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Review

A Mechanistic Narrative Review of Pregnancy Outcomes and Abnormalities of Labor

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Abstract

Objective: To synthesize clinical and mechanistic evidence explaining the association between maternal obesity, adverse pregnancy outcomes, and greater difficulty achieving vaginal delivery. **Mechanism:** A mechanism-oriented narrative synthesis was organized around uterine contractile activity, cervical remodeling, fetal size and passage, adipokine and inflammatory signaling, and a hypothesized birth canal soft-tissue pathway. **Findings in Brief:** Evidence most consistently links obesity to macrosomia, slower labor progression, prolonged induction, greater medication requirements, and increased rates of intrapartum cesarean delivery. However, current clinical and mechanistic evidence does not support a single universal causal pathway. Direct evidence of intrinsic myometrial dysfunction is inconsistent, molecular evidence for obesity-specific alterations in cervical remodeling remains limited, and obesity-related narrowing of pelvic soft tissue has not been directly demonstrated. **Conclusions:** Maternal obesity is associated with greater complexity during attempted vaginal delivery, but current evidence does not establish a single causal mechanism. Excess fetal growth and impaired fetal passage represent the most directly supported pathways, whereas myometrial, cervical, adipokine, and birth canal mechanisms remain variably supported and require further clinical validation. Preconception weight optimization and individualized obstetric management are reasonable, whereas obesity-specific biomarkers and oxytocin protocols should not be adopted without prospective validation.

Keywords: maternal obesity; overweight; pre-pregnancy body mass index; gestational weight gain; vaginal delivery; uterine contractility; cervical ripening; adipokines

1. Introduction

Maternal obesity is an increasingly important public health and obstetric concern. It affects fertility, pregnancy maintenance, labor progression, mode of delivery, and postpartum health. During pregnancy, higher maternal body mass index (BMI) is associated with gestational diabetes mellitus (GDM), preeclampsia, macrosomia, postpartum hemorrhage, neonatal intensive care unit (NICU) admission, labor induction, and cesarean delivery [1,2,3,4,5,6,7]. These consequences extend beyond delivery: maternal obesity before or during pregnancy contributes to postpartum weight retention and subsequent cardiometabolic risk in the mother, as well as obesity and metabolic disease in offspring [8,9,10,11].

Severe perinatal outcomes further strengthen the public health rationale for this review. A dose-response meta-analysis showed that even modest increases in maternal BMI were associated with higher risks of fetal death, stillbirth, neonatal death, perinatal death, and infant death [12]. Although these outcomes are not determined solely by delivery mechanics, they underscore the need to understand how maternal obesity modifies pregnancy physiology, the timing of labor, fetal growth, and delivery management.

A mechanistic focus provides clinically relevant context for these outcome differences. Current evidence suggests that maternal obesity may influence myometrial contractility and oxytocin responsiveness, delay cervical ripening, alter adipokine and inflammatory signaling, and increase fetal size [13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29]. A fourth, mechanical pathway involving soft tissues of the birth canal has also been proposed, although current evidence remains indirect rather than demonstrative. These pathways may converge to contribute to prolonged induction, labor arrest, operative vaginal delivery, or intrapartum cesarean delivery. This review therefore evaluates each individual mechanism and its potential synergy while distinguishing well-supported clinical associations from preliminary or indirect mechanistic evidence.

2. Literature Search Strategy

This article is a mechanism-oriented narrative review. The primary literature search used the Jinglun Knowledge Service Platform (VIPSlib; https://k.vipslib.com), Foreign Medical Information Resources Retrieval System (FMRS/METSTR; https://www.metstr.com), Chinese Medical Journal Full-text Database (Yiigle; https://www.yiigle.com), Wanfang Med Online (https://med.wanfangdata.com.cn), and China National Knowledge Infrastructure (CNKI; https://www.cnki.net). These searches covered each platform from inception through October 2025. During manuscript revision, PubMed (including MEDLINE-

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indexed records) was additionally searched and updated through September 10, 2026, to identify and verify relevant evidence concerning maternal obesity, stillbirth, fetal macrosomia, labor progression, cervical ripening, induction methods, oxytocin requirements, obesity-related reproductive dysfunction, and the strength and limitations of mechanistic evidence. MEDLINE was therefore not searched through a separate interface. Candidate references identified during the supplementary search were individually assessed for relevance and retrievability, and the original articles were reviewed before inclusion to ensure that the cited evidence accurately supported the corresponding statements in the manuscript.

Chinese search terms included "obesity", "overweight", "pre-pregnancy BMI", "gestational weight gain", "pregnancy", "pregnant women", "natural delivery", "vaginal delivery", "labor", "labor induction", "oxytocin", "cesarean delivery", "uterine contraction", "cervical ripening", "adipokines", "leptin", "visfatin", "adiponectin", "resistin", "apelin", "progranulin", "birth canal", "pelvic fat", "macrosomia", and "cephalopelvic disproportion". English search terms included "obesity", "overweight", "pre-pregnancy BMI", "gestational weight gain", "pregnancy", "vaginal delivery", "labor progression", "labor induction", "cesarean delivery", "uterine contractility", "myometrial contractility", "cervical ripening", "adipokines", "leptin", "visfatin", "adiponectin", "resistin", "apelin", "progranulin", "birth canal", "pelvic adipose tissue", "macrosomia", "cephalopelvic disproportion", "stillbirth", and "fetal death". Terms were combined using AND/OR as appropriate to each platform.

