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ORIGINAL ARTICLE

Mediating processes underlying the associations between maternal obesity and the likelihood of cesarean birth

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Abstract

Background: Pregnant women with obesity are more likely to experience cesarean birth compared to women without obesity. Yet, little is known about the underlying mechanisms. The objective of this study was therefore to evaluate how mediators contribute to the association between obesity and prelabor/intrapartum cesarean birth.

Methods: We retrospectively analyzed Swiss cohort data from 394,812 singleton, cephalic deliveries between 2005 and 2020. Obesity (BMI ≥ 30 kg/m^2) was defined as the exposure and prelabor or intrapartum cesarean birth as the outcomes. Hypothesized mediators included gestational comorbidities, large-for-gestational-age infant, pregnancy duration >$41^{0/7}$ weeks, slower labor progress, labor induction, and history of cesarean birth. We performed path analyses using generalized structural equation modeling and assessed mediation by a counterfactual approach.

Results: Women with obesity had a cesarean birth rate of 39.36% vs. 24.12% in women without obesity. The path models mainly showed positive direct and indirect associations between obesity and cesarean birth. In the total sample, the mediation models explained up to 39.47% (95% CI 36.92–42.02) of the association between obesity and cesarean birth, and up to 57.13% (95% CI 54.10–60.16) when including history of cesarean birth as mediator in multiparous women. Slower labor progress and history of cesarean birth were found to be the most clinically significant mediators.

Conclusions: This study provides empirical insights into how obesity may increase cesarean birth rates through mediating processes. Particularly allowing for a slower labor progress in women with obesity might reduce cesarean birth rates and prevent subsequent repeat cesarean births in multiparous women.

KEYWORDS

cesarean birth, generalized structural equation modeling, mediation analysis, obesity

1 | INTRODUCTION

Pregnant women with obesity are more likely to experience cesarean birth (CB) compared to women without obesity.[1–5] Even though a CB may be a life-saving intervention and prevent greater harm, it can also entail adverse short and long-term health consequences,[6] especially in women with obesity[5] and in case of repeat CB.[6] Switzerland's overall CB rate of about 32%[7] clearly exceeds the considered ideal rate of 9%–16% at a population level[8] and indicates potentially preventable interventions. Obesity is not a medical indication for performing a CB[9,10] but is considered a risk factor for women to undergo CB.[1,3] Obesity-related comorbidities and complications have been suggested as contributors to increased CB rates in women with obesity.[5,11] Yet, little is known about the mechanisms. To reduce preventable CB, we therefore need to identify the underlying processes that might link obesity and CB through potential intervening factors.

Rogers et al.[11] developed a conceptual framework that has postulated complex mechanisms for how obesity may causally alter CB risk. One part of the framework theoretically addresses the role of obesity as an exposure that might operate through a set of mediators. First, pregnant women with obesity are at higher risk for hypertensive disorder in pregnancy (HDP), gestational diabetes mellitus (GDM), and correspondingly, for large-for-gestational-age infants (LGA).[1–3] These factors might serve as indications for a CB[12–14] by contributing to obstetric complications. In addition, this framework notes that women with obesity tend to have prolonged pregnancies and slower progress of labor, presumably due to hormonal imbalances; situations that may also increase the likelihood of a CB.[15–17] Gestational comorbidities and related conditions, as well as pregnancy duration $>41^{0/7}$ weeks might furthermore increase CB rates indirectly by operating through labor induction.[11] Finally, multiparous women with obesity appear more likely to have had a CB in a prior pregnancy[18,19] which in itself constitutes a risk factor for having a repeat CB in following pregnancies.[20]

To date, the conceptual assumptions of this framework concerning obesity and the risk of CB[11] have not yet been rigorously tested. The objective of this study was therefore to evaluate the proposed underlying processes of how mediators contribute to the association between obesity and prelabor or intrapartum CB, respectively.

2 | METHODS

2.1 | Sample and study design

In this study, we retrospectively analyzed preexisting Swiss cohort data from 394,812 singleton, cephalic deliveries between 22 and 43 weeks of gestation from January 2005 to December 2020. We excluded cases with compelling indication for CB (i.e., placenta previa, placental abruption, uterine rupture, umbilical cord prolapse),[21] as well as women with preexisting diabetes mellitus or chronic hypertension to establish temporal ordering of the observed variables for the mediation analysis.[22]

Data were collected by the Swiss Obstetric Study Group in over 100 obstetric hospitals in Switzerland. Attending clinicians registered data based on medical records and using preexisting standardized data entry forms. Senior clinicians verified accuracy and completeness at the time of discharge, and an independent quality control group reviewed the data thereafter. Hospitals were asked to verify data in case of inconsistencies. Maternal age, parity, gestational age, singleton/multiple pregnancy, and infant birthweight were recorded as continuous variables. All other data were collected using binary variables indicating the codes of the 10th revision of the International Statistical Classification of Diseases and Related Health Problems [ICD-10].[23] In compliance with the Swiss Human Research Act, Article 2,[24] this study did not need ethical approval as all data were anonymized and irreversibly de-identified before being transferred to researchers.

