The association of a low glycaemic index diet in pregnancy with child body composition at 5 years of age: A secondary analysis of the ROLO study

Sophie Callanan | Cara A. Yelverton | Aisling A. Geraghty | Eileen C. O’Brien | Jean M. Donnelly | Elizabeth Larkin | Mary K. Horan | John Mehegan | Fionnuala M. McAuliffe

1UCD Perinatal Research Centre, School of Medicine, University College Dublin, National Maternity Hospital, Dublin, Ireland
2UCD School of Public Health, Physiotherapy and Sports Science, University College Dublin, Dublin, Ireland

Correspondence
Fionnuala M. McAuliffe, UCD Perinatal Research Centre, School of Medicine, University College Dublin, The National Maternity Hospital, Dublin 2, Ireland. Email: fionnuala.mcauliffe@ucd.ie

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Summary

Introduction: Childhood obesity remains a significant global health concern. Early intervention through maternal diet during pregnancy represents a possible mode of improving childhood adiposity.

Aim: To examine the impact of a low glycaemic index diet during pregnancy on offspring anthropometry at 5 years of age.

Methods: This is a secondary analysis of 387 children from the ROLO pregnancy study 5 years’ post-intervention. At the follow-up, BMI, circumferences and skinfold thickness were obtained. A subgroup of 103 children had a DXA scan completed. Statistical analyses included Independent sample t tests, Mann Whitney-U tests and chi-square tests to compare the intervention and control groups. Adjusted analysis using linear regression controlled for significant confounders between participants who returned at follow-up and those that did not.

Results: There were no significant differences in BMI (16.05 kg/m² vs 16.16 kg/m², P = 0.403), general adiposity (36.60 mm vs 36.00 mm, P = 0.920), central adiposity (0.61 mm vs 0.60 mm, P = 0.540), total fat mass (4.91 kg vs 4.71 kg, P = 0.377) or total lean mass (14.29 kg vs 14.56 kg, P = 0.386) between the intervention and control groups, respectively. No associations were observed in 5-year outcomes in adjusted analyses when controlling for maternal age at delivery, maternal early pregnancy BMI, maternal education and gestational age.

Conclusion: Our study found no evidence that a low glycaemic index diet in pregnancy impacts offspring anthropometry 5 years’ post-intervention. Therefore, modulating maternal carbohydrate quality in pregnancy may not be an appropriate approach to improving weight status in childhood. Future research should investigate the impact of other dietary practices in pregnancy on child health.

Abbreviations: BMI, body mass index; DOHaD, developmental origins of health and disease; DXA, dual-energy X-ray absorptiometry; GDM, gestational diabetes mellitus; GI, glycaemic index; IOTF, International Obesity Task Force; IQR, interquartile range; RCT, randomized control trial; ROLO, a randomized control trial of a low glycaemic index diet in pregnancy to prevent macrosomia; WHO, World Health Organization.
1 | INTRODUCTION

Childhood obesity presents a significant health concern and poses a threat to the health of future generations. At a global level, the World Health Organization (WHO) reported that in 2019 approximately 38.2 million children under 5 years of age were living with overweight or obesity. According to the International Obesity Task Force (IOTF) criteria, the prevalence of Irish children with overweight and obesity is 13.9% in boys and 19.2% in girls aged 5-12 years. In 2018, the Growing Up in Ireland study reported that approximately 20% of Irish 5-year-old children had overweight or obesity, with the prevalence of obesity at 5%. Along with the immediate health consequences that can arise from childhood obesity, including insulin resistance, orthopaedic abnormalities and sleep disorders, the impact during childhood can extend into other aspects of life and can negatively impact social development. There is an increased risk of childhood obesity persisting into adulthood, creating a predisposed risk to developing non-communicable diseases, including cardiovascular disease and type 2 diabetes mellitus. As Ireland is expected to have the highest rates of obesity in Europe by 2030, investigations into possible early prevention strategies are essential to improve the long-term health of future generations.

