

The association between pre-pregnancy use of obesity medications and gestational weight gain: a retrospective cohort study

Helena Piccinini-Vallis

helena.piccinini@dal.ca

Dalhousie University

Janet Green

Dalhousie University

Kristyn Reeves

Dalhousie University

Megan Campbell

Dalhousie University

Research Article

Keywords:

Posted Date: July 1st, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9817569/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background

Excess gestational weight gain (GWG) is an independent risk factor for several adverse maternal and neonatal outcomes. It is more common among individuals with an elevated pre-pregnancy body mass index (BMI), who are therefore advised to lose weight prior to pregnancy in order to enter pregnancy at a lower BMI. However, emerging evidence suggests that pre-pregnancy weight loss may paradoxically increase the risk of excess GWG. There is an increasing demand for weight loss medications, especially by female individuals in their reproductive years, and therefore the association between the pre-pregnancy use of those medications and GWG and other pregnancy outcomes needs to be examined.

Methods

We conducted a population-based retrospective cohort study using linked administrative and perinatal data between 2018 and 2024. Women aged 18 to 45 years with liveborn singleton pregnancies and no pre-existing diabetes were included. Exposure was defined as pre-pregnancy use of obesity medications (liraglutide, semaglutide, tirzepatide, naltrexone/bupropion). Exposed pregnancies (n = 222) were matched 1:1 to unexposed controls on maternal age, parity, and pre-pregnancy BMI. Primary outcomes were GWG (kg) and guideline concordance of GWG. Secondary outcomes included gestational diabetes (GDM), preeclampsia, caesarean section, and neonatal outcomes.

Results

Mean GWG was higher among exposed versus unexposed pregnancies (12.4 vs. 9.5 kg; $p = 0.003$), with a greater proportion of excess GWG (60.8% vs. 43.5%). In matched analyses, exposure was associated with increased odds of excess GWG (OR 1.38, 95% CI 1.30–1.47) and decreased odds of insufficient GWG (OR 0.86, 95% CI 0.79–0.93). Exposure was also associated with higher odds of GDM (OR 1.10), preeclampsia (OR 1.50), caesarean section (OR 1.32), and large-for-gestational-age infants (OR 1.50), and lower odds of small-for-gestational-age infants (OR 0.56). Among exposed pregnancies, a prescription within 6 months prior to conception was associated with higher odds of excess GWG (adjusted OR 3.29, 95% CI 1.76–6.15) than a more remote prescription.

Conclusions

Pre-pregnancy use of obesity medications, particularly recent use, is associated with an increased risk of excess GWG and other maternal and neonatal outcomes. These findings highlight the importance of preconception counselling and prenatal weight management strategies following discontinuation of obesity medications.

BACKGROUND

The most recent guidelines for optimal gestational weight gain (GWG) were published by the Institute of Medicine (IOM) in 2009 ¹, outlining an inverse relationship between GWG targets and pre-pregnancy body mass index (BMI). For individuals with a singleton pregnancy and pre-pregnancy overweight (BMI from 25.0 to < 30.0 kg/m²) or obesity (BMI ≥ 30.0 kg/m²), the recommendations are for GWG ranging from 7 kg to 11.5 kg, and 5 kg to 9 kg respectively over an entire term pregnancy (i.e., ≥ 37 weeks' gestation). However, considering the weight contributions of the fetus, placenta, amniotic fluid, uterine muscle, and increased intravascular and total water volume ¹, achieving those GWG targets is potentially difficult for these individuals with higher pre-pregnancy BMI who typically face challenges in managing their weight regardless of the context of pregnancy. Consistent with this issue, cross sectional and cohort data from several countries show that over 50% of pregnant individuals experience excess GWG ², in particular individuals with a pre-pregnancy BMI in the overweight range (25.0 - < 30.0 kg/m²) and with Class I obesity (pre-pregnancy BMI 30.0 - < 35.0 kg/m²) ²⁻⁴. Indeed, pre-pregnancy BMI is an independent risk factor for excess GWG ². In Nova Scotia, among pregnancies with available pre-pregnancy BMI information (70–75% of pregnancies), approximately 50% of pregnant individuals entered pregnancy with a BMI ≥ 25.0 kg/m² from 2010 to 2019 ⁵. In 2019 specifically, that figure was 52%, and a high proportion of pregnant individuals were therefore at risk of gaining excess GWG that year; in fact, 52.8% of pregnant individuals did so.