2.2 | Variables

Maternal prepregnancy *obesity* [E66] was selected as the binary exposure variable, which was recorded in case of a body mass index (BMI) ≥ 30 kg/m^2. The outcome was childbirth by means of *cesarean birth* (CB) [O82]. Since indications for CB tend to vary by timing of the decision, we differentiated between *prelabor* and *intrapartum CB*. *Prelabor CB* was defined as abdominal delivery performed before labor onset, and *intrapartum CB* when performed after labor onset or due to an emergency requiring urgent delivery.

Nine factors within the framework of Roger et al.[11] were included as hypothesized mediators: *Hypertensive disorder in pregnancy* (HDP; including pregnancy-induced hypertension [O13], preeclampsia [O14.0, O14.1], eclampsia [O15.9]), *gestational diabetes mellitus* (GDM) [O24.4], and *large-for-gestational-age infant* (LGA; defined as >90th percentile of gestational age- and sex-specific birthweight) as indicators for gestational comorbidities and related conditions; *pregnancy duration* $>41^{0/7}$ *weeks*; *prolonged first stage of labor* [O63.0], *prolonged second stage of labor* [O63.1], and *non-progressive labor >2 hours* [O63.9] for slower labor progress; and *history of CB* [O34.2]. Information on attempted *labor induction* by prostaglandin application, intravenous oxytocin, or physical measures was included as a hypothesized mediator between

gestational comorbidities, *LGA*, *pregnancy duration >41$^{0/7}$ weeks* and *CB*. All mediators were introduced as binary indicators.

2.3 | Statistical analyses

The data were analyzed using descriptive methods, path modeling, and mediation analyses. We described frequency of events and central tendency in the total sample and stratified by obesity. To compare the stratification groups, rate ratios, the Pearson's χ^2 for binary, and the unpaired t test with unequal variances for continuous variables were used.

We used two separate path models to assess associations between *obesity*, mediators and *prelabor* or *intrapartum CB*, respectively. Systems of structural relationships between the observed variables[25] were assessed with generalized structural equation modeling based on logistic regressions. This allowed for the simultaneous estimation of network dependencies with binary endogenous variables[26] and for exogenous variables to be correlated.[27]

A counterfactual approach for decomposition in logit models[28] was applied to estimate the direct and indirect associations within two sets of mediation models: 1. We assessed the associations between *obesity* and *CB* mediated through *gestational comorbidities*, *LGA*, *pregnancy duration >41$^{0/7}$ weeks*, *prolonged first and second stage of labor*, *non-progressive labor*, *and history of CB*. 2. We investigated the *labor induction* mediated associations between *obesity* and *intrapartum CB*, as well as between *gestational comorbidities*, *LGA*, *pregnancy duration >41$^{0/7}$ weeks* and *intrapartum CB*. For the first set of mediation models, the counterfactual scenario was conceptually based on the probability for the event of a CB for women with obesity if they had the distribution of the mediators of women without obesity.[28] We analogously constructed the counterfactual scenario for the second set of mediation models. Bootstrapping using 1,000 iterations was applied to obtain standard errors.

Inferential analyses were performed in the total sample for the outcome of *prelabor CB*, and for the outcome of *intrapartum CB* in a subsample including women who attempted trial of labor (i.e., excluding women with *prelabor CB*). To evaluate the role of *history of CB* within the associations, we only included multiparous women into the respective subgroup analyses.

All analyses were conducted using Stata version 15.1.[29]

3 | RESULTS

Among the 394,812 deliveries, women had obesity in 6.89% and experienced CB in 25.17% of cases (Table 1). Compared to women without obesity, women with obesity had 1.75- and 1.69-times the rates of prelabor CB (20.06% vs. 11.47%, $p \leq 0.001$) and intrapartum CB (24.13% vs. 14.27%, $p \leq 0.001$), respectively. They had proportionally higher rates of all mediators except prolonged second stage of labor. Most distinctly, their pregnancies were 3.66- and 3.01-times more often complicated by HDP (6.99% vs. 1.91%, $p \leq 0.001$) and GDM (15.71% vs. 5.22%, $p \leq 0.001$), and LGA occurred 2.01-times more frequently (18.68% vs. 9.27%, $p \leq 0.001$).

