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Systematic Review

A Scoping Review of Perinatal Outcomes for Canadian Individuals with Obesity 1980–2020

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Abstract

Background: Pre-pregnancy obesity has long been associated with a higher risk of antepartum, intrapartum, and postpartum complications, leading to classifying all patients within the WHO obese body mass index range (BMI $\geq$30 kg/m^2) as high-risk. Excessive risk classification can lead to over-treatment and iatrogenic harm. Research supporting these increased risks comes from a variety of countries with different baseline population health and perinatal health care systems. Recent research documents that parturients with high pre-pregnancy BMI can have uncomplicated pregnancies and births. Here, the relationship between obesity and perinatal outcomes in a Canadian population is investigated using Canada as a model of a country with stable nutrition sufficiency and universal access to health care. **Methods**: We searched electronic databases PubMed, Ovid MEDLINE, and CINAHL for peer-reviewed articles in English that examined perinatal outcomes of singleton pregnancies in Canada between 1980 and 2020 based on pre-pregnancy obesity as the exposure of interest. **Results**: The search yielded 1946 results. After full-text screening, 21 articles met criteria for analysis. The impact of obesity on preterm labour, preterm birth, post-term delivery, labour induction, post-partum hemorrhage, and parturient morbidity and mortality in Canada remains inconclusive. The risk of hypertensive disorders, gestational diabetes, shoulder dystocia, and cesarean section delivery was significantly elevated as BMI increased. Operative vaginal delivery was less common in those with pre-pregnancy obesity. **Conclusions**: Though certain adverse perinatal outcomes are associated with increasing pre-pregnancy BMI, given the wide range of results and range of risk values, further research is required to better delineate comorbid risk factors that contribute to poorer outcomes. Nation-specific outcomes that reflect the accessibility of perinatal care and population health are a necessary baseline for the development of accurate health management guidelines.

Keywords: obesity; perinatal; antenatal; intrapartum; postpartum; preterm labor; cesarean section; postpartum hemorrhage; gestational diabetes

1. Introduction

Rising rates of obesity in North America since 1980 have increased rates of perinatal medical complications. Canada is an example of a country with wide variations in obesity rates between provinces and rural and urban areas. The incidence of obesity has increased in Canadian women of childbearing age for more than 30 years. In Nova Scotia, the proportion of women with obesity (pre-pregnancy weight $\geq$90 kg) increased from 4.1% to 10.7% from 1988 to 2001 [1]. According to 2018 Canadian population statistics, the rate of obesity in women of childbearing age (18–49 years old) was 22.7% [2,3], with the overall adult obesity rate averaging 26.8% [4]. Ontario's adult obesity rate met the national average at 26.1%; however, provincial rates ranged from 40.2% in Newfoundland and Labrador and 23.1% in British Columbia [4].

The Word Health Organization defines obesity as a body mass index (BMI) $\geq$30 (kg/m^2) and as abnormal or excessive fat accumulation that presents a risk to health. Obesity is further divided into classes (Table 1) [5]. Pre-pregnancy body mass index (BMI) is associated with perinatal outcomes. Numerous studies from different countries have shown that individuals with pre-pregnancy obesity are at a higher risk for hypertensive disorders (such as pre-eclampsia), parturient glucose disturbances (impaired glucose tolerance, gestational diabetes), prolonged stages of labor, complicated labour (shoulder dystocia, postpartum hemorrhage), caesarean section, and adverse neonatal outcomes (macrosomia, NICU admission) [6–8]. Attempting to avoid morbidity, many national medical guidelines including Canadian, British, and American, classify all individuals with pre-pregnancy obesity as high-risk patients. This can lead to utilizing medical treatments that are unnecessary, cause iatrogenic harm, and restrict patient's choice during pregnancy [9–11]. Recent research has shown that many individuals with high pre-pregnancy BMI can have uncomplicated pregnancies and births [12–15].

To address the conflicting information, a comprehensive approach must be taken to examine the impact of obesity on perinatal outcomes. Understanding the full scope of outcomes that are associated with high pre-pregnancy BMI is important for clinicians and policymakers in establish-

Table 1. WHO Obesity Classification.

Obesity class	BMI range
Class I	30–35.9
Class II	36–39.9
Class III	>40

ing evidence-based guidelines. The relationship between obesity with adverse perinatal outcomes in a Canadian context has not been reviewed comprehensively. This scoping review focuses on the relationship between pre-pregnancy obesity with adverse perinatal outcomes in Canadian patients.

2. Methods

This scoping review was based on the framework described by Arksey and O'Malley to explore the broad literature on maternal obesity and perinatal outcomes in Canadian populations [16]. A scoping review is unlike a systematic review in that it focuses on a broader research question and includes a variety of study designs to map essential concepts in an area of literature. Arksey and O'Malley outlined five key stages of conducting a scoping review: (1) identifying the research question, (2) finding the relevant studies, (3) selecting suitable studies, (4) charting the data, and (5) collating, summarizing, and reporting results [16]. The framework described, though stepwise in nature, was an iterative process that allowed the refinement of keywords and search terms as familiarity was gained with the literature.

The aim was to review the literature that examined the impact of high pre-pregnancy BMI on perinatal outcomes. The research question was, "What are the perinatal outcomes of individuals with pre-pregnancy obesity in Canada?". Perinatal outcomes included pregnancy outcomes such as gestational diabetes mellitus (GDM), gestational hypertension, miscarriage; intrapartum and postpartum outcomes like prolonged labor, shoulder dystocia, anesthesia requirement; and neonatal outcomes including Apgar score, stillbirth, and macrosomia. Pre-pregnancy obesity was defined according to the World Health Organization's classification of BMI of $\geq$30 kg/m^2. In studies comparing outcomes for individuals with obesity to those of normal weight, normal weight was defined as a BMI range of 18.5–24.9 kg/m^2.

A literature search to find relevant articles was completed on the electronic databases PubMed, Ovid MEDLINE, and CINAHL. The manuscripts were restricted to peer-reviewed studies that were available in English. The inclusion criteria were all primary descriptive research studies that examined perinatal outcomes in singleton pregnancies in Canada from January 1, 1980, to April 30, 2020, based on obesity as an exposure of interest. Studies on multiple gestations, bariatric surgery, long-term outcomes, or assisted reproduction were excluded. Key terms selected for the search included "obesity" and "body mass index" (and their variations such as obese, obes*, BMI) in combination with the pregnancy complications outlined above. The combined search terms were further combined with all Canadian provinces and territories as well as "Canada" (and their respective variations). All key terms were adapted to the field headings and MeSH terms suitable for each database.

