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Clinical Profile and Predictors of Childhood Overweight Following In-Utero Exposure to Gestational Diabetes Mellitus: A Retrospective Cohort Study in Guyana

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Abstract *Children exposed to gestational diabetes mellitus (GDM) in utero are at increased long-term risk of obesity and dysglycaemia, yet Caribbean data remain scarce. This retrospective cohort analysis characterised the clinical profile and examined predictors of abnormal body mass index (BMI) among GDM-exposed children in Guyana. Perinatal data were extracted from the records of 33 mothers diagnosed with GDM during the index pregnancy (January–June 2017); informed consent was then obtained and structured maternal telephone interviews ascertained current child parameters. Anthropometric status was classified using the United States Centers for Disease Control and Prevention (CDC) BMI-for-age reference. Associations with overweight/obesity were assessed using Fisher's exact test (with unadjusted odds ratios [OR] and 95% confidence intervals [CI]), the Mann–Whitney U test and the Spearman rank correlation. Overall, 13 children (39.4%, 95% CI 24.7–56.3) were overweight or obese and 5 (15.2%) were obese, while 8 (24.2%) had been macrosomic at birth. No factor was a statistically significant independent predictor of overweight/obesity. The strongest, non-significant signals were a higher number of daily meals (OR 5.62, 95% CI 0.89–35.39; $p = 0.084$) and lower birth weight (OR 3.43, 95% CI 0.73–16.09; $p = 0.139$); girls had a higher median BMI than boys (17.9 vs 16.6 kg/m²; $p = 0.169$). Birth weight was not positively correlated with current BMI ($\rho = -0.18$; $p = 0.33$). This exploratory, underpowered analysis supports routine longitudinal surveillance of GDM-exposed children and adequately powered comparative studies to inform maternal and child health policy in Guyana.*

Keywords *Body Mass Index, Childhood Obesity, Developmental Origins of Health and Disease, Guyana, Gestational Diabetes Mellitus, Retrospective Cohort.*

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Introduction

Gestational diabetes mellitus (GDM), defined as hyperglycaemia first recognised during pregnancy, is among the most common medical complications of pregnancy, and its frequency is rising worldwide alongside obesity and type 2 diabetes [4, 5]. The landmark Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study established a continuous association between maternal glycaemia and adverse perinatal outcomes [2, 20], informing the diagnostic thresholds subsequently recommended by the International Association of Diabetes and Pregnancy Study Groups (IADPSG) and the World Health Organization (WHO) [1, 3].

Beyond the perinatal period, the developmental origins of health and disease (DOHaD) framework holds that the intrauterine environment programmes long-term metabolic risk in the offspring [6, 24]. Considerable evidence links in-utero exposure to GDM with later childhood overweight, obesity, adiposity and impaired glucose metabolism [7-10], adding to the global burden of childhood obesity [23]. Studies of discordant sibships and longitudinal cohorts such as EPOCH and the HAPO Follow-up Study (HAPO FUS) support an at-least-partly intrauterine contribution that is not fully explained by maternal body mass index (BMI) [11-14]. Mothers with GDM also carry a high risk of progression to type 2 diabetes, and their offspring show elevated rates of dysglycaemia, compounding the intergenerational cycle of cardiometabolic disease [15, 22].

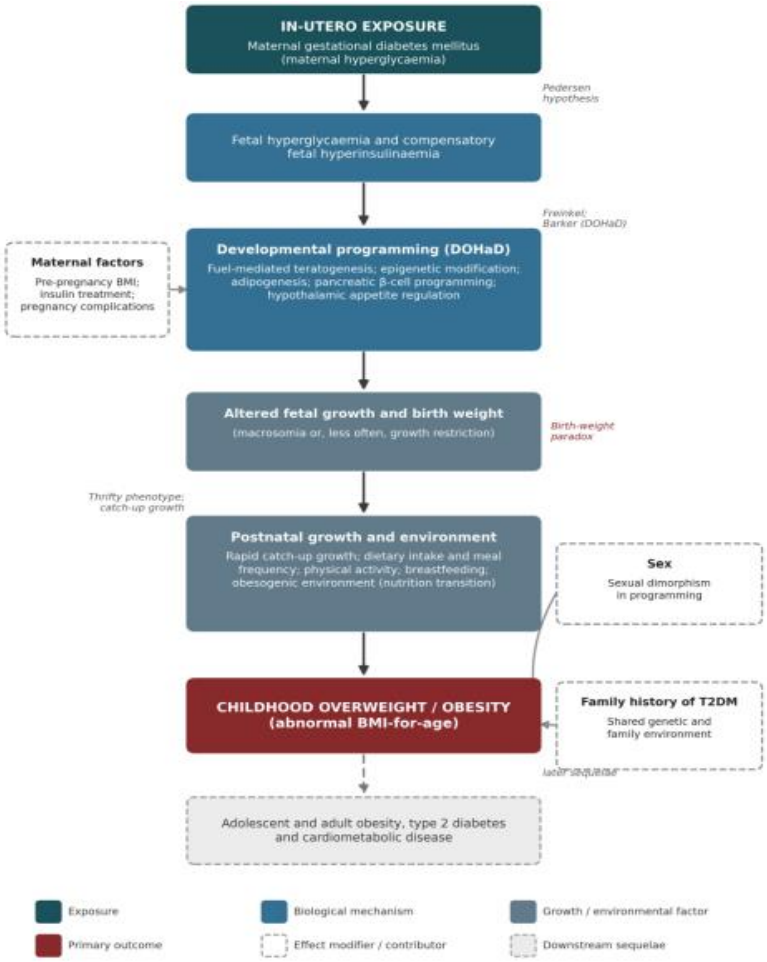

Figure 1. Conceptual Framework Illustrating the Hypothesized Pathways Linking In-utero Exposure to Gestational Diabetes Mellitus and Childhood Overweight/Obesity

Most evidence, however, derives from high-income or large multinational cohorts; data describing GDM-exposed children in the Caribbean, and in Guyana specifically, are sparse. Characterising the clinical profile of these children and identifying early, potentially modifiable predictors of abnormal weight status is a prerequisite for targeted follow-up and prevention [16, 21]. The present study therefore aimed to (i) describe the clinical, perinatal and lifestyle profile of a cohort of GDM-exposed children in Guyana, and (ii) examine perinatal, lifestyle, maternal and family-history predictors of abnormal (overweight/obese) BMI category. A conceptual framework summarising the hypothesised pathways is shown in Figure 1.