The path models mainly showed positive direct and indirect associations between obesity and CB (Figure 1A,B). Women with obesity were significantly more likely to give birth by prelabor (aOR 1.97) or intrapartum CB (aOR 1.63) than women without obesity, even when adjusted for all mediator and control variables. Individuals with obesity had a higher probability of HDM (aOR 4.01), GDM (aOR 3.68), LGA (aOR 2.31), pregnancy duration >41$^{0/7}$ weeks (aOR 1.09), prolonged first stage of labor (aOR 1.43), non-progressive labor (aOR 1.64) and history of CB (aOR 1.73) compared to birthing people without obesity. Labor induction was significantly more likely in women with vs. without obesity (aOR 1.58), even when adjusted for all mediator and control variables. The presence of HDP, GDM, or LGA was associated with an increased likelihood of labor induction (HDP: aOR 4.02, GDM: aOR 2.55, LGA: aOR 1.15), prelabor CB (HDP: aOR 3.96, GDM: aOR 1.60, LGA: aOR 1.42), and intrapartum CB (HDP: aOR 2.08, GDM: aOR 1.11, LGA: aOR 1.36). Women undergoing labor induction were more likely to experience an intrapartum CB (aOR 1.69) compared to those not induced. Intrapartum CB was considerably associated with prolonged first stage (aOR 4.19) and non-progressive labor (aOR 18.72). Women with prolonged second stage of labor were less likely to have an intrapartum CB (aOR 0.21). History of CB was substantially related to prelabor CB (aOR 19.56) and intrapartum CB (aOR 14.65) in multiparous women.

In the total sample, applicable mediators collectively accounted for 18.41% and 39.47% of the total association of obesity with prelabor or intrapartum CB, respectively, and for 57.13% and 46.72% when including history of CB as mediator in the subgroup of multiparous women (Table 2). Increased likelihoods attributable to processes independently operating through HDP, GDM, and LGA in the total sample were similar between prelabor and intrapartum CB (HDP: 10.41%/10.15%, GDM: 4.91%/3.83%, LGA: 5.52%/5.20%). With respect to intrapartum CB, 4.63% were mediated through prolonged first stage and 29.27% by non-progressive labor. For multiparous women, mediating processes through history of CB contributed 51.33% to the total relationship from obesity to prelabor CB and 26.19% to intrapartum CB.

TABLE 1 Characteristics of the study sample in total and stratified by obesity ($N = 394,812$).

Characteristic (m)	Statistic	Total sample ($N = 394,812$)	No obesity ($n = 367,627$, 93.11%)	Obesity ($n = 27,185$, 6.89%)	Rate ratio
Main outcomes					
Any cesarean birth (785)	*n* (%)	99,162 (25.17)	88,481 (24.12)	10,681 (39.36)	1.63
Prelabor cesarean birth (849)	*n* (%)	47,516 (12.06)	42,074 (11.47)	5442 (20.06)	1.75
Intrapartum cesarean birth (849)	*n* (%)	51,582 (13.09; 14.89[a])	46,350 (12.64; 14.27[a])	5232 (19.29; 24.13[a])	1.53; 1.69[a]
Mediators					
Hypertensive disorder in pregnancy (0)	*n* (%)	8918 (2.26)	7018 (1.91)	1900 (6.99)	3.66
Gestational diabetes mellitus (0)	*n* (%)	23,466 (5.94)	19,195 (5.22)	4271 (15.71)	3.01
Large-for-gestational-age infant (73)	*n* (%)	39,154 (9.92)	34,077 (9.27)	5077 (18.68)	2.01
Pregnancy duration >$41^{0/7}$ weeks (0)	*n* (%)	44,780 (11.34)	41,512 (11.29)	3268 (12.02)	1.06
Prolonged first stage of labor (0)	*n* (%)	10,106 (2.56; 2.91[a])	9243 (2.51; 2.84[a])	863 (3.17; 3.97[a])	1.26; 1.40[a]
Prolonged second stage of labor (0)	*n* (%)	34,889 (8.84; 10.05[a])	32,725 (8.90; 10.05[a]*)	2164 (7.96; 9.95[a]*)	0.89; 0.99[a]
Non-progressive labor (0)	*n* (%)	26,213 (6.64; 7.55[a])	23,807 (6.48; 7.31[a])	2406 (8.85; 11.07[a])	1.37; 1.51[a]
History of cesarean birth (0)	*n* (%)	51,991 (13.17; 25.16[b])	46,812 (12.73; 24.38[b])	5179 (19.05; 35.40[b])	1.50; 1.45[b]
Labor induction (0)	*n* (%)	79,122 (20.04)	70,756 (19.25)	8366 (30.77)	1.60
Control variables					
Maternal age (in years) (52)	Mean (SD)	31.20 (5.03)	31.23 (5.03)	30.87 (5.09)	NA
Primiparity (60)	*n* (%)	188,103 (47.65)	175,551 (47.76)	12,552 (46.18)	0.97
White ethnicity (97)	*n* (%)	363,268 (92.03)	337,580 (91.85)	25,688 (94.50)	1.03
Smoking status (0)	*n* (%)	23,150 (5.86)	20,103 (5.47)	3047 (11.21)	2.05