The search yielded 1946 articles (MEDLINE = 479, PubMed = 1206, CINAHL = 261) and 1157 articles remained after removing duplicates, which went through title and abstract screening. From there, 145 articles underwent full-text screening, and 39 studies were selected to include in the review. Two reviewers screened the articles independently for both screening steps. Studies that only examined neonatal outcomes were removed from the analysis, which left 21 studies for full analysis. Conflicts were resolved through discussion until consensus was reached. For the charting process, all articles meeting the inclusion criteria were reviewed and relevant data pertaining to study design, study setting, study population, obesity classification, and perinatal outcomes was extracted. The studies are summarized in Table 2 (Ref. [17–35]). Perinatal outcomes (except neonatal outcomes) that were significantly associated with pre-pregnancy obesity were extracted from each paper and are presented in Table 3 (Ref. [17–35]).

3. Results

A Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flowchart is presented in Fig. 1. Of the 21 articles included in the full analysis, 7 (33.3%) were in Ontario [17–22,36]; 5 (23.8%) in Quebec [23–27]; 3 (14.3%) in Alberta [28–30]; 1 (4.8%) in Newfoundland and Labrador [31]; 1 (4.8%) in British Columbia [32]; 1 (4.8%) in Nova Scotia [33]; 1 (4.8%) in Saskatchewan [37]; and 2 (9.5%) were Canada-wide [34,35]. Fifteen of the 21 studies reported the obesity rate in their cohort, which ranged from 6.1% to 22%; one study looked at extreme obesity (BMI $\geq$50) and reported a 0.6% obesity rate [31]. Two studies [36,37] that did not directly compare perinatal outcomes between parturients of normal weight and those with pre-pregnancy obesity were not included in Tables 2,3, but were mentioned in the results as they covered the rare outcome of maternal morbidity and mortality and stillbirth. Across the 21 studies sample sizes ranged from 180 stillbirth cases in one Canadian tertiary care centre [37] to 506,483 cases that included singleton livebirths and stillbirths in Ontario [17]. One study was a case series, five were prospective cohorts, and 16 were designed as retrospective cohort studies.

The most frequently used measure of perinatal outcomes risk was adjusted odds ratios (ORs) [19,22,23,25,26,28–31,34,35], followed by prevalence of outcomes [18,20,27,31,32], and adjusted relative risks (RRs) [17,21,33]. One study used incidence of outcome [24] and another study used a composite score of obesity-related complica-

Table 2. Study Characteristics.

Study	N, Years included, Location	Study design	Measure of risk
Abenhaim *et al*., 2007 [23]	18,643 deliveries, 1987–1997, at a tertiary care centre in Quebec	Retrospective cohort	(1) AOR for categorical variable; adjusted for maternal age, smoking, parity, pre-existing DM (2) *t*-test for continuous variables
Berger *et al*., 2020 [17][a]	506,483 singleton livebirths, 2012–2016, in Ontario	Retrospective cohort	ARR and 95% CI
Crane *et al*., 2013 [31][b]	12,891 singleton deliveries, 2002–2011, in Newfoundland and Labrador	Retrospective Cohort	(1) Prevalence of perinatal outcomes (2) AORs and 95% CI
Davenport *et al*., 2010 [18][c]	27,986 singleton deliveries, 2000–2009 in London, Ontario	Retrospective cohort	(1) AORs and 95% CI (2) Prevalence of metabolic disorders
Dzakpasu *et al*., 2014 [34]	5591 singleton live births–2006, pan-Canadian survey data	Retrospective cohort	AORs and 95% CI
Dzakpasu *et al*., 2015 [35]	5930 singleton live births, 2005–2006, pan-Canadian survey data	Retrospective cohort	(1) AORs and 95% CI (2) APAF and 95% CI
El-Chaar *et al*., 2013 [19]	6674 singleton deliveries, 2007–2010 in a tertiary care centre in Ottawa, Ontario	Retrospective cohort	(1) AORs and CI 95% Adjusted for maternal age, parity, education quintiles, family income quintiles
Gasse *et al*., 2019 [24]	4683 deliveries, 2015–2016 in Quebec	Prospective cohort	Incidence of adverse outcomes
MacInnis *et al*., 2016 [33]	66,689 singleton deliveries, 2004–2014 in Nova Scotia	Retrospective cohort	ARRs and 95% CI Adjusted for maternal age, area-level income quintile, area of residence, parity
Richardson *et al*., 2017 [20]	29,212 live singleton deliveries 1999–2010, in London, Ontario	Retrospective cohort	Prevalence of each outcome in each BMI group
Shen *et al*., 2017 [21]	76,333 singleton deliveries 2002–2009 that had induction of labour in a tertiary care centre in Toronto, Ontario	Retrospective cohort	AOR, 95% CI
Ronzoni *et al*., 2015 [22]	7543 singleton deliveries 2003–2010 that had induction of labour in a tertiary care centre in Ontario	Retrospective cohort	(1) AOR (2) Prevalence (%) of outcomes in each group
Thuot *et al*., 2013 [25]	1386 pregnancies to women with asthma in Quebec	Retrospective cohort	(1) AORs with 95% CI
Schummers *et al*., 2015 [32][d]	226,958 singleton deliveries between 2004–2012 in British Columbia	Retrospective cohort	Absolute risk of outcome
Sherrard *et al*., 2007 [26]	63,390 singleton deliveries between 1978 to 2001 in a tertiary care centre in Quebec	Retrospective cohort	AOR and 95% CI
Verret-Chalifour *et al*., 2015 [27]	6592 live singleton deliveries between 2005–2010 in Quebec	Retrospective cohort	Prevalence (%) of outcomes
Vinturache *et al*., 2014 [28]	1996 term, singleton, cephalic deliveries between May 2008 and December 2010 in Alberta	Prospective cohort	AOR and 95% CI
Vinturache *et al*., 2015 [29]	1996 term, singleton, cephalic deliveries between May 2008 and December 2010 in Alberta	Prospective cohort	AOR and 95% CI
Vinturache *et al*., 2017 [30]	1996 term, singleton, cephalic deliveries between May 2008 and December 2010 in Alberta	Prospective cohort	Prevalence (%)

[a]Reference group did not have obesity, diabetes, or hypertension; [b]Comparison group BMI $\geq$50; [c]Control group: BMI $\leq$25; comparison group BMI $\geq$30; [d] Pregnancies complicated with pre-existing HTN or DM were excluded.