To our knowledge, this is the first retrospective cohort study from Guyana and one of the very few studies from the Caribbean evaluating childhood anthropometric outcomes following in-utero exposure to gestational diabetes mellitus. The findings provide locally generated evidence to inform long-term follow-up strategies for mothers with GDM and their children.

Materials and Methods

Study Design and Setting

This was a retrospective cohort analysis conducted within the public health service in Guyana, with participants managed at the Georgetown Public Hospital Corporation (GPHC). The cohort was assembled in two phases. In the first phase, between January and June 2017, data were extracted from the records of mothers diagnosed with GDM during the index pregnancy, from which 33 GDM mother–child pairs were selected. In the second phase, informed consent was subsequently obtained from the parents (mothers) of the index children, and structured telephone interviews were then conducted with the mothers to ascertain key parameters concerning the child, including current growth and anthropometry, infant feeding, diet, lifestyle, physical activity, sleep and family history. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidance for cohort studies.

Study Population and Sampling

The source population comprised all women who gave birth at the GPHC between 1 January and 30 June 2017. Eligible participants were those with a diagnosis of GDM confirmed in the hospital records of the index pregnancy, and the study population therefore comprised the children born to these mothers. GDM had been diagnosed in accordance with the GPHC Guidelines for the Diagnosis and Treatment of Diabetes in Pregnancy (updated November 2013): a fasting blood glucose ≥ 92 mg/dL (5.1 mmol/L) or, on a 2-hour oral glucose tolerance test, any one of a fasting value ≥ 92 mg/dL (5.1 mmol/L), a 1-hour value ≥ 180 mg/dL (10.0 mmol/L), or a 2-hour value ≥ 155 mg/dL (8.6 mmol/L) [28]. Of the 42 women with GDM who met the inclusion criteria during this period, complete records permitting full case extraction were available for 33 (33/42; 78.6%), and these mother–child pairs formed the study cohort; sampling was therefore by total (consecutive) enumeration of eligible records rather than probability sampling.

Inclusion and Exclusion Criteria

Children were eligible for inclusion if their mother had a documented diagnosis of GDM in the index pregnancy during January–June 2017, if birth and perinatal records were available, and if the mother provided informed consent to participate in the follow-up telephone interview. Children were excluded if the mother had pre-gestational (type 1 or type 2) diabetes diagnosed before the index pregnancy, if core birth records were missing, or if the mother could not be contacted or declined to provide consent. The resulting participant flow is summarised in Figure 2.

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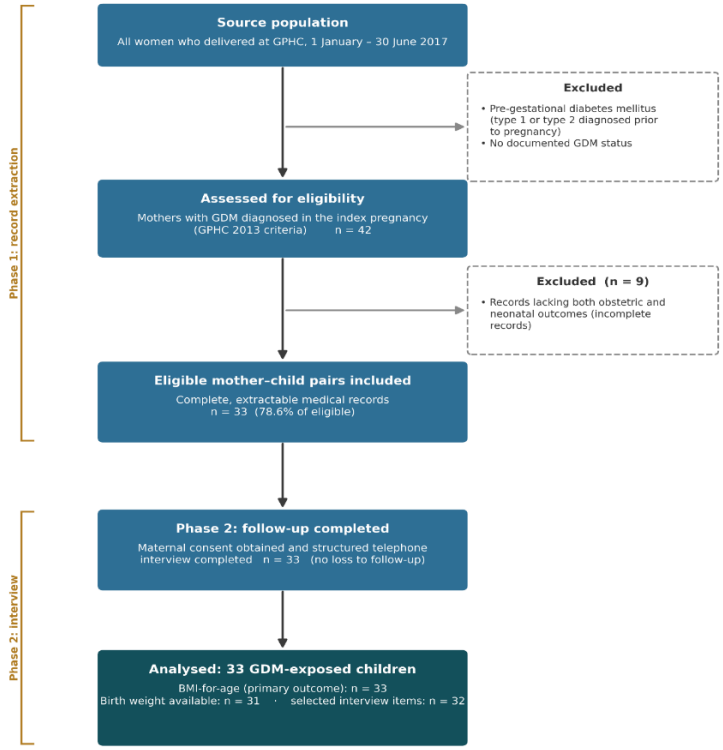

Figure 2. Flow of Participants through the Study, from the Source Population to the Analysed Cohort

Data Collection and Variables

Perinatal and birth variables (birth weight, gestational age, five-minute Apgar score and complications at birth) were abstracted from maternal and child clinical records during the 2017 extraction phase. Current child variables were obtained through the structured maternal telephone interviews and supplemented by available clinic measurements; these comprised anthropometry (weight and height), infant-feeding history (ever breastfed, exclusive and total breastfeeding duration, age at introduction of formula and solids), dietary patterns (meals and snacks per day, and frequency of sugary drinks and other foods), physical activity, screen time, sleep, and family history of diabetes and hypertension. Maternal variables (GDM treatment, pregnancy complications and height) were recorded from the clinical records. Current weight and height were, for most children, reported by the mother during the telephone interview rather than measured directly by the study team, with clinic measurements used where available.

Variable Definitions

The exposure was in-utero GDM (all participants exposed). The primary outcome was an abnormal high BMI category (overweight or obesity) at assessment. BMI (kg/m²) was calculated from measured weight and height and classified for age and sex using the CDC BMI-for-age reference: overweight, 85th–<95th percentile; obesity, ≥95th percentile; and severe obesity, ≥120% of the 95th percentile [17, 18]. Macrosomia was defined as birth weight ≥4000 g, low birth weight as <2500 g, and preterm birth as delivery before 37 completed weeks. Exclusive breastfeeding referred to breast milk only, without other liquids or solids. Family history was defined as a first-degree relative with the condition.

