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Associations between maternal race, socioeconomic status and shoulder dystocia: a population-based retrospective cohort study in Ontario Canada

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Abstract

Background Shoulder dystocia is a severe and emergent adverse perinatal complication. Existing literature suggests that the risk of shoulder dystocia varies by race and socioeconomic status, but research in this area is lacking in Canada.

Objectives To examine variations of shoulder dystocia by maternal race and maternal socioeconomic status and evaluate the interaction between race and macrosomia on the occurrence of shoulder dystocia.

Study design We conducted a population-based retrospective cohort study including White and Black pregnant people who participated in prenatal screening and had singleton stillbirths or live hospital births, excluding those with no-labour caesarean deliveries, from January 1, 2013 to March 31, 2021 in Ontario, Canada. The BORN Information System and Canadian Institute for Health Information databases were linked for analysis. We used multivariable Poisson regression with robust error variance models to examine the association between maternal race, neighbourhood household median income and educational level, and shoulder dystocia, while adjusting for maternal age, parity, previous caesarean section, obesity, substance use, pre-existing physical and mental health conditions, gestational diabetes, obstetrician in antenatal care team, hospital level of maternal care, gestational age, infant sex and rural residence. The interaction between maternal race and fetal macrosomia on the outcome of shoulder dystocia was evaluated.

Results Among the total cohort of 422,580 pregnant individuals, 10.2% were Black individuals and 89.8% were White individuals. The incidence of shoulder dystocia was 4.4% ($N=18,592$) among the entire cohort, 4.4% ($N=16,739$) among White individuals and 4.3% ($N=1,853$) among Black individuals. There was a statistically significant interaction ($P<0.001$) between maternal race and infant macrosomia on shoulder dystocia. Among the sub-cohort of pregnant individuals giving birth to infants with macrosomia, Black individuals had a 17% higher risk of experiencing shoulder dystocia (aRR: 1.17, 95% CI: 1.08–1.28) than White individuals. Individuals living in the lowest-income and least

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educated neighborhoods had a 19% and 25% higher risk of experiencing shoulder dystocia, respectively, compared to those in the wealthiest or most educated neighborhoods. However, no significant racial or household income differences on shoulder dystocia were observed among those giving birth to non-macrosomic infants.

Conclusion The higher risk of shoulder dystocia among birthing individuals in lower socioeconomic status neighborhoods, coupled with the elevated risk for Black individuals at the same socioeconomic status level, suggests a double burden faced by Black individuals under Ontario's universal healthcare system. The relationships between race, socioeconomic status, and macrosomia in shoulder dystocia are complex, highlighting the need for further studies to investigate potential racial disparities and discrimination. There is a need for policies and interventions that specifically address these inequities among Black communities to improve healthcare in Ontario.

Keywords Mother, Pregnancy, Race, Socioeconomic status, Infant, Shoulder dystocia, Macrosomia

Introduction

Shoulder dystocia (SD) is defined as an emergent peripartum event where the infant becomes obstructed in the birth canal after the delivery of the fetal head would be warranted [1]. Complex maneuvers by a skilled birth attendant are necessary to relieve the obstruction and allow the infant to deliver [2]. Published studies have reported an incidence rate ranging from 0.15% to 4.4% in the general pregnant population [3–6]. SD can cause severe maternal complications and may lead to severe neonatal adverse outcomes [2].

Although the mechanism of SD is not understood, the most well-known risk factors include fetal macrosomia – defined as a birth weight exceeding 4000 g, as well as maternal pre-pregnancy obesity and gestational diabetes mellitus (GDM) [3, 7, 8]. In addition, variation in the incidence of SD may be influenced by race (White and Black) and socioeconomic status (SES) [9–11]. Researchers in the U.S. have reported that Black pregnant people had higher odds of SD compared to White pregnant people [9, 10]. Studies also reported that pregnant people with lower SES experience an increased risk of SD compared to those with higher SES [10, 11].

Among various factors, macrosomia (high birth weight) has been reported as the most significant contributor to the risk of SD [12]. High birth weight is typically associated with larger infants, which increases the likelihood of obstructed labor and, consequently, SD. In contrast, SD is relatively uncommon among non-macrosomia infants. In our pilot study, we observed a significantly higher incidence of SD among Black individuals compared to White individuals within the macrosomia sub-cohort ($p < 0.05$), whereas no significant racial difference was found in the overall cohort ($p > 0.05$). These findings suggest a potential interaction between race and macrosomia in relation to the occurrence of SD.

No published studies have examined the complex relationships between race, SES, macrosomia and the risk of SD. Furthermore, Canadian research in this area is lacking. In previous studies, we identified health inequities in numerous maternal and child adverse outcomes within the Canadian universal health system [13–15]. Whether such inequities exist for SD in the province of Ontario, Canada, is unknown. To address this gap, we conducted a study to examine variations of SD by maternal race and maternal SES and evaluate the interaction between race and macrosomia on the occurrence of SD.

Methods

Study design

This is a population-based retrospective cohort study.

Study setting and data sources

Study population

In Ontario, all pregnant people are routinely offered a multiple marker screening (MMS) test in the first or second trimester of pregnancy and can access the test free of charge [13]. Participants' race (likely self-identified as White, Asian, Black, Indigenous Peoples, or Other) is collected during the MMS requisition process [13]. We included White and Black pregnant people who participated in this prenatal screening program and had singleton livebirths or stillbirths hospital births in the province of Ontario between January 1, 2013 to March 31, 2021 in Ontario, Canada. We excluded birthing individuals who experienced no-labour caesarean deliveries. We excluded birthing individuals who underwent caesarean deliveries without labour, as SD is unlikely to occur in the absence of labour [12]. The study cohort included 422,580 pregnant individuals of whom 10.2% were Black individuals and 89.8% were White individuals. Please see Fig. 1 for details of the cohort generation process.