An early risk factor for childhood obesity is foetal macrosomia, defined as having a birth weight greater than 4.0 kg. Early intervention during pregnancy presents an opportunity to prevent childhood obesity, as has been recently highlighted in perinatal research. One area for intervention during pregnancy is maternal diet, particularly glucose levels, due to its importance in foetal growth. Recent research has investigated the impact of controlling maternal glycaemia by trialling a low glycaemic index (GI) diet during pregnancy on neonatal and infant outcomes; however, results remain ambiguous. The ROLO (A Randomized cOntrol trial of a LOw glycaemic index diet in pregnancy to prevent macrosomia) study did not achieve a reduction in birthweight following a low GI dietary intervention in pregnancy; however, more subtle changes in epigenetic status were identified suggesting a possible lasting impact of the intervention on offspring health. The on-going UPBEAT (UK Pregnancies Better Eating and Activity Trial) trial succeeded in reducing gestational dietary GI, however, there were no significant reductions observed in birthweight. Similar to the ROLO cohort, this longitudinal study is assessing potential lasting impacts of behavioural pregnancy intervention, recently reporting a positive reduction in adiposity among the intervention children at 3 years of age.

There is a growing need for longitudinal follow-up of cohorts following pregnancy intervention trials, due to the potential effects that can arise from foetal programming. Foetal programming occurs when the foetus is exposed to adverse environmental stimuli that can cause permanent epigenetic alterations to physiology and metabolism. The Barker hypothesis suggests that adverse nutritional conditions during intrauterine development can increase susceptibility to the development of the metabolic syndrome in adulthood. The maternal environment plays a role in determining the growth trajectory that an individual will follow throughout pregnancy, leading into infancy and childhood. Similarly, the developmental origins of health and disease (DOHaD) concept supports the influence of foetal programming on long-term health suggesting early adverse exposures can influence growth patterns and the incidence of chronic disease. Therefore, pregnancy may be a critical timepoint for the prevention of childhood obesity.

The effects of an altered environment in utero may become evident at different time points within an individual’s life, depending on the mechanisms of epigenetic alterations. A recent study observed that adherence to the Mediterranean diet in early pregnancy resulted in significantly lower BMI z-score, waist circumference and sum of subscapular and triceps skinfold thickness measurements among children at 4 and 7 years of age, which was not mediated by birthweight. Multiple recent studies have observed similar findings further supporting the Barker hypothesis and suggesting that maternal nutritional conditions may influence childhood weight status.

As previously mentioned, the quality and quantity of carbohydrate intake is an aspect of the maternal diet that receives significant attention during pregnancy. While the 5-year postnatal ROLO follow-up found no long-term effect of a low GI pregnancy intervention on maternal weight status, there is potential for impact on the offspring due to the influence of glucose on foetal growth. This has been observed in the Southampton Women's Survey, which showed maternal dietary glycaemic load (GL) and GI in early pregnancy were associated with child fat mass at 4 and 6 years of age. The Lifeways-Cross Generational Cohort study found that when maternal macronutrient consumption was analysed during pregnancy, sugar intake was strongly predictive of child weight status at 5 years of age. These significant observational outcomes support the potential long-term impact of glucose during pregnancy based on the mechanisms of foetal programming. The research available to support this theory is currently observational; a randomized control trial (RCT) is required to further prove the importance of nutrients in pregnancy for child growth.

The aim of this study is to determine the effect of a low GI diet throughout pregnancy on body composition of the offspring at 5 years of age. We hypothesize that there will be an evident impact of this diet at 5 years in the form of lower weight status and adiposity among children in the intervention group.

2 | STUDY DESIGN AND METHODOLOGY

2.1 | Study design and population

This secondary analysis was carried out using data available on 387 children who attended a 5-year post-intervention follow-up from...
the ROLO pregnancy study. The ROLO pregnancy study was published previously. In brief, it was a dietary intervention study conducted in the National Maternity Hospital, Dublin, between 2007 and 2011 to reduce recurrence of macrosomia. Ethical approval for the study was obtained from the Ethics Committee of the National Maternity Hospital. Healthy, secundigravida women who had previously delivered a macrosomic infant were recruited at their first antenatal appointment. A total of 800 women were randomly assigned into either the intervention or control arm; the intervention group received low GI dietary advice from early pregnancy in the form of 2-hour dietary education sessions with a research dietitian at 15, 28 and 34 weeks' gestation. The control group received routine antenatal care, which did not include any formal dietary advice. Data were collected for 759 mother-child dyads at delivery. The ROLO study has since become a longitudinal birth cohort follow-up study with follow-up at 3 months, 6 months, 2 years and 5 years postnatal. A study flow chart detailing the progression of the ROLO study and participant numbers at subsequent follow-ups can be seen in Figure 1.

