

RESEARCH

Open Access



Exploring maternal health, lifestyle, and socioeconomic influences on childhood obesity in Australia

Nasrin Begum^{1,2*}, Enamul Kabir^{3*}, Rasheda Khanam⁴ and Kabir Ahmad⁵

Abstract

Background and objective Childhood obesity has become a significant public health challenge, with its prevalence rising globally. Obesity is defined as a body mass index at or above the 95th percentile for children of the same age and sex. This study aimed to group maternal characteristics during pregnancy and assess their association with childhood obesity from ages 2 to 15 years.

Methods Data from 4,060 mothers in the B cohort (wave 1, children aged 0–1 year) of the Longitudinal Study of Australian Children (LSAC) were analysed to examine maternal characteristics during pregnancy and their association with childhood obesity across waves 2 to 8 (ages 2–15). Latent class analysis (LCA) was employed to identify distinct clusters of maternal health, lifestyle, and dietary factors as exposure variables. Associations between these clusters and childhood obesity, defined using the U.S. Centers for Disease Control and Prevention (CDC) BMI $\geq$ 95th percentile, were assessed using Chi-square tests and multinomial logistic regression were employed to examine the identified clusters impact on childhood obesity by adjusting maternal factors (employment, education, income,) and child-specific factors (physical activity, diet, energy drink consumption).

Results Five clusters emerged: (1) Health Issues with High Mental Health, Medical Needs, and Substance Use, (2) Healthiest Profile with Minimal Dietary Exclusions and Low Medical Risks, (3) Moderate Health Risks with High Smoking Prevalence, (4) Nutritional Exclusions and High Incidence of Other Birth Types, and (5) Severe Health Risks with High Obesity and Medical Dependency. Cluster 5 exhibited the highest risk of childhood obesity, followed by Clusters 3, 4, and 1. Cluster 2 consistently showed the lowest obesity risk. Socioeconomic and child factors mediated the obesity risks in Clusters 1 and 4, with risks persisting for Cluster 3, particularly in later childhood.

Conclusion This study highlights the utility of LCA in identifying maternal factors influencing childhood obesity and underscores the importance of promoting maternal health, lifestyle, and dietary improvements to mitigate obesity risks in children. Targeted interventions addressing high-risk maternal profiles could be instrumental in reducing childhood obesity prevalence.

Keywords LSAC, Childhood obesity, LCA, Clustering, Multinomial regression model

*Correspondence:

Nasrin Begum
nasrin.begum@unisq.edu.au
Enamul Kabir
Enamul.Kabir@unisq.edu.au

Full list of author information is available at the end of the article



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

Background

Childhood obesity has emerged as a significant public health challenge, with its prevalence rising globally [1]. Obesity is characterized by an excessive accumulation of body fat, presenting a risk to health. It is defined as a body mass index (BMI) at or above the 95th percentile, whereas childhood overweight is defined as a BMI between the 85th and 95th percentiles for children of the same age and sex [2]. In Australia, childhood obesity constitutes a growing epidemic, identified as the second leading risk factor contributing to 8.4% of the national disease burden and the top risk factor for non-fatal diseases in 2018 [3, 4]. This crisis not only jeopardizes the health and well-being of affected individuals but also significantly increases healthcare costs [4]. The causes of childhood obesity are multifaceted, resulting from a combination of biological, genetic, maternal, child, and environmental factors, all influenced by demographic, lifestyle, socioeconomic, and regional disparities [4].

Children Behavioural factors such as reducing physical activity [5], excessive screen time [6], and poor feeding practices, such as going to bed with a bottle are causes of childhood obesity [7]. Besides this, maternal factors-encompassing demographic characteristics (education, ethnicity), lifestyle behaviours (smoking, alcohol consumption, physical activity) and health conditions (diabetes, asthma, anxiety)- are strongly associated with childhood obesity risk [8–10]. Maternal pre-pregnancy BMI, gestational weight gain, and gestational diabetes have been consistently linked to childhood obesity. For instance, maternal obesity pre-pregnancy increased the odds of childhood obesity by 3.64 times, while maternal overweight raised the odds by 1.89 times, compared to mothers with normal BMI [11]. Other studies highlight that gestational hypertension, maternal depression, and early pregnancy obesity, particularly in cases of high birth weight or large-for-gestational-age infants, further elevate the risk of childhood and adolescent obesity [12, 13].