Data sources and data linkage

BORN database The Better Outcomes Registry & Network (BORN) is a comprehensive registry collecting, managing, protecting, and sharing data on every pregnancy and birth in Ontario, through the BORN Information System (BIS), since April 1, 2012 [16, 17]. The BIS captures a wide range of data elements, including maternal demographics and health behaviors; pre-existing maternal health problems; prenatal screening results;

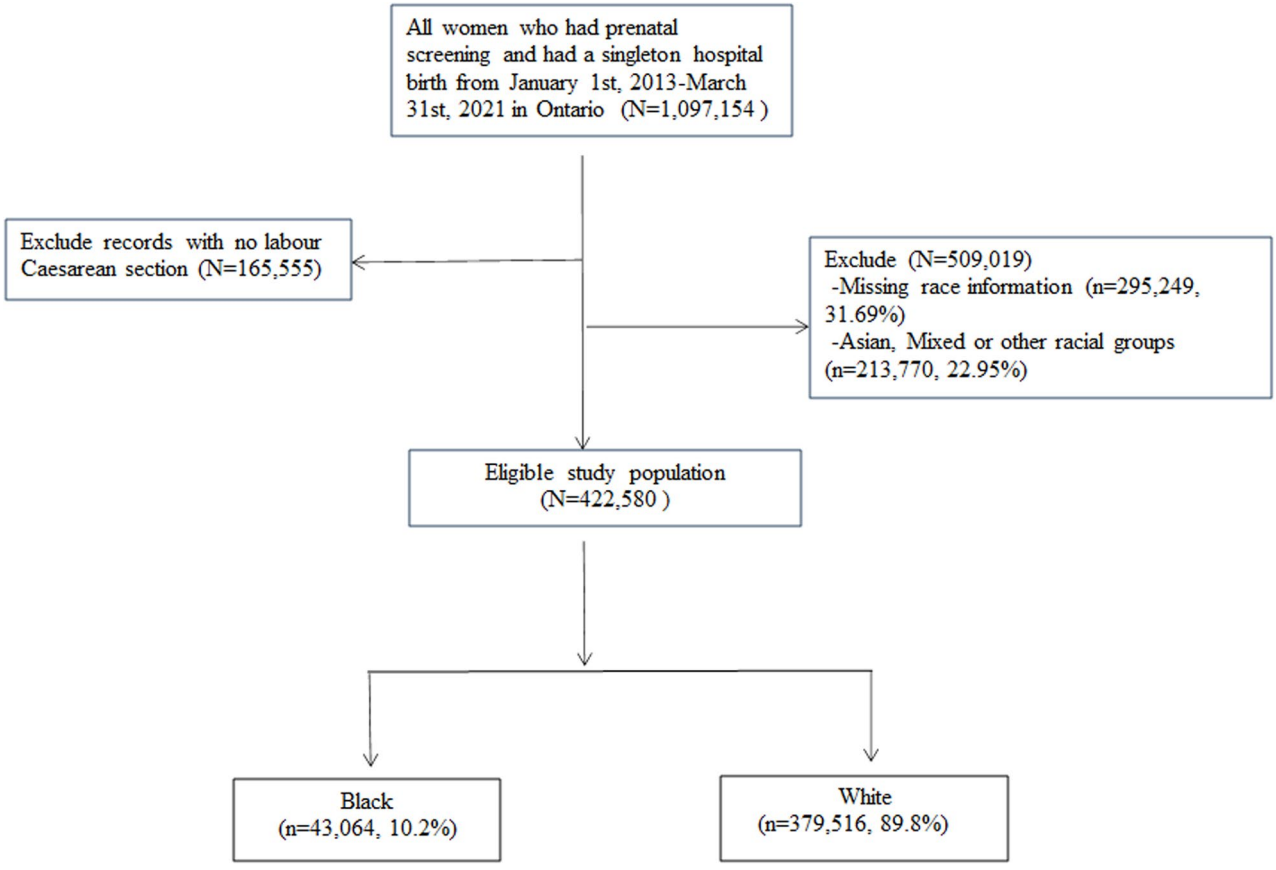

Fig. 1 Flowchart of data sources and data linkage for study cohort (January 1, 2013 - March 31, 2021)

obstetric complications; intra-partum interventions; birth and neonatal outcomes [16, 17].

The Discharge Abstract Database (DAD) Maintained by Canadian Institute for Health Information (CIHI) collects patients' information from acute hospitals' discharge abstraction [18, 19]. BORN receives CIHI-DAD maternal, newborn, and infant (≤ 1 year) records from acute care facilities in Ontario [18]. By linking the DAD with the BIS, we can identify SD in two different sources [17].

The Canadian Census collects extensive sociodemographic information at different levels of geography [20]. The Postal Code Conversion File Plus (PCCF+) version 7B contains the postal codes and their corresponding geographic Dissemination Areas (DAs), the smallest standard geographic area in Canada [21–23]. We linked the study cohort to the 2016 Census data via the PCCF+ file using the BIS maternal residential postal codes. We obtained maternal neighborhood level SES indicators from the 2016 Census data including median after-tax household income and the percentage of people aged 25–64 with a university degree or higher at the DA level in Ontario, Canada [13–15].

Exposure variables

Maternal race (Black/White) was self-reported and recorded on prenatal screening requisition forms by care providers.

As we mentioned above, we derived two commonly used SES indicators as SES exposure variables from Census data in this study. Neighborhood income and education level were categorized into quintiles (Q1 lowest - Q5 highest).