Abbreviations used in table: AOR, adjusted odds ratio; ARR, adjusted relative risk; CI, confidence interval; APAF, adjusted population attributable fraction.

Table 3. Summary of Canadian Studies of Perinatal Outcomes for Individuals with Obesity.

Article title	Obesity rates	Pregnancy outcomes	Intrapartum and postpartum outcomes
Abenhaim *et al*., 2007 [23]		Obese vs normal weight (OR; 95% CI) Preeclampsia (4.65; 3.71–5.83) GDM (3.22; 2.68–3.87) Gestational HTN (2.01; 1.64–2.45) Preterm labour (1.19; 0.98–1.15) PTB <32 weeks (1.20; 0.77–1.87) PTB 32–36 weeks (1.60; 1.32–1.94) Pregnancy > 42 weeks (0.84; 0.55–1.28) Morbidly obese vs normal weight (OR; 95% CI) Preeclampsia (6.26; 3.48–11.26) GDM (4.71; 2.89–7.67) Gestational HTN (2.77; 1.60–4.78) Preterm labour (1.67; 0.99–2.84) PTB 32–36 weeks (2.43;1.46–4.05) PTB <32 weeks (2.14; 0.77–5.95) Pregnancy >42 weeks (0.76; 0.19–3.10)	Obese vs normal weight (OR; 95% CI) Induction of labour (1.60; 1.40–1.82) C–section (1.85; 1.62–2.11) Shoulder dystocia (1.89; 1.65–2.16) Postpartum hemorrhage (1.24; 0.91–1.69) OVD (0.66; 0.50–0.86) Morbidly obese vs normal weight (OR; 95% CI) Induction of labour (2.06; 1.38–3.07) C-section (2.92; 1.97–4.34) Shoulder dystocia (2.6; 0.92–7.31) Postpartum hemorrhage (3.14; 1.65–5.97) OVD (0.57; 0.20–1.58)
Berger *et al*., 2020 [17]	17.8% obesity prevalence 2.70% co-occurrence with DM 3.09% co-occurrence with HTN	Obesity only vs no DOH (ARR; 95%CI) PTB <34 weeks (1.23; 1.16–1.31) Provider–initiated PTB <34 weeks (1.49; 1.35–1.65) Spontaneous PTB <34 weeks (1.10; 1.02–1.19) PTB <37 weeks (1.14; 1.10–1.17) Provider–initiated PTB <37 weeks (1.43; 1.37–1.49) Spontaneous PTB <37 weeks (0.97; 0.93–1.01) PTB <37 weeks + preeclampsia (1.91; 1.68–2.16) PTB <37 weeks + SGA (1.01; 0.88–1.16) PTB <37 weeks + LGA (2.69; 2.41–2.99)	
Crane *et al*., 2013 [31]	Extreme obesity 0.6%	Extreme Obesity vs Normal Weight **Gestational HTN (19.7% vs 4.8%) (AOR 1.56; 1.33–0.82)** **GDM (21.1% vs 1.5%) (AOR 2.04; 1.74–2.38)** PTB <37 weeks (8.5% vs 7.1%) PTB <34 weeks (1.4% vs 1.8%)	Extreme Obesity vs Normal Weight Labour induction (39.0% vs 30.5%) **Non-elective CS (31.7% vs 13.2%)** **All CS (60.6% vs 25.0%) (AOR 1.46; 1.29–1.65)** **Shoulder dystocia (7.1% vs 1.4%) (AOR 1.51; 1.05–2.19)** Post-partum hemorrhage (9.9% vs 4.9%) Maternal death (1.4% vs 0%)

Table 3. Continued.

Article title	Obesity rates	Pregnancy outcomes	Intrapartum and postpartum outcomes
Davenport *et al*., 2010 [18]		Normal weight (%) GDM (2.0) T2DM (0.3) IGT (0.7) Class I obesity (%) GDM (7.5) T2DM (1.1) IGT (1.4) Class II obesity (%) GDM (0.4) T2DM (2.2) IGT (1.6) Class III obesity (%) GDM (17.0) T2DM (4.1) IGT (2.0) Prevalence of prepregnancy overweight and obesity did not explain the increase in maternal glucose disorders.	
Dzakpasu *et al*., 2014 [34]	Prevalence of obesity 13.3%		Obese vs Normal Weight (AOR, 95% CI) **All CS (1.95; 1.61–2.36)** **Unplanned CS (2.29; 1.77–2.96)** **Planned CS (1.45; 1.13–1.85)**
Dzakpasu *et al*., 2015 [35]	Prevalence of obesity 13.5%	Obese vs Normal Weight (AOR, 95% CI) PTB <37 weeks (1.02; 0.73–1.42)	
El-Chaar *et al*., 2013 [19]		Class I obesity vs normal weight (AOR, 95% CI) Preeclampsia (1.91; 1.27–2.90) Gestational HTN (3.07; 2.10–4.49) GDM (2.84; 2.00–4.02) Preterm labour (0.61; 0.39–0.96) Class II obesity vs normal weight (AOR, 95% CI) Preeclampsia (2.48; 1.42–4.32) Gestational HTN (4.80; 3.00–7:70) GDM (4.04; 2.57–6.35) Preterm labour (0.75; 0.40–1.40)	Class I obesity vs normal weight (AOR, 95% CI) OVD (0.59; 0.41–0.85) CS (1.41; 1.27–1.54) Elective CS (1.40; 1.19–1.64) Emergency CS (1.45; 1.26–1.67) Primary CS (1.35; 1.19–1.53) Previous CS (1.03; 0.97–1.09) Labour induction (1.29; 1.15–1.46) Class II obesity vs normal weight (AOR, 95% CI) OVD (0.80; 0.48–1.34)

Table 3. Continued.