Note: All $p \leq 0.001$, except * with $p > 0.05$ for differences between women with and without obesity from Pearson's χ^2 for binary and from unpaired *t* test for continuous variables.

Abbreviations: %, percentage; m, number of missing values; *n*, number of observations; NA, Not applicable; SD, standard deviation.

[a]Excluding women with prelabor cesarean birth ($N = 347,296$).

[b]Subgroup of multiparous women only ($N = 206,649$).

Labor induction mediated 14.10% of the total association between obesity and intrapartum CB, even when adjusted for gestational comorbidities, LGA, and pregnancy duration >$41^{0/7}$ weeks (Table 3). Furthermore, labor induction as subsequent mediator accounted in descending order for 53.79%, 33.23%, 25.50%, and 3.89% of the total association between GDM, pregnancy duration >$41^{0/7}$ weeks, HDP, or LGA, respectively, with intrapartum CB.

4 | DISCUSSION

This study provides the first empirical insights into mediating processes between obesity and an increased likelihood of cesarean birth (CB). It confirms known risk factors for CB, and more importantly reinforces their relevance by showing their contribution to the potential underlying mediation between obesity and CB. We found that obesity may influence childbirth by CB mainly through slower labor progress and history of CB. More detailed analyses of the processes considered are discussed thematically below.

4.1 | Gestational comorbidities and related conditions

In line with the underlying conceptual framework,[11] our study confirms earlier evidence of obesity as a risk factor for HDP, GDM, and LGA,[1–3] which might serve as an indication for CB.[12–14,30] Similarly, several other studies reported an up to 4.5-times higher risk for women with obesity to suffer from gestational comorbidities like HDP and GDM, as well as about a doubled risk for LGA when compared to women without obesity.[1,2] CB are, in turn, commonly encountered by women with comorbidities such as HDP or GDM, in particular for women with high infant weight.[12] Women with HDP were nearly 33% more likely to undergo CB than normotensive women after controlling for obesity and diabetes in a large U.S. cohort study[30] while a cumulative trend towards a higher probability of CB in case of GDM and/or LGA – separately or combined – has been shown in another study.[14] We extended this prior knowledge by adding a quantification of the proposed mediating processes between obesity and prelabor and intrapartum CB through gestational