Outcome

SD cases were captured from the data element of Labour and Birth Complications in the Labour and Birth encounter data in BIS [16]. Additional SD diagnosed cases were identified from CIHI-DAD, which was coded as O66.0 using the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Canadian adaptation (ICD-10-CA) [24, 25].

Covariates and interaction term

The covariates included maternal age at delivery (≥ 35 or < 35 years old), parity, previous caesarean delivery, obesity (pre-pregnancy body mass index [BMI ≥ 30 kg/m^2]), pre-existing maternal health condition during pregnancy, GDM, mental health status pre-pregnancy and during

pregnancy (a composite measure of depression and anxiety), substance use/alcohol exposure/smoking during pregnancy (yes or no), gestational age at birth (≥ 37 or < 37 weeks), obstetrician in antenatal care team, hospital level of maternal care, maternal residence in urban or rural area and infant sex. Covariates and potential confounders were selected based on a literature review, their clinical relevance and experts' input from the research team [13, 14, 26, 27].

Macrosomia was assessed as a potential interaction using the birth weight data element from the BORN database. Infants with a birth weight of > 4000 g were classified as having macrosomia, while those weighing < = 4000 grams at birth were classified as non-macrosomia.

Ethics

This study received ethical approval from the Children's Hospital of Eastern Ontario Research Ethics Board (CHEOREB# 20/15PE). Since we use the registry and health administrative data for the analysis, patient consent is not required according to the law in Ontario Canada.

Statistical analysis

We conducted a descriptive analysis showing the distributions of maternal and infant characteristics. We evaluated the interaction between maternal race and infant macrosomia on the outcome of SD in a multivariable logistic regression model while adjusting for maternal age, parity, previous caesarean delivery, obesity, substance use, pre-existing physical and mental health conditions, gestational diabetes, obstetrician in antenatal care team, hospital level of maternal care, maternal rural residence, gestational age at birth ≥ 37 weeks and infant sex, and two neighborhood SES indicators.

We used multivariable Poisson regression with robust error variance models to examine the association between maternal race, neighborhood income and education level and SD while adjusting for maternal age at birth, parity, previous caesarean delivery, obesity, substance use, pre-existing physical and mental health conditions, GDM, obstetrician in antenatal care team, hospital level of maternal care, maternal rural residence, gestational age at birth, and infant sex among macrosomia and non-macrosomia sub-cohorts. Modified Poisson regression with robust error variance is a valid and widely accepted method for estimating relative risk in studies with binary outcomes, particularly when the outcome is common [28]. In the analysis stratified by macrosomia status, the incidence of SD exceeds 10% among the macrosomia sub-cohort, indicating that it is not a rare outcome. Therefore, we applied a modified Poisson regression model to estimate relative risks (RRs).

In addition, the additive interaction effect was evaluated by combining macrosomia status and race into one variable, categorizing it into four groups: Black mother delivering a macrosomic infant, Black mother delivering a non-macrosomic infant, White mother delivering a macrosomic infant, and White mother delivering a non-macrosomic infant [29]. We assessed this effect and SES factors in a multivariable logistic regression model while adjusting for the same covariates described above in the whole cohort which included pregnant people who gave birth to infants with and without macrosomia [13]. Given that SD is a rare outcome, with a low incidence rate (4.4%) in the whole cohort, the odds ratios (ORs) estimated from a logistic regression model closely approximate RRs that would be obtained from a Poisson regression model [30].

In the multivariable regression models, missing values for obesity (defined by BMI) were treated as a separate category. For other covariates, cases with missing data were excluded from the analysis, as the overall proportion of missingness was around 5%, and its impact on the results was considered minimal.

We also conducted intersectional analysis to unveil the complex relationships between race, two SES indicators and the outcome of SD in a Canadian healthcare system [31].

We used SAS 9.4 (SAS Institute Inc., Cary NC) to perform all data management and analysis [32].

Results

The incidence of SD was 4.4% (N = 18,592) among the entire cohort, 4.4% (N = 16,739) among White individuals and 4.3% (N = 1,853) among Black individuals. The incidence of SD was 2.8% (N = 41) among White people and 2.6% (N = 10) among Black people within the stillbirth subpopulation (appendix Tables 1 and 2). In the macrosomia sub-cohort, the incidence of SD was 14.3% (N = 6,283) among White individuals and 18.0% (N = 581) among Black individuals. In the non-macrosomia cohort, the incidence of SD was 3.1% (N = 10,363) and 3.2% (N = 1,260) among White and Black individuals, respectively. A statistically significant interaction (P < 0.001) was found between maternal race and infant macrosomia on SD.

Table 1 shows the distributions of characteristics among White and Black pregnant people. Compared to White people, more Black people lived in the lowest household income quintile (36.9% vs. 17.3%) while more Black people lived in the highest education level quintiles (11.9% vs. 16.5%). Additionally, a higher proportion of Black people had obesity (23.3% vs. 19.2% at BMI ≥ 30 kg/m^2).