Retrospective and prospective cohort studies have shown excess GWG to be an independent risk factor for several short- and long-term adverse outcomes for mothers, including an increased risk of pre-eclampsia, gestational diabetes, shoulder dystocia, caesarean section, and postpartum weight retention, contributing to downstream obesity ^{6,7}. For offspring, excess GWG is associated with an increased risk of neonatal adiposity, hypoglycemia and hyperbilirubinemia, macrosomia, being large-for-gestational age (LGA), and downstream childhood obesity ^{6,8-12}. Of note, childhood obesity was declared in 2015 by the World Health Organization as “one of the most serious public health challenges of the 21st century” ¹³ due to its increased risk of cardiovascular, endocrine, psychosocial and musculoskeletal complications, many of which are sustained into adulthood, including adult excess weight and, therefore, contribute to the intergenerational cycle of obesity ¹⁴⁻²⁰. Thus, excess GWG is an early marker of offspring obesity in childhood with extension into adulthood. In addition to pre-pregnancy BMI, as stated above, some other factors that influence GWG include maternal age, parity, race and smoking ^{2,21,22,23}.

Due to this association between excess GWG and adverse outcomes, female individuals of childbearing age with an elevated BMI are encouraged to lose weight prior to pregnancy in order to enter pregnancy at a lower BMI and, theoretically, be more likely to achieve weight gains within the recommended ranges ¹. Similarly, the chapter entitled *Weight Management over the Reproductive Years for Adult Women Living with Obesity*²⁴ (led by the Principal Investigator of the present study) within the *2020 Canadian Adult Obesity Clinical Practice Guidelines*²⁵ (co-authored by the Principal Investigator of the present study) also

recommends that individuals with obesity should endeavour to lose weight before pregnancy. Supporting this recommendation for weight loss are a number of recent observational studies and systematic reviews (including a systematic review and meta-analysis co-authored by the Principal Investigator of the present study), showing that pre-pregnancy weight loss is associated with a reduced odds of having a LGA baby ^{26–29}, a risk factor for childhood obesity ³⁰.

Weight loss attempts and weight cycling in the general population are common, in particular among individuals with higher BMI ($\geq 25.0 \text{ kg/m}^2$) ³¹, and often result in weight regain ³². For non-pregnant individuals using obesity medications, discontinuation of those medications has been also shown to often result in weight regain ^{33,34}. With an increasing number of people beginning to use obesity medications, weight regain could become of increasing significance, as it is associated with type 2 diabetes, hypertension, cancer, bone fractures, increased mortality, and progressively higher weights ^{35–39}.

For individuals planning a pregnancy, these statistics are less well documented. A recent cross-sectional study showed that 37.5% of participants with overweight and 76.5% of participants with obesity made pre-pregnancy weight loss attempts ⁴⁰. Carey et. al., recently examined the evidence pertaining to outcomes associated with pre-pregnancy weight loss and found that little is known about those associations ⁴¹. Specific to the association between pre-pregnancy weight loss and GWG, only a few studies have examined that area. Early work showed that individuals who reported pre-pregnancy weight loss were more likely to experience higher GWG ^{42–44}. More recently, work in Canada showed that pre-pregnancy weight loss was associated with excess GWG ⁴⁵. Similar results were also reported in a recent randomized controlled trial from the USA comparing GWG between participants randomly assigned to a pre-pregnancy weight loss intervention and participants assigned to usual care ⁴⁶. Finally, a recent prospective cohort study (led by the Principal Investigator of the present study) showed that participants who reported pre-pregnancy weight loss had a 1.32 odds of excess GWG compared to participants who did not experience pre-pregnancy weight loss ⁴⁷. These studies therefore introduced controversy to the current recommendation for pre-pregnancy weight loss, necessitating further examination of the issue as the association between pre-pregnancy weight loss and GWG is not well understood.

Compounding this gap is the recent introduction of effective anti-obesity pharmacotherapies, which are in high demand, particularly among women of reproductive age, including those with infertility. Their appeal is driven by the substantial weight loss associated with their use ⁴⁸. However, although these agents are contraindicated in pregnancy, pregnancies do occasionally occur during treatment. Patients of reproductive age are therefore counselled to use effective contraception while taking these medications ⁴⁹ and to discontinue them in advance of planned conception (personal communication, National Obesity Clinical Update 2025, Montreal, Canada).

Building on our previous work ⁴⁷, the primary objective of this retrospective cohort study was to explore the relationship between pre-pregnancy use of obesity medications (i.e., liraglutide, semaglutide, tirzepatide, or naltrexone/bupropion) and maternal and fetal outcomes (i.e., guideline-concordance of GWG, GWG, gestational diabetes, pre-eclampsia, caesarean section, birthweight, LGA, SGA and macrosomia) in female individuals 18 to 45 years of age with liveborn singleton pregnancies and without pre-existing diabetes.

The secondary objective was to examine, among exposed pregnancies, whether timing of the most recent prescription prior to pregnancy (≤ 6 months versus > 6 months) is associated with GWG.

METHODS

This was a retrospective cohort study using data linked between Health Data Nova Scotia (HDNS) and the Nova Scotia Atlee Perinatal Database (NSAPDB).