Socioeconomic factors significantly influence maternal health behaviours and consequently, childhood obesity risk. Maternal lifestyle and health behaviours are pivotal to childhood obesity risk, yet these behaviours are often shaped by broader family factors, such as socioeconomic status. For instance, lower-income families may have limited access to nutritious foods and safe physical environments, which can restrict maternal health behaviours and, in turn, impact children's obesity risk [14]. Thus Considering family socioeconomic context provides a more comprehensive understanding of the factors influencing maternal and child health outcomes. Moreover, children from low-income

families are disproportionately at risk of developing obesity compared to their higher-income counterparts [15]. Parental age also plays a role, with both younger and older mothers being linked to higher childhood obesity prevalence [16].

Maternal lifestyle behaviours during pregnancy, including smoking and dietary habits, are critical. Smoking during pregnancy doubles the likelihood of childhood obesity [17]. These maternal lifestyle factors had also contributed to being obesity in adolescent [18]. Maternal dietary exclusions also have been associated with increased obesity risk in children. A recent study reported that the exclusion of fish and eggs during pregnancy was specifically associated with an increased risk of mild to moderate obesity in children at ages 6–7 years [19]. These findings align with the Developmental Origins of Health and Disease (DOHaD) and Fetal Origins Hypothesis, which posit that maternal health behaviours during pregnancy influence fetal growth trajectories and metabolic programming through mechanisms like hormonal dysregulation, fetal overnutrition, and epigenetic modifications [20, 21].

Although substantial research has examined individual maternal factors such as smoking, alcohol consumption and chronic conditions, most studies analyse these factors separately, rather than in combination. While cluster analyses are more commonly applied to child behaviours, as for example, prior research on Australian adolescents identified clusters of behaviours such as physical activity, diet, and sedentary habits, and their associations with obesity, self-rated health, and quality of life [22]. But limited studies explore clustering of maternal health and lifestyle characteristics, particularly in the context of childhood obesity [8, 23–26]. So, there is a research gap. This gap highlights the need for a comprehensive understanding of maternal health-related clusters and their cumulative impact on childhood obesity risk.

To our knowledge, no studies have explored the use of cluster analysis to examine maternal characteristics during pregnancy and their association with offspring obesity. These characteristics include BMI, birth method and long-term health conditions (diabetes, asthma and hypertension), mental health conditions (anxiety and stress), socio-demographic factors (age and ethnicity), dietary factors (exclusion of certain foods) and lifestyle factors (smoking and alcohol consumption). This study aims to identify cluster patterns based on maternal health, lifestyle, dietary, and socio-demographic factors, and to assess their association with childhood obesity, measured by BMI percentile. It also examines the longitudinal impact of these patterns on obesity in children aged 2 to 15 years.

Materials and methods

Data source and study setting

In this study, we focused on specific variables directly related to maternal health, lifestyle, and dietary factors to precisely assess their unique impact on childhood obesity outcomes. Maternal diet [27] and prenatal care were included for their roles in shaping fetal development and potential obesity risk. Data were used from the longitudinal Study of Australian Children (LSAC). The LSAC is a nationwide household survey in Australia that began in 2004. It gathers data on the health and developmental progress of Australian children and adolescents [28]. In the LSAC study, the respondents were selected using a multistage stratified cluster sampling technique. The household is a primary sampling unit, and data was collected from children and their corresponding mother's. Details regarding the LSAC survey design and methodology are available elsewhere [28]. This survey has two cohorts: Birth cohort (born in March 2003–February 2004) and Kindergarten cohort (born in 1999–February 2000). However, this study used B/cohort (Birth cohort who were born in March 2003–February 2004) that means the children were 0–1 years in wave 1. The data were collected biennially, meaning the children were 2–3 years old in wave 2, 4–5 years old in wave 3 and so on. Maternal characteristics during pregnancy in wave 1 were collected based on various factors such as BMI, birth method, long-term health related factors (diabetics, hypertension and asthma), mental depression (stress, anxiety), demographic factors (age, ethnicity), dietary factors (exclusion of food) lifestyle factors of mother (alcohol consumption, smoking). The study focused on measuring obesity in children aged 2–15. In wave 1, the children were under 2 years old, so this age group was excluded from the study, as measuring obesity based on BMI at this stage is not considered meaningful for this cohort. Most of the relevant variables for this study were unavailable in wave 9 due to data collection being impacted by COVID-19. As a result, the study focused on data from waves 2 to 8. The ages of children in each wave were as follows: wave 2 (2–3 years), wave 3 (4–5 years), wave 4 (6–7 years), wave 5 (8–9 years), wave 6 (10–11 years), wave 7 (12–13 years) and wave 8 (14–15 years). The investigation focused on exploring the association between childhood obesity at different ages and the maternal factors during pregnancy measured in wave 1.