Eligible publications included clinical studies, cohort studies, case-control studies, systematic reviews, meta-analyses, guidelines, expert consensus statements, and experimental studies addressing maternal obesity, gestational weight gain, pregnancy outcomes, labor progression, vaginal delivery, or potential underlying mechanisms. Studies published within the past 10 years were prioritized, while seminal studies with important clinical or mechanistic relevance were retained. Articles with limited relevance, duplicate publications, unavailable full texts, incomplete information, or conference abstracts lacking sufficient study details were excluded. The included literature was narratively synthesized according to the following framework: epidemiological background, pregnancy outcomes, labor manifestations, potential mechanisms, and clinical implications.

Because this is a narrative review, no formal risk-of-bias or Grading of Recommendations Assessment, Development and Evaluation (GRADE) assessment was undertaken. To avoid overstating causality, the evidence was interpreted according to study design: systematic reviews, guidelines, and large clinical studies were prioritized for clinical associations; randomized or comparative clinical studies were used to address management questions; and human tissue, animal, and *in vitro* studies were used to support mechanistic plausibility. Statements based predominantly on experimental or indirect evidence were explicitly framed as hypotheses or potential mechanisms rather than established causal pathways.

3. Epidemiological Trends in Maternal Obesity and Regional Differences in Delivery Outcomes

Obesity has become a major public health issue worldwide. A systematic review and meta-analysis estimated that the global prevalence of obesity among pregnant women was approximately 16.30% from 2010 to 2019, while the combined prevalence of overweight or obesity among pregnant women reached 43.80% [30]. In China, women of reproductive age are also experiencing an increasing burden of overweight and obesity. Wu et al. [31] reported that among Chinese women aged 18–49 years, the prevalence of overweight and obesity was 21.05% and 6.08%, respectively, with both rates increasing from 2013 to 2019. In addition, Du et al. [32] investigated the appropriate range of gestational weight gain among singleton term primiparas in northern China, providing a reference for gestational weight management in this population. Recent systematic reviews and meta-analyses have further demonstrated that increased pre-pregnancy BMI is associated with elevated risks of multiple adverse maternal, fetal, and neonatal outcomes [4,5].

With regard to regional delivery outcomes, the 2025 National Report on Medical Service and Quality Safety (Obstetrics) documented significant provincial variation in 2024. Heilongjiang presented the highest total cesarean delivery rate (70.63%) and primiparous cesarean delivery rate (72.57%) among all provinces. Its intrapartum cesarean delivery rate (13.33%), episiotomy rate among vaginal births (28.15%), and macrosomia incidence (8.11%) were also among the highest reported nationally [33]. These data highlight weight management, macrosomia prevention, labor induction, and intrapartum care as relevant priorities in Northeast China. However, regional quality indicators cannot establish maternal obesity as a direct cause of the high cesarean delivery rate. Rather, they provide clinical context for examining the relationships among maternal BMI, gestational weight gain, fetal size, and failed vaginal delivery.

4. Maternal Obesity-Related Pregnancy Outcomes: Clinical Basis for Vaginal Delivery Risk

4.1 Maternal Outcomes

Obesity is associated with reproductive endocrine and metabolic dysfunction and impaired fertility, particularly in the context of ovulatory dysfunction, insulin resistance, polyendocrine metabolic ovarian syndrome (PMOS; formerly polycystic ovary syndrome [PCOS]) [34], altered oocyte competence, and impaired endometrial receptivity

[35,36,37]. Obesity is also associated with an increased risk of miscarriage after spontaneous conception [38]. During pregnancy, it is an important risk factor for preeclampsia, GDM, and venous thromboembolism [2,7,39,40,41]. A Clinical and Experimental Obstetrics & Gynecology (CEOG) review similarly emphasized that obesity-related morbidity extends across the antenatal, intrapartum, and postpartum periods [7]. These complications may increase the likelihood of medically indicated delivery and intrapartum intervention; however, their effects on delivery mode depend on disease severity, gestational age, cervical status, fetal condition, and obstetric management.

The relationship between obesity and preterm birth is not entirely unidirectional. Some studies have reported lower rates of spontaneous preterm birth among women with obesity than among women with normal weight. As BMI increases, medically indicated deliveries account for a greater proportion of preterm births, whereas older term cohorts reported higher incidence of post-term pregnancy [42,43]. Thus, obesity does not simply increase the risk of preterm birth; rather, it may influence gestational duration and delivery planning through comorbidities, labor-onset patterns, and the timing of obstetric intervention.

4.2 Fetal and Neonatal Outcomes

Maternal obesity may affect embryonic and fetal development and is considered an important risk factor for congenital anomalies, including neural tube defects [44,45]. Previous studies have also suggested that maternal obesity is associated with an increased risk of several birth defects, including omphalocele and cardiac defects, although the magnitude of this risk varies by defect type [46]. In addition, obesity can reduce the quality and completeness of fetal structural visualization during prenatal ultrasonography, particularly of the fetal heart, spine, and umbilical cord, potentially impairing diagnostic assessment and increasing the likelihood of incomplete visualization [47].

