Maternal Region of Origin and Small for Gestational Age: A Cross-sectional Analysis of Victorian Perinatal Data.

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Research Article

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Abstract

Background

Being born small for gestational age is a strong predictor of the short- and long-term health of the neonate, child, and adult. Variation in the rates of small for gestational age have been identified across population groups in high income countries, including Australia. Understanding the factors contributing to these population group differences may assist clinicians to reduce the morbidity and mortality associated with being born small. Victoria, in addition to New South Wales, accounts for the largest proportion of net overseas migration and births in Australia. The aim of this research was to analyse how migration was associated with small for gestational age in Victoria.

Methods This was a cross sectional population health study of singleton births in Victoria from 2009 to 2018 (n = 708,475). The prevalence of being born small for gestational age (SGA; < 10th centile) was determined for maternal country of origin groups. Multivariate logistic regression analysis was used to analyse the association between maternal region of origin and SGA.

Results Maternal region of origin was an independent risk factor for SGA in Victoria (p < .001), with a prevalence of SGA for migrant women of 11.3% (n = 27,815) and 7.3% for Australian born women (n = 33,749). Women from South East Asia, South Central Asia, or Sub Saharan Africa, OR 1.75 (95%CI: 1.70 to 1.8), women from North and North East Africa, Middle East, OR 1.40 (95%CI: 1.35 to 1.45) and migrant women from the Americas, Europe, and Oceania, OR1.06 (95%CI: 1.02 to 1.12) more likely to birth an SGA child in comparison to women born in Australia.

Conclusions Victorian woman’s region of origin was an independent risk factor for SGA. Variation in the rates of SGA between maternal regions of origin indicates additional factors such as, a woman’s pre migration exposures, the context of the migration journey, settlement conditions and the social environment post migration impact the potential for SGA. These findings highlight the importance of intergenerational improvements to the wellbeing of migrant women and their children. Further research is required to identify modifiable elements that contribute to birthweight differences across population groups.

Background

Improving the wellbeing of women and children is essential if we are to achieve progress on the sustainable development goals, reduce inequality and create a more inclusive future for all [1]. Every person’s life potential is shaped during the critical periods of growth and human development that are associated with conception, pregnancy, and birth. Growing small for gestational age (SGA; < 10th centile) in utero more than doubles the risk of stillbirth [2], increases the child’s risk for neonatal death [3], postnatal growth stunting [4], and reduces learning potential in comparison to a child born appropriate for gestational age (AGA) [5, 6]. In adulthood, individuals who were born SGA are predisposed to chronic...
health problems, resulting in a decreased earning capacity [7], reduced productivity, and increased economic costs for the broader population [8].

Reducing preventable perinatal death by achieving a reduction to 10% in the prevalence of SGA by 2035 is a target of the Every Newborn Action Plan [9] endorsed by the World Health Organization. Children born in low- and middle-income countries are more likely to be SGA, and there are also differences between population groups in high income countries [10]. In Australia, 11.9% of migrant children were born SGA for the year 2017 compared to 9.7% of those to Australian born women [11], raising questions of what factors are driving differences in the prevalence of SGA across population groups.

The causes of growth restriction during the antenatal period are multifactorial, ranging from fetal malformations, infections, placental and umbilical cord abnormalities; to maternal factors, such as maternal preeclampsia [12] and anaemia [13]. In the absence of intrauterine pathology, SGA is socially influenced by the context the mother inhabits [14–16]. Being a younger woman [17], not in a relationship [18, 19], or being the woman's first baby [20] are associated with higher rates of SGA, as are smoking [21], being underweight, or having low gestational weight gain [22, 23]. In contrast, being overweight [24] and high gestational weight gain decreased the risk of SGA [25]. Gestational weight gain is primarily influenced by food security [26]. However, certain cultural factors are also associated with SGA through the influence of body image [27], food taboos, and dietary misconceptions [28, 29] on gestational weight gain. Environmental factors such as socioeconomic disadvantage [30, 31], natural disasters, famine, and conflict also increase the risk of SGA via pathways of reduced access to clean water, sanitation, food security, and health care [32–34].

Variation in the risk of SGA in high income countries, including Australia, has been associated with a woman's socioeconomic status [31, 35], via factors such as education [36], income [37], and living conditions [18] that influence a woman's access to resources in her social context. Socioeconomic status and the risk for SGA has been found to be influenced by a woman's racial classification [38] or ethnic group [39], as race modifies exposure to racially determined disadvantage and systemic racism [40]. Meta-analyses of the factors associated with SGA for migrant women in high income countries have confirmed that a woman's region of origin and migration status [41] increased her risk of SGA via pathways of access and barriers to social resources.

Australia was founded in the context of migration. The history of colonisation of First Nations country has resulted in Australia being home to people from over 270 diverse ancestry groups [42]. By 2018, close to 40% of women birthing in Australia were born overseas, with the largest proportion of women arriving from India and China [43]. Victoria and New South Wales account for both the largest proportion of net overseas migration and births in Australia [42, 44]. Maternal region of birth was found to be an independent risk factor for stillbirth and this risk increased 2.3-fold when SGA was diagnosed and 4.3-fold when SGA was not diagnosed [45]. Previous research into migrant health in Victoria has identified poorer perinatal health outcomes for some migrant population groups [46]. Population group differences in the prevalence of SGA have been identified for migrant women in high income countries. However,
possible differences in rates of SGA across population groups in Victoria remains unknown. This lack of knowledge is potentially contributing to a higher risk of stillbirth for women from certain regions of birth. Therefore, the aim of this study was to determine how a woman's position as a migrant is associated with the prevalence of being SGA at birth in Victoria.