Study participants

The study aimed to determine the appropriate number of clusters and examine their potential associations with obesity using LSAC data. Initially, there were 5107 pregnant mothers in wave 1 of the B cohort. However, the key

maternal indicator, BMI, which was self-reported in the LSAC data and available for only 4,060 mothers. Consequently, the study reduced the sample to 4060 pregnant mothers aged 15–49 years whose data were available in the LSAC from the B birth cohort (March 2003–February 2004). Data from approximately 20% (1/5 of the sample) of mothers for whom BMI information was unavailable were omitted from the final analysis to ensure the accuracy and relevance of the findings.

Ethics approval and consent to participate

This study used secondary data from the LSAC survey dataset, which received ethical approval from the Australian Institute of Family Studies Ethics Committee. Written informed consent was obtained from all adolescents and/or their legal guardians by the LSAC authorities. The de-identified unit record dataset was provided to us at the University of Southern Queensland for the purposes of this doctoral research. To access the data, we completed and signed the Confidentiality Deed Poll, which was submitted to both NCLD (ncldresearch@dss.gov.au) and ADA (ada@anu.edu.au). All procedures in this study were followed in accordance with the relevant guidelines and regulations.

Independent variables

The study selected maternal characteristics during pregnancy based on existing studies [9–11, 13, 17, 29]. In our study, we have selected indigenous' variable instead of ethnicity variable because indigenous Australian people face higher rates of chronic conditions, including obesity compared to non-indigenous Australian people due to range of historical, social, and economic factors that have influenced health outcomes [30]. A detailed list of the selected maternal characteristics, question types and their categorizations are described in Table 1.

Obesity based outcome variable

In this study, obesity was the key outcome variable, measured using BMI percentiles from the U.S. Centers for Disease Control and Prevention (CDC). BMI percentile data from the LSAC dataset were used, and interviewers measured respondents' weight following standard protocols [31]. LSAC measured children BMI as per the CDC definitions, where overweight was defined as BMI-for-age $\geq$ 85th and $<$ 95th percentile, and obesity as $\geq$ 95th percentile. The study used the CDC cutoffs because they align with the LSAC dataset's methodology and are widely used in similar population health studies. The LSAC team categorized BMI scores as follows: underweight III; underweight II; underweight I; normal weight; overweight; and obesity. Following that, the three underweight categories were merged into one to simplify the

Table 1 List of the selected maternal characteristics during pregnancy, question types and their categorizations