Maternal obesity is an important risk factor for excessive fetal growth and macrosomia [2,3,4,5,7]. A recent umbrella review identified pre-pregnancy obesity and excessive weight gain before or during pregnancy as having suggestive evidence for fetal macrosomia [48]. Similarly, a 2026 retrospective study conducted in China and published in CEOG reported associations between macrosomia and maternal weight or BMI, gestational weight gain, and GDM-related factors, although its observational design does not establish causality [49]. Definitions of macrosomia vary across studies and should be interpreted according to the threshold applied by each source. Excessive fetal size increases the risks of shoulder dystocia, severe perineal laceration, chorioamnionitis, postpartum hemorrhage, and cesarean delivery [50]. Neonates born to women with obesity are also more likely to require neonatal intensive care [4,5,6]. These fetal and neonatal factors may reduce the likelihood of uncomplicated vaginal delivery by increasing the risk of cephalopelvic disproportion, difficult fetal passage, or the need for expedited delivery.

4.3 Labor Progression and Mode of Delivery

Women with obesity more often require intrapartum interventions and may experience slower cervical dilation, prolonged labor, or cesarean delivery following induction [4,5,7,13,19,25,51]. Akgol et al. [52] specifically reported an association between reduced spontaneous labor at term and a greater frequency of planned delivery, but did not demonstrate a difference in delivery mode. Jensen et al. [53] reported increased oxytocin use, earlier amniotomy, and primary uterine inertia with higher pre-pregnancy BMI; however, this study should not be cited as evidence of obesity-related pregnancy complications or a higher cesarean delivery rate. Frey et al. [15] further showed that the maximum oxytocin dose is multifactorial and may reflect diabetes, macrosomia, fever, magnesium exposure, or induction rather than BMI alone. More direct evidence supporting prolonged induction and greater medication requirements is provided by Pevzner et al. [16], the systematic review by Ellis et al. [19], and a contemporary review focused on labor induction in pregnant individuals with obesity [54]. In a secondary analysis of two multicenter randomized trials, Croll et al. [55] found no statistically significant differences in cesarean rates among women with obesity receiving a Foley catheter, oral misoprostol, or vaginal Prostaglandin E2 (PGE2), although failed Foley catheter placement and switching to another induction method were more frequent in the Foley catheter group. These findings support a more demanding induction process as a clinical phenotype but do not identify a single obesity-specific pharmacologic or mechanical defect.

Recent evidence on the timing of labor induction in women with obesity does not support the simplistic view that obesity should prompt clinicians to avoid induction. A systematic review and meta-analysis by Krogh et al. [56] suggested that, compared with expectant management, induction of labor at term in women with obesity may reduce the risk of cesarean delivery; however, the certainty of the evidence was low to very low, and the risk of instrumental vaginal delivery may be increased. In addition, a study of dinoprostone-induced vaginal delivery suggested that higher maternal BMI was associated with a prolonged active phase and longer total labor duration [57]. A 2025 systematic review and meta-analysis of interventions for the management of maternal obesity did not show a clear reduction in rates of induction or overall cesarean delivery, reinforcing the distinction between general weight-management benefits and evidence for altered labor mechanics [58].

Obesity is also associated with greater complexity of obstetric anesthesia and perioperative management. Early studies of massively or morbidly obese parturients reported higher rates of epidural placement or replacement failure.

Table 1. Adverse pregnancy outcomes associated with maternal obesity and their potential effects on vaginal delivery.

Outcome	Direction of risk	Potential mechanism	Specific effect on vaginal delivery
GDM	Increased	Insulin resistance and hyperglycemia promote excessive fetal growth	May increase macrosomia and fetal-passage difficulty and can complicate induction [1,2,4,5,48]
Preeclampsia	Increased	Endothelial dysfunction, inflammation, and placental disease	May require medically indicated delivery before spontaneous labor; route depends on severity and fetal status [8,39]
Venous thromboembolism	Increased	Hypercoagulability, reduced mobility, and inflammation	Does not directly obstruct vaginal birth but complicates anticoagulation, anesthesia, and surgical contingency planning [8,40]
Post-term pregnancy or reduced spontaneous labor at term	Possibly increased	The mechanism is uncertain; cervical readiness and labor-onset signaling may contribute	May increase planned delivery, cervical ripening, or induction; evidence does not establish a single causal pathway [19,43,52,56]
Macrosomia	Increased	Maternal hyperglycemic and dyslipidemic states promote fetal overgrowth	Increases cephalopelvic disproportion and shoulder dystocia, directly hindering fetal descent and passage [1,3,48,50]
Adverse neonatal outcomes	Increased	Metabolic abnormalities and perinatal complications	May prompt continuous monitoring or expedited delivery; effects on route depend on the underlying complication [1,4,5,6]
Fetal death or stillbirth	Increased	Placental dysfunction and metabolic comorbidity may contribute; mechanisms remain incompletely defined	Not a mechanical cause of difficult vaginal delivery, but it strengthens surveillance and delivery-timing considerations [3,12]

GDM, gestational diabetes mellitus.

Difficult tracheal intubation was also more frequent in a small subgroup, although this difference did not reach conventional statistical significance [59,60]. These findings support early anesthetic assessment, particularly among women with severe obesity, but should not be generalized to all women with obesity. Cesarean delivery therefore does not remove obesity-related risk. In the absence of an obstetric contraindication, optimizing the conditions for safe vaginal delivery remains clinically important. Table 1 (Ref. [1,2,3,4,5,6,8,12,19,39,40,43,48,50,52,56]) summarizes how major obesity-associated pregnancy outcomes may affect vaginal delivery through distinct clinical or mechanical pathways. Together, these clinical phenotypes define the observations requiring mechanistic explanation. Section 5 therefore examines uterine contractility, cervical remodeling, fetal passage, and the hypothesized contribution of birth canal soft tissues, as well as the interactions among these factors.