Methods

Study design and population

A cross-sectional study of routinely collected population data on all singleton births in Victoria between January 2009 to December 2018 was undertaken. The quality of the Victorian Perinatal Data Collection (VPDC) is regularly audited for accuracy which supports the validity of the findings in this research study [47]. Data were available for the total population of women (n = 708,475), providing a sufficiently large study population for analysis across subgroups. Exclusion criteria were: fetal deaths, stillbirths, congenital abnormalities, births at gestations of less than 20 or greater than 43 weeks, and unknown neonatal sex, gestation, or birthweight. Multiple births were also excluded as they are more likely to be confounded by prematurity and maternal pregnancy conditions. After data cleaning, the sample captured 98.9% of women birthing a singleton baby in Victoria during the time period.

Low risk ethics approval was granted in December 2019 for secondary analysis of routinely collected perinatal data. All research methods were performed in accordance with the relevant guidelines and regulations for the analyses of secondary population data. The Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM) provided formal access to the deidentified data according to regulation 10 of the Public Health and Wellbeing Regulations 2009, ensuring respect for the privacy, confidentiality and cultural sensitivities of the women included in the study. Ethical review applied the Victorian Health Records Act Statutory Guidelines for research and due to the deidentified nature of the data a waiver for consent was approved. The research data were stored according to quality procedures for the storage of research data, including data encryption for transfer and storage.

Measurement of Key Variables

Small for gestational age.

Small for gestational age was the dependent variable, defined as a birthweight < 10th centile, adjusted for sex and gestational age [48]. Birthweight centiles were determined using the Australian national birthweight percentiles to define the point of significance [49] and were added by the VPDC prior to release to the researchers. The population standard was chosen as it has been validated to be representative of the Australian population [49], is the standard used in Victoria for population health research [50], and does not assume that race or ethnicity generates genetic differences in birthweight [51].

Maternal region of origin.
The independent variable, maternal region of origin, was a composite categorical variable provided by the Victorian Agency for Health Information (VAHI) and grouped according to the United Nations M49 Standard geoscheme \[52\]. Country of birth is a self-reported indicator routinely collected during the provision of maternity care in Victoria \[53\]. In preparation for analysis, we consolidated maternal country of birth into two categories, non-migrant women born in Australia and migrant women who were born in a country other than Australia. The variable region of origin was further grouped according to the unadjusted 10th centile birthweight clusters into four regional groupings: Americas, Europe, Oceania; Australia; North East Africa, North Africa, Middle East; and South East Asia, South Central Asia, Sub-Saharan Africa.

**Potential confounding variables.**

Socioeconomic Indexes for Areas (SEIFA) was used as the measure for socioeconomic status. SEIFA is a composite measure that includes a range of factors from employment status, household income, level of education, disability, single parenthood, and rental or mortgage status \[54\]. The SEIFA quintiles were consolidated from 5 levels into SEIFA advantaged (quintiles 4 and 5) average (quintile 3) and disadvantaged (quintiles 1 and 2) for statistical analysis. Additional variables controlled for in the analyses included maternal age in years, parity, relationship status, body mass index, height, smoking status before and after 20 weeks gestation, gestational age at first pregnancy visit. Maternal medical conditions and pregnancy complications included anaemia, diabetes, gestational diabetes, hypertensive disorders, pre-eclampsia, HELLP syndrome (haemolysis, elevated liver enzymes, low platelet count), and suspected fetal growth restriction.

**Statistical Analysis**

The statistical data editor software program Statistical Product and Services Solution version 26 (SPSS V.26) was used for data analyses. Data were checked using frequencies, errors, and outliers in preparation for analyses. Of the total study population, 704 cases (0.01%) were missing birthweight and these cases were deleted. Bivariate analyses using the Chi-square test for independence between the dependant variable SGA and the independent variable were performed, including examining the potential confounding of mediating variables identified in the literature review. Statistically significant unadjusted associations were identified in preparation for binary logistic regression analysis. Hierarchical logistic regression models were built to measure the independent association [presented as adjusted odds ratio (aOR) with 95% confidence intervals (95%CI)] between SGA and maternal region of origin, whilst controlling for effects of the identified confounding variables. Statistical significance was set at p value < 0.05.

**Results**

Between 2009 and 2018 there were 708,475 women who birthed a singleton baby in Victoria. The demographics of the study population are presented in the Additional file 1, Table 1. Most women were born in Australia (67%; n = 461,903). The remaining women were migrants from South East Asia and Sub-
Saharan Africa (17.9%; n = 126,640); the Americas, Europe, and Oceania (9.1%; n = 64,195); and North East Africa, North Africa, and the Middle East (7.9%; n = 55,737). The mean birthweight for the Victorian study population (3387.39g, SD = 543.4g) was 59.4g (95%CI: 58.1 to 60.7, \( p < .001 \)) higher than the Australian 2017 population mean birthweight of 3328g [44].

A smaller proportion of migrant women than Australian born women were not in a relationship (6.7% vs 14.6% respectively); under the age of 20 years (0.8% vs 2.5%); and SEIFA advantaged (36.9% vs 41.6%). Migrant women were also more likely than Australian born women to be birthing their first baby (45.3% vs 43% respectively); classified as short height (12.5% vs 28%); develop more gestational diabetes managed with diet (9.5% vs 4.0) and insulin (6.1% vs 2.8%); and experience more suspected fetal growth restriction (5.8% vs 4.3%). The majority of women in the study population were non-smokers. Those that did smoke were more likely to be Australian born women rather than migrant women, both before 20 weeks gestation (3.8% vs 13.4%); and after 20 weeks gestation (1.9% vs 8.5%). These findings were statistically significant at \( p < 0.001 \), and are presented in the Additional file 1, Table 2.