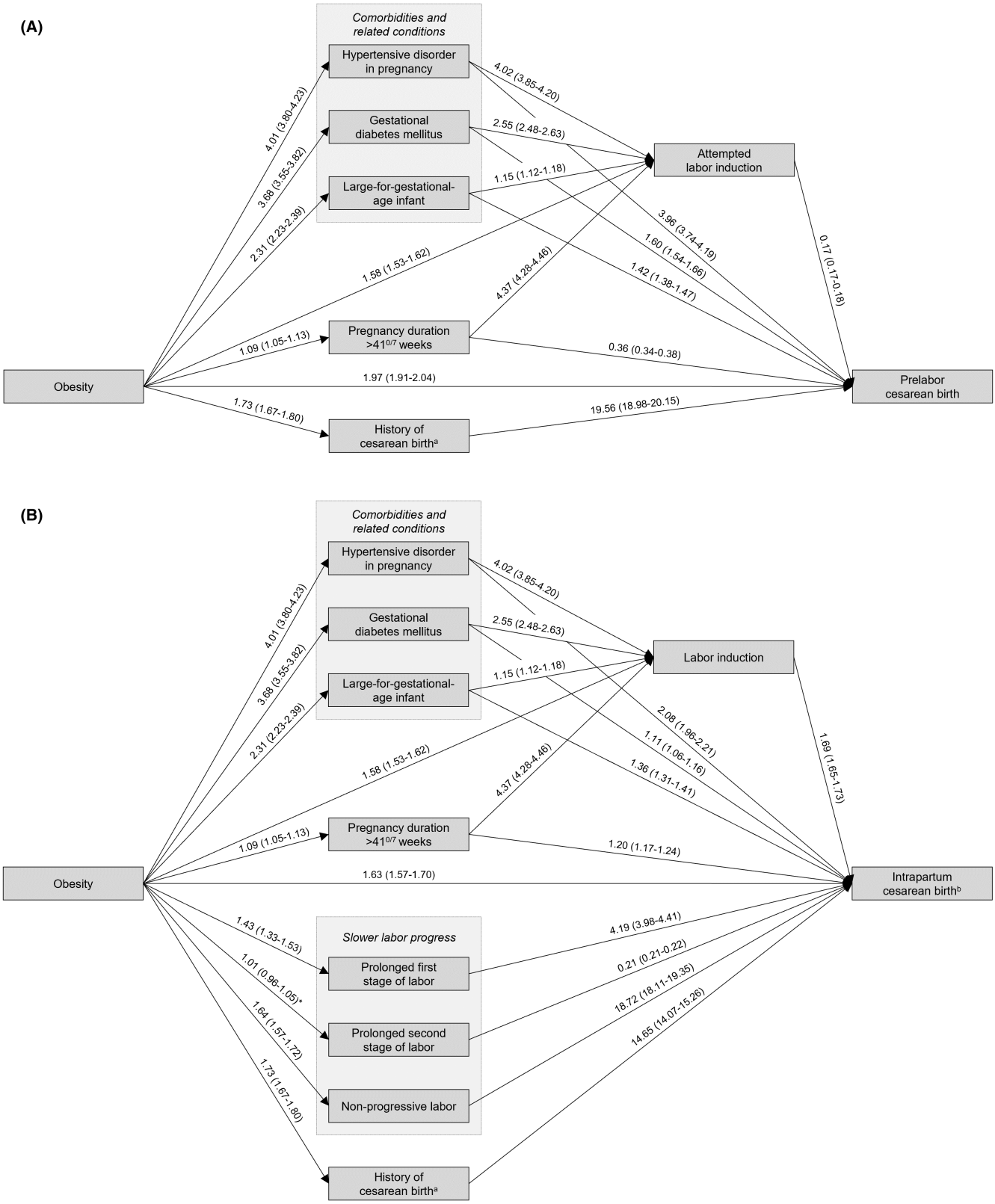

FIGURE 1 Path associations between obesity, mediators and (A) prelabor cesarean birth and (B) intrapartum cesarean birth, adjusted odds ratios (aOR) and 95% confidence intervals (CI). Analyses from full cases based on generalized structural equation modeling using logistic regression. Adjusted for control variables (*maternal age*, *primiparity*, *ethnicity*, *smoking status*, and *year of delivery*). (A) All $p \leq 0.001$; p values from Wald χ^2 tests. (B) All $p \leq 0.001$, except * with $p > 0.05$; p values from Wald χ^2 tests. (A, B) [a]Subgroup analyses of multiparous women only ($N = 206{,}649$). (B) [b]Excluding women with prelabor cesarean birth ($N = 347{,}296$). Arrows do not imply causal pathways but denote proposed processes.[11]

TABLE 2 Mediated associations between obesity and cesarean birth, adjusted odds ratios (aOR), and relative sizes of indirect to total associations in percentages (%) and 95% confidence intervals (CI).