5. Potential Mechanisms by Which Maternal Obesity Affects Vaginal Delivery

Successful vaginal delivery depends on uterine contractility, the birth canal, fetal size and position, and maternal and clinical factors. Maternal obesity may influence several of these domains simultaneously rather than through a single pathway. Fig. 1 provides a conceptual framework linking maternal obesity to uterine contractility, cervical remodeling, fetal growth, labor abnormalities, and the hypothesized contribution of birth canal soft tissues.

5.1 Impaired Myometrial Contractility

Effective uterine contraction is the primary force driving vaginal delivery. *In vitro* studies suggest that obesity may impair myometrial contractility, reducing both spontaneous and oxytocin-induced contractions of uterine smooth muscle [14]. Uterine contraction depends on excitation-contraction coupling in myometrial smooth muscle cells, calcium influx, myosin light-chain phosphorylation, and the coordinated expression of multiple contraction-associated proteins. Dyslipidemia, a high-cholesterol environment, and chronic low-grade inflammation in obesity may interfere with these processes. Zhang et al. [17] found that modulation of cholesterol levels can affect human myometrial contractility and calcium signaling; a high-cholesterol state may alter the lipid microenvironment of cell membranes, lipid rafts, and calcium signaling, thereby affecting myometrial responses to spontaneous and oxytocin-induced stimulation [18].

Recent mechanistic evidence suggests that findings in this field remain inconsistent. Lammers et al. [26] compared *in vitro* contractile parameters and oxytocin receptor expression in myometrial samples from term cesarean deliveries across BMI groups and found no significant differences between women with obesity and women with normal weight in spontaneous contractions, oxytocin-

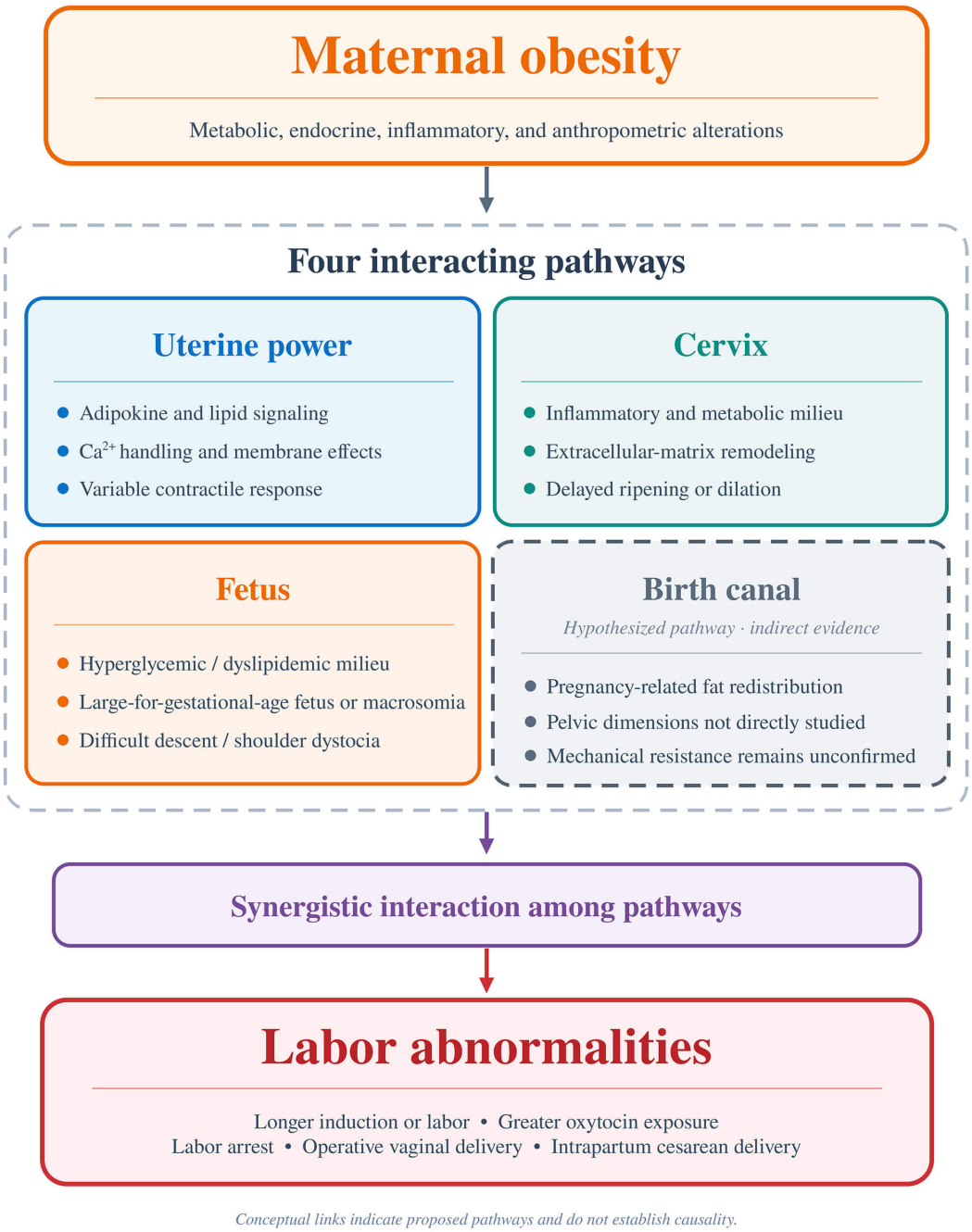


Fig. 1. Conceptual framework linking maternal obesity with uterine power, cervical remodeling, fetal size and passage, a hypothesized birth canal soft-tissue pathway, and labor abnormalities. The birth canal pathway is shown with a dashed border as direct evidence of obesity-related pelvic adiposity or narrowing is lacking. Arrows indicate a conceptual synthesis of proposed associations and should not be interpreted as evidence of causality. The figure is an original synthesis created by the authors based on the evidence cited in this review.

induced contractions, or oxytocin receptor (OXTR) expression. Therefore, difficulty with vaginal delivery in the context of obesity should not be attributed solely to intrinsic impairment of myometrial contractility; cervical conditions, fetal size, mechanical resistance, and clinical interventions should also be considered.