Article title	Obesity rates	Pregnancy outcomes	Intrapartum and postpartum outcomes
			CS (1.50; 1.31–1.72)
		Class III obesity vs normal weight (AOR, 95% CI)	Elective CS (1.63; 1.31–2.02),
		Preeclampsia (5.19; 3.32–8.13)	Emergency CS (1.40; 1.13–1.75)
		Gestational HTN (3.72; 2.22–6.25)	Primary CS (1.38; 1.13–1.67)
		GDM (5.70; 3.73–8.70)	Previous CS (1.07; 1.00–1.15)
		Preterm labour (0.75; 0.39–1.45)	Labour induction (1.62; 1.39–1.88)
			Class III obesity vs normal weight (AOR, 95% CI)
			OVD (0.52; 0.28–0.97)
			CS (1.49; 1.30–1.70)
			Elective CS (1.64; 1.31–2.04)
			Emergency CS (1.32; 1.06–1.65)
			Primary CS (1.46; 1.23–1.73)
			Previous CS (1.03; 0.95–1.12)
			Labour induction (1.67; 1.43–1.93)
Gasse *et al*., 2019 [24]		Normal weight (%)	
		HDP (8.5)	
		Preeclampsia (4.3)	
		Preterm preeclampsia (0.6)	
		Obesity (%)	
		HDP (22.5)	
		Preeclampsia (10.2)	
		Preterm preeclampsia (1.6)	
MacInnis *et al*., 2016 [33]	Prevalence of obesity 22%	Obese class I vs Normal weight (ARR, 95% CI)	Obese class I vs Normal weight (ARR, 95% CI)
		GDM (4.82; 4.23–5.49)	Labour induction (1.44; 1.38–1.49)
		HDP (2.20; 1.81–2.66)	CS (1.51; 1.44–1.58)
		Obese class II vs Normal weight (ARR, 95% CI)	Obese class II vs Normal weight (ARR, 95% CI)
		GDM (7.17; 6.20–8.30)	Labour induction (1.61; 1.53–1.69)
		HDP (2.22; 1.73–2.86)	CS (1.69; 1.60–1.70)
		Obese class III vs Normal weight (ARR, 95% CI)	Obese class III vs Normal weight (ARR, 95% CI)
		GDM (8.96; 7.65–10.49)	Labour induction (1.69; 1.60–1.79)
		HDP (3.90; 3.04–5.00)	CS (2.05; 1.94–2.19)

Table 3. Continued.

Article title	Obesity rates	Pregnancy outcomes	Intrapartum and postpartum outcomes
Richardson *et al*., 2017 [20]	Prevalence of obesity 16%	Normal weight vs Obese (%): **Gestational HTN (4.1% vs 14.5%)** **Preeclampsia/eclampsia (2.9% vs 7.8%)** **GDM (2.1% vs 10.9%)** **PTB <37 weeks (6.5% vs 9.2%)**	Normal weight vs Obese (%): **CS (18.9% vs 34.7%)**
Shen *et al*., 2017 [21]	Prevalence of obesity 12.9%	Obese (ARR, 95% CI) **Gestational HTN 2.81 (2.07–3.81)** **Preeclampsia 3.38 (2.40–4.76)**	
Ronzoni *et al*., 2015 [22]	Prevalence of obesity of 12.9%		Normal weight (%) Vaginal delivery (75.5) OVD (27.6), CS (24.7) Obese (%) **Vaginal delivery (63.1)** **OVD (18.7)** **CS (36.9)** **AOR of CS for Obese group = 2.307**
Thuot *et al*., 2013 [25]	Prevalence of obesity 16.2%	Obese vs Normal Weight PTB <37 weeks (0.7; 0.4–1.2)	
Schummers *et al*., 2015 [32]	Prevalence of obesity 12.0%	Normal weight PTB <37 weeks (7.1) Preeclampsia (3.4) GDM (6.1) PTB <32 weeks (0.6) Medically indicated birth <37 weeks (1.6) Class I obesity PTB <37 weeks (8.4) **Preeclampsia (10.0)** **GDM (13.7)** PTB <32 weeks (0.6) **Medically indicated birth <37 weeks (2.5)** Class II obesity PTB <37 weeks (8.8) **Preeclampsia (12.8)**	Normal weight Shoulder dystocia (3.5) Birth injury secondary to shoulder dystocia (0.1) CS (26.5) Postpartum hemorrhage (0.7) Maternal mortality or severe morbidity (0.6) Obesity class I **Shoulder dystocia (4.1)** **Birth injury secondary to shoulder dystocia (0.1)** **CS (38.2)** Postpartum hemorrhage (0.8) Maternal mortality or severe morbidity (0.6) Obesity class II **Shoulder dystocia (4.4)** **Birth injury secondary to shoulder dystocia (0.2)**

Table 3. Continued.

Article title	Obesity rates	Pregnancy outcomes	Intrapartum and postpartum outcomes
		GDM (16.6) PTB <32 weeks (0.6) **Medically indicated birth <37 weeks (3.4)** Class III obesity PTB <37 weeks (10.3) **Preeclampsia (16.3)** **GDM (20.8)** PTB <32 weeks (0.5) **Medically indicated birth <37 weeks (4.1)**	**CS (43.1)** Postpartum hemorrhage (0.7) Maternal mortality or severe morbidity (0.5) Obesity class III **Shoulder dystocia (6.1)** **Birth injury secondary to shoulder dystocia (0.3)** **CS (49.7)** Postpartum hemorrhage (0.3) Maternal mortality or severe morbidity (0.6)
Sherrard *et al*., 2007 [26]			Obese vs Normal Weight (AOR, 95% CI) Primary CS before labour (2.01; 1.39–2.90) Primary CS after labour onset (2.12; 1.86–2.42) Repeat CS before labour (1.85; 1.44–2.37) Repeat CS after labour onset (1.96; 1.11–3.47)
Verret-Chalifour *et al*., 2015 [27]	Prevalence of obesity 12.6%	Obese vs Normal Weight **GDM (15.9% vs 3.5%)** **HDP (23.0% vs 7.2%)**	Obese vs Normal Weight **Elective CS (15.0% vs 9.0%)** **Urgent CS (16.4% vs 10.3%)**
Vinturache *et al*., 2014 [28]	Prevalence of obesity 10%	Obese vs Normal Weight (aOR, 95% CI) **Gestational HTN (5.7; 3.7–8.8)** **Preeclampsia (5.3; 3.3–8.5)** **Eclampsia (10.6; 2.5–44.6)** **GDM (6.5; 3.7–11.2)**	Obese vs Normal Weight (AOR, 95% CI) **Labour induction (1.3; 1.0–1.7)**
Vinturache *et al*., 2015 [29]	Prevalence of obesity 10%	Obese vs Normal Weight (AOR, 95% CI) **Pregnancy complications**[1] **(3.0; 2.1–4.2)**	Obese vs Normal Weight (AOR, 95% CI) Vaginal delivery (0.7; 0.5–1.0) **OVD (0.4; 0.2–0.8)** **Emergency CS (2.5; 1.6–3.8)**
Vinturache *et al*., 2017 [30]	Prevalence of obesity 10%	Macrosomic vs non-macrosomic babies (AOR, 95% CI) **Maternal obesity (15.7% vs 10.0%)** Obese vs Normal Weight (mothers of macrosomic babies) (aOR, 95% CI) **Gestational HTN (19.4% vs 1.9%)** **Pre-eclampsia (16.1% vs 0.9)** GDM (6.5% vs 3.7%)	Obese vs Normal Weight (mothers of macrosomic babies) (AOR, 95% CI) Labour induction (48.4% vs 37.4%) Emergency CS (35.5% vs 22.4%) OVD (3.2% vs 4.7%)