Factors	Characteristics	Questions	Categorization
Health	BMI	Categorical representation of BMI	1 = Underweight; 2 = Normal Weight; 3 = Overweight; and 4 = Obesity
	Diabetes	During this pregnancy, did you have diabetes?	1 = Yes and 2 = No
	High blood pressure	During this pregnancy, did you have high blood pressure needing treatment (admission to hospital or medication)?	1 = Yes and 2 = No
	Other physical problem	During this pregnancy, did you have other physical health problems?	1 = Yes and 2 = No
	Mental health problem	During this pregnancy, did you have problems with stress, anxiety or depression?	1 = Yes and 2 = No
	Use any medical conditions	Does Mother have any medical conditions or disabilities that have lasted, or are likely to last, for six months or more?	1 = Yes and 2 = No
	Use asthma medication 'over-the-counter' prescribed	What 'over-the-counter' medications were used? Asthma medications (Ventolin etc.)	1 = Yes and 0 = No
		What prescribed medicines or tablets were taken for asthma?	1 = Yes and 0 = No
	Combined		1 = Yes and 0 = No
	Use diabetics medication	What prescribed medicines or tablets were taken for diabetes?	1 = Yes and 0 = No
	Use blood pressure tablets	What prescribed medicines or tablets were taken? Blood pressure tablets	1 = Yes and 0 = No
Dietary	Anti-depressants	What prescribed medicines or tablets were taken? Anti-depressants	1 = Yes and 0 = No
	Exclude meat consumption	did (you/child's mother) not eat any of the following: (Include foods excluded permanently, regardless of pregnancy, as well as foods excluded specifically during pregnancy) Meat?	1 = Yes and 0 = No
	Exclude fish	did (you/child's mother) not eat any of the following: (Include foods excluded permanently, regardless of pregnancy, as well as foods excluded specifically during pregnancy) Fish?	1 = Yes and 0 = No
	Exclude dairy foods consumption	did (you/child's mother) not eat any of the following: (Include foods excluded permanently, regardless of pregnancy, as well as foods excluded specifically during pregnancy) Dairy foods (milk, cheese, yoghurt etc.)?	1 = Yes and 0 = No
	Exclude eggs	During the pregnancy with child, did (you/child's mother) not eat any of the following: (Include foods excluded permanently, regardless of pregnancy, as well as foods excluded specifically during pregnancy) Eggs?	1 = Yes and 0 = No
Healthcare	Medical visits before birth	About how many medical visits or check-ups did (you/child's mother) have in total before child was born? (That is, during the pregnancy)	1 = 1–3 Visits; 2 = 4–6 visits; 3 = 7–9 visits; and 4 = 10 or more visits
	Revived medical care	Who did (you/child's mother) mainly go to for medical care during the pregnancy?	1 = General practitioner GP; 2 = Obstetrician; 3 = Midwife or nurse; 4 = Formal shared care arrangement; and 5 = Other
Sociodemographic	Indigenous	Is Mother of Aboriginal or Torres Strait Islander origin?	1 = Indigenous and 0 = Non-indigenous
	Maternal age	What was Mother's age last birthday?	1 = 15–19 years; 2 = 20–35 years; and 3 = 36–45 years, 4 = 46 + years
	Birth type	What type of birth, or delivery, was it?	0 = Normal; 1 = Caesarean and 3 = Others

Table 1 (continued)

Factors	Characteristics	Questions	Categorization
Lifestyle	Alcohol	During the pregnancy, did you smoke alcohol?	1 = Yes and 0 = No
	Smoking Status	During the pregnancy, did you smoke cigarettes?	1 = Yes and 0 = No

analysis and focus on the broader objective of examining maternal and socio-demographic factors influencing childhood obesity. This approach reduces complexity and allows for a more robust analysis of the key factors affecting weight status across the spectrum. Finally, this study categorized BMI as follows: Underweight: Less than the 5th percentile; Normal Weight: 5th percentile to less than the 85th percentile; Overweight: above the 85th percentile but below the 95th percentile; Obesity: 95th percentile or greater.

Statistical analysis

This study performed all statistical analyses using STATA (version 17). Data are expressed as frequency (%) for categorical variables. Moreover, the bivariate analysis is also performed to determine the distribution of maternal characteristics and assess their associations with obesity. To perform this association, we implemented a chi-square test. LCA was implemented to make cluster patterns based on the characteristics of maternal BMI, birth method, long-term health, dietary, health care and sociodemographic factors. Furthermore, multinomial logistic regression model was used to investigate the associations between identified clusters and obesity in offspring at the children aged from 2 to 15 years and p -value < 0.05 determines the statistical significance.