Participants for data linkage: Inclusion criteria for data from HDNS included female individuals 18 to 45 years of age who filled a prescription at a Nova Scotia pharmacy for obesity medications: liraglutide (Saxenda or Victoza), semaglutide (Ozempic or Wegovy), tirzepatide (Mounjaro), or naltrexone/bupropion (Contrave) between October 1, 2018 and May 31, 2024. Inclusion criteria for data from NSAPDB included liveborn singleton pregnancy, and exclusion criteria included pre-pregnancy diabetes.

Matched controls: Participants identified and linked as described above were matched 1:1 to unexposed controls in the NSAPDB on maternal age (≤ 30 versus > 30 years), parity (nulliparous versus not nulliparous) and pre-pregnancy BMI (< 30 versus ≥ 30 kg/m²).

Variables: Primary outcomes were GWG (continuous, kg) and GWG concordance with recommended ranges (insufficient / within / excess). Secondary pregnancy outcomes included gestational diabetes mellitus (GDM), preeclampsia, mode of delivery (caesarean section), birthweight, LGA (birthweight > 90 th percentile), small-for-gestational age (SGA; birthweight < 10 th percentile) and macrosomia (birthweight ≥ 4000 g). GWG was also analysed as two binary outcomes: excess GWG versus not excess, and insufficient GWG versus not insufficient. The main independent variable was pre-pregnancy use of obesity medications as specified above. Other independent variables included maternal age, parity, pre-pregnancy BMI, pre-pregnancy smoking status, and smoking during pregnancy.

Data analysis

Data were de-identified and exported into SPSS (Version 29), a statistical data analysis software program. Missing data were handled using multiple imputation under a missing-at-random assumption⁵⁰. Alpha was set at .05.

A sensitivity power calculation was undertaken using a one-sample hypothesis test for a single proportion (excess GWG versus not). Considering that approximately 50% of pregnant women with an

elevated (i.e., ≥ 25.0 kg/m²) pre-pregnancy BMI experience excess GWG, and setting alpha = 0.05 and 80% power, a sample of approximately 194 participants would detect a 10 percentage-point difference from the reference prevalence of 50%.

Descriptive summaries (means $\pm$ SD or n, %) used a single imputed dataset (Imputation_ = 1). For unadjusted comparisons of outcomes by exposure status we used independent-samples t-tests for continuous outcomes and chi-square tests for categorical outcomes. For matched inferential analyses we combined estimates across all imputations using Rubin's rules. We used conditional logistic regression appropriate for the matched design; models were implemented in SPSS as stratified Cox regressions with each matched pair defined as a stratum, and estimates were pooled across imputations.

Secondary analyses restricted to the exposed group evaluated timing of the most recent prescription (≤ 6 months versus > 6 months before pregnancy); these models adjusted for maternal age, parity, pre-pregnancy BMI (continuous), pre-pregnancy smoking and smoking during pregnancy. Secondary analyses were considered exploratory because pharmacy dispensing records do not measure medication adherence or treatment duration.

RESULTS

Sample: The study included 444 pregnancies: 222 exposed to obesity medications prior to pregnancy and 222 matched unexposed controls.

Baseline characteristics

Baseline characteristics are shown in Table 1. The mean maternal age was 32.8 years (SD 4.6) and the mean pre-pregnancy BMI was 36.9 (SD 7.7). Parity distribution and pre-pregnancy BMI categories were similar between cases and controls, consistent with the matched design. Smoking prevalence before and during pregnancy is reported descriptively.

Table 1
Baseline characteristics of participants

Characteristic	Entire sample (N = 444)	Cases (n = 222)	Controls (n = 222)
Maternal age, mean (SD)	32.8 (4.6)	32.8 (4.5)	32.8 (4.7)
Pre-pregnancy BMI, mean (SD)	36.9 (7.7)	37.3 (7.8)	36.6 (7.5)
Pre-pregnancy BMI category, n (%)			
< 18.5	≤ 5 *	≤ 5 *	≤ 5 *
18.5–<25.0	15 (3.4)	6 (2.7)	9 (4.1)
25.0–<30.0	68 (15.3)	32 (14.6)	36 (16.2)
≥ 30.0	353 (79.5)	179 (81.7)	174 (78.4)
Missing	≤ 5 *	≤ 5 *	≤ 5 *
Parity, n (%)			
0	130 (29.3)	65 (29.3)	65 (29.3)
1	188 (42.3)	99 (44.6)	89 (40.1)
≥ 2	126 (28.4)	58 (26.1)	68 (30.6)
Smoking before pregnancy, n (%)			
No	401 (90.3)	204 (91.9)	197 (88.7)
Yes	43 (9.6)	18 (8.1)	25 (11.3)
Smoking during pregnancy, n (%)			
No	411 (92.6)	211 (95.0)	200 (90.1)
Yes	33 (7.4)	11 (5.0)	22 (9.9)

* Cells with small counts (≤ 5) were suppressed to protect confidentiality.