[1]Pregnancy-induced hypertension, preeclampsia, eclampsia, diabetes mellitus and placenta previa.

Bolded outcomes indicate statistically significant findings.

Abbreviations used in table: AOR, adjusted odds ratio; ARR, adjusted relative risk; CI, confidence interval; CS, caesarean section; DM, diabetes mellitus; GDM, gestational diabetes mellitus; HDP, hypertensive disorders of pregnancies; HTN, hypertension; IGT, impaired glucose tolerance; OVD, operative vaginal delivery; PTB, preterm birth; T2DM, type 2 diabetes mellitus.

tions that included gestational diabetes and hypertensive disorders of pregnancy [29]. Each study adjusted for a different set of factors, but age, parity, and smoking status were frequently used. It is important to note that each study had a slightly different range of BMI for the control group; however, most studies used the WHO normal BMI class (18.5–24.9 kg/m^2) as the control group. Additionally, some studies did not report whether the values they had calculated were significantly different. The differing variables and comparators confound comparisons between studies as would be done in a meta-analysis. This review examined the following outcomes: preterm labour, preterm birth, post-term pregnancy, hypertensive disorders of pregnancy, gestational diabetes, labour induction, mode of delivery (Caesarean section, operative vaginal delivery), shoulder dystocia, and postpartum hemorrhage, because they were the most commonly reported in the analyzed manuscripts.

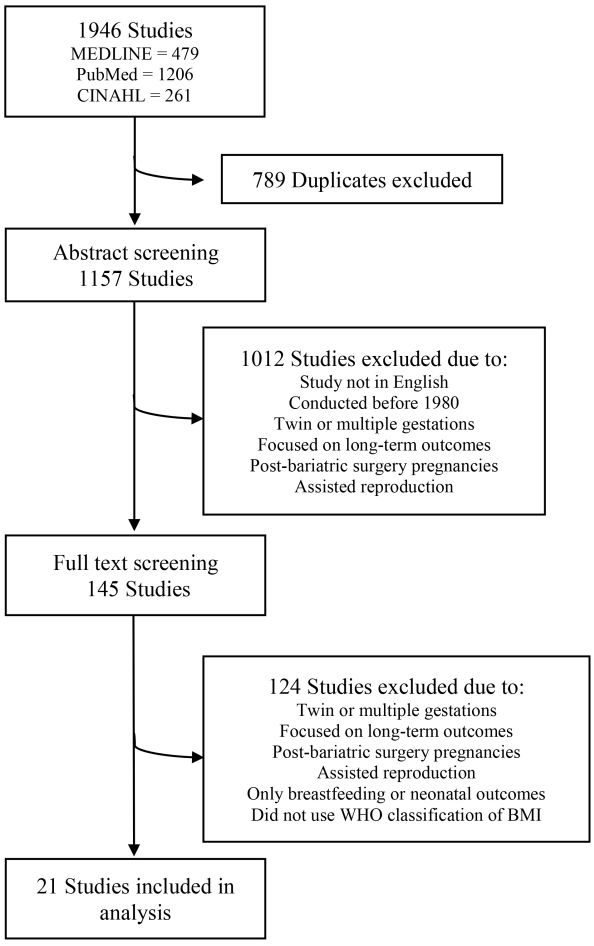

Fig. 1. PRISMA Chart.

3.1 Pregnancy Outcomes

3.1.1 Preterm Labour

Preterm labour (PTL) is defined as labour before 37 weeks of pregnancy with one study reporting preterm births between 32 and 36-weeks gestation [23]. The effect of obesity on PTL was inconclusive. One of the two studies that examined PTL found slightly decreased ORs for all three classes of obesity against the reference normal BMI group, though it was not a significant difference for any of the groups [19]. Abenhaim *et al.* [23] found slightly increased ORs for PTL. However, the confidence intervals of the ORs from the two studies overlap. Additionally, no study found significant differences between the ORs of PTL in different classes of obesity compared to the reference BMI.

3.1.2 Preterm Birth

Preterm birth (PTB) is defined as birth before 37 weeks of pregnancy. Different studies reviewed used differing definitions and some studies further subcategorized PTB into early and late. There were mixed results out of the 7 studies that reported on PTB [17,20,23,25,31,32,35]. Four of the studies found varying levels of increased risk of PTB in patients with obesity [17,20,23,31], only 1 of which was a significant increase from the control normal BMI group [20]. One study reported no difference in risk between the obese and control BMI groups for preterm birth [35]. The final study, which specifically looked at parturients with asthma, reported a slightly decreased but insignificant risk of PTB in those with obesity (AOR, 95% CI 0.7; 0.4–1.2) [25]. Further subcategorizing obesity into the WHO-defined classes, Abenhaim *et al.* [23] reported that the adjusted ORs for PTB for those with obesity class I and II (PTB $<$32 weeks 1.20, 0.77–1.87; PTB 32–36 weeks 1.60, 1.32–1.94) were lower than that of individuals with obesity class III (PTB $<$32 weeks 2.14, 0.77–5.95; PTB 32–36 weeks 2.43, 1.46–4.05), although the 95% confidence intervals overlapped. Neither group had a significantly increased risk for PTB compared to the control group. Similarly, Crane *et al.* [31] did not find a significant difference in rates of PTB between the control and BMI $>$50 groups. Some studies also reported risks of PTB less than 32 and 34 weeks, and PTB between 32–36 weeks [17,23]. There was not a significant difference in rates of PTB between gestational periods in those studies.

