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Overweight/obesity and other predictors of gestational diabetes among Aboriginal and non-Aboriginal women in Western Australia

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ABSTRACT

This population-based study investigated the association of BMI and other predictors with gestational diabetes mellitus (GDM) among Australian Aboriginal and non-Aboriginal mothers. We conducted a state-wide retrospective cohort study that included all singleton births in Western Australia (n = 134,552) between 2012 and 2015 using population health datasets linked by the Western Australian Data Linkage Branch. Associations between GDM and its predictors were estimated as adjusted relative risks (aRRs) from multivariable generalised linear models. Adjusted ratio of relative risks (aRRRs) compared RRs in Aboriginal and non-Aboriginal mothers. Adjusted population attributable fractions estimated the contribution of overweight/obesity to GDM burden, and adjusted predicted probabilities for GDM were plotted against BMI levels. The following predictors had stronger associations with GDM in Aboriginal, compared to non-Aboriginal, mothers: maternal obesity (aRR [95% CI] 3.16 [2.54–3.93]; aRRR 1.57 [1.26–1.94]), previous LGA (aRR 1.70 [1.37–2.12]; aRRR 1.41 [1.13–1.76]) and previous macrosomia (birthweight ≥ 4 kg) (aRR 1.55 [1.24–1.94]; aRRR 1.53 [1.22–1.91]). 46.1% (95% CI: 36.6–54.1) of GDM cases in Aboriginal women (23.3% in non-Aboriginal mothers, 95% CI: 21.6–25.1) were attributed to overweight/obesity. Compared to non-Aboriginal mothers, adjusted GDM probabilities were higher at all BMI levels and showed greater increase with BMI. Overweight/obesity is a key driver of GDM among Aboriginal women. Association between BMI and GDM is stronger in Aboriginal, compared to non-Aboriginal, women especially at higher BMI.

1. Introduction

Gestational diabetes mellitus (GDM) is the most common pregnancy complication, and its increasing trend is becoming a global concern (International Diabetes Federation, 2019). GDM has many negative implications for the neonate. It increases the risk of fetal overgrowth, respiratory distress syndrome, neonatal hypoglycaemia, preterm birth, hyperbilirubinemia and polyhydramnios (Denney and Quinn, 2018). GDM is also associated with many maternal complications including pre-eclampsia, gestational hypertension, caesarean section, shoulder dystocia and vaginal laceration (Denney and Quinn, 2018). Moreover, GDM has negative long-term implications for both the offspring and mother. It increases the risk of diabetes in the offspring (Dabelea et al., 2008), and the future risk of type 2 diabetes mellitus (T2DM) in the mother by nearly ten-fold (Vounzoulaki et al., 2020).

Globally, the burden of GDM is higher among Indigenous

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populations (Voaklander et al., 2020). In Australia, GDM prevalence is consistently higher among Aboriginal women (Australian Institute of Health and Welfare, 2010; Hare et al., 2020). There has been a greater increase in the burden of GDM in the Aboriginal population over time (Hare et al., 2020), and Aboriginal mothers whose pregnancies are complicated by GDM have more than four-fold greater risk of post-partum T2DM, when compared to their non-Aboriginal counterparts (Chamberlain et al., 2016). Moreover, Aboriginal babies born to mothers with GDM have more adverse neonatal outcomes than non-Aboriginal babies (Duong et al., 2015).

The Australian Aboriginal population shows different magnitude and extent of cardiometabolic disease and its risk profile. The rapid shift from traditional to modern lifestyle and health behavioural factors originating from social disadvantage are believed to be the main drivers of the heavier burden and distinct pattern of metabolic disease in the Aboriginal population (O'Dea, 1992; Daniel et al., 2011). For example, Aboriginal Australians have: different, generally higher, prevalence of cardiometabolic health risk factors (Australian Institute of Health and Welfare, 2020); different age- and sex-specific prevalence of diabetes (Minges et al., 2011); heavier burden of cardiometabolic disease at considerably younger ages (Maple-Brown, 2011); and differential strength of association between cardiometabolic disease and certain predictors (Maple-Brown et al., 2013).

The prevalence of obesity, which is a well-established risk factor for GDM (Paulo et al., 2021), is considerably higher among the Aboriginal population and its burden is continuously growing (Hutchins and Nev-erauskas, 2014). Evidence has shown that race/ethnicity modifies the effect of obesity on GDM (Kim et al., 2012). The population attributable fraction (PAF) of overweight/obesity for GDM also differs by ethnicity (Makgoba et al., 2012). However, the relationship between GDM and overweight/obesity has not been sufficiently studied within an Aboriginal context.

A more nuanced understanding of factors contributing to GDM risk, using population-based datasets, can offer opportunities to identify targets and guide prevention efforts for GDM. The aim of the present study was to explore the predictors of GDM; estimate PAFs associated with its most well-established risk factor, overweight/obesity; and to investigate the relationship between body mass index (BMI) and GDM among the Aboriginal and non-Aboriginal women in Western Australia using state-wide data.