Latent class analysis

Latent class analysis is a subset of the structural equation model that can easily handle large datasets and categorical variables to determine subgroups from a set of variables. These subgroups are known as “latent classes”, “class memberships”, or “clusters”. Nowadays, LCA is widely used in various domains [32–34]. In fact, as clusters are defined primarily by using the Latent Class Analysis and it's an unattended automated process, we get automatically generated mutually exclusive clusters with different sizes. The selected classes should have enough observations to provide a representative class of a population [35]. In practice, clusters with a size of less than 5% will not be retained, as indicated in similar studies [36]. This study also employed LCA-based model to determine the cluster patterns (classes) based on maternal health, lifestyle, dietary and sociodemographic based factors. This function provides various statistical evaluation

parameters such as log likelihood, degrees of freedom (df), Akaike information criterion (AIC), Bayesian information criterion (BIC), and likelihood ratio [χ^2]. These parameters were used to determine the optimal clusters. Usually, the purpose of this analysis is to determine the models that minimize BIC, χ^2 and maximize the likelihood. We estimated the LCA based models for one to seven clusters and the model is repeated five to ten times for each cluster. To increase precision, this study repeated the analyses into four times, starting with a two-class model and gradually adding subgroups.

Regression modelling

After identifying clusters, we performed a bivariate analysis to investigate the relationship between childhood obesity and clusters. Since the childhood weight status (outcome variable) had more than two groups, multinomial logistic regression (MLR) model was used to show how the cluster patterns influenced on childhood obesity over different waves. In this study, we performed MLR based model from two viewpoints. One was without adjusting any covariates and another was with adjusting for family income, mother's education and mother's employment status and additionally, as children age and progress through different developmental stages, other factors, such as diet and physical activity, become increasingly significant in the development of obesity. To reflect these variations, the study incorporated child-specific variables where data were available in the LSAC dataset. For Waves 2, 3, 4, and 5, the analysis controlled for child physical activity, maternal education, maternal employment, and weekly family income. For Waves 6 and 7, the model included child physical activity, diet, maternal education, maternal employment, and weekly family income. In Wave 8, additional variables such as energy drink consumption were also included alongside child physical activity, diet, maternal education, maternal employment, and weekly family income. Adjusting for these covariates helps us to understanding the independent effect of cluster patterns on the childhood obesity. Moreover, this approach provides a comprehensive framework for evaluating the impact of cluster patterns on obesity across different waves while controlling these covariates.

Results

Baseline characteristics

(Comparative analysis of baseline characteristic between mothers with and without reported BMI data).

In the LSAC data, approximately 20% of mothers were not reported their BMI. To understand the influence of this missing people within the context of our results, we conducted a comparative analysis of baseline characteristics between mothers with and without reported BMI data (see additional Table 1). This analysis revealed significant differences between two groups. Mothers without BMI data were less likely report to adverse health conditions, such as diabetes, high blood pressure, and mental health problems and exhibited health behaviour, including lower smoking and alcohol consumption rates. Additionally, sociodemographic differences, such as a higher proportion of indigenous and younger maternal age, were observed in the excluded group. These findings provide valuable context regarding the potential influence of missing BMI data.

An overview of maternal characteristics stratified by children's BMI categories (underweight, normal weight, overweight, and obesity) in Wave 2:

Table 2 presents the distribution of maternal characteristics across children's BMI categories, highlighting the prevalence and association of maternal health, behaviour, dietary factors, healthcare access, and sociodemographic variables. Statistical significance for differences between BMI groups is indicated by p -values (<0.05).

Maternal BMI significantly varied across child BMI categories ($p<0.001$), with the prevalence of maternal obesity notably higher among children in the obesity group (35.2%) compared to other categories. Mothers of underweight children were more likely to be underweight themselves (20.0%). High blood pressure was significantly associated with higher child BMI ($p<0.001$), with 17.6% of mothers of obesity children reporting high blood pressure, compared to 5.0% in the underweight category. Mental health issues also displayed a significant association ($p=0.004$), being more prevalent among mothers of obesity children (25.8%). Maternal smoking and alcohol consumption demonstrated significant associations with child BMI. Smoking was most common among mothers of obesity children (25.8%; $p=0.001$). In contrast, alcohol consumption was less frequent among mothers of obesity children (28.9%) compared to those in other BMI categories ($p=0.027$). No significant associations were observed between maternal dietary exclusions (meat, fish, dairy, or eggs) and child BMI categories. However, some minor trends, such as higher exclusion of eggs among mothers of obesity children (7.6%), warrant further exploration. The number of medical visits before childbirth was marginally associated with child BMI ($p=0.050$). Mothers of

obesity children were slightly more likely to report fewer than four visits (3.8%). Healthcare provider type showed no significant differences across groups, although mothers of obesity children were more likely to report receiving care from general practitioners (34.0%) and less likely from obstetricians (38.4%). No significant association was found between maternal age or Indigenous status and child BMI categories. Most mothers fell within the 20–35 age group across all BMI categories. Birth type was also not significantly associated, with normal deliveries being the most common across all groups.