3.1.3 Post-Term Delivery

Post-term delivery (PTD) was defined as a birth in a pregnancy longer than 42 weeks [23]. Only one study calculated an adjusted OR for PTD, finding a slightly decreased but non-significant risk. Additionally, the AOR confidence intervals were large and crossed 1 for all obese patients (0.84; 0.55–1.28) and those labeled morbidly obese (0.76; 0.19–3.10) [23]. Given the lack of significance and low number of studies examining this outcome, the impact of obesity on PTD remains inconclusive.

3.1.4 Hypertensive Disorders of Pregnancy

Hypertensive disorders of pregnancy (HDP) include pre-existing hypertension, gestational hypertension, pre-

eclampsia, eclampsia, and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome. From the 12 studies that examined these outcomes [19–21,23,24,27–33], 11 found significantly elevated odds or prevalence of hypertensive disorders in individuals with obesity [19–21,24,27–32]. Three studies further subcategorized obesity into its WHO classifications and examined the prevalence of HDP compared to the reference group [19,23,33]. All three studies found that the odds of developing HDP increased with increasing class of obesity with AORs ranging from 2.01 for class I obesity to 3.90 for class III obesity (Table 3) [19,23,33]. Vinturache *et al*. [29] examined a composite outcome of pregnancy complications, which included gestational hypertension, pre-eclampsia, eclampsia, diabetes, and placenta previa and found significantly increased odds of that composite in patients with obesity. In a subgroup analysis of mothers of infants with macrosomia, defined as birth weight $\geq$4000 g, Vinturache *et al*. [30] found that the obesity rate was 50% higher for mothers of infants with macrosomia. The prevalence in this subgroup for gestational hypertension was 10 times higher than for women in the normal BMI range with newborns with macrosomia. Crane *et al*. [31] also found that the prevalence of gestational hypertension in those with extreme obesity was over 4 times greater than the control BMI (Table 3).

3.1.5 Gestational Diabetes

Gestational diabetes mellitus (GDM) was defined as diabetes that is first diagnosed during pregnancy. Eleven studies reported the impact of obesity on developing GDM [18–20,23,27–33], with seven finding a significantly increased risk in those with obesity [19,20,27–29,31,32]. One study mentioned above only examined a composite score of pregnancy complications and not GDM as an individual outcome [29]. The five studies that investigated the risk of GDM by subclass of obesity found that the risk increased with increasing BMI [18,19,23,32,33]. Furthermore, the adjusted OR for GDM was significantly higher for those with extreme obesity, defined as BMI >50, compared to the normal weight control group [31]. However, one study that explored the temporal trends of GDM and obesity concluded that the increased prevalence of GDM is not fully explained by pre-pregnancy BMI [18]. Another study found that in mothers of newborns with macrosomia, the group with obesity had an insignificantly higher prevalence of GDM compared to the control group [30].

Berger, *et al*. [17] examined preterm labour and birth related to preeclampsia in Ontario, finding a 2.70% co-occurrence with diabetes and obesity and a 3.09% co-occurrence with hypertension. The overall risk for preterm birth (PTB) <34 weeks was increased (1.23; 1.16–1.31). When this risk is divided into spontaneous PTB <34 weeks (1.10; 1.02–1.19) and provider-initiated PTB <34 weeks (1.49; 1.35–1.65), evidence for iatrogenic PTB emerges. The pattern is clearer for the contribution of hypertension management to PTB when comparing provider-initiated PTB <37 weeks (1.43; 1.37–1.49) to spontaneous PTB <37 weeks (0.97; 0.93–1.01). The risk of PTB <37 weeks complicated by preeclampsia is almost double that for those without obesity and preeclampsia (1.91; 1.68–2.16).

3.2 Intrapartum and Postpartum Outcomes

3.2.1 Labour Induction

Seven studies analysed the effect of obesity on the need for labour induction, defined as the stimulation of uterine contractions before labour has begun spontaneously [19,23,28–31,33]. Five studies found slightly increased but insignificant odds of labour induction [19,23,30,31,33], whereas two studies found a significant increase [28,29]. Odds of induction of labour was not significantly affected by increasing BMI class [19,23,33]. Crane *et al*. [31] found slightly higher but insignificant rates of labour induction in the extreme obesity cohort.

3.2.2 Shoulder Dystocia

Shoulder dystocia is a birth complication where one or more of the neonate's shoulders are impacted against the pubic bone or the sacral promontory requiring special maneuvers moving the fetus to effect delivery. Three studies from Quebec, British Columbia and Newfoundland and Labrador that did not define shoulder dystocia, used retrospective database reviews to conclude that the risk of shoulder dystocia increases with increasing BMI [23,31,32]. Of the three studies, two found a significantly increased risk of shoulder dystocia in those with pre-pregnancy obesity [31,32]. Schummers *et al*. [32] further analysed the risk of shoulder dystocia in each class of obesity compared to the normal BMI range, finding a significant ($p < 0.001$) increase in the occurrence of shoulder dystocia with increasing BMI (BMI 18.5- < 25 = 3.5%, 30- < 35 = 3.8%, 35- < 40 = 4.5%, and >40 = 4.1%). Abenhaim *et al*.'s [23] study showed a more profound increase in odds ratio using those in a normal BMI range as the reference group, finding that those with obesity had an OR for shoulder dystocia of 1.89 (95% CI 1.65–2.16) and an OR of 2.6 for those with a BMI >40 (95% CI 0.92–7.31). Finally, the rate of shoulder dystocia in the extreme obesity cohort (BMI >50) was found to be five times that of the control group (1.4% vs 7.1%) [31]. Birth injuries secondary to shoulder dystocia increased with increasing BMI in one study but were too rare for a detailed analysis [32].