Statistical Analysis

Continuous variables were screened for normality (Shapiro–Wilk test) and summarised as mean ± standard deviation (SD) or median

(interquartile range, IQR) as appropriate; categorical variables are presented as counts and percentages. The 95% CI for the prevalence of overweight/obesity was estimated using the Wilson score method. Associations between candidate exposures and overweight/obesity were examined using Fisher's exact test for categorical variables, with unadjusted odds ratios (OR) and 95% CIs (Haldane–Anscombe correction where a zero cell was present), and the Mann–Whitney U test for continuous variables. Monotonic associations between continuous exposures and current BMI were assessed using the Spearman rank correlation coefficient (ρ). Given the small sample and limited number of outcome events, multivariable modelling was not undertaken; analyses were unadjusted, exploratory and not corrected for multiple comparisons, and a two-sided p-value < 0.05 was considered statistically significant. Analyses were performed in Python (version 3.12; pandas, SciPy and statsmodels packages). Missing data were handled by available-case analysis, with each estimate based on all participants having non-missing data for the relevant variables and the denominator reported throughout; no imputation was undertaken. The parents accompanied the children to the nearest health center where anthropometric measurements were performed by the clinical staff of the corresponding health facility. The anthropometric measurements were then reported via telephone interview in a follow-up interview to specifically collect this data.

Results

Cohort and Participant Flow

Thirty-three GDM-exposed children were included. The mean age was 9.2 ± 0.1 years, and 18 (54.5%) were boys. The cohort was predominantly of African (20, 60.6%) and East Indian (7, 21.2%) ethnicity. Data on sex were complete for all 33 children; a small number of interview items (notably screen time) were incomplete, and denominators are reported for each variable. The flow of participants through the study is shown in Figure 2, and demographic and birth characteristics in Table 1.

Table 1. Demographic and Birth Characteristics of the Cohort (N = 33)

Characteristic	**Value**
Child age, years — mean ± SD	9.16 ± 0.13
Sex, n (%)	
Male	18 (54.5)
Female	15 (45.5)
Ethnicity, n (%)	
East Indian	7 (21.2)
African	20 (60.6)
Mixed	1 (3.0)
Amerindian	1 (3.0)
Other	4 (12.1)
Birth weight, g — mean ± SD	3385.0 ± 678.84
Macrosomia (≥4000 g), n (%)	8 (24.2)
Low birth weight (<2500 g), n (%)	2 (6.1)
Gestational age, weeks — median (IQR)	38.86 (37.64–39.29)
Preterm (<37 weeks), n (%)	6 (18.2)
5-minute APGAR — median (IQR)	10.0 (10.0–10.0)

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Clinical Profile: Anthropometric Status, BMI Classification and Percentiles

At a median age of nine years, the median BMI was 17.1 (IQR 15.4–20.1) kg/m² and the median BMI-for-age percentile was 67.0 (IQR 31.0–90.0). Sixteen children (48.5%) were of healthy weight and four (12.1%) were underweight. In total, 13 children (39.4%, 95% CI 24.7–56.3) were overweight or obese (8 [24.2%] overweight, 3 [9.1%] with obesity and 2 [6.1%] with severe obesity), and 5 (15.2%, 95% CI 6.7–30.9) met the criterion for obesity. At birth, 8 children (24.2%) had been macrosomic. The distribution of BMI categories is shown in Figure 3, the distribution of birth weight in Figure 4, and full anthropometric findings, including percentiles, in Table 2.

Table 2. Anthropometric Status, BMI Classification and Percentiles (N = 33)

Characteristic	Value
Current weight, kg — median (IQR)	30.0 (27.3–35.7)
Current height, cm — mean ± SD	131.65 ± 6.21
BMI, kg/m² — median (IQR)	17.1 (15.4–20.1)
BMI-for-age percentile — median (IQR)	67.0 (31.0–90.0)
BMI-for-age category, n (%)	
Underweight	4 (12.1)
Healthy weight	16 (48.5)
Overweight	8 (24.2)
Obesity	3 (9.1)
Severe obesity	2 (6.1)
Overweight or obese, n (% [95% CI])	13 (39.4 [24.7–56.3])
Obese (incl. severe), n (% [95% CI])	5 (15.2 [6.7–30.9])

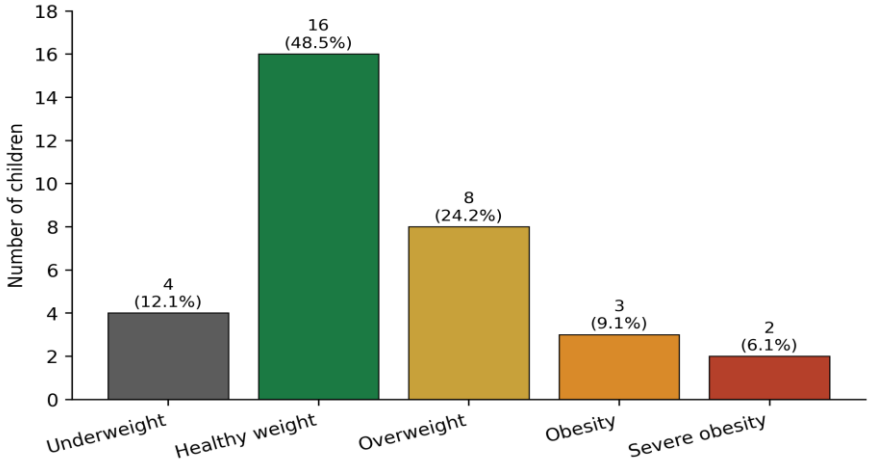

Figure 3. Distribution of BMI-for-Age Categories among the 33 GDM-exposed Children

Clinical Profile: Breastfeeding, Infant Feeding, Diet, Lifestyle, Physical Activity, Sleep and Maternal/Family Characteristics

Almost all children (32, 97.0%) had been breastfed, for a median total duration of 15 (IQR 12–19) months and a median exclusive duration of 5 (IQR 3–6) months. Reported diet and activity patterns were broadly favourable (median 3 meals/day, high reported physical activity, median sleep 10 hours/night, median screen time 4 hours/day). Nearly all mothers had received insulin during the index pregnancy (32, 97.0%), 16 (48.5%) had

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documented gestational or chronic hypertension, and a family history of type 2 diabetes was reported for 17 children (51.5%). Feeding, lifestyle, maternal and family characteristics are presented in Tables 3 and 4.