Mediation paths	Direct aOR (95% CI)	Indirect aOR (95% CI)	Relative size in % (95% CI)
Prelabor cesarean birth			
Total sample			
Obesity → **All applicable mediators**[a] → Prelabor cesarean birth	1.74 (1.68–1.80)	1.13 (1.12–1.14)	18.41 (16.85–19.98)
Obesity → **Hypertensive disorder in pregnancy** → Prelabor cesarean birth	1.76 (1.70–1.81)	1.07 (1.06–1.07)	10.41 (9.33–11.49)
Obesity → **Gestational diabetes mellitus** → Prelabor cesarean birth	1.77 (1.71–1.83)	1.03 (1.03–1.03)	4.91 (4.10–5.71)
Obesity → **Large-for-gestational-age infant** → Prelabor cesarean birth	1.76 (1.71–1.82)	1.03 (1.03–1.04)	5.52 (4.82–6.21)
Obesity → **Pregnancy duration >41$^{0/7}$ weeks** → Prelabor cesarean birth	1.76 (1.70–1.82)	0.99 (0.99–1.00)	−1.13 (−1.76 to 0.49)
Subsample: Multiparous women[b]			
Obesity → **All applicable mediators** → Prelabor cesarean birth	1.35 (1.31–1.39)	1.49 (1.46–1.53)	57.13 (54.10–60.16)
Obesity → **History of cesarean birth** → Prelabor cesarean birth	1.38 (1.33–1.42)	1.40 (1.37–1.43)	51.33 (48.33–54.33)
Intrapartum cesarean birth[c,d]			
Total sample			
Obesity → **All applicable mediators**[a] → Intrapartum cesarean birth	1.51 (1.47–1.56)	1.31 (1.29–1.33)	39.47 (36.92–42.02)
Obesity → **Hypertensive disorder in pregnancy** → Intrapartum cesarean birth	1.71 (1.65–1.78)	1.06 (1.06–1.07)	10.15 (8.95–11.36)
Obesity → **Gestational diabetes mellitus** → Intrapartum cesarean birth	1.72 (1.66–1.79)	1.02 (1.02–1.03)	3.83 (2.93–4.73)
Obesity → **Large-for-gestational-age infant** → Intrapartum cesarean birth	1.72 (1.66–1.79)	1.03 (1.03–1.03)	5.20 (4.43–5.96)
Obesity → **Pregnancy duration >41$^{0/7}$ weeks** → Intrapartum cesarean birth	1.72 (1.66–1.79)	1.01 (1.00–1.01)	1.24 (0.89–1.59)
Obesity → **Prolonged first stage of labor** → Intrapartum cesarean birth	1.70 (1.64–1.77)	1.03 (1.02–1.03)	4.63 (3.56–5.70)
Obesity → **Prolonged second stage of labor** → Intrapartum cesarean birth	1.72 (1.65–1.78)	1.00 (1.00–1.01)*	0.19 (−0.55–0.93)*
Obesity → **Non-progressive labor** → Intrapartum cesarean birth	1.52 (1.47–1.57)	1.19 (1.17–1.21)	29.27 (26.45–32.09)
Subsample: Multiparous women[b]			
Obesity → **All applicable mediators** → Intrapartum cesarean birth	1.36 (1.30–1.43)	1.31 (1.26–1.36)	46.72 (41.24–52.21)
Obesity → **History of cesarean birth** → Intrapartum cesarean birth	1.46 (1.38–1.55)	1.14 (1.11–1.18)	26.19 (21.00–31.38)

Note: Counterfactual approach for decomposing the total associations in logit models into direct and indirect associations (see *Statistical analyses* for further information). Analyses from full cases. All $p \leq 0.001$, except * with $p > 0.05$. Mutually adjusted for mediators (*hypertensive disorder in pregnancy*, *gestational diabetes mellitus*, *large-for-gestational-age infant*, *pregnancy duration >41$^{0/7}$ weeks*) and control variables (*maternal age*, *primiparity*, *ethnicity*, *smoking status*, and *year of delivery*).

[a]Except history of cesarean birth.

[b]Subgroup analyses of multiparous women only ($N = 206{,}649$).

[c]Additionally mutually adjusted for *prolonged first stage of labor*, *prolonged second stage of labor*, and *non-progressive labor*.

[d]Excluding women with prelabor cesarean birth ($N = 347{,}296$).

TABLE 3 Labor induction mediated associations, adjusted odds ratios (aOR) and relative sizes of indirect to total associations in percentages (%) and 95% confidence intervals (CI).

Mediation paths	Direct aOR (95% CI)	Indirect aOR (95% CI)	Relative size in % (95% CI)
Intrapartum cesarean birth[a]			
Obesity → Labor induction → Intrapartum cesarean birth	1.68 (1.63–1.74)	1.09 (1.08–1.10)	14.10 (12.96–15.24)
Hypertensive disorder in pregnancy → Labor induction → Intrapartum cesarean birth	1.82 (1.72–1.92)	1.23 (1.21–1.24)	25.50 (23.31–27.69)
Gestational diabetes mellitus → Labor induction → Intrapartum cesarean birth	1.10 (1.05–1.14)	1.11 (1.11–1.12)	53.79 (41.76–65.81)
Large-for-gestational-age infant → Labor induction → Intrapartum cesarean birth	1.57 (1.52–1.61)	1.02 (1.02–1.02)	3.89 (3.23–4.54)
Pregnancy duration >41$^{0/7}$ weeks → Labor induction → Intrapartum cesarean birth	1.34 (1.30–1.37)	1.16 (1.15–1.16)	33.23 (30.67–35.78)

Note: Counterfactual approach for decomposing the total associations in logit models into direct and indirect associations (see *Statistical analyses* for further information). Analyses from full cases. All $p \leq 0.001$. Mutually adjusted for *obesity*, *hypertensive disorder in pregnancy*, *gestational diabetes mellitus*, *large-for-gestational-age infant*, *pregnancy duration >41$^{0/7}$ weeks*, and control variables (*maternal age*, *primiparity*, *ethnicity*, *smoking status*, and *year of delivery*).

