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Maternal obesity and offspring adiposity trajectories during early childhood: a longitudinal study from the PONCH cohort

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Abstract

Background/Objectives

Maternal obesity and gestational diabetes mellitus (GDM) are increasingly common metabolic conditions during pregnancy, potentially affecting offspring metabolic development and adiposity across childhood. We investigated longitudinal adiposity and anthropometric development in preschool children born to women with normal weight, obesity, or GDM, and explored associations between maternal metabolic markers during pregnancy and offspring adiposity outcomes.

Subjects/Methods

The Pregnancy Obesity Nutrition Child Health (PONCH) cohort is a longitudinal mother–child study including offspring of women with normal weight (NW), obesity (OB), or GDM during pregnancy. Repeated anthropometric measurements and body composition assessments by air-displacement plethysmography were obtained annually between ages 2 and 6 years in 178 children (NW, n = 80; OB, n = 57; GDM, n = 41). Longitudinal differences between maternal groups were evaluated using mixed-effects models.

Results

Children in the OB group exhibited higher adiposity throughout preschool age compared with the NW group, including BMI (16.9 vs 16.2kg/m², $p < 0.001$), body fat percentage (28.0% vs 24.9%, $p < 0.001$), and fat mass (5.22 vs 4.35kg, $p < 0.001$). Differences became more pronounced with age and were generally greater in boys, whereas adiposity profiles remained similar between the GDM and NW groups. Maternal adiposity-related markers showed progressively stronger associations with offspring adiposity across childhood, including fat mass in 6-year-old boys ($r = 0.60$, $p < 0.001$) and girls ($r = 0.40$, $p = 0.04$). Maternal glycaemic markers showed limited associations, primarily observed at 2 years of age in girls, whereas associations at later ages were sparse and inconsistent.

Conclusions

Offspring of women with obesity exhibited progressively greater adiposity throughout preschool age compared with offspring of women with normal weight and GDM. These findings suggest that maternal obesity may represent a more sustained determinant of offspring adiposity development during early childhood and support early preventive strategies. Continued longitudinal follow-up will be important to determine whether these differences persist into later childhood and adolescence.

INTRODUCTION

Childhood obesity is increasing globally and represents a major contributor to early cardiometabolic disease, including type 2 diabetes and cardiovascular disease [1]. Maternal obesity and gestational diabetes mellitus (GDM) are common metabolic conditions during pregnancy and are recognized as important determinants of offspring metabolic health [2–4]. The Developmental Origins of Health and Disease (DOHaD) framework proposes that environmental exposures during critical periods of fetal and early postnatal development may induce long-term alterations in physiological systems regulating adiposity, glucose metabolism, and cardiometabolic risk [5]. Hence, the intrauterine environment and the first years of life are considered as critical windows for the establishment of later metabolic trajectories [6].

A large body of evidence has linked maternal obesity and GDM to increased adiposity and altered metabolic health in offspring already at birth and during infancy [2, 3]. Maternal obesity has consistently been associated with higher birthweight and increased adiposity during infancy and childhood, whereas findings regarding maternal glycaemic dysregulation and early-life adiposity have been more variable [6]. Collectively, findings from birth cohorts and longitudinal follow-up studies suggest that maternal obesity and glycaemic dysregulation may be associated with different developmental patterns of adiposity and metabolic health across childhood [7–10].

Although rapid infant growth is a recognized predictor of later obesity, most studies examining offspring adiposity in relation to maternal metabolic status have focused on birth or infancy [11–14]. Consequently, considerably less is known about how adiposity and body composition trajectories evolve throughout preschool age, particularly using repeated body composition assessments during a developmental period characterised by rapid changes in adiposity distribution and growth trajectories [15]. In particular, it remains unclear whether maternal glycaemic and adiposity-related factors show different patterns of association with adiposity development across early childhood, including potential sex-specific differences [7, 13].

The Pregnancy Obesity Nutrition Child Health (PONCH) study is a longitudinal mother–child cohort established to investigate how maternal metabolic health during pregnancy influences offspring growth, adiposity, and metabolic development from infancy through childhood [16]. In previous analyses from the cohort, we demonstrated that maternal obesity and GDM influenced offspring body composition already during early infancy. Specifically, maternal glycaemic markers were associated with adiposity in infant girls already at 1 week of age, suggesting early sex-specific programming effects [16]. Similar sex-specific associations between maternal insulin resistance and infant adiposity have also been reported in other cohorts using direct body composition measurements [12]. These findings supported the hypothesis that maternal metabolic factors may influence offspring adiposity trajectories from the earliest stages of life.

Whether these early-life differences persist into later childhood remains unclear. Longitudinal follow-up of the PONCH cohort into preschool age provides a unique opportunity to investigate whether early programming signals translate into persistent differences in adiposity trajectories and body composition

development throughout early childhood, and to examine potential cross-generational determinants of child adiposity development. Understanding how maternal obesity and glycaemic dysregulation relate to adiposity development across early childhood may help identify children at increased risk of obesity and inform future preventive strategies.

The primary aim of the present study was to investigate differences in adiposity and anthropometric measures among preschool children aged 2–6 years according to maternal metabolic group (normal weight, obesity, and GDM). Secondary aims were to examine associations between maternal glycaemic and adiposity-related markers during pregnancy and offspring adiposity and anthropometric outcomes across early childhood, including potential age- and sex-specific patterns.

METHODS

Ethical approval

The study was approved by the Regional Ethical Review Board in Gothenburg, Sweden (Dnr 402-08). All participants received oral and written information prior to inclusion, and written informed consent was obtained from all women on behalf of themselves and their children.

Study design and participants

The present study is based on the PONCH cohort, a longitudinal mother–child study including women with normal weight (NW), obesity (OB), GDM, and their offspring. Offspring were categorized according to maternal group (NW, OB, or GDM). Recruitment procedures and maternal study visits have previously been described in detail [17,18].

Briefly, pregnant women with normal weight (BMI 18.5–24.9 kg/m^2) and obesity (BMI ≥30 kg/m^2) were recruited from antenatal care units in the Gothenburg area. In addition, pregnant women diagnosed with GDM, irrespective of BMI, were recruited at Sahlgrenska University Hospital, Gothenburg, Sweden, at the time of diagnosis. During the study period, pregnant women with risk-factors or elevated non-fasting blood glucose levels in routine antenatal care underwent oral glucose tolerance testing (OGTT). GDM diagnosis was based on the European Association for the Study of Diabetes 1991 criteria, including fasting plasma glucose ≥7.0 mmol/L, 2-hour OGTT glucose ≥10.0 mmol/L, or random non-fasting plasma glucose ≥12.2 mmol/L [19].