Figure 2 shows the incidence of SD by neighborhood income quintiles among White and Black birthing

Table 1 Comparison of characteristics between Blacks and Whites

Variables	Black		White		
	N	%*	*N*	%*	*P* value[£]
Maternal Age at delivery (years) (Mean ± SD)	30.5 ± 5.6		30.8 ± 4.9		< 0.01
≥35	10,865	25.23	85,082	22.42	
<35	32,199	74.77	294,434	77.58	
Neighbourhood Median household income after tax					< 0.01
Quintile 1 (lowest)	15,908	36.94	65,716	17.32	
Quintile 2	8541	19.83	74,281	19.57	
Quintile 3	5950	13.82	81,143	21.38	
Quintile 4	7433	17.26	77,913	20.53	
Quintile 5 (highest)	4335	10.07	72,048	18.98	
Missing	897	2.08	8415	2.22	
Neighbourhood education level					< 0.01
Quintile 1 (lowest)	5103	11.85	62,477	16.46	
Quintile 2	9038	20.99	72,569	19.12	
Quintile 3	10,106	23.47	72,812	19.19	
Quintile 4	11,627	27	81,216	21.4	
Quintile 5 (highest)	6312	14.66	82,152	21.65	
Missing	878	2.04	8290	2.18	
Maternal pre-existing health conditions					< 0.01
Yes	3028	7.03	28,454	7.5	
No	40,036	92.97	351,062	92.5	
Mental health Condition before or during pregnancy					< 0.01
Yes	3295	7.65	71,757	18.91	
No	39,769	92.35	307,759	81.09	
Previous caesarean section					< 0.01
Yes	3177	7.38	19,082	5.03	
No	38,364	89.09	350,626	92.39	
Missing	1523	3.54	9808	2.58	
Pre-pregnancy BMI (kg/m^2) (Mean ± SD)	27.1 ± 6.3		26.0 ± 6.1		< 0.01
Underweight (< 18.5)	1253	2.91	11,238	2.96	
Normal (18.5–24.9)	14,514	33.7	176,203	46.43	
Overweight (25.0-29.9)	11,519	26.75	90,361	23.81	
Obese (≥ 30)	10,048	23.33	72,966	19.23	
Missing	5730	13.31	28,748	7.57	
Parity					<0.01
Yes	25,073	58.22	185,310	48.83	
No	17,555	40.76	191,430	50.44	
Missing	436	1.01	2776	0.73	
Smoking, drug or alcohol use during pregnancy					< 0.01
Yes	2724	6.33	48,853	12.87	
No	38,845	90.2	321,779	84.79	
Missing	1495	3.47	8884	2.34	
Gestational diabetes					< 0.01
Yes	2729	6.34	19,737	5.2	
No	40,335	93.66	359,779	94.8	
Maternal residence in rural area					< 0.01
Yes	441	1.02	57,092	15.04	
No	42,471	98.62	320,827	84.54	
Missing	152	0.35	1597	0.42	
Obstetrician in antenatal care team					< 0.01
Yes	33,699	78.25	260,733	68.7	
No	9365	21.75	118,783	31.3	
Hospital level of maternal care					< 0.01

Table 1 (continued)

Variables	Black		White		
	N	%*	*N*	%*	*P* value[£]
level I	1188	2.76	42,590	11.22	
level II	31,540	73.24	234,583	61.81	
level III	9844	22.86	98,886	26.06	
Missing	492	1.14	3457	0.91	
Gestational age at birth (Mean ± SD)	39.0 ± 1.9		38.6 ± 2.6		< 0.01
20–36	3433	7.97	22,142	5.83	
37–38	10,904	25.32	88,127	23.22	
39–40	23,530	54.64	212,029	55.87	
>=41	5174	12.01	57,180	15.07	
Missing	23	0.05	38	0.01	
Infant sex					0.02
Female	21,141	49.09	184,121	48.51	
Male	21,883	50.82	195,120	51.41	
Missing or indeterminate	40	0.09	275	0.07	
Infant birth weight in grams (Mean ± SD)	3249.9 ± 610.5		3414.7 ± 536.3		
Macrosomia					<0.01
Yes	3221	7.48	43,949	11.58	
No	39,476	91.67	332,968	87.73	
Missing	367	0.85	2599	0.68	

* % calculation represents the column percentage, missing values were excluded for % calculation

[£] A Chi-square test was performed for each categorical variable to obtain the corresponding *p*-value