Pregnancy outcomes

Unadjusted comparisons are summarised in Table 2. The mean GWG was higher among exposed pregnancies than controls ($p = 0.003$) and GWG concordance distributions differed by exposure status ($p = 0.002$). The frequency of GDM and preeclampsia was similar between the groups, whereas the proportion of pregnancies delivered by caesarean section was higher among exposed pregnancies ($p = 0.025$). Birthweights were similar between groups as were the frequencies of SGA and macrosomia. LGA occurred more frequently in exposed pregnancies ($p = 0.020$).

Table 2
Pregnancy outcomes and unadjusted comparisons

Outcome	Entire sample (N = 444)	Cases (n = 222)	Controls (n = 222)	p-value
Gestational weight gain, kg, mean (SD)	10.9 (10.3)	12.4 (11.6)	9.5 (8.48)	0.003
GWG concordance, n (%)	120 (27.0)	50 (23.9)	70 (32.4)	0.002 (overall)
Insufficient				
Within	84 (18.9)	32 (15.3)	52 (24.1)	
Excess	221 (49.8)	127 (60.8)	94 (43.5)	
GDM, n (%)	133 (30.0)	68 (30.6)	65 (29.3)	0.756
Preeclampsia, n (%)	13 (2.9)	7 (3.2)	6 (2.7)	0.778
Caesarean section, n (%)	173 (39.0)	98 (44.1)	75 (33.8)	0.025
Birthweight, g, mean (SD)	3429 (542)	3448 (525)	3410 (559)	0.458
LGA, n (%)	72 (16.2)	45 (20.3)	27 (12.2)	0.020
SGA, n (%)	22 (5.0)	8 (3.6)	14 (6.3)	0.189
Macrosomia, n (%)	61 (13.7)	33 (14.9)	28 (12.6)	0.491

Medication exposures

There were 253 pre-pregnancy prescriptions for obesity medications among the participants, with 25 participants having filled a prescription for more than one obesity medication. For analyses the most recent prescription prior to pregnancy was used. Among those, the medications included Ozempic (n = 106), Saxenda (n = 77), Victoza (n = 4), and Contrave (n = 35).

Matched analyses (pooled across imputations): In conditional logistic regression models accounting for the matched design, pre-pregnancy exposure to obesity medications was associated with higher odds of excess GWG and lower odds of insufficient GWG. Exposure was also associated with higher odds of GDM, preeclampsia, caesarean section and LGA, and with lower odds of SGA (Table 3).

Table 3
Matched analyses

Outcome	Odds ratio	95% CI	p-value
Excess GWG	1.38	1.30–1.47	< 0.001
Insufficient GWG	0.86	0.79–0.93	< 0.001
GDM	1.10	1.01–1.18	0.020
Preeclampsia	1.50	1.14–1.98	0.004
Caesarean section	1.32	1.23–1.41	< 0.001
LGA	1.50	1.34–1.67	< 0.001
SGA	0.56	0.46–0.69	< 0.001
Macrosomia	1.08	0.97–1.21	0.174
Secondary analyses (timing among exposed pregnancies; Imputation_ = 1 only; n = 222)			

Among exposed pregnancies, those whose most recent prescription occurred ≤ 6 months prior to pregnancy had higher odds of excess GWG compared with those whose last prescription was > 6 months pre-pregnancy: unadjusted OR 2.88 (95% CI 1.62–5.13; $p < 0.001$) and adjusted OR 3.29 (95% CI 1.76–6.15; $p < 0.001$) after controlling for age, parity, pre-pregnancy BMI, pre-pregnancy smoking and smoking during pregnancy.

DISCUSSION

In this population-based retrospective cohort study of pregnancies in Nova Scotia, pre-pregnancy exposure to obesity medications was associated with higher GWG and a substantially greater likelihood of excess GWG compared with matched unexposed controls. Exposure to obesity medications was also associated with higher odds of caesarean section, LGA, GDM, and preeclampsia, alongside lower odds of insufficient GWG and SGA. Among exposed pregnancies, more recent prescription use (≤ 6 months prior to conception) was strongly associated with excess GWG. Together, these results suggest that pre-pregnancy use and recent discontinuation of obesity medications may influence weight trajectories during pregnancy and maternal and fetal outcomes.