Maternal study visits were performed during pregnancy and included anthropometric and body composition measurements, blood sampling, and lifestyle questionnaires [17,18]. Exclusion criteria for the study were pre-pregnancy diabetes, multiple pregnancies, use of neuroleptic drugs, vegetarianism and veganism. None of the included women reported smoking or alcohol consumption during pregnancy. Children were followed longitudinally from infancy through childhood [16]. The present analysis includes repeated anthropometric and body composition measurements obtained annually between ages 2 and 6 years (**Figure 1**). Data were collected between 2012 and 2025 at Sahlgrenska

University Hospital, Gothenburg, Sweden. In total, 178 children were included in the study: 80 in the NW group (39 girls, 41 boys), 57 in the OB group (22 girls, 35 boys), and 41 in the GDM group (23 girls, 18 boys).

The main reasons for missed follow-up visits were child illness at the scheduled study visit, which according to protocol was performed within a limited time window around each birthday (± 10 days). Reasons for study drop-out included lack of time and relocation outside the Gothenburg area. A higher proportion of missing body composition measurements was observed at the 2- and 3-year follow-ups due to practical challenges associated with body composition assessments in younger children (Figure 1), including difficulty remaining still during Bod Pod measurements and reluctance to enter the chamber.

Maternal and child visits

Maternal pregnancy visits have previously been described in detail [18]. Maternal assessments were performed in the morning after an overnight fast and included anthropometric measurements, venous blood sampling and assessment of body composition by air-displacement plethysmography, performed with Bod Pod Gold standard system (Bod Pod 2007 A, Life Measurement, Concord, CA and software version 5.4.0. Cosmed).

Maternal adiposity-related markers included body weight, BMI, body fat percentage and total fat mass. Glycaemic markers included fasting plasma glucose, HbA1c, insulin, and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). HOMA-IR was calculated according to the standard formula: fasting insulin (mU/L) × fasting glucose (mmol/L) / 22.5. HbA1c, glucose and insulin analyses were performed at the accredited Clinical Chemistry Laboratory, Sahlgrenska University Hospital. First-trimester body weight and BMI were measured at study visits in women with normal weight and obesity, whereas corresponding data for women with GDM were obtained from medical records because study visits were initiated after diagnosis. Educational level was classified according to highest attained education level.

Child anthropometric and body composition measurements were obtained annually between ages 2 and 6 years. Body weight was measured using calibrated scales and height using a stadiometer. BMI was calculated as weight (kg)/height (m^2). Hip and waist circumferences were measured by standard protocols to the nearest 0.5 cm. Body composition, including body fat percentage and fat mass, was assessed at maternal pregnancy visits and all child follow-up visits by Bod Pod. For children, the paediatric Bod Pod option was used, measurements were performed in triplicate, and body composition was calculated using the Lohman equation.

Statistical methods

Repeated measurements from all available visits were included in the longitudinal analyses. Linear mixed-effects models were used to account for within-subject correlations and allowed inclusion of

participants with varying numbers of follow-up visits under the assumption that data were missing at random. Missing data were not imputed.

Maternal baseline characteristics were compared between groups using one-way analysis of variance (ANOVA) for continuous variables, including maternal age, BMI, and glycaemic measures. Pairwise group comparisons were evaluated using Tukey-adjusted p-values. Categorical variables, including educational level and ethnicity, were compared using Pearson's chi-square test.

Primary outcomes of child adiposity and anthropometric measures were analysed longitudinally across ages 2–6 years using linear mixed-effects models. Models included maternal group (NW, OB, and GDM), child age, and child sex as fixed effects, with a random intercept for child ID to account for repeated measurements within individuals. Sex-stratified analyses were additionally performed by including interaction terms between maternal group and child sex. Data derived from mixed-effects models are presented as estimated marginal means ± SEM unless otherwise stated. Pairwise comparisons between maternal groups were performed using Tukey-adjusted contrasts. Assumptions of normality and homoscedasticity were evaluated by visual inspection of Q–Q plots and residual plots for all statistical models. Homogeneity of variance was additionally assessed using Bartlett's test where appropriate.

Associations between maternal glycaemic and adiposity-related markers during pregnancy and offspring adiposity and anthropometric outcomes were evaluated using Pearson correlation analyses. Correlation matrices were generated separately by child age and stratified by sex where specified.

To summarize the overall strength of associations across ages, mean absolute correlation coefficients (|r|) were calculated as the average of the absolute correlation coefficients across all maternal–offspring variable pairs within each category of maternal markers (adiposity-related and glycaemic markers). This approach provided a summary measure of overall association strength.

P-values for associations involving maternal adiposity-related variables were adjusted for multiple testing using the Benjamini–Hochberg false discovery rate (FDR) procedure. Associations involving maternal glycaemic markers are presented using unadjusted p-values and should therefore be interpreted with caution.

Unless otherwise stated, a two-sided p-value <0.05 was considered statistically significant. All statistical analyses and data visualizations were performed in R version 4.5.3 using packages including lme4 (1.1-38), emmeans (2.0.2), devtools (2.4.6), and ggplot2 (4.0.2).

RESULTS

Maternal characteristics during pregnancy

Maternal BMI in early pregnancy was highest among women with obesity, intermediate among women with GDM, and lowest among women with normal weight (all pairwise comparisons $p < 0.001$) (**Table 1**). Glycaemic measures in late pregnancy were elevated among women with GDM, with higher blood

glucose and HbA1c compared with women with normal weight and obesity, consistent with the diagnostic criteria for GDM. Gestational weight gain was lower among women with obesity and GDM than among women with normal weight (both $p < 0.001$), whereas gestational age was slightly lower in the GDM group than in the normal-weight group.

Educational level differed between groups with a higher proportion of women with ≥3 years of university education in the normal-weight group (73.1%) than in the obesity group (46.4%). Ethnicity also differed between groups, with a higher proportion of women with non-Nordic ethnic background in the GDM group (23%) than in the normal-weight (6%) and obesity (10%) groups.