**Maternal Region of Origin and SGA**

There was a total of 61,564 SGA neonates in the study population, resulting in an overall SGA rate of 8.7%. A higher proportion of SGA was identified for migrant women at 11.3% (n=27,815) compared to 7.3% (n=33,749) for Australian born women. We found significant variation in the distribution of SGA across maternal regions of origin (\( p < 0.001 \)). The proportion of SGA for migrant women from South East, South Central Asia, and Sub-Saharan Africa 14% (n= 17,687); North East, North Africa, and the Middle East 10% (n=5,563) was higher than for women from Australia 7.3% (n=33,749); the Americas, Europe, and Oceania 7.1% (n=4,565). After controlling for confounding factors, regression analysis identified migrant women were 1.48 times more likely to birth an SGA baby than women born in Australia (95%CI: 1.45 to 1.51, \( p < .001 \)), see Table 1.

**Table 1.**

*Adjusted odds ratio (95%CI) independent association between SGA and migrant/non migrant women.*
| Variable                              | SGA aOR (95%CI) |
|--------------------------------------|-----------------|
| **Marital Status**                   |                 |
| In a Relationship                    | 1               |
| Not in a Relationship                | 1.06 (1.03 to 1.10) *** |
| **Age Group (years)**                |                 |
| < 20                                 | 0.78 (0.72 to 0.85) *** |
| 20 to 35                             | 0.91 (0.89 to 0.94) *** |
| 35+                                  | 1               |
| **Parity**                           |                 |
| Primiparous                          | 2.05 (2.01 to 2.10) *** |
| Multiparous                          | 1               |
| **BMI**                              |                 |
| Underweight                          | 2.06 (1.96 to 2.17) *** |
| Healthy Weight                       | 1.28 (1.26 to 1.31) *** |
| Overweight                           | 1               |
| **Height**                           |                 |
| Tall                                 | 1               |
| Average                              | 1.63 (1.59 to 1.68) *** |
| Short                                | 2.77 (2.69 to 2.85) *** |
| **Smoking before 20wks**             |                 |
| Yes                                  | 1.13 (1.06 to 1.20) *** |
| No                                   | 1               |
| **Smoking after 20wks.**             |                 |
| Yes                                  | 2.20 (2.05 to 2.37) *** |
| No                                   | 1               |
| **Gestational Age at 1st Visit**     |                 |
| Before 11 weeks                      | 1               |
| 12 to 23 weeks                       | 1.07 (1.04 to 1.09) *** |
| 24 weeks /no care                    | 1.25 (1.21 to 1.30) *** |
| **Type 1 Diabetes**                  |                 |
| Yes                                  | 1               |
| No                                   | 2.98 (2.29 to 3.89) *** |
| **Pre-existing Hypertension**        |                 |
| Yes                                  | 1.26 (1.15 to 1.37) *** |
| No                                   | 1               |
| **Gestational Diabetes - Insulin**   |                 |
| Yes                                  | 1               |
| No                                   | 1.29 (1.22 to 1.36) *** |
| **Preeclampsia**                     |                 |
| Yes                                  | 1.44 (1.35 to 1.53) *** |
| No                                   | 1               |
| **HELLP Syndrome**                   |                 |
| Yes                                  | 1.68 (1.38 to 2.04) *** |
| No                                   | 1               |
| **Suspected FGR**                    |                 |
| Yes                                  | 10.25 (9.97 to 10.54) *** |
| No                                   | 1               |
Maternal SEIFA

|                           | advantaged  |     |
|---------------------------|-------------|-----|
|                           | Reference   | 1   |
| Average                   | 1.05 (1.02 to 1.08) | ** |
| Disadvantage              | 1.05 (1.02 to 1.07) | *** |

Maternal Region of Origin

|                             | Australia Born Women  |     |
|-----------------------------|-----------------------|-----|
|                             | Reference             | 1   |
| Migrant Women               | 1.48 (1.45 to 1.51)   | *** |

Note. 1. Birthweight Adjusted for Sex and Gestational Age. 2. r = reference. 3. ***p < 0.001, ** p < 0.01.

Independent association between SGA and maternal region of origin.

Multivariate modelling was performed to identify the differences in SGA between maternal regions of origin groups, see Table 2. A statistically significant independent association between SGA and maternal region of origin groups was identified. In comparison to Australian born women, migrant women from the Americas, Europe, and Oceania were more likely to birth an SGA child (aOR 1.06, 95%CI: 1.02 to 1.12, p = .003); women from North Africa, North East Africa, and the Middle East were 1.4 times more likely to birth an SGA child (95%CI: 1.35 to 1.45, p < .001) and women from South East Asia, South Central Asia, and Sub-Saharan Africa were 1.75 times more likely to birth an SGA child (95%CI: 1.70 to 1.80, p < .001).

Table 2:

*adjusted odds ratio (95%CI) independent association between SGA and maternal region of origin.*

| Maternal Region of Origin                                             | SGA aOR (95%CI) |
|-----------------------------------------------------------------------|-----------------|
| Australia r                                                            | 1               |
| Americas, Europe, Oceania                                             | 1.06 (1.02 to 1.12) | *   |
| North & North East Africa, Middle East                                | 1.40 (1.35 to 1.45) | *** |
| South East, South Central Asia, Sub Saharan Africa                    | 1.75 (1.70 to 1.80) | *** |

Note. 1. Birthweight Adjusted for Sex and Gestational Age. 2. r = reference. 3. ***p < 0.001. * p < 0.05. 4. Confounding variables included, marital status, maternal age group, parity, BMI, height group, smoking before or after 20 weeks gestation, gestational age at first visit, type 1 diabetes pre-existing hypertension, gestational diabetes -insulin, pre-eclampsia, HELLP syndrome, suspected FGR.

Socioeconomic status has previously been identified as a strong influencing factor in the association with SGA. Therefore, additional regression analysis was undertaken to assess for collinearity with the
independent variable region of origin. A series of modelling stratified by SEIFA advantaged, average, and disadvantaged was performed to confirm the relationships between SGA and region of origin (see Table 3). The independent association between maternal region of origin and SGA was statistically significant across all SEIFA groups except for average and disadvantaged women from the Americas, Europe, and Oceania. These findings indicate for migrant women in Victoria, the risk of SGA did not follow a classic socioeconomic gradient of disadvantage, confirming maternal region of origin was a much stronger predictor of SGA than maternal socioeconomic status.

