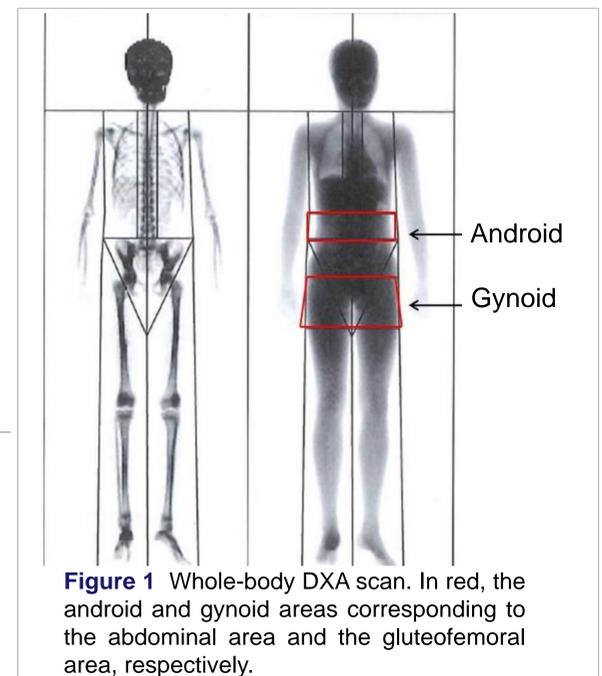


Figure 1 Whole-body DXA scan. In red, the android and gynoid areas corresponding to the abdominal area and the gluteofemoral area, respectively.

Δ Growth SDS	Total fat% (SDS)	Android-to-Gynoid Fat Ratio
	β (95% CI)	β % (95% CI)
Weight 0-3 months	0.20 (0.11, 0.28)**	2.9 (0.2, 5.6)*
Weight 3-18 months	0.25 (0.16, 0.34)**	2.0 (-0.7, 4.8)
Weight 18-36 months	0.38 (0.21, 0.56)**	6.9 (1.1, 13.1)*
Height 0-3 months	0.08 (-0.02, 0.17)	1.0 (-2.0, 4.1)
Height 3-18 months	0.07 (-0.03, 0.17)	1.8 (-1.2, 4.8)
Height 18-36 months	0.23 (0.06, 0.41)*	4.4 (-1.3, 10.4)
Abd.cir. 0-3 months	0.14 (0.08, 0.20)**	2.8 (0.8, 4.8)*
Abd.cir. 3-18 months	0.09 (0.02, 0.16)*	2.4 (0.1, 4.7)*
Abd.cir. 18-36 months	0.12 (0.02, 0.22)*	4.3 (1.0, 7.8)*

Table 1 Associations between changes in selected growth parameters and DXA fat outcomes at 8-15 years of age, adjusted for previous growth, birth weight SDS, maternal pre-pregnancy BMI and Tanner Stage. Abd.cir: Abdominal circumference * $p < 0.05$ ** $p < 0.01$

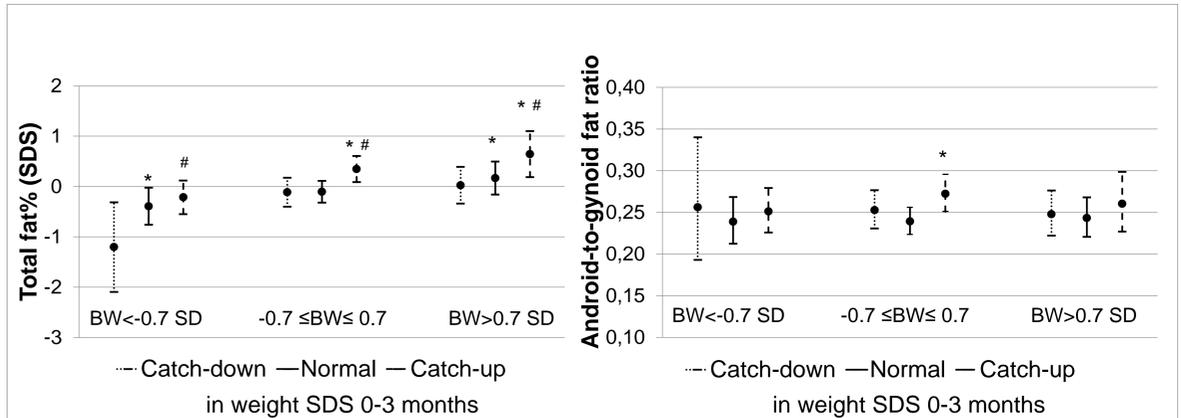


Figure 2 Effect of catch-down (dotted lines), normal growth (solid lines) and catch-up (dashed lines) in weight SDS 0-3 months on total fat% SDS (left) and android-to-gynoid ratio (right) stratified by BW SDS quartiles (lower, intermediate and upper). Dots: adjusted estimated mean; Error bars: 95% CI. * $p < 0.05$ for difference using intermediate quartile BW SDS and normal weight change as reference. # $p < 0.05$ for linear trend

Circulating adiponectin was inversely associated with android fat% SDS (boys: $\beta = 11\%$ [-21%; -2%], girls: $\beta = -22\%$ [-31%; -12%]) and positively associated with gynoid fat% SDS (boys: $\beta = 18\%$ [0%; 38%], girls: $\beta = 23\%$ [5%; 45%]) when adjusting for total fat% SDS, reflecting different metabolic function of abdominal and gluteofemoral fat. However, infant growth was not associated with circulating adiponectin.

Circulating leptin was positively associated with total fat% SDS (boys: $\beta = 72\%$ [64; 80%], girls: $\beta = 73\%$ [64%; 80%]), but not with regional fat% when adjusting for total fat% SDS. Change in weight SDS 3-18 months (girls) and 18-36 months (both sexes) were positively associated with circulating leptin, (girls: $\beta = 15\%$ [4%; 27%] and $\beta = 28\%$ [2%; 59%], respectively; boys $\beta = 21\%$ [1%; 41%]), reflecting the strong correlation between adipose tissue and leptin.

Conclusion

This longitudinal birth cohort made it possible to show that prenatal and postnatal growth has independent and differential effects on pubertal fat distribution.

References

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