Table 1. Maternal characteristics during pregnancy by maternal group

	NW		OB		GDM	P (between groups)		
	n	Mean (SD)	**n**	Mean (SD)	**n**	Mean (SD)	NW vs OB	NW vs GDM	OB vs GDM
Maternal data									
Gestational age (weeks)	80	40.2 ± 1.1	57	40.0 ± 1.6	41	39.6 ±1.2	0.70	**0.049**	0.28
Maternal age (y), T1	80	31.4 ± 3.7	57	31.1 ± 3.6	41	32.1 ± 4.6	0.87	0.65	0.42
Maternal BMI (kg/m^2), T1	80	22.3 ± 1.6	57	35.0 ± 3.9	41	28.2 ± 7.2	**<0.001**	**<0.001**	**<0.001**
HbA1c (mmol/mol), T3	79	29.6 ± 2.8	55	30.7 ± 3.1	39	33.9 ± 4.3	0.13	**<0.001**	**<0.001**
Plasma glucose (mmol/l), T3	78	4.5 ± 0.4	55	4.9 ± 0.4	39	5.2 ± 0.8	**<0.001**	**<0.001**	**0.04**
Gestational weight gain (kg)	80	11.4 ± 3.1	51	7.9 ± 5.5	41	8.4 ± 4.8	**<0.001**	**0.001**	0.89
Education, n (%)									
≤2 years high school						2 (5.4%)	**0.002**	0.06	0.20
≥3 years high school		8 (10.3%)		18 (32.1%)		8 (21.6%)			
≤3 years university		13 (16.7%)		12 (21.4%)		6 (16.2%)			
≥3 years university		57 (73.1%)		26 (46.4%)		21 (56.8%)			
Ethnicity, n (%)									
Sweden		75 (93.8%)		51 (89.5%)		31 (77.5%)	0.58	**0.01**	**0.02**
Nordic countries		1 (1.3%)		3 (5.3%)					
Europe		3 (3.8%)		2 (3.5%)		2 (5.0%)			
Outside Europe		1 (1.3%)		1 (1.8%)		7 (17.5%)			

Values are presented as mean (SD) for continuous variables and n (%) for categorical variables. P-values < 0.05 were considered statistically significant and are presented in bold. Continuous variables were compared between groups using one-way analysis of variance (ANOVA), with pairwise group comparisons evaluated using Tukey-adjusted p-values. Categorical variables were compared using Pearson's chi-square test. BMI, body mass index; GDM, gestational diabetes mellitus; NW, normal weight; OB, obesity; T1, trimester one; T3, trimester three.

Longitudinal adiposity and anthropometric measures (2-6 years)

In longitudinal mixed-model analyses including repeated measurements across ages 2–6 years, children born to mothers with obesity exhibited higher adiposity and anthropometric measures than children born to mothers with normal weight (**Figure 2**). These differences were observed across multiple outcomes, including BMI, body fat percentage, fat mass, body weight, and hip circumference. For example, estimated BMI, fat mass, and body fat percentage were all higher in the OB group than in the NW group (BMI: 16.9 vs 16.2 kg/m^2; fat mass: 5.22 vs 4.35 kg; body fat percentage: 28.0% vs 24.9%; all $p < 0.001$).

Several outcomes additionally differed between children in the OB and GDM groups. BMI, fat mass, and hip circumference were higher in the OB group than in the GDM group (BMI: 16.9 vs 16.4 kg/m^2, $p = 0.003$; fat mass: 5.22 vs 4.43 kg, $p = 0.003$; hip circumference: 58.1 vs 56.7 cm, $p = 0.010$). Complete model estimates and pairwise comparisons are presented in **Supplementary Table 1**. No significant differences were observed between the GDM and NW groups.

Descriptive longitudinal trajectories demonstrated persistent separation between maternal groups across ages 2–6 years (**Figure 3**). Across outcomes, children in the OB group consistently exhibited higher adiposity and anthropometric measures compared with the NW group, whereas trajectories in the GDM group largely overlapped with those in the NW group. The clearest visual separation was observed for BMI, fat mass, and waist circumference, with differences becoming more pronounced between 5 and 6 years of age. For example, at 6 years of age, mean fat mass was 4.79 kg in the OB group compared with 3.62 kg in the NW group.

Sex-stratified longitudinal analysis

Boys born to mothers with obesity exhibited consistently higher adiposity and anthropometric measures across all outcomes compared with those born to mothers with normal weight (**Figure 4**). For example, boys in the OB group had higher BMI (16.9 vs 15.9 kg/m², $p < 0.001$), fat mass (4.96 vs 3.81 kg, $p < 0.001$), and body fat percentage (26.8% vs 22.3%, $p < 0.001$) compared with boys in the NW group. Several outcomes also differed between the OB and GDM groups, including fat mass (4.96 vs 4.11 kg, $p = 0.042$) and hip circumference (57.9 vs 55.9 cm, $p = 0.025$). In contrast, adiposity and anthropometric measures were generally similar between the GDM and NW groups. Complete model estimates and pairwise comparisons are presented in **Supplementary Table 2**.

Among girls, differences between maternal groups were less pronounced. Girls in the OB group had higher body weight and hip circumference compared with those in the NW group (body weight: 18.5 vs 17.4 kg, p = 0.019; hip circumference: 59.6 vs 57.0 cm, p < 0.001). BMI, body fat percentage, and fat mass also tended to be higher in the OB group, although these differences did not reach statistical significance (Supplementary Table 2). Overall, girls in the GDM group and NW group showed comparable anthropometry and adiposity across outcomes.

Secondary outcome: Cross-generational determinants of child adiposity

Across ages 2 to 6 years, maternal adiposity-related markers including body weight, BMI, body fat percentage, and total fat mass showed progressively increasing associations with child adiposity and anthropometric outcomes (body weight, BMI, body fat percentage, fat mass, hip and waist circumference). Both the mean absolute correlation coefficients and the number of statistically significant associations increased with age, with the strongest observations at 6 years (**Figure 5**). This pattern was consistent in pooled analyses and when stratified by sex, although associations were generally stronger in boys. Heatmap analyses further illustrated the progressive strengthening and increasing density of associations between maternal adiposity-related markers and offspring adiposity outcomes across childhood (**Supplementary Figure 1**). For example, in 6-year-old boys, maternal body weight was strongly associated with child fat mass (r = 0.66), BMI (r = 0.60), and hip circumference (r = 0.59) (all FDR-adjusted p < 0.001).