2. Methods

2.1. Study design and population

This is a retrospective cohort study that initially included all women who gave birth at ≥ 20 weeks' gestation in the state of Western Australia between 2012 and 2015 (n = 138,382). Multiple pregnancies (n = 3,830), women with pre-existing diabetes (n = 1,337) or those with missing GDM status (n = 1,584) were excluded from the final study cohort (n = 131,631) (Fig. 1).

2.2. Data sources

The study used population health datasets linked by the Western Australian Data Linkage System. This system uses probabilistic matching (Holman et al., 1999) to link data from different sources, and the researchers receive data with no identifying fields. The datasets used included the Midwives Notification System (MNS), Hospital Morbidity Data Collection (HMDC), Western Australian Register of Developmental Anomalies (WARDA) and WA Registry of Births, Deaths and Marriages. The MNS and HMDC were our primary sources of data. The MNS records neonatal and maternal details of all births at 20 weeks or more, and HMDC provides information on infants' and mothers' diagnoses and

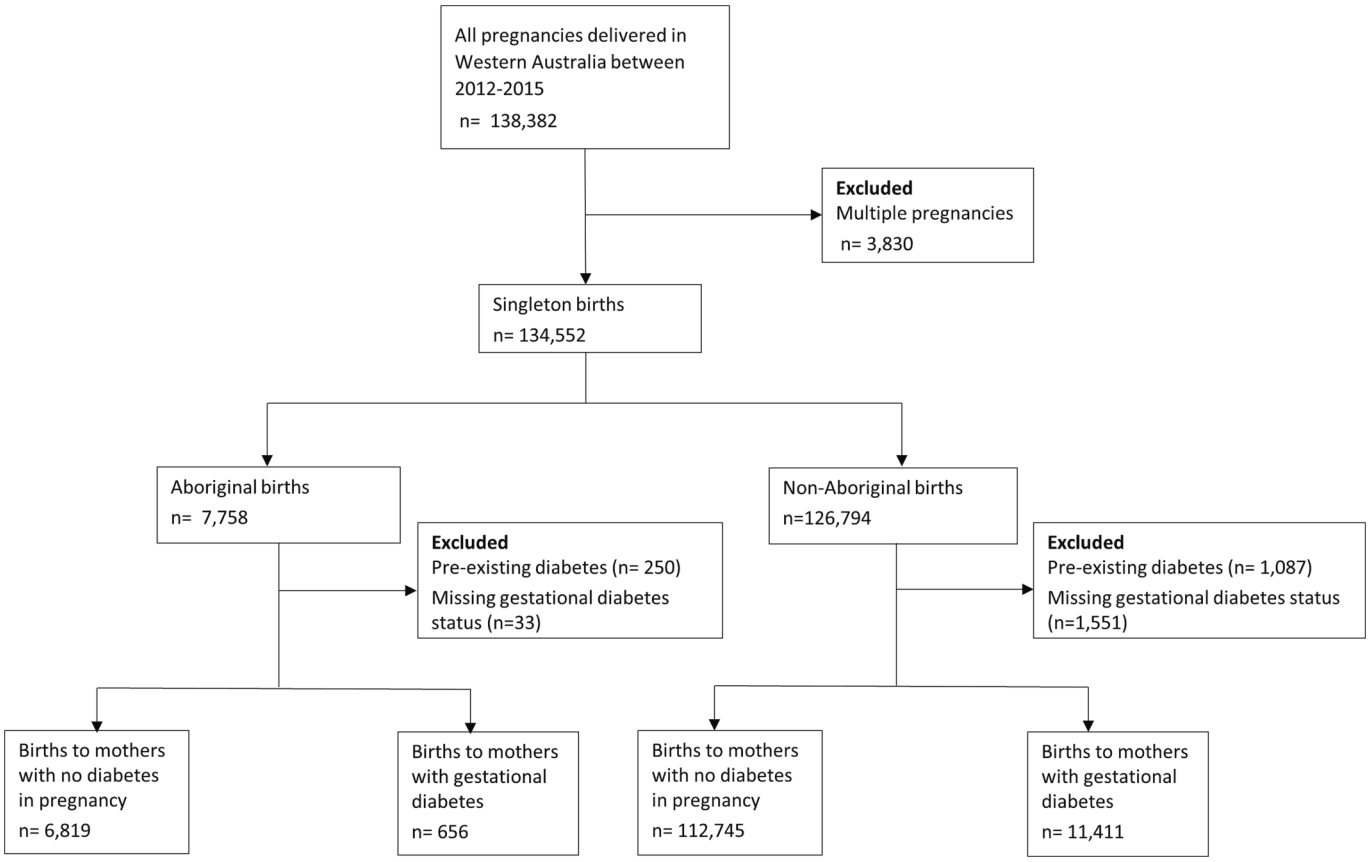

Fig. 1. Flow chart showing the study population.

procedures during hospitalisation.