Table 3.

Independent association between SGA and maternal region of origin stratified SEIFA advantage, average, disadvantaged.

| Regional Region of Origin                             | SGA OR (95%CI) | SEIFA Advantaged | SEIFA Average | SEIFA Disadvantaged |
|------------------------------------------------------|----------------|------------------|---------------|---------------------|
| ralia r                                               | 1              | 1                | 1             | 1                   |
| ricas, Europe, Oceania                               | 1.15 (1.08 to 1.22) *** | 1.01 (0.92 to 1.11) | 0.99 (0.92 to 1.06) |
| h & North East Africa, Middle                         | 1.38 (1.30 to 1.47) *** | 1.40 (1.29 to 1.53) *** | 1.40 (1.32 to 1.49) *** |
| h East, South Central Asia, Sub-Saharan Africa        | 1.83 (1.75 to 1.91) *** | 1.84 (1.73 to 1.95) *** | 1.67 (1.61 to 1.74) *** |

Note. 1. Birthweight Adjusted for Sex and Gestational Age. 2. r = reference. 3. ***p < 0.001. 4. Confounding variables included, marital status, maternal age group, parity, BMI, height group, smoking before or after 20 weeks gestation, gestational age at first visit, type 1 diabetes pre-existing hypertension, gestational diabetes -insulin, pre-eclampsia, HELLP syndrome, suspected FGR.

Being born SGA was also associated with several maternal characteristics, medical conditions, and pregnancy complications, see Table 1. For the total study population, lower risks of SGA were associated with younger age: a risk reduction of 23% at < 20 years (aOR 0.78, 95%CI: 0.72 to 0.85, p < 0.001), and 11% at age 20 to 35 years (aOR 0.91, 95%CI: 0.89 to 0.94, p < .001), compared with women over 35 years of age. Women birthing their first baby were twice as likely (aOR 2.05, 95%CI: 2.01 to 2.10, p <.001) to birth an SGA child than women birthing subsequent babies. Shorter women (< 25th centile of the study population) were 2.77 times more likely (95%CI: 2.69 to 2.85, p < .001), and average height women (add the centile range here to make it clear) 1.63 times more likely (95%CI: 1.59 to 1.68, p < .001), to birth an
SGA baby than tall women (> 75th centile of the study population). Being underweight doubled a woman’s odds of birthing an SGA baby (aOR 2.06, 95%CI: 1.96 to 2.17, p < .001) compared to women who were overweight or obese.

Women not accessing care until after 24 weeks gestational or not at all were 25% more likely to birth an SGA child than women accessing maternity care before 12 weeks gestation (95%CI: 1.21 to 1.30, p < .001). The risk of SGA increased in proportion to the severity of hypertensive disorders from 26% more likely with pre-existing hypertension, 44% more likely with pre-eclampsia, to 68% more likely with HELLP syndrome (p < .001). Both type 1 diabetes (aOR 2.98, 95%CI: 2.29 to 3.89, p < .001), and gestational diabetes treated with Insulin (aOR 1.29, 95%CI: 1.22 to 1.36, p < .001), were protective, decreasing the odds of SGA compared to women without these conditions. Women with suspected fetal growth restriction were over 10.25 times more likely to birth an SGA child than women not suspected of fetal growth restriction (95%CI: 9.97 – 10.54, p < .001).

Discussion

The prevalence of SGA in the population of Victorian women who gave birth between 2009 and 2018 was higher for migrant women than women born in Australia. The highest prevalence of SGA was for migrant women from the regions South East Asia, South Central Asia, and Sub-Saharan Africa, followed by migrant women from North Africa, North East Africa, and the Middle East, and was lowest for migrant women from the America’s, Europe, and Oceania. These findings were consistent after adjustment for potential confounding factors indicating a woman’s position as a migrant was a strong predictor of SGA in Victoria.

Women in our study population from regions with a higher proportion of low- and middle-income countries, such as, South East Asia and Sub-Saharan Africa had a higher prevalence of SGA compared to women from high income regions such as Americas, Europe, or Australia. These findings confirm a woman’s region of origin is associated with her risk for SGA. A potential explanation for this associated risk may be a woman’s preconception exposures to conditions that influence her reproductive health and therefore her potential to birth an SGA child. A woman’s context prior to migration influences her wellbeing and sets the scene for her migration journey: growing up in a high-income country allows access to resources, such as universal education, food security and health care, that may not be available to all women in low- and middle-income countries.

The level of gender equality, conflict and stability of her environment influences a woman’s reason for migration: a voluntary migration for employment or education is a very different journey to one that is forced due to conflict or natural disaster [55]. In 2018, over 70 million people were escaping persecution and conflict, a forced migration leaving them exposed to human rights abuse, trauma, and human trafficking [56]. How a woman arrives in a new country determines her access to resources such as health care, employment, and freedom of movement, via complex visa systems and associated visa privileges or barriers [57]. Migration becomes a social determinant of SGA via pathways of the migration context [58],
differential access to social resources during settlement [41, 59] and potential exposures to racially determined discrimination in a new country [40, 60, 61].

The risk of SGA for migrant women in our study population was not mediated by socioeconomic advantage, suggesting other factors were stronger predictors of SGA for some women. Previous research has identified the birthweights of migrant children increase over time to align with the birthweights of children born to women from the settlement country, irrespective of a woman's geographical origin or ancestry [62]. Key factors in achieving this birthweight increase were comprehensive settlement policies that were responsive to the needs of migrant women [63]. This adjustment in birthweight after resettlement suggests the birthweight potential of migrant offspring is not fixed according to the woman's ancestry, geographical origin, or preconception exposures. Rather, the context of migration, settlement conditions and the social environment post migration also impact the potential for SGA [63]. Our research was not designed to measure the influence of pre- and post-migration conditions; however, we were able to identify additional factors to region of origin and socioeconomic status that were associated with SGA.