In contrast, maternal glycaemic markers (fasting glucose, insulin, HbA1c, and HOMA-IR) showed the strongest unadjusted associations with child adiposity outcomes at 2 years of age and progressively weaker and less consistent associations from ages 3 to 6 years (Figure 5). Associations with maternal glycaemic markers were primarily observed in girls at younger ages, whereas associations in boys were less frequent and of smaller magnitude. Heatmap analyses demonstrated that associations between maternal glycaemic markers and offspring adiposity outcomes were mainly detectable at 2 years of age and did not remain significant after correction for multiple testing (**Supplementary Figure 2**). In 2-year-old girls, maternal HOMA-IR and insulin during pregnancy showed positive associations with child fat mass (r = 0.53 and r = 0.52 respectively) and body fat percentage (r = 0.48 and r = 0.46 respectively) (all unadjusted p < 0.01).

DİSCUSSİON

In this longitudinal follow-up of the PONCH cohort, offspring of women with obesity exhibited progressively higher adiposity and anthropometric measures throughout preschool age compared with offspring of women with normal weight, with differences becoming increasingly apparent at later ages. In contrast, offspring of women with GDM showed adiposity profiles largely comparable to those of offspring of women with normal weight. Notably, obesity-related differences were more pronounced in boys, whereas associations with maternal glycaemic markers were primarily observed in girls at younger

ages. Together, these findings indicate that maternal obesity and maternal glycaemic dysregulation are associated with different patterns of adiposity development across early childhood.

From infancy to preschool age: developmental divergence of maternal metabolic influences

The present findings extend our previous observations in the same cohort during infancy, where maternal obesity and GDM influenced offspring body composition already during the first months of life [16]. In that study, infant girls but not boys born to mothers with obesity or GDM exhibited higher body fat percentage, and maternal glycaemic markers were associated with adiposity in girls already at 1 week of age. Similar sex-specific associations between maternal insulin resistance and neonatal adiposity have been reported in other cohorts. Henriksson et al. demonstrated that maternal HOMA-IR during late pregnancy was positively associated with fat mass in infant daughters but not sons, suggesting early sex-specific metabolic sensitivity to maternal insulin resistance [12]. Together, these findings support the concept that intrauterine metabolic exposures contribute to early adiposity programming in a sex-specific manner.

In the current preschool follow-up, similar associations were detectable at 2 years of age, particularly in girls, suggesting that maternal glycaemic influences may persist beyond infancy into early toddlerhood. However, these associations were less pronounced than those observed during infancy and diminished throughout the preschool years. This pattern is broadly consistent with findings from the APPROACH cohort, where maternal glucose homeostasis in women with overweight or obesity during pregnancy was associated with offspring adiposity and metabolic markers at birth, whereas associations were considerably weaker or absent during follow-up at 3 and 5 years of age [20]. Together, these findings suggest that maternal glycaemic influences on adiposity may be most pronounced during fetal life and early childhood, whereas associations become less evident in later childhood.

In contrast, differences between offspring of women with obesity and normal weight became increasingly apparent over time. Longitudinal follow-up during early childhood revealed clearer separation in adiposity and anthropometric trajectories, particularly between 5 and 6 years of age. Children born to mothers with obesity consistently exhibited higher adiposity measures across preschool age, with increasingly greater separation in fat mass and waist circumference during later follow-up, suggesting emerging differences not only in overall adiposity but also in central adiposity distribution.

One of the most notable findings was the apparent shift in maternal metabolic influences across early childhood. Associations between maternal glycaemic markers and offspring adiposity were relatively uncommon and primarily observed at 2 years of age, whereas maternal adiposity markers demonstrated progressively stronger associations involving a larger number of maternal and offspring adiposity variables throughout preschool age, with the strongest observations at 6 years. Previous studies have similarly suggested that maternal insulin resistance and glucose homeostasis may contribute more strongly to fetal and neonatal adiposity, whereas maternal obesity exerts broader and more sustained influences on adiposity trajectories in school-aged children [10,14]. These observations are consistent

with the concept that maternal obesity reflects a broader combination of intrauterine programming, shared genetic susceptibility, familial lifestyle patterns, dietary behaviours, and other postnatal environmental influences that continue to shape adiposity development throughout childhood [7,10]. Early childhood may therefore represent a transitional developmental period during which early metabolic programming increasingly interacts with postnatal lifestyle-related exposures, including dietary patterns, physical activity, sedentary behaviour, and family environment. Unlike intrauterine exposures, these lifestyle factors are potentially modifiable, highlighting early childhood as a critical window for prevention and healthy lifestyle promotion.

The observed sex-specific patterns indicate that the influence of maternal metabolic exposures may vary according to both sex and developmental stage, consistent with experimental and epidemiological evidence suggesting that susceptibility to such exposures differs between sexes and across developmental stages [13,14]. Whereas maternal glycaemic influences appeared most evident in girls during infancy and early childhood, adiposity differences related to maternal obesity became increasingly apparent in boys during preschool age. Together, these findings suggest that sex-specific metabolic vulnerability may evolve throughout development. Around 5–6 years of age, children enter the adiposity rebound period, a developmental stage during which BMI and fat mass trajectories begin to diverge and later obesity risk becomes increasingly apparent [21]. Because girls generally undergo adiposity rebound slightly earlier than boys, the more pronounced obesity-related group differences observed in boys at 5–6 years may partly reflect differences in developmental timing rather than stable sex-specific susceptibility alone. Whether the observed divergence continues into school age and adolescence remains an important question for future longitudinal follow-up.

Cross-generational determinants and future metabolic implications

Although maternal glycaemic markers during pregnancy showed limited associations with preschool adiposity in the present study, detectable associations were observed during infancy and early toddlerhood. This pattern is particularly relevant in light of previous studies demonstrating that maternal glycaemic status during pregnancy may influence offspring glucose–insulin homeostasis independently of offspring adiposity. In the Healthy Start Study, higher maternal HbA1c during pregnancy was associated with higher fasting glucose and lower insulin sensitivity in offspring at 4–7 years of age, whereas these associations were not substantially mediated by adiposity across early childhood [9]. Similarly, follow-up analyses from the HAPO cohort demonstrated associations between maternal glucose levels during pregnancy and later childhood (10–14 years) glucose, independent of offspring adiposity measures [22]. Together, these findings suggest that adiposity may not fully capture the long-term metabolic consequences of maternal glycaemic dysregulation.