2.3. Measurements

2.3.1. Aboriginal identification and gestational diabetes

To identify Aboriginal women, we adopted the algorithm described by the 'Getting Our Story Right' project (Christensen et al., 2014). This project uses a multi-stage median method to generate a single consistent status of Aboriginality for each individual in the linked administrative datasets. The term 'Aboriginal' is used on the basis that about 96% of the Aboriginal and Torres Strait Islander population in Western Australia identify solely as Aboriginal people."

We ascertained GDM status from the MNS (under pregnancy complications) and HMDC. In the HMDC, GDM was captured from the relevant codes (O24.4 and O24.9) of International Classification of Disease version 10 (Australian Modifications) (ICD-10-AM).

2.3.2. Predictors of GDM

We defined overweight as BMI $\geq$ 25 and below 30, and obesity as BMI $\geq$ 30. The remoteness of the mother's residence was determined using the Australian Statistical Geography Standard classification, which divides areas into five broad categories (major cities, inner regional, outer regional, remote and very remote) based on relative access to service (Australian Bureau of Statistics, 2016). We collapsed the categories to remote/very remote (yes/no) in the regression analysis. Socioeconomic status (SES), categorised into tertiles, was determined based on mother's usual area of residence at the time of birth using the census closest to the birth, measured by the Index of Relative Socioeconomic Disadvantage (an aggregate, area-based measure of SES) (Pink, 2013). The index uses certain attributes (income, education, unemployment, housing and occupational skill) to rank areas (average of 400 persons in Statistical Areas Level 1) by the level of socioeconomic disadvantage. Parity was categorised into 0, 1, 2 and 3 or more. Maternal obstetric history variables included previous events: stillbirth, large for gestational age (LGA), macrosomia and congenital anomalies. Stillbirth was defined as birth of a baby with no signs of life at $\geq$20 weeks' gestation or weighing $\geq$400 g where gestation is unknown. LGA was defined as birthweight >90th percentile using sex- and gestational age-specific percentiles of birthweight in Australia (Dobbins et al., 2012). Macrosomia was defined as birthweight $\geq$4000*g* and congenital anomalies were ascertained from WARDA (WA Register of Developmental Anomalies (WARDA), 2021).

2.3.3. Missing data

About 6.9% of BMI and 2.4% of SES values were missing (Table S1). The proportion of missing BMI values was higher in Aboriginal mothers (14.9%) compared to non-Aboriginal mothers (6.4%). Missingness for other variables ranged from 0 to 1.3%.

2.4. Ethics approval

Ethics approvals were obtained from the Western Australian Aboriginal Health Ethics Committee (Project 797), the University of Western Australia Human Research Ethics Committee (RA/4/20/6161) and the Western Australian Department of Health Human Research Ethics Committee (RGS0000003168).

2.5. Statistical analysis

Characteristics of pregnancies complicated by GDM in Aboriginal and non-Aboriginal mothers were summarised using descriptive statistics and compared using Pearson's chi-squared test (for binary and categorical variables) and Wilcoxon–Mann–Whitney test (for continuous variables).

Exploring GDM predictors and assessing their importance in the Aboriginal, relative to non-Aboriginal, mothers were performed by entering the predictors (chosen on clinical and biological plausibility grounds) into univariable and multivariable generalised linear models (GLMs) to estimate the relative risks (RRs) and 95% confidence intervals (CI). For all GLMs, we used log as the link function, Poisson as the family of distribution of GDM and cluster-robust variance estimation to account for the clustering effect resulting from multiple birth events from the same mother in our longitudinal cohort. Multicollinearity between predictors was assessed by variance inflation factor. As both LGA and macrosomia are defined by fetal weight, their RRs were estimated from separate multivariable models. RRs for maternal obstetric history predictors were estimated from models restricted to parous women. To compare the relative strength of GDM predictors in Aboriginal vs non-Aboriginal mothers, we incorporated an interaction term between Aboriginal status and each predictor (in separate models). The interaction term represented the ratio of RRs (RRR) for the predictor in the Aboriginal relative to non-Aboriginal mothers.

The PAF for the effects of overweight/obesity (in relation to healthy BMI) on GDM was estimated as a percentage. Since PAF has a causal interpretation, it was estimated from GLMs adjusted for potential confounders. Thus, the models, stratified by Aboriginal status, included GDM as the outcome variable, and BMI, maternal age (continuous), smoking (binary), remoteness, SES, and parity as the independent variables. To calculate the adjusted PAFs and their 95% CI, we used Stata post-regression command '*punaf*' (Newson, 2013), which utilises methods described by Greenland and Drescher (1993). *punaf* compares a counterfactual scenario in which the exposure of interest is set to zero (maternal BMI < 25) with the real-world scenario.

To describe the relationship between BMI and the risk of GDM in the Aboriginal and non-Aboriginal populations, we fitted GLMs that included potential confounders (included in the PAF models above). The assumption of the linear relationship between BMI and GDM within GLMs was tested by Box–Tidwell transformation (which identified the best power of BMI for model fit), using Stata module '*boxtid*' (Royston, 2013). The Stata Post-estimation commands '*margins*' and '*marginsplot*' estimated and plotted the adjusted probability and 95% CI of GDM along the BMI continuum. An interaction term between Aboriginal status and BMI (in continuous form) assessed whether the BMI-GDM relationship significantly differed between the two populations.