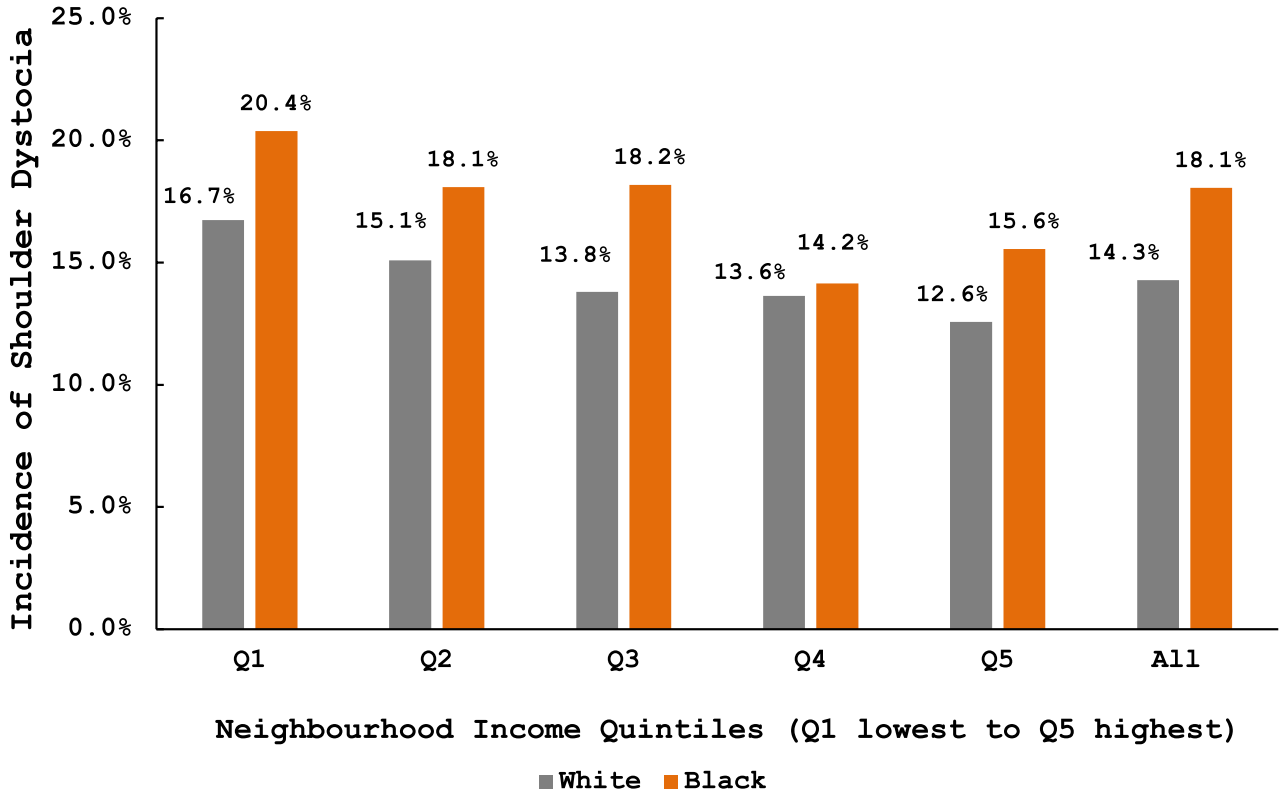

Fig. 2 Incidence of shoulder dystocia by neighborhood income quintiles among White and Black birthing individuals who gave birth to babies with macrosomia
Data source: BORN Ontario, January 1 2013 to March 31 2021

individuals who gave birth to babies with macrosomia. Compared to White people, Black pregnant individuals had a higher incidence of SD across all income and education quintiles; the incidence of SD was higher among people living in the lowest-income neighbourhoods compared to people living in the highest-income neighbourhoods. Similarly, all Black birthing individuals had higher SD incidence across all neighbourhood education quintiles (Fig. 3).

Table 2 shows associations between race, SES and risk of SD stratified by macrosomia and non-macrosomia sub-cohorts. Among the sub-cohort of macrosomia, compared to White pregnant people, Black pregnant people had a 17% higher risk of experiencing SD (aRR: 1.17, 95% CI: 1.08–1.28). Those living in the lowest household income neighborhoods had a 19% higher risk of experiencing SD (aRR: 1.19, 95% CI: 1.1–1.3) and those living in neighborhoods with the least level of education had a 25% higher risk of SD (aRR: 1.25, 95% CI: 1.14–1.37) compared to those living in the wealthiest or most educated neighborhoods respectively.

Among sub-cohort of non-macrosomia (Table 2), compared to people living in the highest educated neighborhoods, people living in the least educated neighborhoods had a 29% higher SD (aRR: 1.29, 95% CI: 1.2–1.38). No difference in SD risk was detected by race and neighbourhood household income.

Table 3 shows the additive interaction effect of SD among Black pregnant individuals with macrosomic infants. Compared to White pregnant individuals with non-macrosomic infants, Black pregnant individuals with macrosomia infants had six times the odds of experiencing SD. White pregnant individuals with macrosomic infants had four times the odds of experiencing SD, while Black pregnant individuals with non-macrosomic infants showed no difference in the odds of experiencing SD.

Discussion

Principal findings

We found an interaction between fetal macrosomia and race on the risk of SD. In the sub-cohort of individuals who gave birth to infants with fetal macrosomia, Black individuals had a higher risk of SD than White individuals, whereas no such difference was observed in the non-macrosomia group. The combination of living in lower SES neighborhoods and being a Black pregnant individual is associated with an increased risk of SD.

Results in the context of what is known

The incidence ranges of SD in the entire birth cohort and among individuals who experienced macrosomia were