The progressively stronger associations observed between maternal adiposity-related markers and offspring adiposity throughout preschool age could reflect shared familial and environmental influences which may become increasingly important in shaping adiposity trajectories throughout childhood [7,9]. Such influences may contribute to the gradually diverging adiposity trajectories observed across

maternal groups during early childhood. Longitudinal follow-up of the PONCH cohort into school age and adolescence will therefore be important to determine whether early adiposity trajectories and metabolic regulation continue to diverge during later developmental stages characterised by increasing insulin resistance, pubertal maturation, and greater behavioural autonomy.

Strengths and limitations

Major strengths of the present study include the longitudinal design, repeated measurements across multiple developmental stages, and the use of direct body composition assessments rather than BMI alone. The repeated use of air-displacement plethysmography in both mothers and children provided a uniquely detailed characterization of adiposity development throughout preschool age, allowing assessment of longitudinal changes in body composition beyond anthropometric measures alone. Furthermore, the well-characterised mother–child cohort, including comparison groups representing distinct prenatal metabolic exposures (normal weight, obesity, and GDM), enabled investigation of how different maternal metabolic environments relate to offspring adiposity development over time.

Several limitations should also be acknowledged. Sample size was moderate, particularly in sex-stratified analyses. The observational design precludes causal inference, and the contribution of unmeasured genetic, socioeconomic, and lifestyle-related factors cannot be excluded. Educational levels differed between groups and may partly reflect broader socioeconomic differences associated with obesity risk. However, detailed lifestyle-related data during childhood were not included in the present analyses. Associations involving maternal glycaemic markers were exploratory and based on unadjusted analyses. Furthermore, the relatively good glycaemic control achieved among women with GDM may partly explain the limited differences observed between the GDM and NW groups.

CONCLUSION

Offspring of women with obesity exhibited progressively greater adiposity throughout preschool age than offspring of women with normal weight, whereas associations between maternal glycaemic markers and offspring adiposity were primarily observed during infancy and early childhood. These findings suggest that maternal obesity may represent a more sustained influence on offspring adiposity development during early childhood. The present findings highlight the importance of maternal metabolic health during pregnancy and may help identify opportunities for early monitoring and preventive strategies in children at increased risk of obesity. Continued longitudinal follow-up will be important to determine whether these differences persist into later childhood and adolescence.

Abbreviations

HbA1c, glycated haemoglobin

HOMA-IR, Homeostatic Model Assessment of Insulin Resistance

GDM, gestational diabetes mellitus

NW, normal weight

OB, obesity

OGTT, oral glucose tolerance test

PONCH, Pregnancy Obesity Nutrition Child Health

Declarations

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Author Contributions:

S.Å. contributed to data collection, was responsible for data processing and curation, performed the statistical analyses and data visualization, and drafted the manuscript. U.A.H. contributed to study design, methodology, funding acquisition, data collection, interpretation of the results, and critical revision of the manuscript. E.K. contributed to data collection, interpretation of the results, and critical revision of the manuscript. A.H. conceived and designed the study, contributed to methodology and funding acquisition, interpreted the results, and critically revised the manuscript. All authors contributed to interpretation of the data, critically revised the manuscript, and approved the final version for publication.

A.H. is the guarantor of this work and, as such, had full access to all data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Data Availability Statement:

The data that support the findings of this study are not publicly available. Data may be made available from the corresponding author upon reasonable request and subject to applicable ethical and legal restrictions.