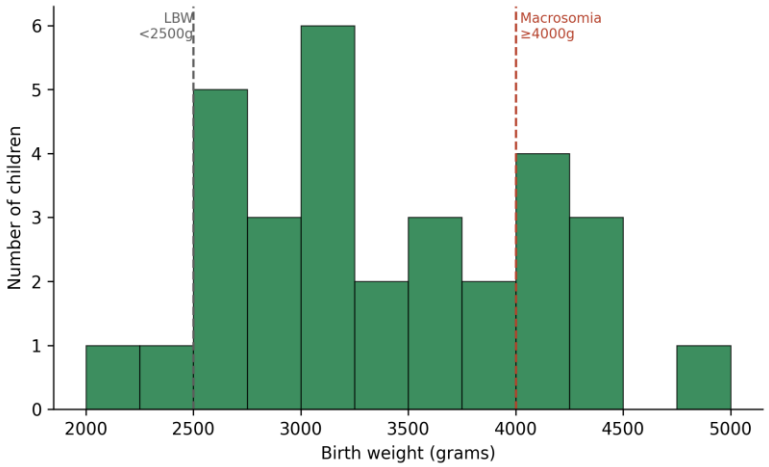

Figure 4. Distribution of Birth Weight (n = 31); Dashed Lines Mark the Low-birth-Weight (<2,500 g) and Macrosomia (≥4,000 g) Thresholds

Table 3. Breastfeeding, Infant Feeding, Diet, Lifestyle, Physical Activity and Sleep (N = 33)

Characteristic	Value
Ever breastfed, n (%)	32 (97.0)
Exclusive breastfeeding, months — median (IQR)	5.0 (3.0–6.0)
Total breastfeeding, months — median (IQR)	15.0 (12.0–19.0)
Age formula introduced, months — median (IQR)	5.0 (3.0–6.0)
Age solids introduced, months — median (IQR)	7.0 (7.0–8.0)
Meals per day — median (IQR)	3.0 (3.0–3.0)
Snacks per day — median (IQR)	3.5 (3.0–4.0)
Eats breakfast daily, n (%)	32 (97.0)
Daily fresh fruit, n (%)	29 (87.9)
Daily fresh vegetables, n (%)	19 (57.6)
Sugary drinks daily, n (%)	11 (33.3)
Fast food weekly+, n (%)	13 (39.4)
Sweets weekly+, n (%)	6 (18.2)
Physical activity, days/week — median (IQR)	7.0 (5.75–7.0)
Physical activity, hours/day — median (IQR)	3.5 (3.0–4.0)
Screen time, hours/day — median (IQR)	4.0 (3.0–4.12)
Sleep, hours/night — median (IQR)	10.0 (9.5–10.0)

Table 4. Maternal and Family Characteristics (N = 33)

Characteristic	Value
Maternal GDM treatment, n (%)	
Insulin (±other)	32 (97.0)
Metformin only	1 (3.0)
Insulin exposure in utero, n (%)	32 (97.0)
Maternal height, cm — mean ± SD	160.08 ± 7.17

Any maternal pregnancy complication, n (%)	28 (84.8)
Gestational/chronic hypertension, n (%)	16 (48.5)
Family history of type 2 diabetes, n (%)	17 (51.5)
Family history of hypertension, n (%)	10 (30.3)
Mother's education ≥ secondary, n (%)	31 (93.9)

Bivariate Associations and Predictors of Overweight/Obesity

Bivariate associations between candidate exposures and overweight/obesity are summarised in Table 5, and Spearman correlations between continuous exposures and current BMI in Table 6. No exposure was significantly associated with overweight/obesity. The unadjusted odds ratios with 95% confidence intervals for the categorical predictors are presented in Table 7 and displayed in Figure 6. All confidence intervals were wide and included the null, consistent with limited statistical power. The strongest signals, none reaching significance, were a higher number of daily meals (≥4 meals/day: OR 5.62, 95% CI 0.89–35.39; p = 0.084) and lower birth weight (<3000 g: OR 3.43, 95% CI 0.73–16.09; p = 0.139), each associated with higher odds of overweight/obesity. The presence of any maternal pregnancy complication was associated with lower odds (OR 0.12, 95% CI 0.01–1.22; p = 0.066). Female sex and a family history of type 2 diabetes were each associated with approximately two-fold higher odds, again non-significant.

Table 5. Bivariate Associations between Candidate Exposures and Overweight/Obesity (Overweight/Obese vs not)

Exposure	**Test**	**p-value**
Sex (female vs male)	Fisher exact	0.493
Macrosomia (yes vs no)	Fisher exact	0.431
Preterm (yes vs no)	Fisher exact	0.364
Ever breastfed	Fisher exact	1.000
Family history T2DM	Fisher exact	0.481
Family history HTN	Fisher exact	0.461
Maternal hypertension	Fisher exact	0.481
Any maternal complication	Fisher exact	0.066
Participates in organized sport	Fisher exact	1.000
Birth weight †	Mann–Whitney U	0.060
Gestational age †	Mann–Whitney U	0.845
Exclusive breastfeeding †	Mann–Whitney U	0.635
Total breastfeeding †	Mann–Whitney U	0.949
Screen time †	Mann–Whitney U	0.238
Physical activity (h/day) †	Mann–Whitney U	0.284
Sleep duration †	Mann–Whitney U	0.162
Maternal height †	Mann–Whitney U	0.205

p-values from Fisher's exact test (categorical exposures) or the Mann–Whitney U test (continuous exposures). Directional effect sizes (unadjusted odds ratios with 95% confidence intervals) for the categorical predictors are presented in Table 7.