All analyses were conducted using Stata version 15.1 (StataCorp 2017).

2.6. Sensitivity analysis

As the proportion of missing values for BMI, the primary predictor of interest, was relatively high and varied by Aboriginal status, we conducted sensitivity analyses to assess whether the exclusion of mothers with missing BMI may have biased the obtained estimates. Two extreme-case scenarios were used as tools of bias analysis to check whether the PAF estimates from complete case analyses have been impacted by the missingness. Aboriginal mothers with missing BMI values were considered as having healthy BMI and non-Aboriginal mothers with missing BMI as overweight/obese in the first scenario, and values were reversed in the second scenario. We also added a missing data category to the BMI category variable, entered the new variable in the GLMs and used the resultant RRs and RRRs to assess the impact of missing BMI data.

2.7. Aboriginal involvement

The study design and interpretation of findings were guided by the Kaadaninny Aboriginal Advisory Committee and Ngangk Yira Council of Elders, with broader Cultural Governance provided by the Ngangk Yira Institute for Change. These groups will continue to be involved in future communications, dissemination and translational work.

3. Results

There were 134,552 singleton pregnancies in Western Australia between 2012 and 2015, of which 7,758 (5.8%) were Aboriginal (Fig. 1). About 656 (8.5)% of singleton births to Aboriginal mothers were complicated by GDM compared to 11,411 (9.0%) of their non-Aboriginal counterparts.

Compared to non-Aboriginal women, Aboriginal mothers with GDM had significantly higher median BMI, and were much more likely to be obese and less likely to be in the healthy BMI category (Table 1). Aboriginal mothers were also more likely to: be younger, have higher parity, have smoked during pregnancy and to live in remote/very remote areas. The likelihood of gestational hypertension, eclampsia/pre-eclampsia and adverse obstetric history events (previous: LGA, macrosomia, stillbirth and birth defects) were higher in Aboriginal mothers than in non-Aboriginal mothers.

Table 1
Characteristics of singleton births complicated by gestational diabetes in Aboriginal and non-Aboriginal mothers in Western Australia, 2012–2015.

Characteristics		Aboriginal mothers (%) (n = 656)	Non-Aboriginal mothers (%) (n = 11,411)	p-value[1]
BMI category	below 25	121 (20.5)	4298 (39.7)	<0.001
	>=25 & <30	148 (25.0)	3197 (29.6)	
	>=30	322 (54.5)	3321 (30.7)	
	Missing values	65 (9.9)	595 (5.2)	
BMI, median (IQR)		30.8 (25.7, 35.9)	26.6 (23.0, 31.2)	<0.001
Maternal age group, years	25 or below	260 (39.6)	1206 (10.6)	<0.001
	>25 to 35	296 (45.1)	7389 (64.8)	
	above 35	100 (15.2)	2816 (24.7)	
Maternal age (years), median (IQR)		28.0 (23.0, 32.5)	32.0 (28.0, 35.0)	<0.001
Parity group	0	175 (26.7)	5024 (44.3)	<0.001
	1	143 (21.8)	3757 (33.1)	
	2	127 (19.4)	1587 (14.0)	
	3 plus	210 (32.1)	967 (8.5)	
Smoking during pregnancy		276 (42.2)	762 (6.8)	<0.001
SES tertile	1st (most disadvantaged)	445 (70.6)	4006 (36.0)	<0.001
	2nd	139 (22.1)	3818 (34.3)	
	3rd (least disadvantaged)	46 (7.3)	3300 (29.7)	
Remoteness	Major cities	267 (40.8)	9576 (85.0)	<0.001
	Inner regional	31 (4.7)	689 (6.1)	
	Outer regional	111 (17.0)	589 (5.2)	
	Remote	151 (23.1)	328 (2.9)	
	Very remote	94 (14.4)	86 (0.8)	
Remote/very remote		245 (37.5)	414 (3.7)	<0.001
Mother not married or defacto		337 (51.5)	1309 (11.5)	<0.001
Female sex of baby		298 (45.4)	5509 (48.3)	0.160
Existing hypertension		18 (2.8)	236 (2.1)	0.260
Caesarean delivery		257 (39.3)	4836 (42.9)	0.070
Gestational hypertension		51 (7.8)	567 (5.0)	0.002
Preeclampsia or eclampsia		38 (5.8)	475 (4.2)	0.050
Previous LGA		101 (15.4)	852 (7.5)	<0.001
Previous LGA restricting to parity > 0		101 (21.0)	852 (13.3)	<0.001
Previous macrosomia		94 (14.3)	816 (7.2)	<0.001
Previous macrosomia restricting to parity > 0		94 (19.5)	816 (12.8)	<0.001
Previous stillbirth		24 (3.7)	126 (1.1)	<0.001
Previous stillbirth restricting to parity > 0		24 (5.0)	126 (2.0)	<0.001
Previous congenital anomalies		51 (7.8)	470 (4.1)	<0.001
Previous congenital anomalies restricting to parity > 0		51 (10.6)	470 (7.4)	0.010

BMI, body mass index; IQR, interquartile range; LGA, large for gestational age; SES, socioeconomic status.