As the children of the ROLO pregnancy study turned 5 years of age, they were invited to attend a follow-up appointment. Participants were eligible to return at this point if they had taken part in the ROLO pregnancy study and were between 4.5 and 5.5 years of age (average age 5.1 years). Of the 759 dyads in the ROLO pregnancy study, a total of 387 (50.9%) children were included in this analysis based on available anthropometric data collected at the 5-year follow-up. Of these, 198 children were in the intervention group. The ROLO study was funded by the Health Research Board Ireland, the Health Research Centre for Health and Diet Research, The National Maternity Hospital Medical Fund and the European Union’s Seventh Framework Programme (FP7/2007-2013). Ethical approval for the 5-year follow-up study was granted by the Ethics Committee of the National Maternity Hospital and Our Lady’s Children Hospital, Crumlin. The Current Controlled Trials registration number for the ROLO study was ISRCTN54392969 and the reference number was xGEN/279/12. All eligible individuals provided written, informed consent prior to participating in the research study.

2.2 | Data collection

Information regarding maternal age at delivery, ethnicity, education level and maternal smoking history were taken from hospital records.
Education level was dichotomized in this analysis into those having completed education from a higher education institute (universities, colleges and institutes of technology) and those who did not. Data were also collected on the study infant, including sex, birthweight, gestational age at delivery and breastfeeding status. At the 5-year follow-up, age of the study child at the time of appointment was also recorded. In addition, data were analysed and compared between participants who attended the follow-up 5 years post-intervention and those who did not return.

2.3 | Anthropometry and body composition

A trained research dietitian/nutritionist took a series of anthropometric measurements from the children at the 5-year follow-up appointment. Participants were dressed in light clothing and removed their shoes for weight and height measurements. Weight was measured to the nearest 0.1 kg using the SECA model flat, mobile weighing scales (SECA gmbh & co. Kg. Germany). Height was measured to the nearest 0.1 cm using the SECA 123 portable stadiometer (SECA gmbh & co. Kg. Germany). From this, the body mass index (BMI) was calculated as kg/m² for each participant. Weight and BMI values were converted to standardized scores and centiles, estimated relative to the 1990 UK reference data.34 Each participant was categorized as having a normal weight, having overweight or having obesity according to the UK Growth Charts and WHO cut-off points, respectively. Using the UK Growth Chart cut-off points, having overweight was defined as a BMI greater than or equal to the 91st centile and having obesity was defined as a BMI greater than or equal to the 98th centile.35 Using WHO cut-off points, having overweight was defined as a BMI greater than or equal to the 85th centile and having obesity was defined as having a BMI greater than or equal to the 95th centile.36 Skinfold thickness measurements were taken at four sites, including the biceps, triceps, thigh and subscapular using a Holtain Tanner/Whitehouse skinfold caliper to the nearest 0.1 mm. The sum of all four skinfold measurements was used as a marker of general adiposity. Central adiposity was estimated by dividing the subscapular skinfold measurement by the triceps measurement. The SECA 201 ergonomic circumference measuring tape was used to measure circumferences in centimetres at multiple body sites, including the waist, hip and thigh. In addition, the waist: hip ratio was determined by dividing the abdominal circumference by the hip circumference. In a further analysis, anthropometric data were separated by sex to determine differences between males and females at 5 years of age.

Of the 387 children that attended a follow-up appointment at 5 years, a subgroup of 103 children had a DXA (dual-energy X-ray absorptiometry) scan taken. The DXA was performed using the Lunar iDXA scanner (GE Healthcare, Madison WI) with enCORE v.14.1 software. Measurements taken included bone mineral density (g/cm²), bone mass (kg), fat mass (kg) and lean mass (kg) for each child.

2.4 | Statistical analysis

Statistical analysis was completed using SPSS (Statistical Package for Social Sciences) software version 24. All variables were tested for normality using the Kolmogorov-Smirnov tests and examining simple histograms as a visual aid. Shapiro-Wilk test was used to determine the normality of the DXA variables. Normally distributed variables were reported as mean (SD [SDI]). Non-parametric variables were reported as median [interquartile range (IQR)]. Independent sample t tests were used to compare differences between intervention and control groups in normally distributed continuous variables. Mann Whitney-U tests were carried out for all non-parametric variables. Chi-square tests were used to calculate frequencies and to determine significance for all categorical variables. Categorical variables were reported as n (%). Adjusted linear regression analyses were performed between RCT group and child weight/body composition outcomes to adjust for confounders between participants that returned to the follow-up visit at 5 years and those that did not return. A P value of less than 0.05 was considered statistically significant for all analyses.