Table 6. Spearman Rank Correlations between Continuous Exposures and Current BMI

Exposure	n	Spearman ρ	p-value
Birth weight (g)	31	-0.18	0.329
Gestational age (weeks)	32	0.12	0.518
Exclusive breastfeeding (months)	29	-0.14	0.455
Total breastfeeding (months)	30	-0.24	0.208
Meals per day	33	0.30	0.085
Snacks per day	30	0.30	0.106
Physical activity (days/week)	32	0.16	0.389
Screen time (hours/day)	32	-0.20	0.260
Sleep (hours/night)	32	0.14	0.459
Maternal height (cm)	28	-0.20	0.315

Table 7. Unadjusted Associations between Candidate Predictors and Overweight/Obesity (n = 33; 13 Events)

Predictor (index vs reference)	Index, n/N (%)	Reference, n/N (%)	OR (95% CI)	p
Female sex	7/15 (46.7%)	6/18 (33.3%)	1.75 (0.43–7.17)	0.493
Macrosomia at birth (≥4000 g)	2/8 (25%)	11/25 (44%)	0.42 (0.07–2.53)	0.431
Birth weight <3000 g	6/10 (60%)	7/23 (30.4%)	3.43 (0.73–16.09)	0.139
Preterm birth (<37 wks)	1/6 (16.7%)	12/27 (44.4%)	0.25 (0.03–2.44)	0.364
Not breastfed	0/1 (0%)	13/32 (40.6%)	0.48 (0.02–12.73)	1
Family history of type 2 diabetes	8/17 (47.1%)	5/16 (31.2%)	1.96 (0.47–8.11)	0.481
Family history of hypertension	5/10 (50%)	8/23 (34.8%)	1.88 (0.42–8.47)	0.461
Maternal hypertension in pregnancy	5/16 (31.2%)	8/17 (47.1%)	0.51 (0.12–2.12)	0.481
Any maternal pregnancy complication	9/28 (32.1%)	4/5 (80%)	0.12 (0.01–1.22)	0.066
Maternal insulin treatment	13/32 (40.6%)	0/1 (0%)	2.08 (0.08–54.92)	1
No participation in organized sport	0/1 (0%)	13/32 (40.6%)	0.48 (0.02–12.73)	1
Daily sugary-drink consumption	2/11 (18.2%)	11/22 (50%)	0.22 (0.04–1.27)	0.132
Screen time ≥4 h/day	7/19 (36.8%)	6/14 (42.9%)	0.78 (0.19–3.19)	1
≥4 meals/day	5/7 (71.4%)	8/26 (30.8%)	5.62 (0.89–35.39)	0.084

OR, odds ratio; CI, confidence interval. Odds ratios are unadjusted; the Haldane–Anscombe correction (+0.5) was applied where a zero cell was present. p-values from Fisher's exact test.

The comparison of continuous characteristics by weight status was consistent with these findings (Table 8). Children who were overweight or obese had a lower median birth weight than their non-overweight peers (3,120 vs 3,550 g; p = 0.060) and a marginally higher number of daily meals (p = 0.052). Birth weight was not positively correlated with current BMI (ρ = −0.18; p = 0.33; Figure 5). The non-significant inverse association between daily sugary-drink consumption and overweight (OR 0.22) is most plausibly attributable to small-sample noise or reverse causation and is not interpreted further.

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Table 8. Continuous Characteristics by Weight Status, Median (IQR)

Variable	Overweight/obese (n≈13)	Not overweight (n≈20)	p
Birth weight (g)	3120 (2700–3200)	3550 (3095–4105)	0.060
Gestational age (weeks)	38.9 (38.3–39.3)	38.7 (36.8–39.3)	0.845
Meals per day	3 (3–4)	3 (3–3)	0.052
Total breastfeeding (months)	15 (14–18)	15 (12–23)	0.949
Sleep (hours/night)	10.0 (9.7–10.0)	10.0 (9.5–10.0)	0.162
Maternal height (cm)	159 (156–163)	164 (155–168)	0.205

p-values from the Mann–Whitney U test. Group sizes vary by item owing to missing data.

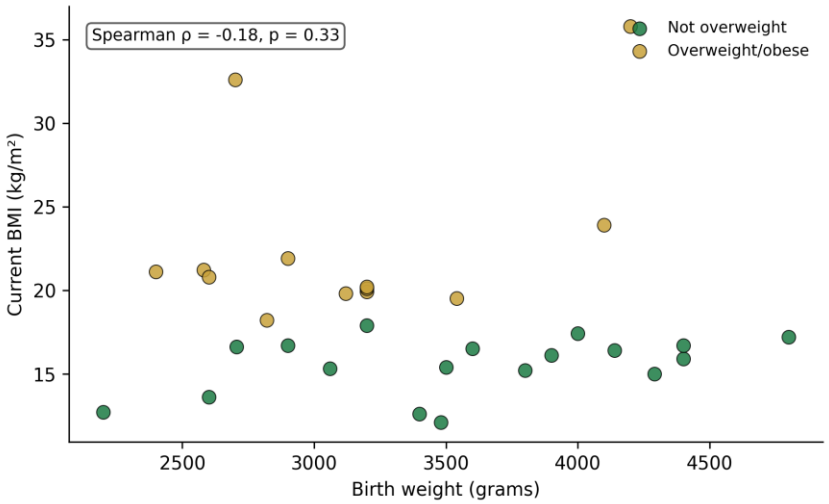

Figure 5. Birth Weight Versus Current BMI (n = 31). The Spearman Correlation was Weak and Non-significant