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Figures

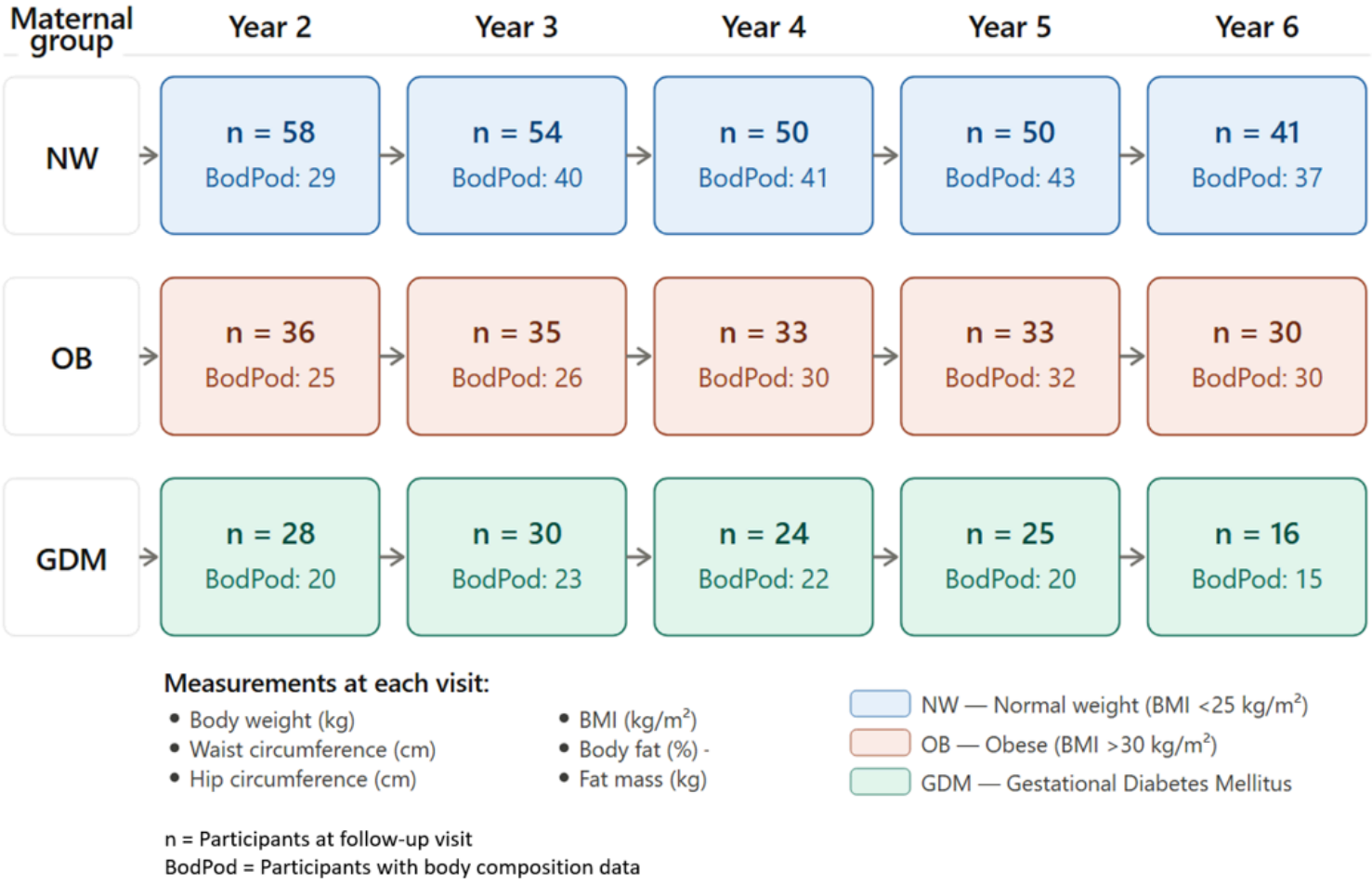


Figure 1

Study design and participant flow through the longitudinal follow-up.

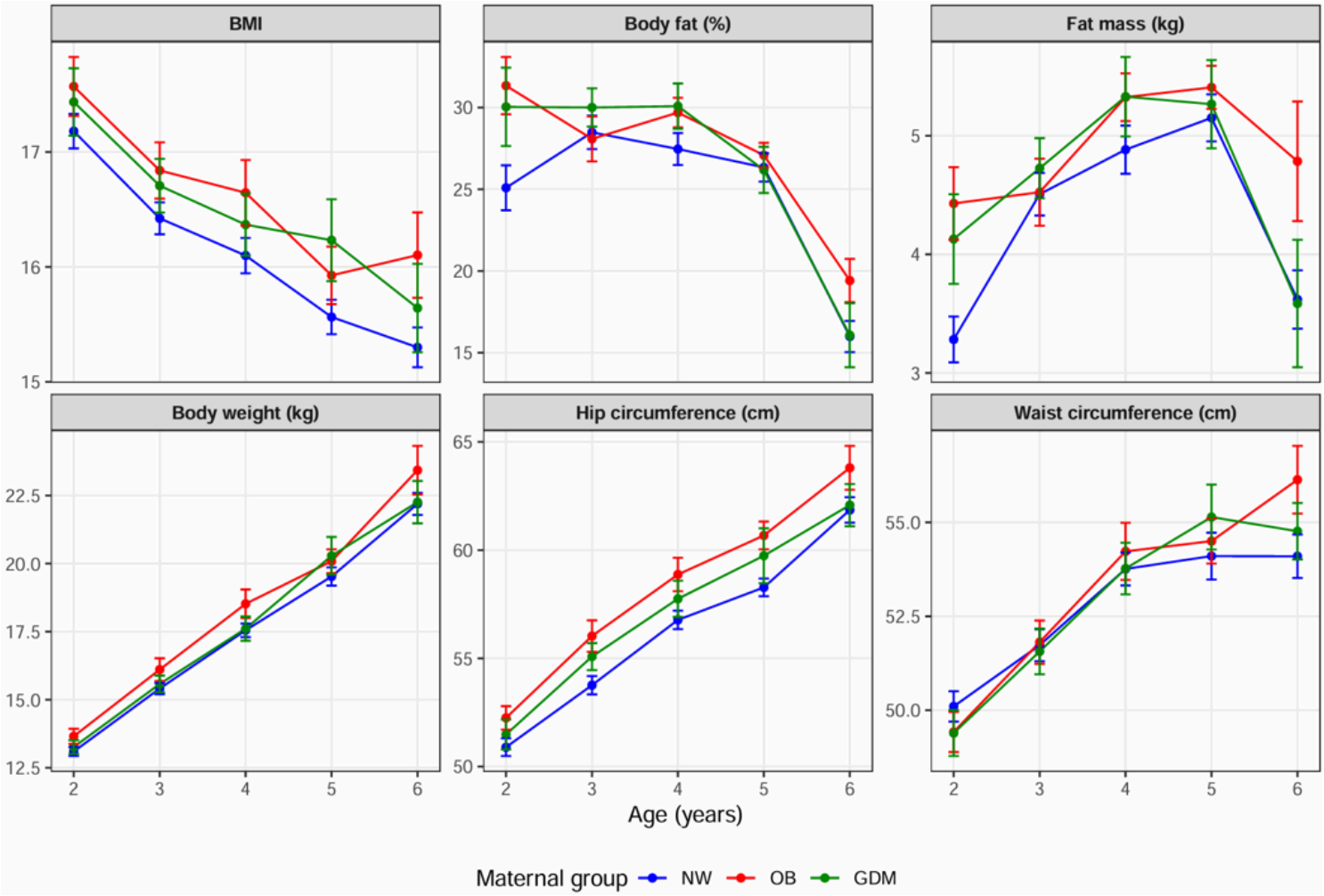


Figure 2

Descriptive age-specific trajectories of child adiposity and anthropometric outcomes from ages 2 to 6 years according to maternal metabolic status during pregnancy: normal weight (NW, n=68), obesity (OB, n=41), and gestational diabetes mellitus (GDM, n=36). Data are presented as observed mean ± SD at each age.