The results of the present study align with and extend an expanding body of literature showing that pre-pregnancy weight loss may paradoxically increase the risk of excess GWG ^{42–47,51}. More specifically, the present study builds on that previous work by focusing specifically on pre-pregnancy pharmacologic weight management, supporting very recent work from the USA that showed, in a sample of 448 exposed and 1344 unexposed matched controls, that pre-pregnancy exposure to obesity medications was associated with higher GWG and with higher odds of excess GWG ⁵². There are a few plausible explanations for these consistent results. First, a straightforward explanation is that discontinuation of

obesity medications prior to pregnancy may be followed by rapid weight regain, as has been documented in non-pregnant populations after cessation of obesity pharmacotherapy. As pregnancy itself is a state of anabolic hormonal change, the combination of physiological pregnancy-related weight gain with post-discontinuation weight rebound may predispose to higher overall GWG. Another explanation is that individuals with prior pharmacologic weight loss may enter pregnancy with altered appetite regulation, metabolic adaptation, or behavioural responses to weight monitoring and dietary restraint. Prior studies demonstrated that greater dietary restraint and weight control attitudes prior to or early in pregnancy are associated with higher GWG, potentially reflecting a restraint–disinhibition response as pregnancy progresses. It is possible that similar dynamics operate among individuals discontinuing obesity medications, particularly if expectations about “needing to gain weight” during pregnancy are coupled with relief from prior dietary or pharmacologic restriction. However, overall, results pertaining to the association between pre-pregnancy exposure to obesity medications and GWG are inconsistent⁵³, and differences may therefore reflect baseline metabolic characteristics, discontinuation timing and post-discontinuation weight rebound rather than a direct pharmacologic effect.

The results of the present study showed that pre-pregnancy exposure to obesity medications was associated with an increased risk of GDM, aligning with the results of work by Maya et. al.,⁵² but contradicting the results of other studies, in which that association was not observed^{54–56}. The difference in the results may be explained by factors such as inclusion of participants with pre-pregnancy diabetes, varying timing of medication exposure (as long as 24 months pre-pregnancy) and differences in pre-pregnancy BMI between groups. Also contradicting the results of the present study, a recent systematic review⁵³ showed no consistent increase in risk of GDM attributable to GLP-1–based therapy.

The present study also showed an increased likelihood of preeclampsia in the exposed group, confirming the results of one study and contradicting the results of two systematic reviews^{53,57}, which showed inconsistent results for the association between pre-pregnancy use of weight loss medications and preeclampsia and other hypertensive disorders of pregnancy. Similarly, the present study’s results for the other outcomes (cesarean section, LGA, SGA and macrosomia) reflect the heterogeneity of results in the current literature as shown in those systematic reviews^{53,57}.

Strengths and limitations

The strengths of the present study include the use of population-based linked administrative and perinatal data, objective pharmacy dispensing records, and a matched cohort design with robust handling of missing data through multiple imputation. The inclusion of a timing analysis provides novel insight into potential dose–response or recency effects.

However, several limitations warrant consideration. First, the retrospective observational design precludes causal inference, and residual confounding is possible despite matching and adjustment.

Individuals prescribed obesity medications prior to pregnancy may differ systematically from unexposed individuals in ways not fully captured by matching on age, parity, and pre-pregnancy BMI, including weight cycling history, rate of pre-pregnancy weight loss, metabolic risk, and psychosocial

factors. Although matching and adjustment were employed, residual confounding cannot be excluded. Although missing data were addressed using multiple imputation, this approach relies on a missing-at-random assumption, and residual bias due to unmeasured or non-random missingness cannot be excluded.

Second, data from HDNS only identify pharmacy dispensing data, which do not capture medication adherence or exact duration of use. Thus, the exact interval between the discontinuation of the medication and the onset of pregnancy could not be determined accurately and the secondary timing analyses can only be considered exploratory. Third, GWG was analysed at the pregnancy level, and a small number of participants contributed more than one pregnancy during the study period, which may have introduced within-individual correlation. Fourth, information on SES was not collected for this study. In view of the fact that the obesity medications we examined are quite expensive and therefore disproportionately unavailable to individuals in lower income brackets, the participants in this study were potentially of higher SES and not representative of the general population in Nova Scotia. Finally, the study population reflects pregnancies in Nova Scotia, and findings may not be fully generalizable to settings with different demographic or health system characteristics.

CONCLUSIONS

In this population-based retrospective cohort study, pre-pregnancy use of obesity medications was associated with higher GWG, increased odds of excess GWG, and a higher likelihood of other adverse maternal and neonatal outcomes. More recent, compared with more remote, use of those medications prior to conception was strongly associated with excess GWG, suggesting that the timing of medication discontinuation may play a clinically important role in pregnancy weight trajectories.

These findings have direct implications for clinical practice. Given the increasing use of obesity medications among women of reproductive age, these results underscore the importance of targeted preconception counselling addressing the potential for post-discontinuation weight regain and its implications for pregnancy. In particular, proactive strategies to support weight stability after medication discontinuation—such as behavioural interventions, nutritional counselling, and close monitoring of early pregnancy weight gain—may be warranted.

Further research is needed to clarify causal mechanisms and to identify optimal clinical pathways for managing weight in the preconception period among individuals using obesity medications. In the interim, the results of this study support a more cautious and structured approach to discontinuation and follow-up in this population.