Cluster analysis and their associated characteristics

Model selection using LCA

The model fit statistics using LCA model is presented in Table 3. The cluster results showed that class-five yielded the lowest value of BIC, lower value of AIC, likelihood ratio (χ^2) and log-likelihood values compared to other class models. Therefore, the class-five model was chosen as better model which were used for further analysis.

Characteristics of the selected factors by cluster-wise

The distribution of selected health, lifestyle, and pregnancy-related factors across five identified clusters of pregnant women in Australia (see additional Table 1). These clusters were categorized based on the severity of health risks and lifestyle factors observed during pregnancy. This study found that there were 12.2% of women belongs to cluster 1, 55.0% of women in cluster 2, 20.3% of women in cluster 3, 6% of women in cluster 4, and the 6.2% of women belonged to cluster 5, respectively. This study also found that the cluster 1 had the highest levels of obesity, substance use, and mental health issues, healthcare needs, with the greatest number of medical visits and substantial use of asthma medications. Whereas the cluster 2 had the lowest obesity rates, minimal health complications, and low levels of substance use as well as the most favourable health profile. The cluster 3 was characterized by moderate obesity and underweight, with a high prevalence of smoking and mental health issues. Moreover, this group's diverse health profile points to the need for targeted smoking cessation programs and mental health support for pregnant women at moderate risk. The highest dietary exclusions and diverse birth outcomes, the proportion of "other" birth types and high dietary restrictions were found in cluster 4. This indicates the need for comprehensive nutritional guidance and support for women with unique dietary needs and complex birth experiences. On the other hand, the cluster 5 is marked by severe health issues, including the highest obesity rates, medication use, healthcare needs substantial diabetes, and high blood pressure. This cluster highlights the critical importance of early medical

Table 2 An overview of maternal characteristics stratified by children's BMI categories (underweight, normal weight, overweight, and obesity) in Wave 2

Maternal Factors	Maternal Characteristics	Children's BMI categories				P value
		Underweight	Normal weight	Overweight	Obesity	
Health related	BMI					
	Underweight	40(20.0)	336(12.4)	46(6.9)	11(6.9)	
	Normal weight	91(45.5)	1296(47.8)	249(37.1)	49(30.8)	< 0.001
	Overweight	40(20.0)	658(24.3)	210(31.3)	43(27.0)	
	Obesity	29(14.5)	423(15.6)	167(24.9)	56(35.2)	
	Diabetes					
	No	190(95.0)	2581(95.1)	631(93.9)	145(91.2)	0.120
	Yes	10(5.0)	132(4.9)	41(6.1)	14(8.8)	
	High blood pressure					
	No	190(95.0)	2520(92.9)	619(92.1)	131(82.4)	< 0.001
	Yes	10(5.0)	193(7.1)	53(7.9)	28(17.6)	
	Other physical problem					
	No	165(82.5)	2169(8.0)	523(77.8)	129(81.1)	0.435
	Yes	35(17.5)	544(20.1)	149(22.2)	30(18.9)	
	Mental health problem					
	No	170(85.0)	2263(83.4)	537(79.9)	118(74.2)	0.004
	Yes	30(15.0)	450(16.6)	135(20.1)	41(25.8)	
	Use any medical condition/s					
	No	164(82.0)	2058(75.9)	517(76.9)	113(71.1)	0.094
	Yes	36(18.0)	655(24.1)	155(23.1)	46(28.9)	
	Use asthma medication					
	No	190(95.0)	2524(93.0)	627(93.3)	149(93.7)	0.748
	Yes	10(5.0)	189(7.0)	45(6.7)	10(6.3)	
	Use diabetics medication					
	No	199(99.5)	2680(98.8)	662(98.5)	155(97.5)	0.362
	Yes	1(0.5)	33(1.2)	10(1.5)	4(2.5)	
	Use blood pressure tablets					
	No	199(99.5)	2658(98.0)	659(98.1)	153(96.2)	0.182
	Yes	1(0.5)	55(2.0)	13(1.9)	6(3.8)	
	Anti-depressants					
	No	195(97.5)	2653(97.8)	658(97.9)	157(98.7)	0.856
	Yes	5(2.5)	60(2.2)	14(2.1)	2(1.3)	
Behaviour related	Smoking					
	No	176(88.0)	2323(85.6)	563(83.8)	118(74.2)	0.001
	Yes	24(12.0)	390(14.4)	109(16.2)	41(25.8)	
	Alcohol consumption					
	No	129(64.5)	1624(59.9)	406(60.4)	113(71.1)	0.027
	Yes	71(35.5)	1089(40.1)	266(39.6)	46(28.9)	