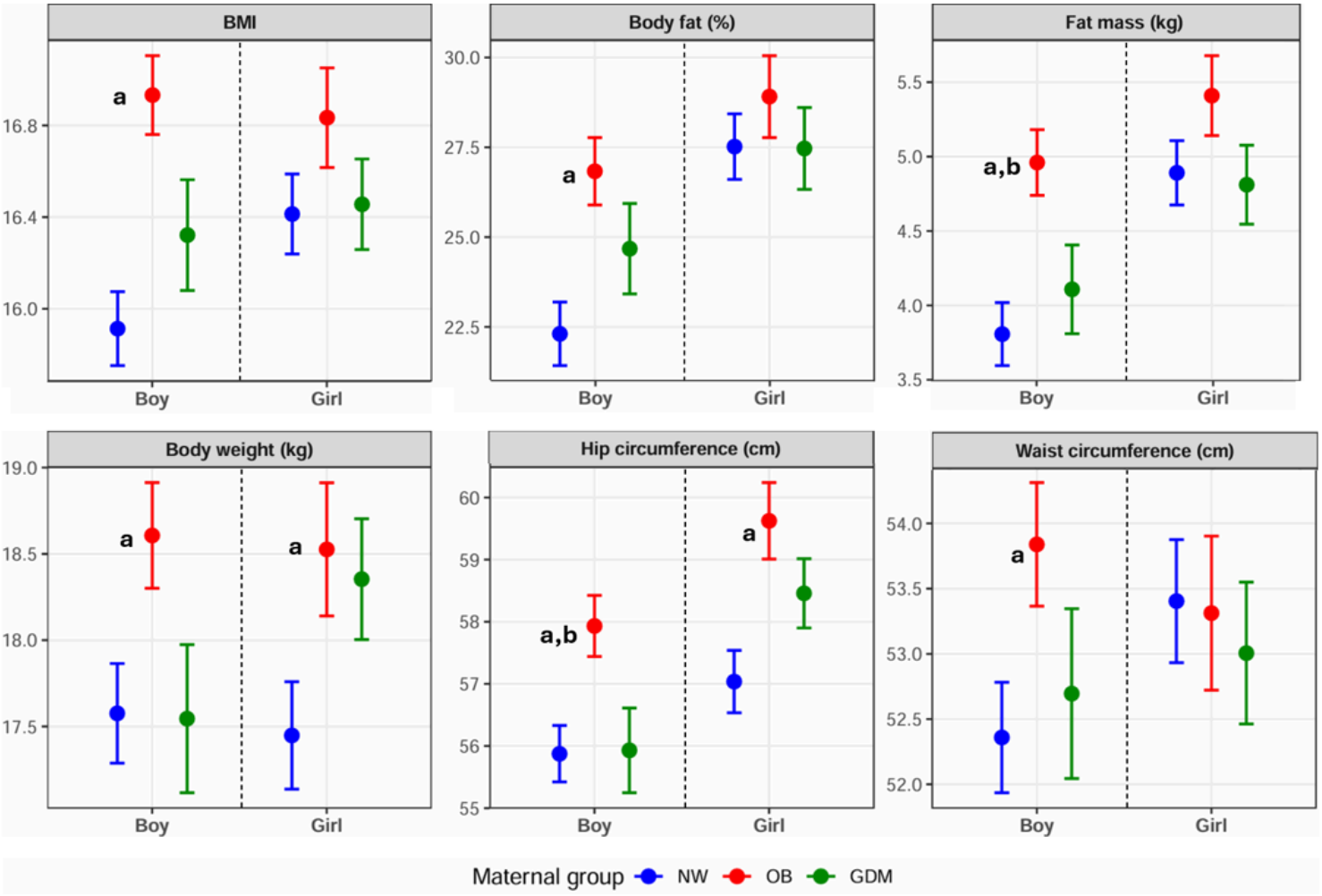


Figure 3

Sex-stratified estimated marginal means for child adiposity and anthropometric outcomes across ages 2–6 years according to maternal metabolic status during pregnancy: normal weight (NW; 30 girls, 38 boys), obesity (OB; 17 girls, 24 boys), and gestational diabetes mellitus (GDM; 20 girls, 16 boys). Estimates were derived from linear mixed-effects models including maternal group, child sex, their interaction, and child age as fixed effects, with child ID included as a random effect. Data are presented as estimated marginal means ± SEM. Pairwise comparisons between maternal groups within each sex were performed using Tukey-adjusted contrasts. Letters indicate statistically significant pairwise differences: a, different from NW; b, different from GDM.