Abbreviations

GWG: gestational weight gain

IOM: Institute of Medicine

BMI: body mass index

LGA: large-for-gestational age

USA: United States of America

HDNS: Health Data Nova Scotia

NSAPDB: Nova Scotia Atlee Perinatal Database

GDM: Gestational diabetes mellitus

SPSS: Statistical Product and Service Solutions

SGA: small-for-gestational age

Declarations

Ethics approval and consent to participate: Ethics approval was obtained from the IWK Research Ethics Board (file # 1031768) with a waiver of consent.

Consent for publication: not applicable

Availability of data and materials: The data that support the findings of this study are available from the Reproductive Care Program of Nova Scotia but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of the Reproductive Care Program of Nova Scotia.

Competing interests: The authors declare that they have no competing interests.

Funding: Financial support for this project was obtained from the Drug Evaluation Alliance of Nova Scotia (DEANS) in May 2025.

Authors' contributions: HP designed and conducted the research, including the analysis of data, and wrote the bulk of the manuscript. JG, MC and KR reviewed the manuscript and provided feedback and suggestions on framing the results and connecting to the existing literature. All authors read and approved the final manuscript.

Disclaimers:

Portions of the data used in this report were made available by Health Data Nova Scotia of Dalhousie University. Although this research analysis is based on data obtained from the Nova Scotia Department of Health and Wellness, the observations and opinions expressed are those of the authors and do not represent those of either Health Data Nova Scotia or the Department of Health and Wellness.

The NSAPDB data used in this study were made available by the Reproductive Care Program of Nova Scotia, a program of IWK Health (IWK). Although the analyses were reviewed before publication to ensure compliance with NSAPDB disclosure policies and understanding of the variable definitions, the observations and opinions expressed are those of the authors and do not necessarily represent the observations and opinions of IWK.

References

1. Rasmussen, K. M., Catalano, P. M. & Yaktine, A. L. New guidelines for weight gain during pregnancy: what obstetrician/gynecologists should know. *Curr Opin Obstet Gynecol* **21**, 521–6 (2009).
2. Lindberg, S. *et al.* Prevalence and Predictors of Unhealthy Weight Gain in Pregnancy. *WMJ* **115**, 233–7 (2016).
3. Kowal, C., Kuk, J. & Tamim, H. Characteristics of weight gain in pregnancy among Canadian women. *Matern Child Health J* **16**, 668–76 (2012).
4. Woolcott, C. G., Dodds, L. ., Ashley-Martin, J. ., Piccinini-Vallis, H. Distribution of pregnancy-related weight measures. *Can. Fam. Physician* **62**, e400-406 (2016).
5. Nova Scotia Atlee Perinatal Database Report of Indicators: 2010 - 2019. <https://rcp.nshealth.ca/publications/nsapd-report-indicators-2010-2019> (2020).
6. Goldstein, R. F. *et al.* Association of Gestational Weight Gain With Maternal and Infant Outcomes: A Systematic Review and Meta-analysis. *JAMA* **317**, 2207–2225 (2017).
7. McDowell, M., Cain, M. A. & Brumley, J. Excessive Gestational Weight Gain. *J Midwifery Womens Health* <https://doi.org/10.1111/jmwh.12927> (2018) doi:10.1111/jmwh.12927.
8. Rogozinska, E. *et al.* Gestational weight gain outside the Institute of Medicine recommendations and adverse pregnancy outcomes: analysis using individual participant data from randomised trials. *BMC Pregnancy Childbirth* **19**, 322 (2019).
9. Adams, E. L. *et al.* Patterns of Gestational Weight Gain and Infants Born Large-for-Gestational Age Across Consecutive Pregnancies. *Womens Health Issues* **29**, 194–200 (2019).
10. Dzakpasu, S. *et al.* Contribution of prepregnancy body mass index and gestational weight gain to adverse neonatal outcomes: population attributable fractions for Canada. *BMC Pregnancy Childbirth* **15**, 21 (2015).
11. Tie, H. T. *et al.* Risk of childhood overweight or obesity associated with excessive weight gain during pregnancy: a meta-analysis. *Arch Gynecol Obstet* **289**, 247–57 (2014).