Clinically, women with obesity are more likely to experience uterine inertia, require higher doses of oxytocin, and have prolonged labor. However, oxytocin requirements are not determined by BMI alone and may also be influenced by diabetes, macrosomia, fever, magnesium use, induction method, and cervical status [15]. Pevzner et al. [16] found that women with a BMI $\geq$40 kg/m^2 may require greater oxytocin doses and longer oxytocin exposure during prostaglandin-induced cervical ripening and labor induction. The systematic review and meta-analysis by El-

lis et al. [19] also supports an association between obesity and prolonged induction, increased medication requirements, and a higher risk of cesarean delivery following induction. Together, these findings support an association between obesity and more demanding labor induction, but do not establish intrinsic myometrial dysfunction as the sole or universal mechanism.

5.2 Increased Oxytocin Requirements and Abnormal Regulation of Uterine Contractions

Oxytocin is an important agent used for labor induction and augmentation. Increased oxytocin requirements in women with obesity may reflect reduced myometrial responsiveness but may also be related to poor cervical status, greater fetal weight, delayed labor onset, or other clinical factors. Because the lipid microenvironment of cell membranes, lipid raft structure, and calcium signaling pathways influence myometrial contractile responses, obesity-related hyperlipidemia and lipotoxicity may alter membrane microdomains and signal transduction efficiency [17,18]. Nevertheless, current evidence does not support simplified statements such as “obesity blocks oxytocin receptors” or “obesity reduces oxytocin receptor expression”.

A more appropriate interpretation is that clinical and *in vitro* studies suggest that increased oxytocin requirements in women with obesity may reflect a combination of myometrial responsiveness, cervical readiness, fetal size, induction method, and comorbidities rather than a single BMI-mediated defect. Prospective studies integrating myometrial tissue, serum metabolic biomarkers, and labor curves are warranted to clarify the mechanisms underlying altered oxytocin requirements in women with obesity.

5.3 Delayed Cervical Ripening and Cervical Dilation

Cervical ripening is a prerequisite for labor onset and successful induction. Nuthalapaty et al. [13] showed that maternal weight was negatively correlated with the rate of cervical dilation and that women with higher body weight experienced more pronounced prolongation of labor. Systematic reviews and meta-analyses also suggest that, compared with women with normal weight, women with obesity have greater difficulty achieving cervical ripening and entering active labor, often requiring higher doses of cervical ripening agents or longer induction times [19].

Mechanistically, cervical ripening involves collagen degradation, extracellular matrix remodeling, local immune cell infiltration, and regulation by prostaglandins and inflammatory mediators. Chronic low-grade inflammation, insulin resistance, and adipokine dysregulation associated with obesity may affect cervical tissue remodeling, thereby delaying cervical softening, shortening, and dilation. Although direct evidence linking obesity to molecular remodeling of the cervix remains limited, the clinical manifestations of prolonged induction, delayed entry into active labor, and slower cervical dilation among women with obesity support impaired cervical ripening as a potentially important component of difficult vaginal delivery. However, contemporary reviews and comparative induction data do not establish a single optimal cervical-ripening method or demonstrate an obesity-specific molecular cervical defect [54,55]. Delayed clinical progression should therefore be interpreted as a phenotype requiring further explanation rather than as direct evidence of molecular cervical dysfunction.

5.4 Adipokines, Chronic Low-Grade Inflammation, and Myometrial Contractility

Adipose tissue is not merely an energy-storage organ but also a metabolically active endocrine tissue. A moderate increase in adipose tissue during pregnancy is a normal physiological adaptation. However, excessive adipose accumulation, particularly visceral adiposity, may disrupt the secretion of adipokines and inflammatory mediators in women with obesity. Previous ultrasonographic studies have shown that adipose tissue distribution during pregnancy may shift from subcutaneous toward preperitoneal or visceral fat accumulation, with a higher abdominal fat index often associated with greater visceral fat deposition [27,28,61]. A recent review on maternal overweight, obesity, and adipokines further reported that maternal overweight and obesity may alter adipokine profiles during pregnancy and lactation. Evidence for increased leptin levels is relatively consistent, whereas changes in adiponectin, ghrelin, obestatin, and resistin remain heterogeneous [29].

Several adipokines may contribute to the regulation of uterine contractility. Leptin inhibits the frequency and amplitude of spontaneous and oxytocin-induced contractions in human myometrium *in vitro* [20]. Similarly, Mumtaz et al. [21] reported that visfatin and leptin inhibited myometrial contractility in human and rat tissues. Apelin also reduced spontaneous and oxytocin-induced contractions in human uterine muscle strips [22]. Progranulin (PGRN), an adipokine associated with obesity and metabolic inflammation, may also link excessive gestational weight gain to altered expression of contraction-associated proteins, including gap junction protein alpha 1 (*GJA1)*/connexin 43 (CX43) [23,24]. These studies identify biologically plausible pathways but do not establish that circulating adipokine levels predict uterine inertia or vaginal-delivery failure in clinical practice. The principal findings and types of evidence are summarized in Table 2 (Ref. [20,21,22,23,24,29]). Table 2 distinguishes experimental effects from serum associations and narrative evidence to avoid interpreting mechanistic plausibility as clinical validation.