Consistent with other studies, we found women birthing their first child were more likely to birth an SGA child compared to women birthing subsequent children [17, 20]. We also identified, underweight women were more likely to birth an SGA child than overweight women [22–24, 26, 64], confirming the importance of food security and appropriate gestational weight gain during pregnancy. We also found women of tall stature were less likely to birth an SGA child, and these tall women were more likely to be SEIFA advantaged and originate from high income regions such as the Americas, Europe, and Australia. Maternal height has previously been reported to align with social advantage [15, 65] and short maternal stature has been identified as an intergenerational response to environmental conditions of malnutrition, disease, and poverty [66], supporting the theory that premigration exposures may influence a woman's potential to birth an SGA child.

The health of the mother is recognised to influence the health of the child through physiological adaptations to pregnancy, and complications associated with adaptation can undermine health [6, 12]. Our finding of women over 35 years being more likely to birth an SGA child contradicted other studies [17], and may be influenced by factors we did not measure, such as the use of assisted reproductive technology [67] or other maternal health and social conditions [34]. The potential for SGA in the Victorian population studied was increased for women with hypertensive disorders and the risk increased with the severity of the disease, which confirms the importance of improving the health and wellbeing of all women. Smoking is a modifiable factor that was associated with an increased risk of SGA, however, the most effective interventions for smoking cessation are still to be identified [68]. Women who accessed maternity care after 24 weeks gestation or not at all were also more likely to birth an SGA child, irrespective of their region of origin, indicating the importance of universal access to culturally safe maternity care for all women [41, 69, 70].

The strength of this study is the large sample size which enabled analyses for the outcome of SGA across smaller population subgroups. Further, the quality of the VPDC has previously been validated for
conservative analysis of associations which strengthens the generalisability of the findings to the population of Victoria. Data was predominantly complete on the key variables SGA and maternal region of origin; therefore, missing data was not a major problem for analysis. The study controlled for a number of confounding factors identified in previous research, which facilitated examination of independent associations. Some of the findings from our research regarding the influence of confounding factors and their association with SGA were similar to those from previous studies. However, the study also had some limitations. Cross sectional study designs do not establish causal relationships; although, they do provide a valuable tool for initial analysis of population health outcomes and associations [71]. Maternal region of origin is a composite self-reported variable that is useful for initial analyses of migrant women's health outcomes. However, the data set used did not permit the identification of which elements of being a migrant woman, or which countries in each region, contributed to the associated higher risk of SGA.

Including all singleton births across all gestational age groups potentially introduced bias as premature babies are more likely to be SGA and spontaneous prematurity is also influenced by a range of sociocultural factors. This is also a recognised limitation when using population growth standards [72]. In response, some authors would advocate for the use of a customised growth standard rather than a population growth standard to negate the confounding effect of prematurity [51]. However, customised growth standards cannot distinguish between the physiological or pathological characteristics of fetal growth [73] and thus may mask pathological growth restriction [72]. In addition, customising fetal growth standards assume a baby may be constitutionally small based on the mother's geographical origins or ethnicity. Whilst there may always be babies born healthy and small, the INTERGROWTH-21st studies [16] have demonstrated improvements in fetal and neonatal wellbeing can be achieved across all population groups. When a woman's health and nutrition needs were met in an environment conducive to wellbeing, only 3.5% of variation in fetal and newborn growth across population groups was due to differences in ancestry [15].

Understanding why some fetal growth adaptions become pathological and others do not continue to be an evolving space of research and public endeavour. This study has addressed an important question regarding SGA that has direct clinical relevance. Understanding the different factors that lead to population group differences in being born SGA is essential if we are to achieve an equitable and inclusive future for all women and children. A balanced approach is required to identify effective preventative strategies that do not contribute to inappropriate intervention in pregnancy for healthy small babies. To our knowledge this is the first study to measure the association between migration and SGA for the total population of women birthing in Victoria.

Conclusions

In summary, we have identified a woman's social position as a migrant is an independent factor associated with birthing an SGA child in Victoria. The increased rate of SGA children born to migrant Australian women varied according to the woman's region of origin, suggesting additional factors associated with migration and settlement contribute to significant differences in the risk associated with
SGA. These findings indicate both short- and long-term commitment to targeted initiatives are essential to achieve intergenerational improvements in the wellbeing of migrant women and their children. Further research is required to determine which aspects of the migration and settlement experiences can be modified to reduce the risk of SGA and therefore avoid the long-term consequences that flow from this.

List Of Abbreviations

AGA: Appropriate for Gestational Age

aOR: Adjusted Odds Ratio

BMI: Body Mass Index

CI: Confidence Interval

EFW: Estimated Fetal Weight

FGR: Fetal Growth Restriction

HELLP: Haemolysis, Elevated Liver Enzymes and Low Platelet Count

INTERGROWTH-21ST: The INTERnational fetal and new-born GROWTH consortium for the 21st century project

SEIFA: Socioeconomic Indexes for Areas

SGA: Small for Gestational Age

SPSS: Statistical Product and Services Solution

VAHI: Victorian Agency for Health Information

VPDC: Victorian Perinatal Data Collection

Declarations

Ethics approval

This study was granted low risk ethics approval by the St Vincent’s Research Ethics Committee. (HREC/59974/SVHM-2019-194857[v1]).

Consent for publication

Not applicable.

Availability of data and materials statement
The data that support findings of this study are under restrictions from the Victorian Perinatal Data Collection. The data were provided by the Victorian Agency for Health Information via their data request hub and are not publicly available. Due to the sensitive nature of public health records, access to the data is by formal application to the Consultative Council on Obstetric and Paediatric Mortality and Morbidity (CCOPMM).