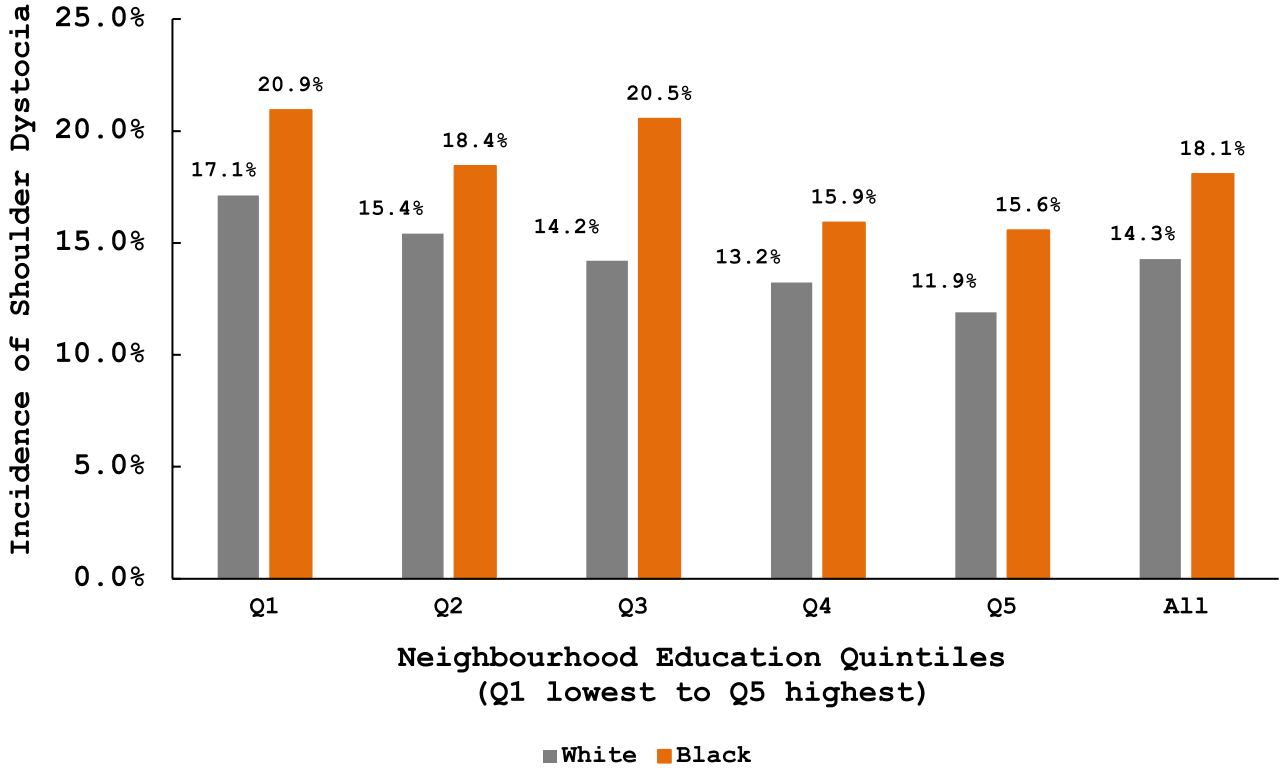

Fig. 3 Incidence of shoulder dystocia by neighborhood education quintiles among White and Black birthing individuals who gave birth to babies with macrosomia
Data source: BORN Ontario, January 1 2013 to March 31 2021

Table 2 Associations between maternal race, maternal socioeconomic status and shoulder dystocia by macrosomia and non-macrosomia

Variable	Macrosomia		Non-macrosomia	
	Crude RR (95% CI)	Adjusted RR (95% CI)	Crude RR (95% CI)	Adjusted RR (95% CI)
Maternal Race				
Black	1.26 (1.17–1.36)	1.17 (1.08–1.28)	1.03 (0.97–1.09)	1.03 (0.97–1.1)
White	Reference	Reference	Reference	Reference
Neighbourhood median household income after tax				
Quintile 1 (lowest)	1.36 (1.27–1.46)	1.19 (1.1–1.3)	1.21 (1.14–1.28)	1.05 (0.98–1.12)
Quintile 2	1.21 (1.12–1.3)	1.11 (1.02–1.2)	1.15 (1.08–1.22)	1 (0.94–1.07)
Quintile 3	1.1 (1.03–1.19)	1.02 (0.94–1.11)	1.15 (1.08–1.22)	1.02 (0.96–1.09)
Quintile 4	1.08 (1-1.16)	1.05 (0.97–1.13)	1.06 (1-1.13)	1 (0.94–1.07)
Quintile 5 (highest)	Reference	Reference	Reference	Reference
Neighbourhood education level				
Quintile 1 (lowest)	1.43 (1.33–1.54)	1.25 (1.14–1.37)	1.41 (1.33–1.49)	1.29 (1.2–1.38)
Quintile 2	1.29 (1.2–1.39)	1.17 (1.07–1.27)	1.29 (1.22–1.37)	1.19 (1.11–1.27)
Quintile 3	1.22 (1.13–1.31)	1.13 (1.04–1.23)	1.15 (1.08–1.22)	1.09 (1.02–1.16)
Quintile 4	1.11 (1.03–1.2)	1.05 (0.97–1.14)	1.09 (1.03–1.15)	1.06 (1-1.13)
Quintile 5 (highest)	Reference	Reference	Reference	Reference

(1) Neighbourhood education level was defined as the percentage of people aged 25–64 who had a university degree or higher at a dissemination area

(2) Neighbourhood income was defined as median household income after tax at a dissemination area

(3) Neighbourhood education level and income were categorized as quintiles. The lowest value is Q1 and highest value is Q5

(4) Macrosomia and non-macrosomia are two sub-cohorts, where two separated Poisson regression with robust error variance models were conducted to examine associations between maternal race, two SES indicators and shoulder dystocia while adjusting for maternal age, parity, previous caesarean delivery, substance use, pre-existing physical and mental health conditions, gestational diabetes, obstetrician in antenatal care team, hospital level of maternal care, maternal rural residence, gestational age at birth ≥ 37 weeks and infant sex

(5) We categorized "Unknown" values of obesity as a separate level. Other variables' missing values were excluded in analysis

(6) We only pulled the data from prenatal screening population to generate the cohort, which does not represent the entire birth population in Ontario. Around 30%-35% of the birth population does not receive prenatal screening

(7) Data source: BORN Ontario, January 1 2013 to March 31 2021

Table 3 Associations between maternal race, maternal socioeconomic status, infant macrosomia, and shoulder dystocia: measurement of additive interaction between race and macrosomia on shoulder dystocia risk

Variables	Crude OR (95% CI)	Adjusted OR (95% CI)
Maternal race and infant macrosomia		
Black pregnant individuals having a macrosomia infant	6.85 (6.25–7.51)	6.19 (5.61–6.83)
Black pregnant individuals having a non-macrosomia infant	1.03 (0.97–1.09)	1.01 (0.94–1.07)
White pregnant individual having a macrosomia infant	5.19 (5.02–5.37)	4.8 (4.64–4.97)
White pregnant individual having a non-macrosomia infant	Reference	Reference
Neighbourhood Median household income after tax		
Quintile 1 (lowest)	1.24 (1.18–1.3)	1.11 (1.04–1.17)
Quintile 2	1.17 (1.11–1.23)	1.04 (0.99–1.1)
Quintile 3	1.15 (1.09–1.21)	1.02 (0.97–1.08)
Quintile 4	1.08 (1.03–1.13)	1.02 (0.97–1.08)
Quintile 5 (highest)	Reference	Reference
Neighbourhood education level		
Quintile 1 (lowest)	1.49 (1.42–1.56)	1.3 (1.22–1.38)
Quintile 2	1.34 (1.27–1.4)	1.2 (1.13–1.27)
Quintile 3	1.21 (1.16–1.27)	1.11 (1.05–1.17)
Quintile 4	1.12 (1.06–1.17)	1.06 (1.01–1.12)
Quintile 5 (highest)	Reference	Reference