Table 2. Potential relationships between adipokines and uterine contractile function.

Adipokine	Change with obesity or excessive gestational weight gain	Potential effect on uterine contractility	Evidence type and key references
Leptin	Usually increased	Inhibits spontaneous and oxytocin-induced contraction frequency and amplitude	Human myometrial and comparative *in vitro* studies [20,21]; narrative review of circulating profiles [29]
Visfatin	May be increased	May inhibit human and animal myometrial contractility	Human and rat *in vitro* study [21]
Apelin	Variable	Reduced spontaneous and oxytocin-induced contraction of uterine muscle strips	Human myometrial *in vitro* study [22]
PGRN	May increase with excessive gestational weight gain	May alter contraction-associated protein expression in uterine smooth muscle cells	Obesity/adipose-tissue mechanistic study [23]; uterine-cell and maternal-serum association study [24]

PGRN, progranulin.

5.5 Birth Canal Soft Tissues and Vaginal Delivery

Vaginal delivery is influenced by the structure and compliance of the birth canal and surrounding soft tissues. In a cohort of term nulliparous women, higher pre-pregnancy BMI was associated with slower labor progression before 6 cm of cervical dilation, with less pronounced differences later in labor [25]. However, this study did not identify a pelvic soft-tissue mechanism. Anthropometric studies indicate that adipose tissue distribution changes during pregnancy and that these patterns may differ according to baseline adiposity [62,63]. However, these studies did not assess pelvic adiposity, birth canal dimensions, fetal descent, or operative vaginal delivery. Therefore, they cannot establish that obesity narrows the birth canal or increases pelvic soft-tissue resistance.

At present, increased pelvic soft-tissue resistance should be regarded as a hypothesis rather than an established mechanism. Associations between maternal BMI and labor progression may reflect myometrial, cervical, fetal, management-related, or unmeasured factors. Future studies using prenatal ultrasonography, magnetic resonance imaging, or three-dimensional pelvic-floor assessment should directly evaluate pelvic soft-tissue distribution, birth canal dimensions, fetal descent, and delivery outcomes before this mechanism is incorporated into clinical risk prediction.

5.6 Macrosomia and Cephalopelvic Disproportion

Maternal obesity and excessive gestational weight gain increase the likelihood of macrosomia [2,3,4,5,48,49]. Greater fetal size can hinder fetal descent and rotation and is associated with cephalopelvic disproportion, shoulder dystocia, severe perineal trauma, and intrapartum cesarean delivery [48,50]. This fetal pathway is better supported than the proposed birth canal soft-tissue pathway because it is supported by large clinical cohorts and multiple evidence syntheses.

5.7 Synergistic Interactions Among Mechanisms

In clinical practice, these mechanisms are likely to interact rather than act independently. Maternal metabolic dysregulation may promote fetal overgrowth, while adipokine and inflammatory changes alter contractile efficiency or cervical remodeling. Poor cervical readiness can prolong induction and increase oxytocin exposure, whereas greater fetal size increases the mechanical demands of descent; any additional effect of birth canal soft tissues remains unconfirmed. Thus, a modest impairment in one domain may become clinically important when several contributing factors coexist. These interacting pathways and their shared labor phenotypes are summarized in Fig. 1.

Evidence is not uniform across these pathways. The association between maternal obesity and excessive fetal growth or fetal-passage difficulty is supported by multiple clinical studies and evidence syntheses, whereas prolonged labor induction, slower labor progression, and greater medication requirements are reproducible clinical phenotypes that do not identify a single underlying mechanism. Direct human mechanistic evidence for intrinsic myometrial dysfunction is mixed [14,26], obesity-specific molecular evidence of cervical remodeling remains limited [13,19,54,55], adipokine effects are supported mainly by experimental or associative studies [20,21,22,23,24,29], and pelvic soft-tissue resistance remains indirect and hypothetical [25,62,63]. Thus, Fig. 2 summarizes the nature and principal limitations of the evidence rather than assigning numerical source counts or formal evidence grades.

6. Clinical Implications

6.1 Preconception and Antenatal Management

Management should begin before pregnancy and continue through the postpartum period. Preconception counseling should address weight, nutrition, physical activity, glucose, lipid levels, blood pressure, fertility, and medica-