**Competing interests**

The authors declare they have no competing interests.

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**Authors contributions**

Conceptualisation: SG, PL, KS, FA.

Methodology: SG, PL.

Data curation: SG.

Formal analysis: SG, PL.

Investigation: SG, PL, KS, FA.

Project administration: SG, FA.

Validation: SG, PL, KS, FA.

Writing original draft: SG

Writing – review & editing: SG, PL, KS, FA.

Visualisation: SG

Supervision: FA

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**References**

1. United Nations. Transforming our world: The 2030 agenda for sustainable development. New York: UN Publishing; 2015.
2. Flenady V, Wojcieszek A, Middleton P, Ellwood D, Erwich J, Coory M, et al. Stillbirths: Recall to action in high-income countries. The Lancet. 2016;387(10019):691–702.
3. Flamant C, Gascoin G. Short-term outcome and small for gestational age newborn management. Journal de gynécologie, obstétrique et biologie de la reproduction. 2013;42(8):985–95.
4. Lee A, Kozuki N, Cousens S, Stevens G, Blencowe H, Silveira M, et al. Estimates of burden and consequences of infants born small for gestational age in low- and middle-income countries with INTERGROWTH-21st standard: Analysis of CHERG datasets. British Medical Journal (Online). 2017;358:j3677.
5. de Bie HMA, Oostrom KJ, Delemarre-van de Waal HA. Brain development, intelligence and cognitive outcome in children born small for gestational age. Hormone Research in Paediatrics. 2010;73(1):6–14.

6. Miller SL, Huppi PS, Mallard C. The consequences of fetal growth restriction on brain structure and neurodevelopmental outcome. The Journal of Physiology. 2016;594(4):807–23.

7. Australian Institute of Health and Welfare. Australia's Health 2018. Canberra: Australian Institute of Health and Welfare; 2018. Report No.: 16.

8. Risnes KR, Vatten LJ, Baker JL, Jameson K, Sovio U, Kajantie E, et al. Birthweight and mortality in adulthood: A systematic review and meta-analysis. International Journal of Epidemiology. 2011;40(3):647–61.

9. Lawn JE, Blencowe H, Oza S, You D, Lee ACC, Waiswa P, et al. Every newborn: Progress, priorities, and potential beyond survival. The Lancet. 2014;384(9938):189–205.

10. World Health Organization. Global nutrition targets 2025: Low birth weight policy brief. World Health Organization; 2014.

11. Data tables: Australia's mothers and baby's 2017. [Internet]. AIHW Website. 2019 [cited 20/12/2020]. Available from: https://www.aihw.gov.au/reports/mothers-babies/australias-mothers-and-babies-2017-in-brief/data.

12. Burton GJ, Redman CW, Roberts JM, Moffett A. Pre-eclampsia: Pathophysiology and clinical implications. British Medical Journal. 2019;366:l2381.

13. Haider BA, Olofin I, Wang M, Spiegelman D, Ezzati M, Fawzi WW. Anaemia, prenatal iron use, and risk of adverse pregnancy outcomes: Systematic review and meta-analysis. British Medical Journal. 2013;346(6):f3443-f.

14. Kelly Y, Panico L, Bartley M, Marmot M, Nazroo J, Sacker A. Why does birthweight vary among ethnic groups in the UK? Findings from the Millennium Cohort Study. Journal of Public Health. 2009;31(1):131–7.

15. Villar J, Papageorghiou AT, Pang R, Ohuma EO, Ismail LC, Barros FC, et al. The likeness of fetal growth and newborn size across non-isolated populations in the INTERGROWTH-21st project: The fetal growth longitudinal study and newborn cross-sectional study. The Lancet Diabetes and Endocrinology. 2014;2(10):781–92.

16. Papageorghiou AT, Kennedy SH, Salomon LJ, Altman DG, Ohuma EO, Stones W, et al. The INTERGROWTH-21st fetal growth standards: toward the global integration of pregnancy and pediatric care. American Journal of Obstetrics and Gynecology. 2018;218(2):S630-S40.

17. Kozuki N, Lee ACC, Silveira MF, Sania A, Vogel JP, Adair LS, et al. The associations of parity and maternal age with small-for-gestational-age, preterm, and neonatal and infant mortality: A meta-analysis. BioMed Central Public Health. 2013;13(3):S2.

18. Shapiro GD, Bushnik T, Wilkins R, Kramer MS, Kaufman JS, Sheppard AJ, et al. Adverse birth outcomes in relation to maternal marital and cohabitation status in Canada. Annals of Epidemiology. 2018;28(8):503-9.e11.
19. Shah PS, Zao J, Ali S. Maternal marital status and birth outcomes: A systematic review and meta-analyses. Maternal and Child Health Journal. 2011;15(7):1097–109.

20. Ciobanu A, Rouvali A, Syngelaki A, Akolekar R, Nicolaides KH. Prediction of small for gestational age neonates: Screening by maternal factors, fetal biometry, and biomarkers at 35–37 weeks’ gestation. American Journal of Obstetrics and Gynecology. 2019;220(5):486–.

21. Blackburn S. Maternal, fetal and neonatal physiology. 4th ed. United States of America: Elsevier Saunders; 2013.

22. Rafei RE, Abbas HA, Alameddine H, Bizri AA, Melki I, Yunis KA. Assessing the risk of having small for gestational age newborns among Lebanese underweight and normal pre-pregnancy weight women. Maternal & Child Health Journal. 2018;22(1):130–6.

23. Pugh SJ, Albert PS, Kim S, Grobman W, Hinkle SN, Newman RB, et al. Patterns of gestational weight gain and birthweight outcomes in the Eunice Kennedy Shriver National Institute of Child Health and Human Development fetal growth studies–singletons: a prospective study. American Journal of Obstetrics and Gynecology. 2017;217(3):346.e1–e11.