3 | RESULTS

3.1 | Maternal and child demographics

There were 387 children included in this study, with 198 born to women in the intervention group (51.2%). The mean maternal age at delivery was 33 years. Majority of the maternal cohort were Caucasian at 91.2%. Of 340 women, 210 (61.8%) had completed third level education. A total of 10 (2.6%) mothers reported smoking during their pregnancy. The mean birthweight of the children was 4.02 kg. Sex was equally distributed amongst the offspring cohort with 186 (48.1%) of the participants being born male. There was a significant difference in gestational age at delivery (284 days vs 282 days, P = 0.013) between the intervention and control groups, respectively. No other significant differences were found in maternal and child baseline characteristics between the intervention and control groups. The comparison of maternal and child descriptive characteristics in the ROILO pregnancy study between the intervention and control groups is presented in Table 1.

3.2 | Child characteristics at 5-year follow-up

Detailed data of anthropometry and body composition were collected for a total of 387 children who attended a follow-up appointment at 5 years of age. When separated by sex, significant differences were observed in weight (20.35 kg vs 19.70 kg, P = 0.020), height (112.30 cm vs 111.20 cm, P = 0.010), general adiposity (35.20 mm vs 37.80 mm, P = 0.001), thigh circumference (32.10 cm vs 33.00 cm, P = 0.000), total fat mass (4.39 kg vs 4.86 kg, P = 0.015) and total lean mass (14.64 kg vs 13.78 kg, P = 0.004) between males and females, respectively. In depth, comparison of child characteristics between the intervention and control groups can be seen in Table 2.
3.3 | Child anthropometry at 5-year follow-up

No significant differences were found in any of the anthropometric measurements between intervention and control groups. There were no significant differences in BMI status between the intervention and control groups when separated into participants with normal weight, participants with overweight or participants with obesity using UK Growth and WHO cut-off points, respectively. The results of the comparison of anthropometry between the intervention and control groups are displayed in Table 3.

3.4 | Child body fat distribution from DXA scans at 5-year follow-up

Of the 387 children that attended a follow-up appointment, 103 (26.6%) children had a DXA scan carried out. Of these, 50 children were from the intervention group. There was no significant difference in measurements obtained using DXA scans between the intervention and control groups. The results of the DXA scan for this subgroup at 5 years of age are displayed in Table 4.

3.5 | Adjusted regression analysis between RCT group and child anthropometry and body composition outcomes at the 5-year follow-up

Independent sample t tests were carried out to determine differences in baseline characteristics in mothers between those that returned at 5 years, and those that did not return at 5 years. Significant differences were found in maternal early pregnancy BMI (25.38 kg/m² vs 26.17 kg/m², P = 0.01), completion of third level education (61.3% vs 48.7%, P = 0.009), maternal age at delivery (33.36 years vs 31.72 years, P = 0.000) and gestational age at delivery (283 days vs 282 days, P = 0.023) in participants that did return to the 5 year follow-up compared to those that did not return, respectively.
other significant differences were found. Therefore, these variables were included in regression analysis as confounders. Models were run with RCT as a predictor, adjusted for the above confounders, with the following outcomes: weight, weight centile, height, height centile, BMI, BMI centile, general adiposity, central adiposity, hip circumference, thigh circumference, waist:hip ratio, bone density, total bone mass, total fat mass and total lean mass. No significant associations were found between pregnancy RCT group and child weight/body composition outcomes. Further details can be seen in Tables 5 and 6.