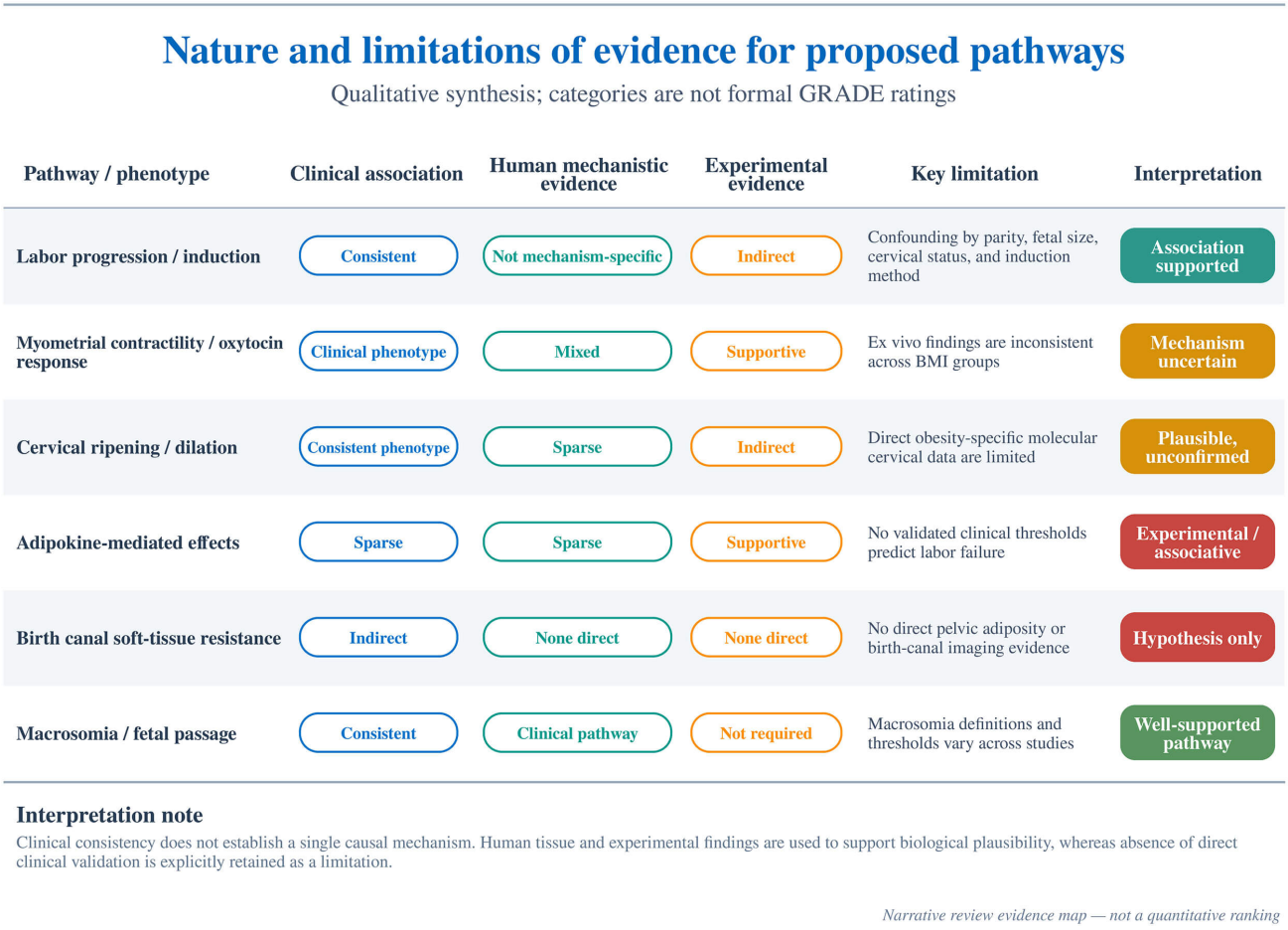


Fig. 2. Qualitative evidence map of the principal pathways considered in this narrative review. Categories describe the dominant type and limitations of the cited evidence and are not formal GRADE ratings. Clinical associations may be consistent even when the underlying biological mechanism remains uncertain, and experimental plausibility should not be interpreted as clinical validation. GRADE, Grading of Recommendations Assessment, Development and Evaluation.

tion safety. During pregnancy, gestational weight-gain targets should be individualized according to pre-pregnancy BMI and clinical context, with attention to fetal growth and obesity-related comorbidities. The American College of Obstetricians and Gynecologists (ACOG), the International Federation of Gynecology and Obstetrics (FIGO), and the European Association for the Study of Obesity (EASO) recommend integrated management across the reproductive life course rather than isolated interventions during labor [8,9,10].

These measures cannot guarantee vaginal delivery but may reduce modifiable contributors such as excessive gestational weight gain, dysglycemia, hypertensive disease, and macrosomia. Evidence that pregnancy-time counseling or lifestyle interventions alone improve major obstetric outcomes remains mixed. In a CEOG longitudinal study of women with overweight or obesity, an awareness intervention was associated with some improvement in weight-control behaviors but did not significantly alter major pregnancy or neonatal outcomes [64]. A 2025 systematic review and meta-analysis similarly found no clear reduction in induction or cesarean delivery rates from maternal obesity management interventions, despite benefits for selected weight-related outcomes [58]. These findings support realistic counseling and preconception optimization rather than assuming that antenatal intervention alone will normalize labor mechanics. Counseling should avoid stigma and incorporate shared decision-making, because BMI alone does not determine labor progression or mode of delivery.

6.2 Timing of Induction and Intrapartum Management

Maternal obesity alone is not a universal indication for delivery before 39 weeks [8]. For otherwise uncomplicated pregnancies at term, elective induction at approximately 39 weeks may be considered after assessment of parity, cervical status, fetal size, comorbidities, previous cesarean delivery, local resources, and patient preferences. Induction outcomes are also influenced by obstetric factors beyond maternal BMI. A multicenter retrospective study of 235 women with Class III obesity undergoing labor induction found that nulliparity (OR 2.81, 95% CI 1.58–4.97) and an unfavorable Bishop score were independently associated

with failed induction [65]. Observational data suggest a lower risk of cesarean delivery with induction at 39 weeks in women with obesity [66], and a systematic review found that term induction may reduce cesarean delivery compared with expectant management [56]. However, the certainty of the evidence was low to very low; therefore, no single gestational age can be recommended for all women with obesity.