(1) Neighbourhood education level was defined as the percentage of people aged 25–64 who had a university degree or higher at a dissemination area

(2) Neighbourhood income was defined as median household income after tax at a dissemination area

(3) Neighbourhood education level and income were categorized as quintiles. The lowest value is Q1 and highest value is Q5

(4) Macrosomia status and maternal race were combined into one variable to evaluate the addictive interaction effect of macrosomia status and maternal race on shoulder dystocia two SES indicators and shoulder dystocia while adjusting for maternal age, parity, previous caesarean delivery, substance use, pre-existing physical and mental health conditions, gestational diabetes, obstetrician in antenatal care team, hospital level of maternal care, maternal rural residence, gestational age at 37 weeks and infant sex

(5) We categorized "Unknown" values of obesity as a separate level. Other variables' missing values were excluded in analysis

consistent with published reports [3–5]. SD incidence in the general birthing population has been reported to range from 0.15% to 4.4%, while among different categories of births with fetal macrosomia, the incidence ranges from 5% to 23% [3–6]. One study conducted in Alberta, Canada reported an SD incidence of 4.4%, including singleton and twin births, and 4.5% for singletons, which is very close to the rate observed in our Ontario-based study [4]. Consistent with published studies, macrosomia is a notable factor influencing the risk of SD [6, 33].

Our finding regarding racial difference in the incidence of SD among Black individuals and White individuals is consistent with published studies in the USA [10, 11]. One study which included 19,236 pregnant people who experienced labour and delivery and were recruited

from five hospitals in the USA between 2010 and 2013, found that the Black birthing individuals were 1.5 times (OR: 1.5, 95% CI: 1.21–1.87) more likely to experience SD compared to the White birthing individuals [11]. Another USA study of 29,612 birthing individuals who had a singleton, vertex, vaginal birth found that the Black birthing individuals had 2.06 times the likelihood of experiencing SD (aOR: 2.06, 1.49–2.88) compared to White birthing individuals, after adjusting for macrosomia and other confounders [10].

Unlike studies conducted in the USA, we found a racial difference in the rate of SD only within the cohort of individuals giving birth to macrosomic infants. One possible explanation is that macrosomia is a dominant contributor to SD in the general birth population, while the proportion of the Black population in Ontario, Canada, is significantly lower than that of the birthing population in the USA [26, 27] As a result, the racial effect was masked when we studied it in the Ontario population, including the non-macrosomia group. This potential mechanism is supported by our analysis of the additive interaction effect of race and macrosomia on SD, where we combined these two factors into a single variable to assess their cumulative impact on SD in a multivariable regression model. The results showed that, compared to White individuals with non-macrosomic infants, Black individuals with macrosomic infants had 6.3 times the odds of experiencing SD, while Black pregnant individuals with non-macrosomic infants had no increased risk of SD. In addition, White individuals with macrosomic infants had 4.8 times higher likelihood of experiencing SD.

In addition to race, we observed differences in the incidence of SD based on SES. This finding is consistent with our previously published studies on other maternal, perinatal, and neonatal outcomes, as well as similar research on SD outcomes conducted in the USA [10, 11, 13, 14]. One USA study found that birthing individuals without a college education had a higher SD rate (2.3%) compared to birthing individuals with some college education (1.4%) [10]. Another large retrospective study from the USA with 9,913,838 records found that people living in wealthier neighbourhoods had a 7% lower likelihood of experiencing SD (adjusted OR: 0.93, 95% CI:0.92–0.94) compared to those living in the lowest-income neighborhoods [3].

Clinical and research implications

Several mechanisms may explain these findings. First, people living in lower SES neighborhoods often have less access to healthcare and face financial barriers to transportation, which can result in delayed or inadequate maternal health care and pregnancy monitoring. This can lead to suboptimal care and an increased risk of SD [14, 15, 30, 31]. Additionally, pregnant people living in poorer and less-educated neighbourhoods with greater deprivation and lower levels of education may have inadequate health literacy, resulting in less awareness about prenatal care and managing obesity, GDM and other conditions [14, 15, 30, 31]. Moreover, limited access to adequate nutritious, physical activities, and safe recreational spaces can contribute to unhealthy lifestyles, which are risk factors for obesity during pregnancy. Obesity, in turn, may increase the risk of macrosomia, further elevating the risk of SD [30, 34].

In the analysis to explore the complex relationships between maternal SES, race, and the risk of SD, we found that compared to White pregnant individuals, Black pregnant individuals had a higher incidence of SD in all education and income quintiles. In addition, we discovered a higher proportion of Black individuals resided in the most materially deprived neighborhoods where they experienced a higher rate of SD across all five education quintile neighborhoods (Table 1, Appendix Fig. 1). In the multivariable regression models, we concluded that living in lower SES neighborhoods was associated with a higher risk of SD, and Black individuals faced a higher risk of SD compared to White individuals, even when residing in neighborhoods with comparable SES.