3.2.3 Operative Vaginal Delivery

Four studies found that individuals with pre-pregnancy obesity had a decreased risk of OVD (a vaginal delivery assisted by forceps or vacuum extractor) [19,22,29,30], with two studies reporting a significant decrease in risk for OVD [22,29]. It is important to note three of the four studies also reported decreased rates of vaginal delivery in the obese cohort [19,22,29], one

of which was significantly lower than the control group [22]. Thus, the lower rate of OVD may be due to the overall decreased rates of vaginal delivery. Only one study reported on the rates of OVD sorted by classes of obesity [19]. All three classes had odds ratios less than 1 when compared to the control cohort, with class II obesity having the highest OR. However, the 95% confidence intervals overlapped significantly for all three classes; thus, the risk of OVD within the classes are relatively consistent.

3.2.4 Caesarean Section Delivery

All studies included both primary CS and repeat elective CS in the elective CS group. Of the 12 studies that analysed the rates of caesarean section (CS) delivery [19,20,22,23,26,27,29–34], seven studies found a significant increase in risk of CS in patients with pre-pregnancy obesity [20,22,27,29,31,32,34]. Furthermore, four studies assessed the rates of elective CS [19,26,27,34], and two described a significant increase in the obese cohort [27,34]. Definitions of emergency (or non-elective) CS varied slightly depending on the study. For example, one defined emergency CS as CS that was planned after onset of labour, whereas another defined it as CS that occurred because of maternal or fetal complications. Six studies specifically examined the risks of emergency or post-labour initiation CS [19,27,29–31,34], with four reporting a significantly higher risk in those with obesity [27,29,31,34]. The risk of elective and emergency CS were similar for those with pre-pregnancy obesity. The four studies that performed an analysis of the obesity classes found that the risk of CS delivery was relatively consistent across the BMI classes [19,23,32,33]; however, in one study, compared to parturients in the 18.5 to 24.99 BMI range (n = 5717), those with extreme obesity (BMI >50, n = 71) were found to have significantly higher rates of all CS (25.0% vs 60.6%) and non-elective CS (13.2% vs 31.7%) [31].

3.2.5 Postpartum Hemorrhage

Studies defined postpartum hemorrhage (PPH) as a >500 mL blood loss in vaginal delivery or >1000 mL blood loss in a caesarean-section. Of the three studies included, two reported an insignificant increase in risk of postpartum hemorrhage in parturients with obesity [23,31], whereas one study found no difference in rates of PPH between any class of obesity and the reference cohort [32]. The rates of PPH were relatively consistent across the obesity classes [32]. PPH in those with extreme obesity (BMI >50) was not significantly increased compared to the reference group in the normal BMI range [31].

3.2.6 Parturient Morbidity and Mortality

Parturient morbidity and mortality included a range of conditions including venous thromboembolic events, myocardial infarction, cardiac arrest, cerebrovascular hemorrhage, and obstetric death. Three studies examined these rare but grave events [31,32,36]; two studies only reported obstetric death [31,36] and one study grouped all severe events together under morbidity and mortality [32]. There was no significant difference in the rates of parturient morbidity and mortality across BMI groups [32]. Crane *et al.* [31] did not find a significantly increased risk of maternal death in those with extreme obesity, though this may be due to the low number of deaths in that cohort. It is important to note that the study by Baghirzada *et al*. [36] is not included in Table 3 as it is a case series.

3.2.7 Stillbirth

Stillbirth is death with a fetal weight of at least 500g after 20 weeks gestation but before birth. Smith *et al.* [37] reviewed 180 stillbirths occurring at a tertiary care center in Saskatchewan from 2011 to 2016 finding that the most common maternal risk factor was obesity (37.6%). The Canadian national stillbirth rate at the time was 0.83% and the Saskatchewan specific rate from 2006 to 2010 was 0.67%, with Smith's analysis finding a 0.87% rate for that tertiary care centre [37,38]. Schummers *et al*. [32] found that the adjusted absolute risk for stillbirth increased significantly with increasing BMI with a 0.3% AAR at a BMI of 25, a 0.4% AAR at a BMI of 36, and a 0.5% AAR at a BMI of 40.

4. Discussion

4.1 Summary of Findings

This review summarizes perinatal outcomes specific to individuals with obesity. This Canadian review provides an example of a country where obesity rates vary widely province to province and health care including perinatal care is available to all citizens, leading to an expectation of good perinatal outcomes. Perinatal obesity may reduce the ease of use and efficacy of obstetrical technology including diagnostic ultrasound and continuous electronic fetal monitoring; however, no Canadian studies examined those technologies. Studies were also lacking on post-cesarean surgical site infection and postpartum thrombosis. The consensus on obesity's impact on preterm labour, preterm birth, post-term delivery, labour induction, post-partum hemorrhage, and parturient morbidity and mortality in Canada remains inconclusive. The risk of hypertensive disorders, gestational diabetes, shoulder dystocia, and cesarean section delivery significantly increased with increasing BMI. Operative vaginal delivery was less common in those with pre-pregnancy obesity.

Canada's 2018 national adult obesity rate was 26.8% [3,4]. Obesity rates in Ontario and Quebec are similar to the national average with Newfoundland and Labrador having the highest obesity rate (40.2%) and British Columbia having the lowest (23%) [3]. Separate rates for women or reproductive aged women are not available. Obesity rates continued to increase across Canada during the last 20 years with accumulating evidence that pre-pregnancy

obesity increases risks for multiple poor reproductive outcomes. Identifying the risks and applying them broadly to all individuals with pre-pregnancy obesity further stigmatizes obesity and has the potential for over-treatment and iatrogenic harm as primary providers attempt to reduce risks.

4.2 Discussion of Findings

Several perinatal complications stem from the same obesity-related hormonal alternations. Leptin, an appetite regulating hormone and a growth hormone in pregnancy, has been shown to inhibit uterine contractions along with other adipokines [39,40]. Inhibiting uterine contractions can decrease preterm labour and preterm birth. Consistent with that physiology, most studies reviewed did not show a significant increase in preterm labor or birth in those with obesity. Management of obesity associated hypertensive disorders of pregnancy could theoretically increase iatrogenic preterm birth; however, most studies did not examine this aspect of preterm birth. Berger *et al.*'s [17] study showed that individuals with both obesity and preeclampsia had an almost doubled risk of provider-initiated preterm birth giving evidence that early induction of labor for hypertensive disorders could be an iatrogenic cause of preterm birth.