[1] Pearson's chi-square test p-values for categorical variable and Wilcoxon–Mann–Whitney test p-values for continuous variable.

In multivariable models (Table 2), obesity was associated with a higher risk of GDM among Aboriginal mothers (RR: 3.16, 95% CI: 2.54–3.93) relative to their non-Aboriginal counterparts (RRR: 1.57, 95% CI: 1.26–1.94). For overweight, RR was 1.71 (95% CI: 1.34–2.19), and RRR was 1.26 (95% CI: 0.98–1.60). Previous LGA (RR: 1.70, 95% CI: 1.37–2.12; RRR: 1.41, 95% CI: 1.13–1.76) and previous macrosomia (RR: 1.55, 95% CI: 1.24–1.94; RRR: 1.53, 95% CI: 1.22–1.91) also exerted greater effects in Aboriginal mothers.

The PAF of overweight/obesity for GDM differed by Aboriginal status. The percentage of GDM attributable to overweight/obesity in the Aboriginal population was 46.1% (95% CI: 36.6–54.1) compared to 23.3% (95% CI: 21.6–25.1) in their non-Aboriginal counterparts (Fig. 2).

Adjusted estimates of the probability of GDM by BMI (as a linear term) and Aboriginal status are presented in Fig. 3. The figure reveals an increased risk of GDM among Aboriginal, relative to non-Aboriginal, women at all BMI points. The gap between the two populations, as represented by the interaction term of Aboriginal status and BMI in the regression model, increases with BMI (p value < 0.001).

The results of the sensitivity analysis suggest that the impact of missing values of BMI on the reported relationship between BMI and GD is unlikely to differ in the Aboriginal and non-Aboriginal mothers. In both extreme case analyses, the PAFs of overweight/obesity for GDM was substantially higher in the Aboriginal, compared to non-Aboriginal, mothers (Table S2). When the BMI was used as a categorical variable with a missing values category in the GLMs, the resultant RRs did not differ between the Aboriginal and non-Aboriginal mothers (Table S3), suggesting that BMI missingness is unlikely to have affected the RRRs from complete case analyses.

4. Discussion

Our findings reported a substantial contribution of overweight/obesity to the burden of GDM differentially among the Aboriginal mothers, and an interaction between Aboriginal status and the diabetogenic effect of overweight/obesity. The PAF estimates suggest that if all pregnant women had a healthy BMI, 46.1% of Aboriginal (and 23.3% of non-Aboriginal) GDM cases could have been prevented. A stronger association between BMI and GDM risk among Aboriginal women was evident at both categorical (as indicated by the RRR) and continuous scales. The RRR, which is the product of dividing the RR of Aboriginal mothers by RR of non-Aboriginal mothers, indicates that obesity poses greater risk for GDM in Aboriginal mothers compared to their non-Aboriginal counterparts.

The higher adjusted PAF of GDM associated with overweight/obesity among Aboriginal women reflects a higher prevalence of overweight/obesity and a stronger association between BMI and GDM risk among this disadvantaged population.

4.1. The heavy burden of overweight/obesity among Aboriginal mothers

Prior to contact with Europeans, Aboriginal Australians led a traditional, healthy lifestyle and were generally lean and physically fit (O'Dea, 1992). The lifestyle of Aboriginal Australians has undergone a rapid transition towards Western lifestyle over a relatively short period of time, and overweight/obesity has become increasingly common (McDermott et al., 2000). Compared with the non-Aboriginal population, Aboriginal Australians have 1.2 times higher risk of BMI above 25 and 1.5 times higher risk of being obese (Australian Institute of Health and Welfare, 2020). This higher risk of obesity is particularly true in Aboriginal women; who are 1.7 times more likely to be obese while Aboriginal men have 1.4 times higher risk, relative to non-Aboriginal women and men, respectively (Hutchins and Neverauskas, 2014). The underlying pathways to such sex-specific differences, which likely contribute to the higher prevalence of T2DM in Aboriginal women (Minges et al., 2011), probably involve limited physical activity among other factors (McDermott et al., 2000). The drivers of the high

Table 2
Predictors of gestational diabetes by Aboriginal status in Western Australia, 2012–2015.