### 4 | DISCUSSION

Our research found no significant evidence to suggest that a low GI diet during pregnancy has a measurable effect on adiposity and

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**Table 3** Comparison of anthropometry in 5-year-old children born into the ROLO pregnancy RCT of a low glycaemic index diet during pregnancy

|                     | Intervention | Control |
|---------------------|--------------|---------|
|                     | N | Mean/median | %/SD/IQR | N | Mean/median | %/SD/IQR | P<sup>a</sup> |
| Weight (kg)<sup>b</sup> | 198 | 19.90 | 3.30 | 189 | 20.00 | 3.60 | 0.619 |
| Weight centile<sup>b</sup> | 198 | 68.00 | 42.00 | 189 | 68.00 | 41.00 | 0.637 |
| Height (cm)         | 197 | 111.52 | 4.45 | 189 | 111.91 | 4.65 | 0.401 |
| Height centile<sup>b</sup> | 197 | 61.00 | 50.00 | 189 | 60.00 | 49.00 | 0.707 |
| BMI (kg/m<sup>2</sup>) | 197 | 16.05 | 1.65 | 189 | 16.16 | 1.85 | 0.403 |
| BMI centile<sup>b</sup> | 197 | 66.00 | 41.00 | 189 | 68.00 | 45.00 | 0.423 |

**BMI categories – UK growth charts<sup>c</sup>**

|                     | Intervention | Control |
|---------------------|--------------|---------|
| Normal (2nd < 91st centile), n (%) | 175 | — | 88.80 | 160 | — | 84.70 | 0.469 |
| Overweight (≥91st centile), n (%) | 18 | — | 9.10 | 23 | — | 12.20 | — |
| Obese (≥98th centile), n (%) | 4 | — | 2.00 | 6 | — | 3.20 | — |

**BMI categories – WHO<sup>d</sup>**

|                     | Intervention | Control |
|---------------------|--------------|---------|
| Normal (≥5th < 85th centile), n (%) | 153 | — | 78.10 | 136 | — | 72.70 | 0.431 |
| Overweight (≥85th centile), n (%) | 33 | — | 16.80 | 37 | — | 19.80 | — |
| Obese (≥95th centile), n (%) | 10 | — | 5.10 | 14 | — | 7.50 | — |
| General adiposity (mm)<sup>a,b</sup> | 178 | 36.60 | 12.18 | 173 | 36.00 | 13.40 | 0.920 |
| Central adiposity (mm)<sup>a,b</sup> | 183 | 0.61 | 0.17 | 178 | 0.60 | 0.14 | 0.540 |
| Hip circumference (cm)<sup>b</sup> | 196 | 60.25 | 4.90 | 189 | 60.70 | 5.60 | 0.580 |
| Thigh circumference (cm)<sup>b</sup> | 196 | 32.60 | 3.50 | 187 | 32.50 | 3.90 | 0.919 |
| Waist:ratio (cm)<sup>a,b</sup> | 196 | 0.91 | 0.06 | 189 | 0.90 | 0.06 | 0.242 |

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**Table 4** Comparison of body composition in 5-year-old children born into the ROLO pregnancy RCT of a low glycaemic index diet during pregnancy using DXA analysis

|                     | Intervention | Control |
|---------------------|--------------|---------|
|                     | N | Mean | SD | N | Mean | SD | P<sup>a</sup> |
| Bone density (g/cm<sup>2</sup>) | 50 | 0.67 | 0.04 | 53 | 0.67 | 0.04 | 0.724 |
| Total bone Mass (kg) | 50 | 0.70 | 0.07 | 53 | 0.71 | 0.08 | 0.522 |
| Total fat mass (kg) | 50 | 4.91 | 1.26 | 53 | 4.71 | 1.28 | 0.377 |
| Total lean mass (kg) | 50 | 14.29 | 1.57 | 53 | 14.56 | 1.52 | 0.386 |

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<sup>a</sup>P values determined using Mann Whitney-U, independent sample t tests or chi-square tests; significance set at P < 0.05; general adiposity estimated as the sum of all skinfold sites (biceps, triceps, subscapular and thigh); central adiposity estimated by dividing the subscapular skinfold measurement by the triceps measurement; waist:hip ratio estimated by dividing the abdominal circumference by the hip circumference.

<sup>b</sup>Median and IQR reported.

<sup>c</sup>UK Growth Chart cut-off points for children aged 2-18 years.35

<sup>d</sup>WHO cut-off points for BMI for children aged 5-19 years.36
weight status of the child at 5 years of age. We hypothesized that the children from the intervention group would have lower measures of adiposity through stabilizing glucose supply during foetal development by targeting maternal diet, however, this was not proven.

The impact of the maternal in-utero environment on childhood growth and development remains to be fully understood, highlighting the importance of this research. In addition, we identified no detrimental impacts of the low GI dietary intervention on child anthropometry at this timepoint in development.