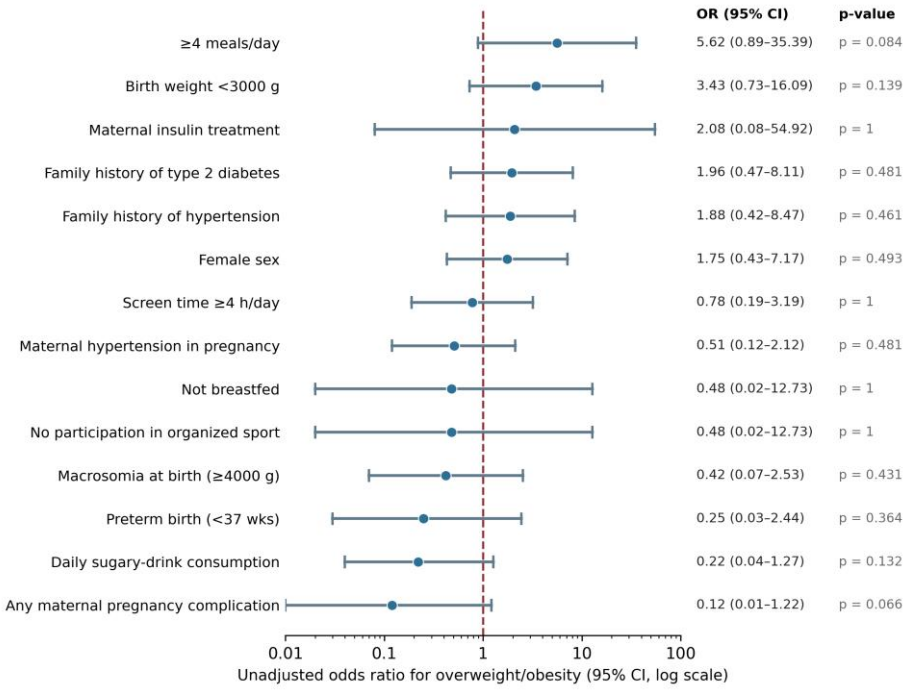

Figure 6. Forest Plot of Unadjusted Odds Ratios (95% Confidence Intervals) for Overweight/Obesity by Candidate Predictor; the Vertical Line Marks the Null (OR = 1)

Sex-stratified Anthropometric Outcomes

Anthropometric outcomes by sex are presented in Table 9 and Figure 7. Girls had a higher median BMI (17.9 vs 16.6 kg/m²; p = 0.169) and BMI-for-age percentile (75.0th vs 57.0th; p = 0.205) than boys, and a higher prevalence of overweight/obesity (7/15, 46.7% vs 6/18, 33.3%; OR 1.75, 95% CI 0.43–7.17; p = 0.49). Birth weight did not differ by sex (median 3,340 vs 3,200 g; p = 0.74). None of these sex differences reached statistical significance, but the direction — a tendency towards greater adiposity among girls — is consistent with reports of sex-specific intrauterine programming considered in the Discussion.

Table 9. Sex-Stratified Anthropometric and Birth Outcomes

Measure	Boys (n = 18)	Girls (n = 15)	p
Current BMI (kg/m²), median (IQR)	16.6 (15.3–19.4)	17.9 (16.5–21.6)	0.169
BMI-for-age percentile, median (IQR)	57.0 (27.3–87.5)	75.0 (53.0–94.0)	0.205
Overweight/obese, n (%)	6 (33.3)	7 (46.7)	0.493
Macrosomia at birth, n (%)	3 (16.7)	5 (33.3)	—
Birth weight (g), median (IQR)	3200 (3060–3800)	3340 (2701–4075)	0.736

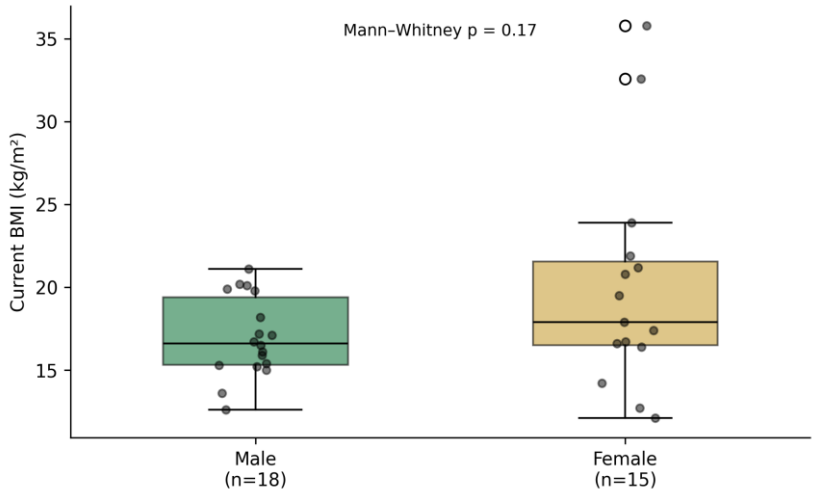

Figure 7. Current BMI by Sex. Boxes show Median and Interquartile Range; Individual Values are Overlaid

Discussion

In this retrospective cohort of GDM-exposed children in Guyana, two in five children (39.4%) were already overweight or obese by a median age of nine years, and one in seven (15.2%) met the criterion for obesity, including 6.1% with severe obesity. Nearly a quarter (24.2%) had been macrosomic at birth. Although no factor emerged as a statistically significant independent predictor of abnormal BMI — unsurprising given the small sample and wide confidence intervals — the most consistent signals pointed to a higher number of daily meals and, paradoxically, lower birth weight as correlates of later overweight, with girls tending towards greater adiposity than boys. Overall, the cohort already carried a considerable cardiometabolic burden in middle childhood. Because the sample was small, these results are better understood as a coherent clinical pattern than as precise risk estimates, and they are consistent with the recognised effects of in-utero hyperglycaemic exposure.

The 39.4% prevalence of overweight or obesity observed here is consistent with, and lies towards the upper end of, the evidence linking in-utero GDM exposure to later childhood adiposity. The systematic review and

meta-analysis by Philipps and colleagues reported that offspring of diabetic pregnancies had a higher mean BMI z-score in childhood, with the association attenuating but persisting after adjustment for maternal BMI [7], and the systematic review by Kim and colleagues similarly concluded that GDM exposure is associated with increased odds of offspring overweight and obesity [8]. Prospective data strengthen this picture: in the HAPO Follow-up Study, higher maternal glycaemia during pregnancy predicted greater childhood adiposity and impaired glucose metabolism in a continuous, threshold-free manner even among mothers who would not have met older diagnostic criteria [13, 14], while Boney and colleagues showed that the combination of large-for-gestational-age birth and maternal GDM markedly increased the risk of childhood metabolic syndrome [9].