24. Denize KM, Acharya N, Prince SA, da Silva DF, Harvey ALJ, Ferraro ZM, et al. Addressing cultural, racial and ethnic discrepancies in guideline discordant gestational weight gain: A systematic review and meta-analysis. Peer Journal. 2018;2018(8):e5407.

25. Anderson NH, Sadler LC, Stewart AW, Fyfe EM, McCowan LME. Independent risk factors for infants who are small for gestational age by customised birthweight centiles in a multi-ethnic New Zealand population. Australian and New Zealand Journal of Obstetrics and Gynaecology. 2013;53(2):136–42.

26. Goldstein RF, Abell SK, Ranasinha S, Misso M, Boyle JA, Black MH, et al. Association of gestational weight gain with maternal and infant outcomes: A systematic review and meta-analysis. Journal of American Medical Association. 2017;317(21):2207–25.

27. Morisaki N, Urayama KY, Yoshii K, Subramanian SV, Yokoya S. Ecological analysis of secular trends in low birth weight births and adult height in Japan. Journal of Epidemiology and Community Health. 2017;71(10):1014–8.

28. Getnet W, Aycheh W, Tessema T. Determinants of food taboos in the pregnant women of the Awabel District, East Gojjam Zone, Amhara Regional State in Ethiopia. Advances in Public Health. 2018;2018:9198076.

29. Zerfu TA, Umeta M, Baye K. Dietary habits, food taboos, and perceptions towards weight gain during pregnancy in Arsi, rural central Ethiopia: A qualitative cross-sectional study. Journal of Health, Population and Nutrition. 2016;35(1):22.

30. Bushnik T, Yang S, Kaufman JS, Kramer MS, Wilkins R. Socioeconomic disparities in small-for-gestational-age birth and preterm birth. Health Reports. 2017;28(11):3–10.

31. Hirst JE, Knight HE, Ohuma EO, Dwyer T, Hennig BD, Papageorghiou AT, et al. Social gradient of birthweight in England assessed using the INTERGROWTH-21st gestational age-specific standard. Archives of Disease in Childhood: Fetal and Neonatal Edition. 2018;104(5):F486-F92.
32. Hendrix NMD, Berghella VMD. Non-placental causes of intrauterine growth restriction. Seminars in Perinatology. 2008;32(3):161–5.
33. Kayode GA, Amoah-Coleman M, Akua Agyepong I, Ansah E, Grobbee DE, Klipstein-Grobusch K. Contextual risk factors for low birth weight: A multilevel analysis. PLoS ONE. 2014;9(10):e109333-e.
34. Cetin I, Mandò C, Calabrese S. Maternal predictors of intrauterine growth restriction. Current Opinion in Clinical Nutrition and Metabolic Care. 2013;16(3):310–9.
35. Bushnik T, Yang S, Kaufman JS, Kramer MS, Wilkins R. Socioeconomic disparities in small-for-gestational-age birth and preterm birth. 2017;28(11):3–10.
36. David M, Borde T, Brenne S, Ramsauer B, Henrich W, Breckenkamp J, et al. Obstetric and perinatal outcomes among immigrant and non-immigrant women in Berlin, Germany. Archives of Gynecology & Obstetrics. 2017;296(4):745–62.
37. Slaughter-Acey JC, Holzman C, Calloway D, Tian Y. Movin’ on up: Socioeconomic mobility and the risk of delivering a small-for-gestational age infant. Maternal and Child Health Journal. 2016;20(3):613–22.
38. Kothari CL, Paul R, Dormitorio B, Ospina F, James A, Lenz D, et al. The interplay of race, socioeconomic status and neighborhood residence upon birth outcomes in a high black infant mortality community. Social Science and Medicine Journal - Population Health. 2016;2:859–67.
39. Eskes M, Abu-Hanna A, Ravelli ACJ, Waelput AJM, Scherjon SA, Bergman KA. Small for gestational age and perinatal mortality at term: An audit in a Dutch national cohort study. European Journal of Obstetrics & Gynecology & Reproductive Biology. 2017;215:62–7.
40. Slaughter-Acey JC, Talley LM, Stevenson HC, Misra DP. Personal versus group experiences of racism and risk of delivering a small-for-gestational age infant in African American women: A life course perspective. Journal of Urban Health. 2019;96(2):181–92.
41. Heslehurst N, Brown H, Pemu A, Coleman H, Rankin J. Perinatal health outcomes and care among asylum seekers and refugees: A systematic review of systematic reviews. BioMed Central Medicine. 2018;16(1):89–25.
42. Migration: Australia [Internet]. ABS. 2020 [cited 20/12/2020]. Available from: https://www.abs.gov.au/statistics/people/population/migration-australia/latest-release#net-overseas-migration.
43. Australia's mothers and babies data visualisations [Internet]. AIHW. 2020 [cited 18/12/2020]. Available from: https://www.aihw.gov.au/reports/mothers-babies/australias-mothers-babies-2017-data-visualisations/contents/demographics-of-mothers-and-their-babies/maternal-country-of-birth.
44. Births, Australia, 2017 [Internet]. ABS Website. 2018 [cited 20/12/2020]. Available from: https://www.abs.gov.au/AUSSTATS/abs@.nsf/Latestproducts/3301.0Main%20Features32017?opendocument&tabname=Summary&prodno=3301.0&issue=2017&num=&view=.
45. Davies-Tuck ML, Davey M-A, Wallace EM. Maternal region of birth and stillbirth in Victoria, Australia 2000–2011: A retrospective cohort study of Victorian perinatal data. PLoS ONE. 2017;12(6):e0178727-e.
46. Belihu FB, Davey M-A, Small R. Perinatal health outcomes of East African immigrant populations in Victoria, Australia: A population based study. BioMed Central Pregnancy and Childbirth. 2016;16(1):86-

47. Davey M-A, Sloan M-L, Palma S, Riley M, King J. Methodological processes in validating and analysing the quality of population-based data: A case study using the Victorian Perinatal Data Collection. Health Information Management Journal. 2013;42(3):12–9.