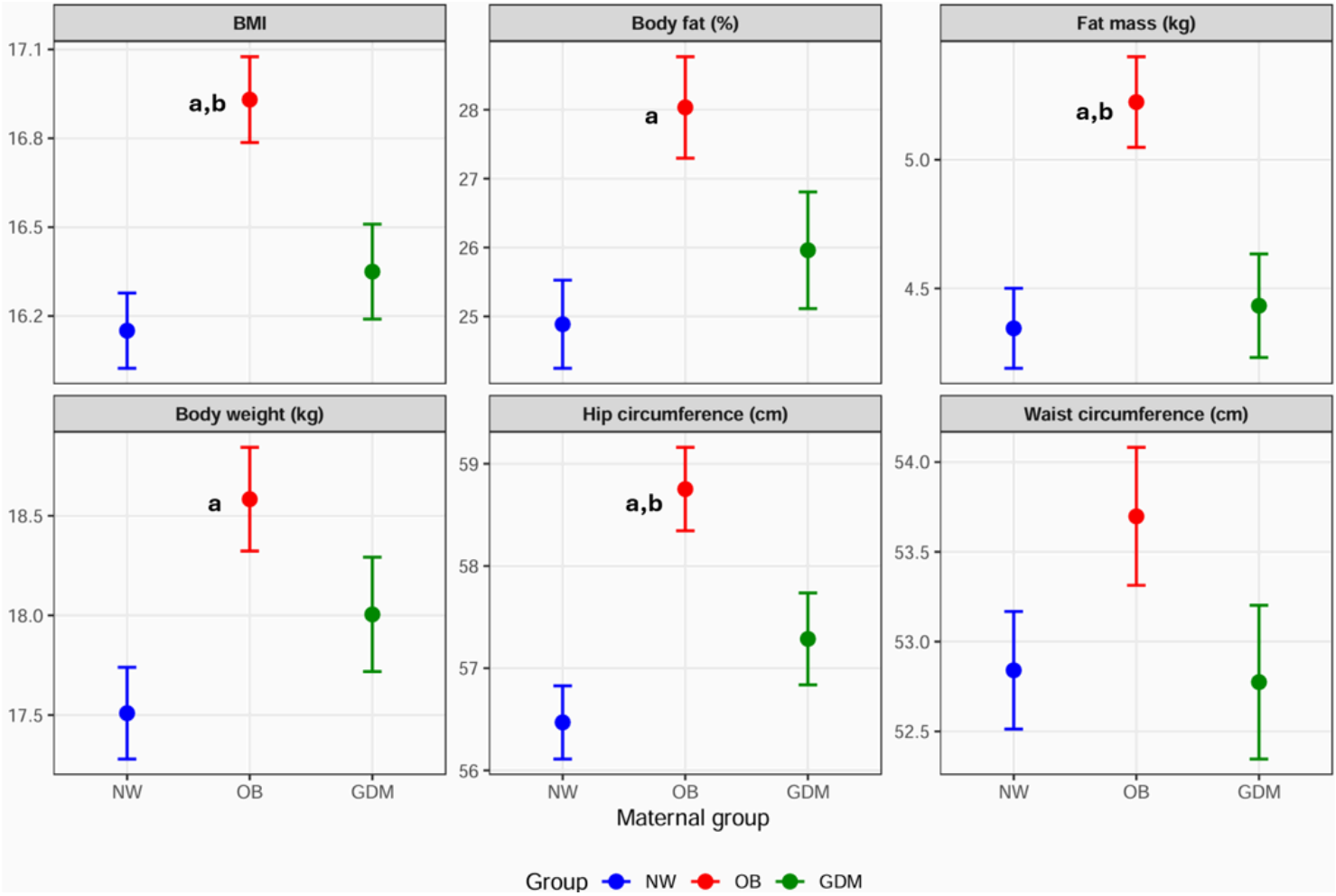


Figure 4

Estimated marginal means for child adiposity and anthropometric outcomes across ages 2–6 years according to maternal metabolic status during pregnancy: normal weight (NW, n=68), obesity (OB, n=41), and gestational diabetes mellitus (GDM, n=36).Estimates were derived from linear mixed-effects models including maternal group, child age, and child sex as fixed effects, with child ID included as a random effect. Data are presented as estimated marginal means ± SEM. Pairwise comparisons between maternal groups were performed using Tukey-adjusted contrasts. Letters indicate statistically significant pairwise differences: a, different from NW; b, different from GDM

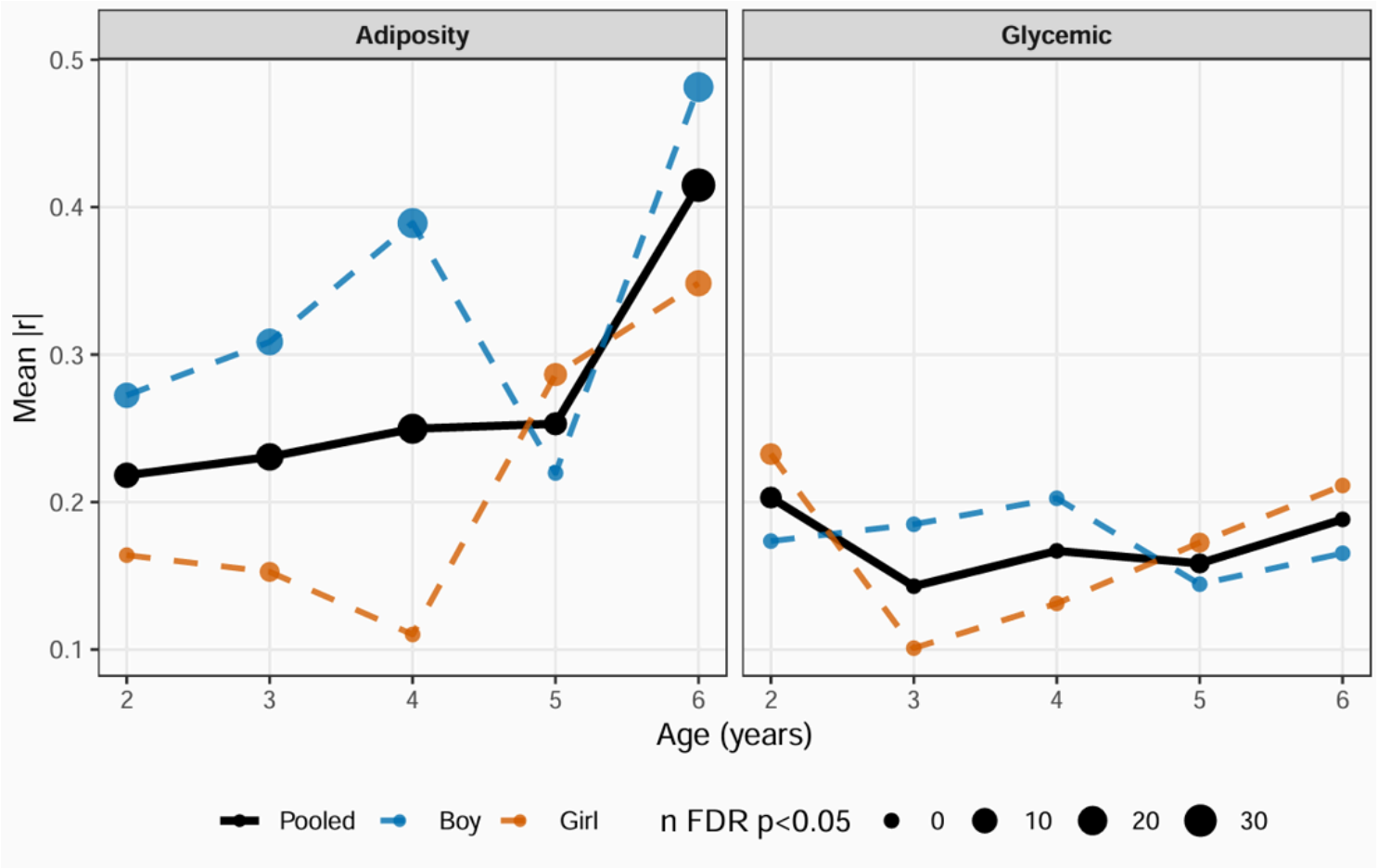


Figure 5

Mean absolute correlation coefficients (|r|) and number of statistically significant associations between maternal metabolic markers and child adiposity and anthropometric outcomes at ages 2–6 years. Maternal adiposity markers include body weight and BMI in early pregnancy (trimester 1), and body fat percentage and total fat mass in late pregnancy (trimester 3). Maternal glycaemic markers include fasting glucose, insulin, HbA1c, and HOMA-IR. Child outcomes include body weight, BMI, body fat percentage, fat mass, and hip and waist circumference. Solid lines represent pooled estimates for the full cohort, and dashed lines represent sex-stratified estimates (boys and girls). Point size reflects the number of statistically significant associations observed at each age, including associations remaining significant after false discovery rate (FDR) correction.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- PONCHMaternalObesityChildAdipositySupplementaryMaterial.docx