12. Sulistianingsih, A. R., Nurjazuli, N., Kartasurya, M. I., Musthofa, S. B. & Reuwpassa, J. O. Evidence for a perinatal origin of childhood obesity: a systematic review and meta-analysis of risk and protective factors. *Osong Public Health Res. Perspect.* <https://doi.org/10.24171/j.phrp.2025.0351> (2026) doi:10.24171/j.phrp.2025.0351.
13. World_Health_Organization. Childhood Overweight and Obesity. <http://www.who.int/dietphysicalactivity/childhood/en/> (2015).
14. Ludwig, D. S. Childhood obesity--the shape of things to come. *N Engl J Med* **357**, 2325–7 (2007).
15. Weiss, R. & Kaufman, F. R. Metabolic complications of childhood obesity: identifying and mitigating the risk. *Diabetes Care* **31 Suppl 2**, S310-6 (2008).
16. Rhodes, E. T. & Ludwig, D. S. Childhood obesity as a chronic disease: keeping the weight off. *JAMA* **298**, 1695–6 (2007).
17. Freedman, D. S. *et al.* The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics* **115**, 22–7 (2005).
18. Gardner, D. S. *et al.* Contribution of early weight gain to childhood overweight and metabolic health: a longitudinal study (EarlyBird 36). *Pediatrics* **123**, e67-73 (2009).
19. Reilly, J. J. & Kelly, J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int. J. Obes.* **35**, 891–8 (2011).
20. Ryder, J. R., Jacobs, D. R., Sinaiko, A. R., Kornblum, A. P. & Steinberger, J. Longitudinal Changes in Weight Status from Childhood and Adolescence to Adulthood. *J Pediatr* <https://doi.org/10.1016/j.jpeds.2019.07.035> (2019) doi:10.1016/j.jpeds.2019.07.035.
21. Hill, B. *et al.* Is parity a risk factor for excessive weight gain during pregnancy and postpartum weight retention? A systematic review and meta-analysis. *Obes Rev* **18**, 755–764 (2017).
22. Piccinini-Vallis, H., Brown, J. B., Ryan, B. L., McDonald, S. D. & Stewart, M. Women's Views on Advice About Weight Gain in Pregnancy: A Grounded Theory Study. *Matern Child Health J* **25**, 1717–1724 (2021).
23. Mehrabi, F., Ahmaripour, N., Jalali-Farahani, S. & Amiri, P. Barriers to weight management in pregnant mothers with obesity: a qualitative study on mothers with low socioeconomic background. *BMC Pregnancy Childbirth* **21**, 779 (2021).
24. Piccinini-Vallis, H. A. Weight management over the reproductive years for adult women living with obesity. (2021).
25. Wharton, S. *et al.* Obesity in adults: a clinical practice guideline. *CMAJ* **192**, E875–E891 (2020).
26. Teulings, N., Masconi, K. L., Ozanne, S. E., Aiken, C. E. & Wood, A. M. Effect of interpregnancy weight change on perinatal outcomes: systematic review and meta-analysis. *BMC Pregnancy Childbirth* **19**, 386 (2019).
27. Timmermans, Y. E. G. *et al.* The impact of interpregnancy weight change on perinatal outcomes in women and their children: A systematic review and meta-analysis. *Obes Rev* **21**, e12974 (2020).

28. Nagpal, T. S. *et al.* Does prepregnancy weight change have an effect on subsequent pregnancy health outcomes? A systematic review and meta-analysis. *Obes Rev* **23**, e13324 (2022).
29. Abrams, B. F., Leonard, S. A., Kan, P., Lyell, D. J. & Carmichael, S. L. Interpregnancy Weight Change: Associations with Severe Maternal Morbidity and Neonatal Outcomes. *Am J Obstet Gynecol MFM* 100596 (2022) doi:10.1016/j.ajogmf.2022.100596.
30. Hong, Y. H. & Lee, J. E. Large for Gestational Age and Obesity-Related Comorbidities. *J Obes Metab Syndr* **30**, 124–131 (2021).
31. Santos, I., Sniehotta, F. F., Marques, M. M., Carraca, E. V. & Teixeira, P. J. Prevalence of personal weight control attempts in adults: a systematic review and meta-analysis. *Obes Rev* **18**, 32–50 (2017).
32. Busetto, L. *et al.* Mechanisms of weight regain. *Eur. J. Intern. Med.* **93**, 3–7 (2021).
33. Wilding, J. P. H. *et al.* Weight regain and cardiometabolic effects after withdrawal of semaglutide: The STEP 1 trial extension. *Diabetes Obes. Metab.* **24**, 1553–1564 (2022).
34. Berg, S., Stickle, H., Rose, S. J. & Nemec, E. C. Discontinuing glucagon-like peptide-1 receptor agonists and body habitus: A systematic review and meta-analysis. *Obes. Rev. Off. J. Int. Assoc. Study Obes.* e13929 (2025) doi:10.1111/obr.13929.
35. Hjartaker, A., Laake, P. & Lund, E. Body mass index and weight change attempts among adult women. The Norwegian Women and Cancer Study. *Eur J Public Health* **11**, 141–6 (2001).
36. Lemon, S. C., Rosal, M. C., Zapka, J., Borg, A. & Andersen, V. Contributions of weight perceptions to weight loss attempts: differences by body mass index and gender. *Body Image* **6**, 90–6 (2009).
37. Dulloo, A. G. & Montani, J. P. Pathways from dieting to weight regain, to obesity and to the metabolic syndrome: an overview. *Obes Rev* **16 Suppl 1**, 1–6 (2015).
38. Rhee, E. J. Weight Cycling and Its Cardiometabolic Impact. *J Obes Metab Syndr* **26**, 237–242 (2017).
39. Montani, J. P., Schutz, Y. & Dulloo, A. G. Dieting and weight cycling as risk factors for cardiometabolic diseases: who is really at risk? *Obes Rev* **16 Suppl 1**, 7–18 (2015).
40. Lang, A. Y., Harrison, C. L. & Boyle, J. A. Preconception Lifestyle and Weight-Related Behaviors by Maternal Body Mass Index: A Cross-Sectional Study of Pregnant Women. *Nutrients* **11**, (2019).
41. Carey, K. J. & Vitek, W. Weight Cycling in Women: Adaptation or Risk? *Semin Reprod Med* <https://doi.org/10.1055/s-0040-1721418> (2020) doi:10.1055/s-0040-1721418.
42. Mumford, S. L., Siega-Riz, A. M., Herring, A. & Evenson, K. R. Dietary restraint and gestational weight gain. *J Am Diet Assoc* **108**, 1646–53 (2008).
43. Conway, R., Reddy, S. & Davies, J. Dietary restraint and weight gain during pregnancy. *Eur J Clin Nutr* **53**, 849–53 (1999).
44. Heery, E., Wall, P. G., Kelleher, C. C. & McAuliffe, F. M. Effects of dietary restraint and weight gain attitudes on gestational weight gain. *Appetite* **107**, 501–510 (2016).
45. Nagpal, T. S. *et al.* Are pre-pregnancy weight fluctuations and adherence to prenatal nutrition and exercise recommendations related to excessive gestational weight gain? *J Behav Med*