When induction is undertaken, clinicians should anticipate that cervical ripening and progression to active labor may take longer [16,19,54,57]. Cervical status, fetal presentation and estimated weight, comorbidities, fetal surveillance, and anesthetic risk should be assessed before induction. Adequate time should be allowed before diagnosing failed induction or labor arrest when maternal and fetal conditions remain reassuring, while prolonged ineffective contractions should prompt reassessment rather than continued management without clear indication. Available comparative data do not establish a universally superior cervical-ripening method for women with obesity [54,55]. Early anesthetic planning is prudent, particularly in women with severe obesity, because neuraxial and airway management may be technically challenging [59,60].

6.3 Adipokines and Individualized Oxytocin Dosing

Adipokine profiles are biologically plausible research biomarkers but are not currently suitable for routine delivery-risk stratification. Leptin, visfatin, apelin, and PGRN have experimental or associative links with myometrial function [20,21,22,23,24], yet circulating concentrations vary according to gestational age, adiposity, metabolic disease, and assay methods [29]. No validated threshold currently predicts induction failure, labor arrest, or intrapartum cesarean delivery; therefore, adipokine measurements should not determine delivery route outside research settings.

Similarly, some women with obesity require longer oxytocin exposure or higher cumulative doses, but available evidence does not establish a validated BMI-specific dosing protocol [15,16,19,26]. Oxytocin should be titrated according to contraction frequency, uterine response, fetal heart rate, cervical change, and institutional safety protocols rather than increased automatically based on maternal BMI. Prospective trials should evaluate whether protocols incorporating BMI, cervical status, parity, metabolic phenotype, and uterine response improve efficacy without increasing tachysystole or fetal compromise.

7. Limitations and Future Directions

This mechanism-oriented narrative review synthesizes clinical outcomes and potential pathways but is not a systematic review or meta-analysis. As such, a Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram and formal risk-of-bias assessment were not performed. The supplementary literature update was extended through September 10, 2026; however, narrative selection may still introduce selection bias. Fig. 2 therefore provides a qualitative map of evidence types and limitations rather than a quantitative ranking or formal GRADE assessment. Systematic reviews and guidelines provide stronger evidence for clinical associations and management, whereas direct clinical validation of the proposed mechanism remains limited.

Several limitations remain. First, obesity exposure is defined inconsistently by pre-pregnancy BMI, BMI at delivery, body composition, or gestational weight gain. Second, associations with labor abnormalities may be confounded by age, parity, diabetes, fetal weight, induction method, cervical status, and intervention thresholds. Third, adipokine and myometrial studies are mostly *in vitro*, tissue-based, or small associative studies. The recent study by Lammers et al. [26] found no BMI-related difference in *ex vivo* contractility or oxytocin receptor expression, while the review by Froń et al. [29] is a narrative review of maternal adipokine profiles rather than a prospective cohort study. Fourth, the cited studies did not directly assess obesity-related pelvic adiposity or birth canal narrowing. Future multicenter cohorts should integrate body composition, metabolic and adipokine profiles, cervical assessments, uterine contraction parameters, complete labor curves, and standardized obstetric management. Clinical trials should also evaluate induction timing and individualized oxytocin strategies while avoiding premature clinical adoption of unvalidated biomarkers.

8. Conclusions

Maternal obesity is consistently associated with adverse pregnancy outcomes and greater complexity during attempted vaginal delivery, but the available literature does not support a single causal mechanism. Excessive fetal growth and impaired fetal passage represent the most directly supported clinical pathways. Slower labor progression, prolonged induction, and greater medication requirements are also supported as clinical phenotypes, but they cannot be attributed to intrinsic myometrial dysfunction alone. Adipokine-mediated effects, obesity-specific molecular cervical remodeling, and altered myometrial signaling remain biologically plausible but incompletely validated in humans, whereas birth canal soft-tissue resistance remains an unconfirmed hypothesis. Current management should therefore emphasize established preconception and antenatal weight optimization, appropriate gestational weight gain, individualized delivery planning, and careful intrapartum assessment rather than unvalidated obesity-specific biomarkers or empiric oxytocin protocols. Prospective human studies integrating metabolic phenotype, cervical assessment, uterine function, fetal size, and standardized labor management are warranted to determine which pathways are causal and clinically relevant.

Author Contributions

HYF conceived the review, developed its overall framework, conducted most of the literature searching and screening, analyzed and synthesized the evidence, and drafted and finalized the manuscript. XS and DHX contributed to the literature search and critically reviewed the manuscript for important intellectual content. NL contributed to the interpretation of the evidence through critical evaluation of the medical arguments and supporting literature, and revised the manuscript's logical structure and wording. All authors have read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflicts of Interest

The authors declare no conflicts of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation and revision of this manuscript, the authors used ChatGPT (GPT-5.6, OpenAI) as an auxiliary tool for English translation, language polishing, improvement of the wording and organization of selected passages, refinement of literature-search strategies, assistance in identifying potentially relevant candidate references, and assistance with the drafting and refinement of figures based on scientific outlines and content structures prepared by the authors. Any candidate references suggested with AI assistance were independently verified by the authors for retrievability, relevance, and accuracy. The original publications were reviewed before inclusion to ensure that the cited evidence appropriately supported the corresponding statements in the manuscript. The scientific interpretation, selection of evidence, conclusions, and final content of the manuscript were reviewed and approved by the authors. The authors take full responsibility for the accuracy, integrity, and scientific content of the manuscript.

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