Discussion: Overall, GT resulted in slightly better results in almost all domains, however without reaching statistical significance. Sustainability in time and cost/benefit need further follow up.

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WHICH PERIODS OF GROWTH IN INFANCY AND CHILDHOOD DETERMINE ADIPOSITY AT AGE 16? THE TRAILS STUDY

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Objective: To assess in which period during infancy and childhood, growth is most associated with overall and abdominal adiposity in adolescence, and with associated metabolic traits. Furthermore, we aimed to assess if these associations differ dependent on smoking during pregnancy.

Population & methods: We obtained repeated anthropometric measurements from birth to mean age of 16.2 years on 772 girls and 708 boys in a population-based cohort. At age 16.2 years, weight, height, skinfold thicknesses, %BF, waist circumference, blood pressure, glucose, insulin, and lipids were measured.

Results: Weight gains between age 2 to 4 and age 4 to 7 years were most strongly associated with higher BMI, sum of skinfolds, %BF and waist circumference at age 16.2 years. Increases were +1.01 to +1.42 SDs per SD in weight gain (all $p< 0.001$). Increases in risks of being overweight or obese at age 16.2 years (odds ratios) were 23.9 to 32.5 per SD in weight gain ($p< 0.001$). Gains in these periods were also associated with a less favorable metabolic syndrome score. If mothers smoked during pregnancy, these weight gains were stronger associated with adiposity measurements at age 16.2 years (0.33 - 0.38 SDS higher; all $p< 0.05$) than if mothers did not smoke during pregnancy.

Conclusions: Strong increases in weight SDS from ages 2 to 7 years predispose to overall and abdominal adiposity and their associated metabolic traits in adolescence. In adolescents whose mothers smoked during pregnancy, associations with weight gain during these years is even more pronounced.

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IMPACT OF MATERNAL BODY MASS INDEX ON OFFSPRING LIVER LIPID DEPENDS ON MODE OF DELIVERY

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Background: Maternal overweight/obesity is associated with increased risk of Caesarean Section (CS) and offspring metabolic syndrome, though the mediating biological pathway is unknown. In an animal model pre-labour CS in comparison with vaginal delivery (VD), is associated with significantly higher offspring intrahepatocellular lipid (IHCL), a central component of the metabolic syndrome (Hyde MJ, 2010).

Aim: To examine the effects of maternal Body Mass Index (BMI) and mode of delivery on neonatal IHCL.

Methods: With research ethics approval and informed written parent consent we employed ^{1}H Magnetic Resonance Spectroscopy of liver during natural sleep in healthy term neonates (Thomas EL, 2008). We used analysis of covariance to explore the effect of maternal BMI, diabetes and mode of delivery, and infant gender, birth weight and postnatal age and their interactions on IHCL (presented as the ratio of lipid CH_2/water).

Results: To date we have studied 57 infants (45 VD, 12 pre-labour CS). IHCL ranged from 0 to 5.5, and maternal BMI from 17 to 35 kg/m^2. There was a significant interaction between maternal BMI and mode of delivery ($p=0.010$). In neonates born by pre-labour CS, IHCL increased by a mean of 29% [95%CI 9-54%] per unit increase in maternal BMI ($p=0.004$). Maternal BMI had no impact on IHCL in VD infants ($p=0.81$).

Conclusion: VD may activate pathways that protect against liver lipid accumulation in the presence of increasing maternal BMI. Pre-labour CS may pose an added risk for the development of the metabolic syndrome in offspring of overweight/obese mothers.