[a]Excluding women with prelabor cesarean birth ($N = 347{,}296$).

comorbidities and LGA. Although about 10%–10.5%, 4%–5% and 5%–5.5% of the total associations between obesity and CB were mediated by HDP, GDM, and LGA, the absolute increase in the likelihood of CB attributable to these factors was rather small. Modifying lifestyle factors directed at a reduction in gestational comorbidities might thus not be the most promising approach for the prevention of CB in women with obesity as HDP, GDM, and LGA appear to play only a minor role in mediating the association. Even though, for example, exercise intervention might be successful in decreasing gestational comorbidities in women with obesity and improve certain outcomes, recent meta-analyses found no significant effect on CB rates.[31,32] Our findings therefore support the common notion that comorbidities and increased infant weight are associated with both obesity and CB, nonetheless they do not seem primarily responsible for high CB rates in birthing people with obesity.

4.2 | Slower labor progress

Current evidence supports our results that women with obesity tend to have a longer duration of the first stage of labor than women without obesity[15,33–35] and are more often diagnosed with non-progressive labor.[15] "Non-progressive labor", "failure to progress in labor" or "labor dystocia", respectively, has been shown to be a frequent indication for CB,[36] especially in women with obesity.[37] We found about 1.5-times higher odds of prolonged first stage, as well as non-progressive labor in women with obesity compared to women without obesity. These factors were in turn associated with a 4- to almost 19-times higher probability of an intrapartum CB. Prolonged first stage and non-progressive labor emerged to be substantial mediators, jointly accounting for about a third of the total association of obesity with intrapartum CB in our study. Explanatory approaches for this strong association mainly assume obesity to be related to hormonal changes that may negatively affect myometrial activity and its oxytocin sensitivity leading to delayed onset of active labor, hypocontractility and reduced labor endurance.[15–17] Cephalopelvic disproportions due to large fetal size and increased maternal pelvic soft tissue have moreover been discussed as obstructive reasons for labor dystocia and the related risk for CB as they may cause a relative narrowing of the birth canal.[3,37,38] In our study however, non-progressive labor remained a highly relevant mediator after adjusting for LGA. In addition, prolonged second stage of labor did not prove to be a relevant mediator. These findings reinforce the assumption that increased deposition of soft tissue in the maternal pelvis was not likely to be a substantial reason for labor dystocia in our sample. Suboptimal myometrial function seems therefore a more likely cause of non-progressive labor and subsequent CB than obstructive reasons in women with obesity.[15,39]

4.3 | History of cesarean birth

For multiparous women, our findings highlight the important role of a previous CB in the association of obesity with a repeat CB as proposed.[11] History of CB has been shown to be more prevalent[18,19] and a more common indication for repeat CB[20] in multiparous women with vs. without obesity. Analogously, we showed that the probability of

having had a CB in a previous pregnancy was almost doubled in women with obesity compared to women without obesity. This fact subsequently increased the odds of prelabor CB by almost 20-times and by nearly 15-times for intrapartum CB. Moreover, history of CB explained over 51% and 26% of the association of obesity with prelabor or intrapartum CB, respectively. Similar results were found by Hermann et al.,[18] where 65.8% of women with obesity and previous CB had a repeat prelabor CB compared with 47.6% for women considered normal weight. Other studies furthermore indicated that obesity decreases the success rate of vaginal birth after previous CB (VBAC)[5,40] leading to more repeat intrapartum CB. These results suggest that the prevention of primary CB is fundamental to lower repeat prelabor CB rates and the number of repeat intrapartum CB due to unsuccessful trial of labor in women with obesity.[5]