Inhibiting contractility may prolong pregnancy beyond 40 weeks gestation increasing the use of induction of labour methods. Poorer quality uterine contractions prolong stages of labour increasing the use of oxytocin augmentation of labor and caesarean section [40]. Although no studies in this review showed a significant increase in post-term pregnancy, research outside of Canada has documented this [41]. A study of 479,864 births occurring between 1990 and 2007 in England demonstrated an adjusted odds ratio of 1.34 for prolonged pregnancy [42]. The low rates of prolonged pregnancy in the Canadian studies may reflect induction of labor before 41 weeks attempting to avoid complications such as shoulder dystocia and stillbirth as is urged by some researchers [43,44]. The elevated odds ratios for caesarean delivery in the reviewed studies may be explained in part by failed early induction of labor.

Leptin is found in abnormally high levels in GDM and its growth stimulating ability may be responsible for the macrosomia associated with GDM [45]. The studies summarized in Table 3 documented gestational diabetes rates ranging from 2.7–19.7% [17,31]. The GDM prevalence in British Columbia was 13.7% in class I obesity and 20.8% in class III obesity indicating increasing risk with increasing BMI. Even in a population with a 20% GDM prevalence, four out five (80%) of pregnant patients are not likely to develop GDM. Clinical support for individuals with obesity in pregnancy must be based on testing measurements and observable symptoms more than generalized risk alone.

Postpartum hemorrhage, the failure of the myometrium to contract around uterine arteries after placental expulsion can also be attributed to the contraction inhibiting effect of adipokines [40]. Results indicating an increased risk for postpartum hemorrhage in those with increasing BMIs were inconsistent. More than the effect of adipokines or other physiologic mechanisms on postpartum bleeding, these results may reflect an increased awareness of the potential for postpartum hemorrhage with providers quickly employing active management of third stage labor with fundal massage and uterotonics.

The studies in Table 3 analyzing shoulder dystocia all found significant increases in the risk for shoulder dystocia, particularly those with BMIs exceeding 40 [23,31,32]. Schummers *et al.* [32] clearly identified an increased risk for shoulder dystocia in each class of obesity compared to the normal BMI range, finding a significant ($p < 0.001$) increase in the occurrence of shoulder dystocia with increasing BMI (BMI 18.5- $<$ 25 = 3.5%, 30- $<$ 35 = 3.8%, 35- $<$ 40 = 4.5%, and $>$40 = 4.1%. Although statistically significant, the clinical significance of a 1% difference in occurrence is negligible. Prevention of shoulder dystocia is often the medical indication for early term induction of labor attempting to limit fetal growth. The potential for shoulder dystocia needs to be weighed against potential iatrogenic harm from early inductions of labor, particularly for multiparas and those with class I obesity.

The increase in hypertensive disorders in pregnancy has been attributed to not only adipokines, but inflammatory cytokines produced in adipose tissue [46]. Almost all the Canadian studies reviewed demonstrated an increase in hypertensive disorders in those with obesity, particularly those with class III obesity. Emerging hypertension and preeclampsia may prompt early induction of labor. The combination of increased risks for macrosomia, shoulder dystocia and hypertensive disorders, particularly if combined with gestational diabetes, may influence obstetrical providers to intervene early with induction of labor or caesarean section to improve perinatal outcomes.

The two most grave obesity associated complications, stillbirth, and maternal death, occur so infrequently that detailed analysis is difficult. Avoiding stillbirth, which is also increased in diabetes, hypertensive disorders, and advanced maternal age, is the goal of many early inductions of labor and may contribute to increased rates of caesarean birth; however, almost all studies summarized found consistent rates of caesarean section across obesity classes. The increased occurrence of caesarean section for those with obesity compared to those in the normal BMI range may indicate a generalization of all obesity-associated risks for poor perinatal outcomes to all individuals with obesity, even when many poor perinatal outcomes don't increase significantly until BMI exceeds 40.

The increased risk that obesity imposes on pregnancy and birth must be balanced with the number of pregnant individuals who will not have obesity related complications. Considering the two most common obesity-related complications to pregnancy, diabetes and hypertensive disorders

of pregnancy which had risk ranges of 7.5–21.1% [18,31] and 8.5–23% [24,27], more individuals with obesity will not have those complications than will have those complications. Individuals with obesity but without co-morbid conditions may demonstrate the concept of metabolically healthy obesity. A recent study from Sweden categorized pregnant patients with obesity into metabolically healthy (MHO) and metabolically unhealthy (MUO) based on first trimester blood pressure, lipoprotein levels and non-fasting blood glucose levels [47]. Researchers found a significantly increased adjusted odds ratio for at least one obesity related perinatal complication (AOR 1.49, 95% confidence interval 1.03–2.15) but concluded that using MUO in risk prediction was no more revealing than using BMI alone. Further research could explore the impact of nutrition and activity education and therapies on both metabolically healthy and unhealthy obesity in pregnancy. The potential to assist pregnant individuals in avoiding gestational diabetes and hypertensive disorders should inspire clinicians to provide the support and resources to learn and use health promoting nutrition and physical activity.

This review was limited by several factors. First, there were few studies of obesity in pregnancy done in Canada. Relying on studies done in the US, where universal health care was not available during the study period and where the prevalence of obesity is higher, may overly bias thinking about obesity-related risks in pregnancy. Studies sometimes grouped all individuals with a BMI $\geq$30 into a single group. Others had insufficient numbers to compare risk across obesity classes, demonstrating different risks as pre-pregnancy BMI increased. Additionally, the mix of risk measures used, including prevalence, relative risk, odds ratios, and adjusted odds ratios makes comparisons across studies difficult.

5. Conclusions

The increased risks for poor perinatal outcomes associated with obesity in pregnancy are documented but poorly understood. Further research that is country specific, analyzed according to WHO BMI classifications, and stratified by obesity class and other variables is needed to improve the understanding required for targeted management guidelines. Obesity class specific risk along with a holistic assessment of health that includes the presence of chronic disease or other risk factors is needed for informed decision making by both clinicians and clients and to avoid the over-application of medical therapies and iatrogenic harm.

Author Contributions

SA and CMJ designed the research study; SA performed the research; SA and CMJ analyzed the data, wrote the manuscript, and read and approved the final manuscript.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

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