Predictors		Aboriginal pregnancies		Non-Aboriginal pregnancies		Adjusted[1] RRR
		Unadjusted RR (95% CI)	Adjusted[1] RR (95% CI)	Unadjusted RR (95% CI)	Adjusted[1] RR (95% CI)	
BMI category	**below 25**	Reference	Reference	Reference	Reference	
	>=25 & <30	1.97 (1.55, 2.50)	1.71 (1.34, 2.19)	1.36 (1.30, 1.42)	1.36 (1.30, 1.42)	1.26 (0.98, 1.60)
	>=30	3.99 (3.24, 4.92)	3.16 (2.54, 3.93)	1.99 (1.91, 2.08)	2.03 (1.94, 2.12)	1.57 (1.26, 1.94)
Maternal age group	**25 or below**	Reference	Reference	Reference	Reference	
	>25 to 35	1.84 (1.55, 2.17)	1.79 (1.46, 2.19)	1.85 (1.74, 1.96)	2.02 (1.90, 2.15)	0.97 (0.80, 1.17)
	above 35	3.80 (3.05, 4.74)	3.30 (2.53, 4.31)	2.77 (2.59, 2.96)	3.10 (2.89, 3.33)	1.23 (0.96, 1.56)
Parity group	**0**	Reference	Reference	Reference	Reference	
	1	1.00 (0.81, 1.22)	0.76 (0.61, 0.95)	0.93 (0.90, 0.97)	0.80 (0.77, 0.83)	1.01 (0.81, 1.26)
	2	1.21 (0.97, 1.50)	0.75 (0.59, 0.96)	0.96 (0.91, 1.01)	0.74 (0.70, 0.78)	1.10 (0.87, 1.40)
	3 plus	1.48 (1.22, 1.80)	0.71 (0.55, 0.91)	1.18 (1.10, 1.26)	0.77 (0.71, 0.83)	1.06 (0.85, 1.31)
Smoking during pregnancy		0.83 (0.71, 0.97)	0.93 (0.78, 1.09)	0.79 (0.74, 0.85)	0.83 (0.77, 0.90)	1.05 (0.88, 1.26)
SES tertiles	**1 (most disadvantaged)**	Reference	Reference	Reference	Reference	
	2	1.10 (0.91, 1.33)	1.01 (0.84, 1.22)	0.89 (0.85, 0.93)	0.85 (0.81, 0.89)	1.17 (0.96, 1.42)
	3 (least disadvantaged)	1.04 (0.77, 1.40)	0.90 (0.67, 1.21)	0.82 (0.79, 0.86)	0.77 (0.73, 0.81)	1.18 (0.88, 1.58)
Remoteness		0.97 (0.83, 1.14)	1.00 (0.84, 1.18)	0.77 (0.69, 0.85)	0.81 (0.73, 0.89)	1.20 (0.99, 1.46)
Previous LGA[2]		2.26 (1.84, 2.79)	1.70 (1.37, 2.12)	1.38 (1.28, 1.48)	1.25 (1.16, 1.34)	1.41 (1.13, 1.76)
Previous macrosomia[2]		2.06 (1.66, 2.56)	1.55 (1.24, 1.94)	1.16 (1.08, 1.24)	1.06 (0.98, 1.14)	1.53 (1.22, 1.91)
Previous stillbirth[2]		1.66 (1.08, 2.55)	1.34 (0.87, 2.08)	1.42 (1.19, 1.68)	1.29 (1.07, 1.54)	1.16 (0.74, 1.83)
Previous congenital anomalies[2]		1.21 (0.91, 1.61)	1.07 (0.80, 1.42)	1.08 (0.98, 1.18)	0.99 (0.90, 1.09)	1.11 (0.83, 1.49)

BMI, body mass index; CI, confidence interval; LGA, large for gestational age; RR, relative risk; RRR, ratio of relative risks; SES, socioeconomic status.
[1] Adjusted RRs were estimated from generalised linear models that included all the predictors (except RRs for previous LGA and previous macrosomia which were estimated from separate models).
[2] RRs for maternal history predictors were estimated from models restricted to parous women.

prevalence of overweight/obesity are believed to be related to the material and social environment of Aboriginal families and the communities in which they live—for example, low income, financial stress, food insecurity and overcrowded housing (Aboriginal and Torres Strait Islander Health Performance Framework, 2017). It is also likely shaped by substantive cultural, historical and macrosocial factors, including issues of dispossession, exclusion, discrimination and marginalisation that have been a feature of post-colonial Aboriginal Australia and are generally accepted as having profoundly negative effects on Aboriginal wellbeing (Shepherd et al., 2012). In particular, racial discrimination has been shown to affect the wellbeing of a substantial proportion of Aboriginal people (Shepherd et al., 2017)—this includes the indirect effects of reduced and unequal access to the range of medical and health promotion resources that are required for good health (Carson et al., 2020). Further, the rapid shift from traditional (nutrient-rich low-energy) to Western (nutrient-poor high-energy) sedentary lifestyle is the proximal explanatory factor of the propensity of the Aboriginal population to obesity (O'Dea, 1992). The mechanism is hypothesised to involve genetic predisposition, although not proven (O'Dea, 1992).