Existing observational evidence suggests maternal GI in pregnancy may have a lasting effect on health outcomes of the next generation. Okubo et al observed GI and GL in early and late pregnancy amongst a subset group of the Southampton Women’s Survey. They found higher maternal glycaemic markers in early pregnancy had a positive association with child adiposity at 4 and 6 years of age, independent of birthweight. The Lifeways Cross-Generation Cohort Study observed the association between maternal macronutrient intake during pregnancy and child weight status at 5 years of age. Maternal sugar intake in pregnancy, independent of total carbohydrate intake, was strongly predictive of child adiposity and weight status at 5 years of age. Interestingly, our RCT did not find any such association between glucose quality and child health, 5 years on.

### Table 5

| Models for intervention | B     | p    | 95% CI   | R² Adj. |
|-------------------------|-------|------|----------|---------|
| Weight (kg)             | -0.250 | 0.370 | -0.800, 0.299 | 0.014   |
| Weight centile          | -0.813 | 0.767 | -6.214, 4.588 | 0.004   |
| Height (cm)             | -0.161 | 0.747 | -1.140, 0.818 | 0.000   |
| Height centile          | 0.210  | 0.945 | -5.772, 6.192 | 0.000   |
| BMI (kg/m²)             | -0.151 | 0.288 | -0.430, 0.128 | 0.051   |
| BMI centile             | -1.905 | 0.418 | -7.219, 3.409 | 0.044   |
| General adiposity (mm)  | -0.698 | 0.580 | -3.174, 1.778 | 0.040   |
| Central adiposity (mm)  | 0.001  | 0.942 | -0.039, 0.042 | -0.012  |
| Hip circumference (cm)  | -0.254 | 0.557 | -1.106, 0.597 | 0.024   |
| Thigh circumference (cm)| -0.172 | 0.586 | -0.794, 0.449 | 0.005   |
| Waist:hip ratio (cm)    | 0.006  | 0.338 | -0.006, 0.018 | -0.011  |

Abbreviations: BMI, body mass index; CI, confidence interval; RCT, randomized control trial.

### Table 6

| Models for intervention | B     | p    | 95% CI   | R² Adj. |
|-------------------------|-------|------|----------|---------|
| Bone density (g/cm²)    | -0.003 | 0.720 | -0.022, 0.016 | -0.029  |
| Total bone mass (kg)    | -0.014 | 0.424 | -0.047, 0.020 | -0.015  |
| Total fat mass (kg)     | 0.164  | 0.545 | -0.371, 0.699 | -0.030  |
| Total lean mass (kg)    | -0.286 | 0.383 | -0.934, 0.362 | -0.014  |

Abbreviations: CI, confidence interval; DXA, dual-energy X-ray absorptiometry; RCT, randomized control trial.
offspring growth. Deierlein et al observed the impact of hyperglycaemia in pregnancy in the absence of GDM on offspring growth. This follow-up of the Pregnancy Infection and Nutrition study found an association between hyperglycaemia in pregnancy and increased risk of having overweight/obesity in the offspring at 3 years of age. Hillier et al aimed to assess the relationship between ranges of maternal glycaemia in pregnancy and the risk of childhood obesity. Elevated hyperglycaemia in pregnancy was associated with an increased risk of childhood obesity at 5-7 years of age. Kubo et al observed the association between maternal hyperglycaemia during pregnancy and adiposity during childhood. Children exposed to the highest quintile of glucose in utero were associated with an increased risk of having a BMI above the 85th percentile. The association between maternal glucose levels and childhood adiposity was irrespective of birthweight. Following the analysis of obesity risk among the ROLO Kids cohort, our results contrast to the findings of these studies. We found no significant differences between the intervention and control group with respect to either the risk of childhood obesity, or the proportions of children with overweight or obesity. This leads to inconsistencies in the literature regarding the true impact of maternal glycaemia on weight status in childhood.

Our results are consistent with previous ROLO follow-ups that have analysed the anthropometry of this cohort at multiple previous time points. At birth, there was no significant difference in neonatal anthropometry between the intervention and control groups except for the finding of a lower thigh circumference in children born to mother’s adhering to the intervention. At the 6-month follow-up there was also no significant difference in anthropometric measurements. Again at 2 years of age there was no evidence of an impact of the low GI diet on anthropometry except for a lower hip-circumference observed in the control group. In contrast, our analysis examined both hip and thigh circumference at 5 years of age with the expectation that these results may have been observed again, however, there was no significant difference found for either measurements at this stage in development.