Table 2 (continued)

Maternal Factors	Maternal Characteristics	Children's BMI categories				P value
		Underweight	Normal weight	Overweight	Obesity	
Dietary related	Exclude meat					
	No	184(92.0)	2506(92.4)	626(93.2)	144(90.6)	0.716
	Yes	16(8.0)	207(7.6)	46(6.9)	15(9.4)	
	Exclude Fish					
	No	176(88.0)	2419(89.2)	603(89.7)	141(88.7)	0.909
	Yes	24(12.0)	294(10.8)	69(10.3)	18(11.3)	
	Exclude Dairy foods					
	No	188(94.0)	2553(94.1)	636(94.6)	152(95.6)	0.836
	Yes	12(6.0)	160(5.9)	36(5.4)	7(4.4)	
	Exclude Eggs					
	No	191(95.5)	2611(96.2)	639(95.1)	147(92.5)	0.083
	Yes	9(4.5)	102(3.8)	33(4.9)	12(7.6)	
Health care related	Medical visits before birth					
	3 visits	3(1.5)	24(0.9)	12(1.8)	6(3.8)	0.050
	4–6 visits	12(6.0)	188(6.9)	45(6.7)	10(6.3)	
	4–6 visits	49(24.5)	571(21.1)	158(23.5)	32(20.1)	
	10 or more visits	136(68.0)	1930(71.1)	457(68.0)	111(69.8)	
	Received medical care					
	GP	63(31.5)	728(26.8)	208(31.0)	54(34.0)	0.177
	Obstetrician	88(44.0)	1289(47.5)	283(42.1)	61(38.4)	
	Midwife or nurse	32(16.0)	478(17.6)	133(19.8)	30(18.9)	
	Formal shared care	12(6.0)	176(6.5)	36(5.4)	11(6.9)	
	Other	5(2.5)	42(1.6)	12(1.8)	3(1.9)	
Sociodemogr-aphic	Indigenous					
	Non-indigenous	196(98.0)	2663(98.2)	658(97.9)	155(97.5)	0.922
	Indigenous	4(2.0)	50(1.8)	14(2.1)	72(1.9)	
	Maternal age					
	15–19 Years	5(2.5)	37(1.4)	8(1.2)	4(2.5)	0.383
	20–35 Years	145(72.5)	2105(77.6)	520(77.4)	127(79.9)	
	36–45 Years	50(25.0)	571(21.1)	144(21.4)	28(17.6)	
	Birth type					
	Normal	128(64.0)	1715(63.2)	399(59.4)	102(64.2)	0.694
	Caesarean	58(29.0)	805(29.7)	221(32.9)	45(28.3)	
	Others	14(7.0)	193(7.1)	52(7.7)	12(7.6)	

interventions to manage obesity, diabetes, and hypertension, which could significantly improve maternal and child health outcomes. The findings emphasized that the importance of personalized care strategies that account for these distinct profiles, particularly focusing on high-risk clusters like Cluster 1 and Cluster 5 that required intensive support and intervention to improve maternal and fetal outcomes.