4.2. *The strong association between BMI and GDM in Aboriginal women*

Aboriginal Australians show stronger association between BMI and T2DM, which has a relatively similar pathophysiology to GDM, and the cause is believed to be their different pattern of fat deposition. Aboriginal people tend to accumulate fat around the body midsections (Piers et al., 2003) and spare the distal extremities (O'Dea et al., 1993). Such pattern of fat distribution is more prominent among females (Adegbija

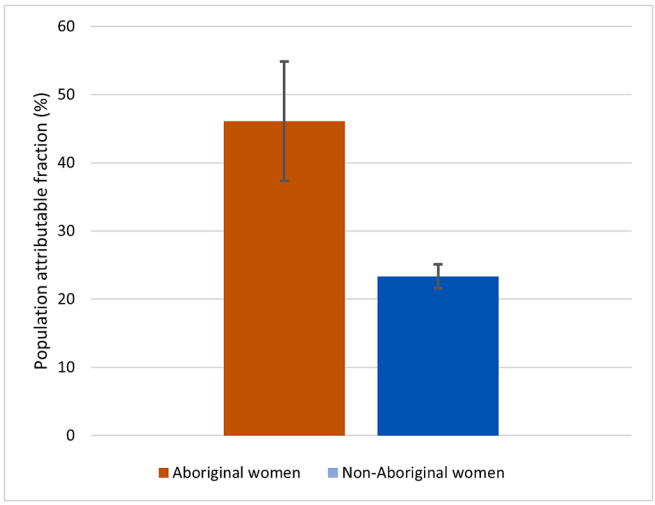

Fig. 2. Adjusted[1] population attributable fraction for gestational diabetes associated with overweight/obesity (BMI > 25) among the Aboriginal and non-Aboriginal populations in Western Australia, 2012–2015. BMI, body mass index. [1]Adjusted for maternal age (continuous), smoking (binary), socioeconomic status (tertiles), remoteness (binary: remote/very remote or not) and parity (categorised into 0, 1, 2 and 3 or more).

and Wang, 2014). Causes of preferential abdominal fat deposition among Aboriginal Australians are believed to be multifactorial, and contributions of lifestyle, genes, infection and inflammation are suggested. (Hoy et al., 2006) Aboriginal Australians were also reported to accumulate higher amounts of body fat when compared to their European counterparts who have similar BMI (Piers et al., 2003; Rutishauser and McKay, 1986) and the healthy BMI range for the Aboriginal population is thus believed to be lower than the conventional threshold (Norgan, 1994; O'Dea, 1996).

Central obesity is a well-established risk factor of insulin resistance, T2DM (Papaetis et al., 2015) and GDM (Zhu et al., 2019). Central obesity in Aboriginal people was associated with lower levels of adiponectin (Hughes et al., 2016), an adipocyte-derived hormone with cardiometabolic protective properties. There is a wealth of literature confirming the low adiponectin levels among individuals with T2DM and GDM (Li et al., 2009; Iliodromiti et al., 2016). The association between central obesity measures and GDM in the Aboriginal population has also been reported. Waist-to-height ratio was the best anthropometric index in predicting GDM among Aboriginal women in the Australian Northern Territory (Sina et al., 2015). Campbell et al. also found that waist circumference was a significant predictor of GDM among Aboriginal women in north Queensland (Campbell et al., 2012).

Janevic et al. hypothesised that a combination of being born low birth weight (which is linked to future GDM (Yeung et al., 2010) and obesogenic environment in adulthood might explain the stronger impact of obesity on the development of GDM in immigrant, compared to US-born, women (Janevic et al., 2018). This may be an additional explanatory factor for the stronger association between BMI and GDM among the Aboriginal Australians who suffer strikingly high rates of low births weight (Ford et al., 2018) and elevated prevalence of obesity (Australian Institute of Health and Welfare, 2017) and obesogenic behaviour (high energy low fibre diet and suboptimal physical activity (Australian Institute of Health and Welfare, 2020).

4.3. Previous evidence

To our knowledge, this is the first whole-population study to directly report on the significant interaction between Aboriginal status and BMI on GDM. Our findings corroborate the results of Thrift and Callaway who reported that 52% of GDM among Aboriginal mothers could be attributed to overweight/obesity, compared to 39% in their non-Aboriginal counterparts (Thrift and Callaway, 2014). In contrast to our study, they found that the RR of GDM increased with overweight/obesity categories with no significant differences between the two populations.