Several factors may explain why the prevalence in this cohort sits at the higher end of published estimates. First, the cohort is uniformly exposed and lacks an internal unexposed comparator, so the figure reflects absolute burden rather than excess risk. Second, Guyana, like much of the Caribbean and the wider Latin American region, is undergoing a rapid nutrition transition in which an obesogenic post-natal environment is superimposed on intrauterine metabolic programming, a convergence that can amplify offspring adiposity beyond that seen in higher-income settings [4, 23]. Third, the modest sample renders the point estimate imprecise. The co-existence of overweight or obesity (39.4%) with underweight (12.1%) in the same small cohort mirrors the double burden of malnutrition increasingly described in transitional economies, and suggests that the high adiposity prevalence cannot be attributed to generalised over-nutrition alone.

These findings are consistent with the wider regional picture. A systematic review of childhood and adolescent overweight and obesity in Latin America and the Caribbean reported combined overweight and obesity prevalences of 18.9% to 36.9% among school-aged children [29], and regional surveillance has documented a sustained rise in childhood overweight across the region over the past two decades [30]. The 39.4% prevalence in the present GDM-exposed cohort lies at or above the upper limit of these general-population estimates, consistent with an additional contribution from in-utero hyperglycaemic exposure superimposed on an already high regional background risk. Direct comparison is limited, however, because the regional figures derive from general paediatric populations rather than GDM-exposed children, and because the present sample is small.

The observed phenotype is biologically consistent with long-standing models of diabetic pregnancy. The Pedersen hypothesis proposed that maternal hyperglycaemia drives fetal hyperglycaemia and compensatory fetal hyperinsulinaemia, promoting excessive fetal growth and fat accretion [26]. Freinkel subsequently broadened this to the concept of fuel-mediated teratogenesis, in which not only glucose but the entire mixture of maternal fuels reaching the fetus during sensitive developmental windows exerts durable, anthropometric and metabolic consequences on the offspring [25]. Fetal hyperinsulinaemia is both a growth signal in utero and a plausible programming stimulus for later adipogenesis, altered appetite regulation and impaired β-cell reserve, providing a mechanistic bridge between intrauterine exposure and the elevated childhood adiposity and dysglycaemia documented in exposed cohorts [13, 14].

These proximal endocrine effects are embedded within the broader developmental origins of health and disease (DOHaD) framework, which holds that environmental exposures during critical periods of plasticity permanently shaped tissue structure, metabolic set-points and disease susceptibility [6]. Candidate molecular substrates include epigenetic modification of genes governing

energy balance, insulin signalling and adipocyte differentiation, as well as the programming of hypothalamic appetite circuits. Evidence from discordant-sibship designs, in which siblings born before and after the mother developed diabetes are compared, supports a specifically intrauterine contribution that is not wholly explained by shared genes or family environment [11]. The near-universal exposure of the present cohort to maternal insulin therapy (97.0%) indicates clinically significant maternal hyperglycaemia, consistent with a meaningful fuel-mediated programming stimulus, even though treatment itself cannot be disentangled from disease severity in these data.

A counter-intuitive finding was the absence of a positive birth-weight–current-BMI association ($\rho = -0.18$; $p = 0.33$) and the signal that lower birth weight, rather than macrosomia, accompanied later overweight (birth weight <3000 g: OR 3.43, 95% CI 0.73–16.09). Although GDM is classically linked to macrosomia, the relationship between birth weight and later adiposity across populations is frequently U-shaped, with risk elevated at both extremes of the birth-weight distribution. The thrifty phenotype hypothesis offers one explanation: fetal nutritional constraint programmes a metabolically thrifty physiology that becomes maladaptive, and obesogenic, when followed by post-natal nutritional abundance [24]. Often it is the post-natal growth trajectory, rather than birth size, that determines later adiposity: rapid infant catch-up growth has been prospectively associated with childhood obesity [27], and longitudinal modelling in the EPOCH study showed that diabetes-exposed offspring diverge in BMI trajectory during childhood rather than at birth [12].

In this light, the present cohort's pattern — weak or inverse birth-weight associations alongside high mid-childhood adiposity — is compatible with a scenario in which the principal driver of overweight is accelerated post-natal weight gain superimposed on intrauterine programming, rather than birth size per se. This interpretation is necessarily tentative given the wide confidence intervals, but it shows why birth weight alone is an inadequate screening criterion for later metabolic risk in GDM-exposed children and why growth-trajectory surveillance is preferable to single time-point anthropometry.

Macrosomia at birth was not associated with later overweight or obesity in this cohort. Although GDM increases the risk of macrosomia, and macrosomia is itself a recognised marker of later adiposity, the relationship between the two is neither necessary nor direct. Several factors may explain the absent association here. The number of macrosomic children was small ($n = 8$), giving very limited power to detect a difference; maternal insulin treatment was near-universal (97%) and may have attenuated fetal overgrowth, weakening any macrosomia signal; and, as noted, later adiposity in programmed children is often driven more by post-natal growth trajectory than by birth size. The finding that lower, rather than higher, birth weight accompanied later overweight in these data is consistent with this post-natal, catch-up-growth interpretation.

Among modifiable post-natal factors, a higher number of daily meals showed the strongest (though non-significant) association with overweight/obesity (≥4 meals/day: OR 5.62, 95% CI 0.89–35.39) and the largest positive correlation with BMI ($\rho = 0.31$), consistent with the recognised contribution of the post-natal dietary environment to weight trajectories in programmed children [9, 19]. The counter-intuitive inverse association between daily sugary-drink consumption and overweight is almost certainly an artefact of the small sample or of reverse causation — families of heavier children restricting sugary drinks — and is not interpreted further. The tendency towards greater adiposity in girls (median BMI 17.9 vs 16.6 kg/m²; overweight/obesity 46.7%

vs 33.3%) aligns with reports of sexually dimorphic intrauterine programming, in which female offspring of diabetic pregnancies may be more susceptible to adiposity-related outcomes [7]; however, the analysis was underpowered to confirm a sex difference and this observation should be regarded as hypothesis-generating. Finally, a family history of type 2 diabetes was present for over half the cohort (51.5%), reflecting the shared genetic and environmental milieu that compounds intrauterine programming, while the paradoxical inverse association between maternal pregnancy complications and child overweight (OR 0.12) rests on very small cell counts and should not be over-interpreted.