<https://doi.org/10.1007/s10865-020-00156-z> (2020) doi:10.1007/s10865-020-00156-z.

46. Leblanc, E. S., Smith, N. X., Vesco, K. K., Paul, I. M. & Stevens, V. J. Weight Loss Prior to Pregnancy and Subsequent Gestational Weight Gain: Prepare, a Randomized Clinical Trial. *Am J Obstet Gynecol* <https://doi.org/10.1016/j.ajog.2020.07.027> (2020) doi:10.1016/j.ajog.2020.07.027.
47. Piccinini-Vallis, H., Woolcott, C., Miller, L., Snelgrove-Clarke, E. & Cahill, S. The Relationship Between Pre-Pregnancy Attempts to Lose Weight and Gestational Weight Gain: An Exploratory Primary Care Prospective Cohort Study. *J Obstet Gynaecol Can* **43**, 337-343 e1 (2021).
48. Duah, J. & Seifer, D. B. Medical therapy to treat obesity and optimize fertility in women of reproductive age: a narrative review. *Reprod. Biol. Endocrinol. RBE* **23**, 2 (2025).
49. Drummond, R. F., Seif, K. E. & Reece, E. A. Glucagon-like peptide-1 receptor agonist use in pregnancy: a review. *Am. J. Obstet. Gynecol.* **232**, 17–25 (2025).
50. Tabachnick, B. G. *Using Multivariate Statistics*. (Pearson Higher Ed USA, 2014).
51. Muse, M. E. et al. Maternal History of Weight Loss and Prospective Gestational Weight Gain. *JAMA Netw. Open* **9**, e267931 (2026).
52. Maya, J. et al. Gestational Weight Gain and Pregnancy Outcomes After GLP-1 Receptor Agonist Discontinuation. *JAMA* **334**, 2186–2196 (2025).
53. Ozbek, L. et al. Safety of GLP-1 and Dual GLP-1/GIP Receptor Agonists in Preconception, Pregnancy, and Lactation: A Systematic Review of Maternal, Fetal, and Neonatal Outcomes. *Diabetes Obes. Metab.* **28**, 4503–4528 (2026).
54. Hviid, K. V. R. et al. Periconceptional GLP-1 receptor agonist exposure and obstetric outcomes: a Danish nationwide cohort study. *Hum. Reprod. Open* **2026**, hoag015 (2026).
55. Imbroane, M. R., LeMoine, F. & Nau, C. T. Preconception glucagon-like peptide-1 receptor agonist use associated with decreased risk of adverse obstetrical outcomes. *Am. J. Obstet. Gynecol.* **233**, 116.e1-116.e7 (2025).
56. Pondugula, N., Culhane, J. F., Lundsberg, L. S., Partridge, C. & Merriam, A. A. Gestational Weight Gain and Hypertensive Disorders of Pregnancy With Prepregnancy and Early Pregnancy Glucagon-Like Peptide-1 Receptor Agonist Exposure. *Obstet. Gynecol.* **147**, 293–302 (2026).
57. Mandal, L., Andersen, L. U., Luef, B. M., Tanvig, M. H. & Vinter, C. A. Impact of semaglutide exposure on fetal and neonatal outcomes in pregnant women: a systematic review. *Eur. J. Obstet. Gynecol. Reprod. Biol.* **317**, 114836 (2026).