48. Lausman A, Kingdom J. Intrauterine growth restriction: Screening, diagnosis, and management. Journal of Obstetrics and Gynaecology Canada. 2013;35(8):741–8.

49. Dobbins TA, Sullivan EA, Roberts CL, Simpson JM. Australian national birthweight percentiles by sex and gestational age, 1998–2007. Medical Journal of Australia. 2012;197(5):291–4.

50. Selvaratnam RJ, Davey M-A, Wallace EM. The pitfalls of using birthweight centile charts to audit care. PloS one. 2020;15(6):e0235113.

51. Gardosi J, Francis A, Turner S, Williams M. Customized growth charts: rationale, validation and clinical benefits. American Journal of Obstetrics and Gynecology. 2018;218(2):S609-S18.

52. Standard Country or Area Codes For Statistical Use (M49) [Internet]. 2011 [cited 11/07/2020]. Available from: https://unstats.un.org/unsd/methodology/m49/.

53. Victorian Government. Victorian Perinatal Data Collection Manual, Version 7.0. Melbourne: Department of Health and Human Services; 2019.

54. Australian Bureau of Statistics. Technical paper: Socio-economic indexes for areas (SEIFA) 2016. ACT: Commonwealth of Australia; 2018.

55. United Nations. International migration 2019: Highlights. Department of Economic and Social Affairs PD; 2019.

56. Sustainable Development Goals [Internet]. United Nations. 2019. Available from: https://www.un.org/sustainabledevelopment/.

57. Australian Human Rights Commision. Immigration detention and human rights 2016 [Available from: https://humanrights.gov.au/our-work/asylum-seekers-and-refugees/projects/immigration-detention-and-human-rights.

58. Villalonga-Olives E, Kawachi I, von Steinbüchel N. Pregnancy and birth outcomes among immigrant women in the US and Europe: A systematic review. Journal of Immigrant and Minority Health. 2016;19(6):1469–87.

59. Pangas J, Ogunsiji O, Elmir R, Raman S, Liamputtong P, Burns E, et al. Refugee women's experiences negotiating motherhood and maternity care in a new country: A meta-ethnographic review. International Journal of Nursing Studies. 2019;90:31–45.

60. Boucher A. Measuring migrant worker rights violations in practice: The example of temporary skilled visas in Australia. Journal of Industrial Relations. 2019;61(2):277–301.

61. Kearns AW, Egan, M. Tabbner, C. & Tannahill, C. Healthy migrants in an unhealthy city? The effects of time on the health of migrants living in deprived areas of Glasgow. Journal of International
62. Eskild A, Sommerfelt S, Skau I, Gyrten J. Offspring birthweight and placental weight in immigrant women from conflict-zone countries; does length of residence in the host country matter? A population study in Norway. Acta obstetricia et gynecologica Scandinavica. 2019;99(5):615–22.

63. Sørbye IK, Vangen S, Juarez SP, Bolumar F, Morisaki N, Gissler M, et al. Birthweight of babies born to migrant mothers - What role do integration policies play? Social Science and Medicine. 2019;9.

64. Reiss K, Breckenkamp J, Borde T, Brenne S, David M, Razum O. Contribution of overweight and obesity to adverse pregnancy outcomes among immigrant and non-immigrant women in Berlin, Germany. European Journal of Public Health. 2015;25(5):839–44.

65. Subramanian SV, Özaltin E, Finlay JE. Height of nations: A socioeconomic analysis of cohort differences and patterns among women in 54 low- to middle-income countries. PloS one. 2011;6(4):e18962.

66. Hayward I, Malcoe LH, Cleathero LA, Janssen PA, Lanphear BP, Hayes MV, et al. Investigating maternal risk factors as potential targets of intervention to reduce socioeconomic inequality in small for gestational age: A population-based study. BioMed Central Public Health. 2012;12(1):333-.

67. Fujimoto VY, Luke B, Brown MB, Jain T, Armstrong A, Grainger DA, et al. Racial and ethnic disparities in assisted reproductive technology outcomes in the United States. Fertility and sterility. 2010;93(2):382–90.

68. McDonnell BP, Dicker P, Keogan S, Clancy L, Regan C. Smoking cessation through optimisation of clinical care in pregnancy: the STOP randomised controlled trial. Clinical Trials. 2019;20(1):550–9.

69. Yelland J, Riggs E, Wahidi S, Fouladi F, Casey S, Szwarc J, et al. How do Australian maternity and early childhood health services identify and respond to the settlement experience and social context of refugee background families? BioMed Central Pregnancy and Childbirth. 2014;14(1):348-.

70. Puthussery SMSW. Perinatal outcomes among migrant mothers in the United Kingdom: Is it a matter of biology, behaviour, policy, social determinants or access to health care? Best Practice & Research: Clinical Obstetrics & Gynaecology. 2015;32:39–49.

71. Webb P, Bain C, Page A. Essential epidemiology: an introduction for students and health professionals. Third ed. Cambridge, United Kingdom: Cambridge University Press; 2017.

72. MacDonald TM, McCarthy EA, Walker SP. Shining light in dark corners: Diagnosis and management of late-onset fetal growth restriction. Australian and New Zealand Journal of Obstetrics and Gynaecology. 2015;55(1):3–10.

73. Henrichs J, Verfaille V, Jellema P, Viester L, Pajkrt E, Wilschut J, et al. Effectiveness of routine third trimester ultrasonography to reduce adverse perinatal outcomes in low risk pregnancy (the IRIS study): nationwide, pragmatic, multicentre, stepped wedge cluster randomised trial. British Medical Journal. 2019.