4.4. Implications and recommendations

The present study adds evidence on the substantial public health

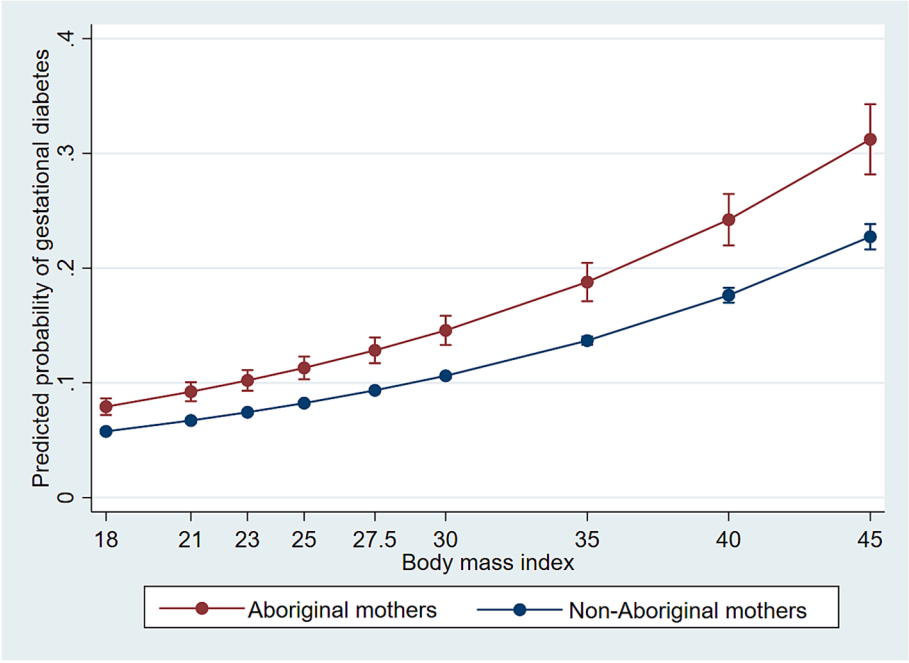

Fig. 3. Adjusted[1] probability of gestational diabetes by BMI and Aboriginal status in Western Australia, 2012–2015. BMI, body mass index. [1]Adjusted for maternal age (continuous), smoking (binary), socioeconomic status (tertiles), remoteness (binary: remote/very remote or not) and parity (categorised into 0, 1, 2 and 3 or more).

burden of overweight/obesity in the Aboriginal population. Given the evidence that BMI underestimates the extent of adiposity in the Aboriginal population, our results may have underestimated this burden on GDM. Our findings also suggest a likely sizable contribution of overweight/obesity to the heavy burden of T2DM in the Aboriginal population (since GDM and T2DM share similar pathophysiological features (Robitaille and Grant, 2008). Absence on whole-population data on T2DM and the considerably higher rates of progression from GDM to T2DM in Aboriginal mothers (Chamberlain et al., 2016) add an additional layer of importance to our work. An indirect, but important, implication of our findings is the role of maternal obesity in the intergenerational cycle of diabetes among the Aboriginal population. Exposure to intrauterine hyperglycemia increases the future risk of diabetes and obesity in the offspring (Shou et al., 2019). Such intergenerational impact of diabetes during pregnancy has been described in Indigenous populations in North America (Sellers et al., 2016; Dabelea et al., 2000).

This study suggests several implications. Preventing overweight/obesity remains a priority among the Aboriginal population, and can have short- and long-term public health benefits. Tackling the problem requires Aboriginal-specific initiatives that focus on improving nutrition and increasing physical activity, positioned within a broader holistic approach to address socioeconomic disadvantage (Burns and Thomson, 2006). Our findings point to the importance of screening for GDM, and counselling Aboriginal women about its risk even within conventionally healthy or slightly elevated BMI ranges.

4.5. Strengths and limitations

Utilising whole-population data produced by merging datasets from different sources is a strength of the present study. This removes selection bias, maximises representativeness and allows following pregnant women between facilities. Moreover, we used an Aboriginal status variable created through robust methods to provide accurate classification and minimise missingness.

The present study has several limitations. First, the study used data collected for administrative, not for research, purposes. Second, the data had relatively high levels of missing BMI values and the proportions missing varied by Aboriginal status. However, sensitivity analyses showed that the missingness is unlikely to have impacted the main findings. Third, we did not have data on gestational weight gain (GWG). Excessive GWG, which is linked to both pre-pregnancy obesity and GDM (McDowell et al., 2019), may partly explain the stronger relationship between BMI and GDM in Aboriginal mothers. Fourth, underdiagnosis of GDM reported among Aboriginal Western Australians (Kirke et al., 2019) could have underestimated the association between BMI and GDM. Fifth, maternal weight was recorded at the booking appointment, or taken as the self-reported weight if not recorded by 20 weeks gestation (Government of Western Australia Department of Health, 2021). Given the higher risk of late presentation to antenatal care in Aboriginal women, they are more likely to self-report their weights and thus to have their BMI underestimated (Kim et al., 2012). This may bias the estimates (PAF and Aboriginality-BMI interaction) towards the null.

5. Conclusions

Overweight/obesity is a key driver of GDM among the Aboriginal population. The risk of GDM increases with BMI levels to a greater extent in Aboriginal, relative to non-Aboriginal, mothers, and the disparities are more prominent at higher BMI values. Developing strategies in partnership with Aboriginal community members to optimise weight pre-conception (and across the life course) should be prioritised.

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CRediT authorship contribution statement

Marwan Awad Ahmed: Conceptualization, Data curation, Formal analysis, Methodology, Visualization, Writing – original draft. **Helen D. Bailey:** Data curation, Methodology, Supervision, Writing – review & editing. **Gavin Pereira:** Methodology, Writing – review & editing. **Scott W. White:** Supervision, Writing – review & editing. **Matthew J.L. Hare:** Methodology, Visualization, Writing – review & editing. **Kingsley Wong:** Methodology, Writing – review & editing. **Rhonda Marriott:** Methodology, Writing – review & editing. **Carrington C.J. Shepherd:** Conceptualization, Methodology, Supervision, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors do not have permission to share data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pmedr.2023.102444.

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