These findings suggest that the elevated risk of SD among Black individuals may be due to multiple factors. First, the higher proportion of Black individuals in more deprived neighborhoods, implies that more Black people face barriers to accessing adequate maternal healthcare in Ontario. Second, even when Black and White individuals lived in neighborhoods with similar SES, Black individuals in the macrosomia cohort exhibited a higher risk of SD, indicating that factors beyond SES and that related to other social determinants of health are contributing to the disparity [31]. Third, although both Black and White individuals in higher SES neighborhoods likely have better access to healthcare and resources, Black individuals may experience systemic discrimination within healthcare settings, further contributing to their increased risk of SD. Black individuals may face additional stressors related to racial discrimination and implicit bias in medical care which can exacerbate obesity and, in turn, increase the risk of SD, independent of SES [13].

Future research should integrate quantitative and qualitative methods to evaluate the hypothesis and underlying mechanisms thoroughly and identify effective strategies for improvement. There is a need for policies and interventions that specifically address these inequities among Black communities to improve maternal and childbirth care in Ontario.

Like other provinces and territories in Canada, Ontario operates a universal, publicly funded healthcare system that provides residents with free access to maternal and childbirth services, including prenatal care, labour

and delivery, postpartum care, and midwifery services [35]. Given the similarities in healthcare delivery models and the comparable backgrounds of Black communities across Canadian jurisdictions, the findings from Ontario may be generalizable to other provinces and territories. However, caution is warranted when considering the applicability of these findings to other countries with universal healthcare systems, such as the United Kingdom's National Health Service or Australia's Medicare. Differences in healthcare infrastructure, demographic compositions, and broader social, political, and economic contexts may influence maternal and perinatal health outcomes. Therefore, conducting similar studies in these international settings would be valuable to explore both similarities and variations.

Strengths and limitations

This study has several notable strengths. It is the first study to examine the relationship between shoulder dystocia, race, and SES in Canada. Globally, to our knowledge, no published studies have examined the additive interaction effects of race on the risk of shoulder dystocia associated with macrosomia. We are also the first to examine the effect of racial variation on SD and potential racial discrimination or systemic bias using an intersectional analysis method, while also considering neighbourhood-level household median income and educational attainment. Furthermore, we used the Ontario pregnancy and birth registry data, which includes a large population, to examine the rare outcome of SD at the population level [13]. As a result, our study has sufficient statistical power, enhancing the generalizability of our findings. Despite the above strengths, there are also several limitations to consider. SES indicators, median household income and education level, were captured at the neighbourhood level. SES at the individual level may differ from the neighbourhood level [30]. Additionally, the study cohort was derived from the prenatal screening population, which represents about 70% of the entire birthing population in Ontario. The social demographic distribution may be different between prenatal screening population and the general birth population in Ontario [13]. Prenatal screening uptake tends to be higher among pregnant individuals residing in urban areas and higher-income neighborhoods [13]. As a result, our study population may reflect a more socioeconomically advantaged group compared to the general population [13]. In addition, potential residual confounding factors including quality of prenatal care and abnormal pelvic structure, may also potentially impact the observed associations [6, 13]. Finally, we were not able to consider newcomer status, religion and other social demographic factors among the Black and White pregnant people due to data limitations [13]. There is a potential for misclassification of race due to self-reported race and non-differential misclassification of SD using birth registry and health administrative databases [13].

Conclusions

Pregnant individuals in lower income and/or lower education quintiles and those who were Black had a higher incidence of SD. Notably, among the macrosomia subcohort, Black individuals experienced significantly higher SD incidence across all SES quintiles. The interactional analysis indicated that Black pregnant individuals faced elevated SD rates across all education quintiles in the lowest income neighborhoods. Additionally, more Black individuals lived in the materially deprived neighbourhoods, where they also experienced disproportionately higher SD rates across all education quintiles. This suggests a compounded burden, as both lower SES and racial disparities contribute to the increased risk of SD among Black individuals.

Abbreviations

SD	Shoulder dystocia
SES	Socioeconomic status
DA	Dissemination area
OR	Odds ratio
aOR	adjusted odds ratio
RR	Relative risk
aRR	adjusted relative risk
CIHI	Canadian Institute for Health Information
DAD	Discharge Abstract Database
BORN	Better Outcomes Registry & Network
BIS	BORN information system
BMI	Body mass index

Supplementary Information

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Supplementary Material 1. Fig S1. SD rate by education quintiles and maternal race among women having infants with macrosomia and living in the lowest-income neighbourhood, BORN Ontario, January 1, 2013 to March 31, 2021. Table S1. Race distribution by livebirth or stillbirth. Table S2. Incidence of SD by livebirth or stillbirth and race.

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Disclaimer

Parts of this material are based on data and information compiled and provided by CIHI. However, the analyses, conclusions, opinions, and statements expressed herein are those of the author(s), and not necessarily CIHI.

Authors' contributions

QM: project development, data management and analysis, manuscript writing and editing. YG: project development, code review, results interpretation, manuscript review and revision. SW, CM, AH, ADH, TK and MW: project development, results interpretation, manuscript review and revision. All authors read and approved the final manuscript.

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Data availability
The data analyzed for this study are held securely at the prescribed registry, BORN Ontario. Data sharing regulations prevent these data from being made available publicly due to the personal health information in the datasets. Enquiries regarding BORN data must be directed to BORN Ontario (Science@BORNOntario.ca).

Declarations

Ethics approval and consent to participate
Our study was approved by the Research Ethics Board (REB) from the Children's Hospital of Eastern Ontario and the Ottawa Health Science Network (File number: 20180078). Informed consent to participate has been waived by the Children's Hospital of Eastern Ontario ethical committee. All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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