The central public-health message is that a large proportion of GDM-exposed children in this setting already carry an abnormally high BMI by middle childhood, identifying them as a concrete, identifiable group for early intervention. This reinforces the case for structured, long-term anthropometric and metabolic surveillance of GDM-exposed children, ideally beginning in infancy to capture growth trajectory rather than relying on birth weight or a single later measurement. Such follow-up is most efficient when coupled with maternal postpartum care, given that women with GDM carry a high lifetime risk of progression to type 2 diabetes [22] and that their offspring show elevated dysglycaemia risk [15], a connected intergenerational pathway that one coordinated screening programme could address. Embedding these activities within a standardised national GDM registry would enable prospective ascertainment, linkage of mother and child records, and equitable follow-up across Guyana's regionalised health system, consistent with international guidance on the continuum of diabetes-in-pregnancy care [16, 21].

From an implementation perspective, several concrete steps follow for Guyana and comparable settings. Long-term follow-up of GDM-exposed children could be integrated into existing maternal diabetes and antenatal services, so that mother and child are reviewed within a single care pathway rather than separately. Routine measurement of BMI-for-age at child-health and immunisation visits would allow early identification of children moving onto an obesogenic trajectory. Lifestyle and nutritional counselling begun during the index pregnancy and continued post-partum could address both maternal progression to type 2 diabetes and the child's early growth environment. A standardised national GDM registry would link maternal and child records, support recall for follow-up, and generate the prospective data needed to quantify risk locally. These activities align with existing non-communicable disease (NCD) prevention programmes, and embedding GDM-offspring follow-up within the national NCD framework would make efficient use of limited resources.

The absence of statistically significant predictors warrants careful interpretation and should not be read as evidence of no association. With 33 children and only 13 outcome events, the study was substantially underpowered to detect the modest effect sizes typical of this field; the rule of thumb of approximately ten events per variable permits essentially no multivariable adjustment, so all estimates are unadjusted and confounding cannot be excluded. The wide confidence intervals, several spanning an order of magnitude, quantify this imprecision directly, and the consistent direction of several effects (meal frequency, lower birth weight, female sex) is arguably more informative than the individual p-values. No correction for multiple comparisons was applied, given the exploratory, hypothesis-generating intent. These considerations frame the present analysis as a clinically grounded characterisation that should inform the design of an adequately powered, comparative successor study rather than as a source of definitive risk estimates.

Limitations

Several limitations should be considered when interpreting these findings. The sample was small (n = 33) with only 13 outcome events, yielding wide confidence intervals and precluding multivariable adjustment; analyses were therefore unadjusted, exploratory and not corrected for multiple comparisons. The single-arm design, without a contemporaneous non-exposed comparison group, means the excess risk attributable to GDM cannot be quantified. Because current child parameters were obtained by maternal telephone interview, some variables are subject to recall and reporting bias, and a small number of items had missing data. In addition, the cohort was restricted to the 33 of 42 eligible women (78.6%) for whom complete records were available, and the nine pregnancies without extractable records may differ systematically from those analysed, raising the possibility of selection bias. Finally, as a single-jurisdiction cohort assembled over a defined six-month window, the findings may not be generalisable to all GDM-exposed children. Residual confounding is also likely, since several maternal variables known to influence offspring adiposity (notably pre-pregnancy body mass index, gestational weight gain and socioeconomic status) were not available for adjustment. Because current weight and height were largely maternally reported during the telephone interview rather than measured directly, some anthropometric misclassification is also possible. These findings should accordingly be regarded as hypothesis-generating.

Recommendations for Future Research

Several priorities for future research follow from this work. The findings should be confirmed and extended through adequately powered, prospective multicentre cohort studies, since recruitment across several sites would improve both precision and generalisability. Such studies should ideally incorporate a contemporaneous non-GDM comparison group, without which the excess risk attributable to in-utero exposure cannot be quantified. They would also benefit from richer phenotyping than was possible here: measurement of insulin-resistance biomarkers (for example, fasting insulin and HOMA-IR) together with HbA1c would allow early dysglycaemia to be detected before it becomes clinically apparent, while waist circumference and a direct estimate of body-fat percentage would characterise adiposity more completely than body mass index alone. Analyses should also account for pubertal status, which strongly influences body composition in middle childhood and adolescence. Finally, longitudinal growth trajectories captured from birth, rather than single time-point anthropometry, would clarify whether later adiposity in GDM-exposed children is driven more by the pattern of post-natal growth, particularly rapid catch-up growth, than by birth size. Together, these additions would provide the stronger, comparative evidence needed to guide screening and early intervention in this population.

Conclusion

In this retrospective cohort of children exposed to gestational diabetes mellitus (GDM) in utero in Guyana, nearly 40% were overweight or obese by nine years of age, while one-quarter had been macrosomic at birth, highlighting a considerable early cardiometabolic burden. Although no independent predictors of abnormal BMI were identified, likely due to the limited sample size, higher meal frequency, lower birth weight, and female sex emerged as important trends warranting further investigation. These findings support routine, long-term anthropometric and metabolic monitoring of GDM-exposed children and highlight the need for larger comparative studies to identify modifiable risk factors. Integrating such follow-up into maternal and child health

services in Guyana and similar resource-constrained settings could facilitate early intervention and help reduce the future burden of obesity and cardiometabolic disease.

Conflict of Interest

The authors declared that there is no conflict of interest.

Ethical Approval

This study was conducted as part of a PhD research programme approved by the Ministry of Health Guyana Institutional Review Board, the GPHC Research Committee, and governed by Texila American University institutional research oversight. All data was de-identified; study IDs replaced hospital medical record numbers in all analytical datasets. No participant intervention was involved in this retrospective record review. The study was conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from the mothers of participating children prior to the telephone interviews, and all data was de-identified before analysis.

Author Contributions

Dr. Samantha N.A. Kennedy conceived and designed the study, acquired, analysed and interpreted the data, and drafted the manuscript. Dr. Michael Olabode Tomori provided PhD supervision, academic oversight, critical manuscript review, and interpretation of findings. Dr. Abiodun Olaiya Paul contributed to the methodology, scientific review, and manuscript editing. All authors approved the final manuscript and accepted responsibility for its content.

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Data Availability

The de-identified dataset supporting the findings of this study is available from the corresponding author on reasonable request, subject to Ministry of Health data-governance approval.

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