When looking beyond gestational glycaemia, a lack of evidence reporting the impact of the maternal environment in utero on child weight status at this timepoint has been reported in other dietary pregnancy studies. Brei et al investigated the impact of high-dose long chain polyunsaturated fatty acid supplementation during pregnancy and lactation on child body composition up to 5 years of age. There were no significant differences in anthropometry among children between the intervention and control group up to 5 years of age. A follow-up of the Generation R study observed the association between maternal dietary patterns in pregnancy and child body composition at 6 years of age. Following adjustment for sociodemographic and lifestyle factors, the initial associations between higher adherence to specific dietary patterns and lower BMI among the children were no longer significant suggesting the lack of influence of maternal diet during pregnancy. Furthermore, there was a lack of association between sugar dietary pattern and child body composition. The outcomes of these studies further support our findings, hence further longitudinal investigation into different aspects of the maternal diet during pregnancy is required to gain more understanding of the role specific nutrients play in determining weight status of offspring throughout childhood.

Our analysis found that there were significant differences in several anthropometric measurements when the cohort was dichotomized by sex. Males were found to be taller, weighed more and had more lean body mass than females, this is an early reflection of typical male body composition in adulthood. In addition, females had a higher thigh circumference, higher general adiposity measures and greater fat mass than males in this cohort, similarly these findings highlight early adiposity patterns that are expected in female adults for reproductive purposes. Growth throughout childhood is not a linear process, children undergo multiple periods of rapid growth in which their organs and skeleton enlarge to adult capacity and undergo body composition remodelling. Hence, the differences observed in our analysis may be attributed to varying growth rates and patterns between males and females throughout childhood and into adolescence.

Our findings of no relationship between a low GI diet in pregnancy and child body composition are further confirmed by our sub-analysis using DXA scans. The DXA is considered a gold standard in measuring body composition providing readings for body fat composition for this subgroup of children. This study is unique in that it is the first to use DXA scans to assess childhood adiposity as a means of supporting the outcome of anthropometric measurements 5 years after trialling a low GI intervention during pregnancy.

Our research has a number of strengths, the first being it is a large longitudinal study with novel outcomes that have not been previously investigated, contributing to the existing gap in interventional research. Our study was an RCT, which can infer causality compared to observational studies that are limited to identifying potential associations. Although our findings were not significant, this unique aspect of our study combined with the observational support of the impact of gestational glycaemia on childhood adiposity fills the current gap of interventional research in this area. The infants included in this study were those at risk of having a high birthweight; potentially the effect of previously giving birth to infants with macrosomia is stronger than that of diet, hence we did not find any associations. The consistent methods of assessing the anthropometric measurements, including skinfold thickness are robust and repeatedly used as routine measures of adiposity in children. However, this study is not without its limitations. A potential weakness of the study is that the children of the ROLO pregnancy study were at a higher risk of being born with macrosomia as there is an increased risk for recurrent macrosomia with each successive pregnancy. Therefore, the study methodology and outcomes may be considered less applicable to the general population. It is possible that the high level of education amongst the mothers in this cohort may cause potential bias due to the association with being a more health conscious population, potentially reducing the difference in body weight between the two groups. Finally, another limitation of this research to highlight is the small sample size attributed to the loss to follow-up that was experienced 5 years’ post-intervention, suggesting that the study may not have been sufficiently powered to show significant associations.
CONCLUSION

In summary, our findings do not support the hypothesis that a low GI diet in pregnancy has an impact on child body composition at 5 years of age. However, we identified no detrimental impacts of this intervention on the anthropometry of the children. Our findings contribute to the current gap in perinatal research by examining the impact of a pregnancy intervention on childhood growth. Although our analysis found that a low GI diet in pregnancy may not suffice to improve weight status at 5 years of age, existing evidence justifies further longitudinal investigation as the effects of foetal programming may arise at a later timepoint in development. In addition, future research may investigate the lasting impact of other nutrients or dietary patterns throughout pregnancy that may support the Barker hypothesis of a lasting impact on child health. Identifying effective prevention strategies for childhood obesity in pregnancy will not only be of direct benefit to the offspring but will improve the health of future generations by breaking the detrimental intergenerational cycle of obesity.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

ORCID

Fionnuala M. McAuliffe https://orcid.org/0000-0002-3477-6494

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