4.4 | Induction of labor

The results of this study further substantiate the already distinct evidence that women with obesity are more likely to have their labor induced compared to women without obesity,[4,15,19,41] even after accounting for potential medical indications for labor induction such as gestational comorbidities, LGA, or pregnancy duration $>41^{0/7}$ weeks.[15,41] These unexplained labor inductions accounted for about one in seven intrapartum CB in women with obesity in our study. Moreover, our findings corroborate that longer pregnancy duration, HDP, GDM, and LGA are frequent reasons for labor induction[18] and that especially comorbidities and LGA lead to higher CB rates.[13,14] Further analyses revealed that almost 26% to 54% of the association between the gestational comorbidities HDP and GDM, and intrapartum CB were mediated by performing a labor induction. However, our results regarding the nearly 1.7-times increased probability of intrapartum CB in all individuals undergoing labor induction are opposed to findings from previous research.[42–44] Cochrane reviews found no increased CB rates when comparing planned early delivery with expectant management beyond 34 or 37 gestational weeks in the general pregnant population,[42] in women with hypertensive disorders,[44] or GDM.[43] Yet, the evidence from the general pregnant population with or without comorbidities might not necessarily be applicable when considering labor induction in women with obesity.[45] Above-mentioned pathophysiological changes in labor function associated with obesity might similarly impede the success of labor induction, by making it longer and more difficult.[46] Women with obesity appear more likely to end labor induction with CB in some studies,[15,20,46,47] while other studies did not find labor induction to affect the CB rate in women with a BMI ≥ 35 kg/m^2,[48,49] or even found reduced odds of CB in case of term elective induction.[50,51] Given these inconclusive findings, more research is needed to draw conclusions on labor induction as a mediator linking obesity, gestational comorbidities, and/or pregnancy duration $>41^{0/7}$ weeks with CB.

4.5 | Strengths and limitations

Our study is the first to test the conceptual assumptions of a theoretically well-founded framework[11] using a large preexisting Swiss cohort data set. The results provide important empirical insights into mediating processes linking obesity and CB. As a limitation, no definitive conclusions can be drawn regarding the proposed causality of the models due to the retrospective observational nature of our data. We implemented multiple measures to strengthen these conclusions, however. First, we tried to establish temporal ordering[22] of exposure, mediators and outcomes by excluding women with preexisting diabetes mellitus or chronic hypertension from our sample. It is therefore likely that the temporal ordering holds, even with respect to potential history of CB considering the prevailing length of birth intervals and the time dimension of developing obesity. Second, the tested assumptions of mediation were derived a priori from an existing framework.[11] Nevertheless, there might be other reasonable models on how the factors may be related. Further, there may be potential confounding factors that we were unable to consider in our analyses due to institutional privacy reasons or data unavailability (e.g., hospital type, socioeconomic status). Since a substantial direct link between obesity and CB remained unexplained by the proposed mediators in our study, broader context-level factors may also play a role in the association by interacting with the obesity status of women to modify the risk for medical childbirth intervention.[11] Evidence indeed exists for high variation in CB rates within and across settings.[e.g.,52–54] Maternal preferences for the mode of childbirth might be shaped by cultural factors, fear of pain, past experiences, or interaction with clinicians.[55] Clinicians' characteristics like gender, age, education, and experience,[56,57] and clinicians' values, beliefs, and attitudes likely influence thresholds to perform operative deliveries.[56,58] Decision-making might moreover be modified by medicolegal considerations, financial incentives, institutional factors, as well as resources and management policies, among others.[e.g.,54,56,57] Yet, the interaction of the broader context with obesity still needs to be studied to understand its relevance for childbirth

outcomes. Our results thus encourage further prospective studies including context-level factors to substantiate the causality of the processes underlying the associations between maternal obesity and the likelihood of CB.

4.6 | Implications and conclusions

Providing empirical insights into how obesity may increase CB rates, this study suggests the reduction of primary and subsequent repeat CB among women with obesity by preventive actions targeting the proposed mediating processes.

Given the significance of prolonged first stage and non-progressive labor as joint mediators, clinicians might allow for a slower labor progress in women with obesity and tolerate longer stagnation before diagnosing "non-progressive labor", "failure to progress", "labor arrest", or "labor dystocia"[4,5,10,17,20,33,34] as long as there is no compelling medical indication for an intrapartum CB. The traditional definition of non-progressive labor based on Friedman's work[59] has been challenged[60] and contemporary findings from non-medicalized birthing practice have prompted a refinement in understanding physiological labor durations and time thresholds.[35] An emphasis on care characterized by *watchful waiting* such as midwife-led models of care and/or continuous labor support may acknowledge individual childbirth processes[61] and decrease intervention rates while increasing satisfaction and being safe for mother and child.[62–64] Moreover, decisions to perform labor induction in women with obesity ought to be discussed in the context of possible consequences of a CB, alongside maternal and fetal risks and benefits for planned earlier delivery,[46] particularly since maternal obesity alone is not considered a medical indication for labor induction.[10,65] Evidence-based and person-centered childbirth care may improve known short- and long-term health outcomes for women and children[6] by safely preventing primary CB and subsequently reducing repeat CB in women with obesity.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interest.

DATA AVAILABILITY STATEMENT

Intellectual property rights of the data used for this study are owned by the Swiss Obstetric Study Group (Arbeitsgemeinschaft Schweizerischer Frauenkliniken, Switzerland) and are available for third parties on reasonable request.

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