Prevalence of childhood weight status across different clusters over time

Table 4 outlines the distribution of weight status (underweight, normal weight, overweight, and obesity) based on BMI among children across five clusters from Wave 2 to Wave 8. The data reveals significant differences in

Table 3 Model fit statistics using latent class analysis model

Model	N	Log likelihood	df	AIC	BIC	Likelihood ratio (X^2)
One class	4,060	-39,256.6	32	78,577.2	78,779.0	18,700.0
Two class	4,060	-38,723.5	65	77,577.0	77,987.1	17,633.8
Three class	4,060	-38,429.4	98	77,054.7	77,673.0	17,045.5
Four class	4,060	-38,313.3	127	76,880.6	77,681.8	16,813.3
Five class	4,060	-37,996.0	163	76,318.0	77,346.4	16,178.8
Six class	4,060	-37,883.0	197	76,160.0	77,402.9	15,952.8
Seven class	4,060	-37,834.7	223	76,115.3	77,522.2	15,856.1

AIC Akaike's information criterion, BIC Bayesian information criterion, df Degrees of freedom, X^2 Chi-square goodness of fit, N Number of participants

Table 4 Distribution of childhood weight status by cluster across waves 2–8

Outcome variable	Cluster 1	Cluster 2	Cluster 3	Cluster 4	Cluster 5	P-value*
Wave 2 (n = 3744)	n (%)	n (%)	n (%)	n (%)	n (%)	
Underweight	20(4.4)	112(5.3)	47(6.4)	14(6.3)	7(3.0)	< 0.001
Normal weight	336(73.7)	1560(74.3)	492(67.3)	165(74.0)	160(68.4)	
Overweight	77(16.9)	366(17.4)	146(20.0)	36(16.1)	47(20.1)	
Obesity	23(5.0)	62(3.0)	46(6.3)	8(3.6)	20(8.6)	
Wave 3 (n = 3625)						
Underweight	30(6.8)	123(6.0)	52(7.4)	10(4.5)	11(4.9)	0.017
Normal weight	315(71.3)	1482(72.7)	479(68.5)	164(73.9)	144(64.3)	
Overweight	76(17.2)	349(17.1)	127(18.2)	34(15.3)	47(21.0)	
Obesity	21(4.8)	84(4.1)	41(5.9)	14(6.3)	22(9.8)	
Wave 4 (n = 3515)						
Underweight	19(4.4)	98(4.9)	39(5.9)	10(4.8)	9(4.1)	0.001
Normal weight	323(74.4)	1561(78.4)	471(71.4)	154(74.0)	155(70.1)	
Overweight	66(15.2)	258(13.0)	100(15.2)	32(15.4)	37(16.7)	
Obesity	26(6.0)	75(3.8)	50(7.6)	12(5.8)	20(9.1)	
Wave 5 (n = 3378)						
Underweight	15(3.6)	107(5.5)	26(4.2)	6(2.9)	11(5.2)	< 0.001
Normal weight	300(72.6)	1464(75.8)	428(69.6)	151(73.0)	130(61.6)	
Overweight	73(17.7)	281(14.5)	106(17.2)	37(17.9)	42(19.9)	
Obesity	73(17.7)	281(14.5)	106(17.2)	37(17.9)	28(13.2)	
Wave 6 (n = 3057)						
Underweight	17(4.5)	138(7.8)	29(5.2)	8(4.5)	12(6.5)	< 0.001
Normal weight	266(70.0)	1275(72.3)	359(64.9)	124(70.1)	110(59.8)	
Overweight	77(20.3)	285(16.2)	118(21.3)	33(18.6)	39(21.2)	
Obesity	20(5.3)	65(3.7)	47(8.5)	12(6.8)	23(12.5)	
Wave 7 (n = 2727)						
Underweight	14(4.1)	117(7.3)	34(7.4)	11(7.0)	8(4.8)	< 0.001
Normal weight	240(69.8)	1155(72.2)	286(62.3)	107(67.7)	92(55.4)	
Overweight	67(19.5)	270(16.9)	107(23.3)	30(19.0)	44(26.5)	
Obesity	23(6.7)	58(3.6)	32(7.0)	10(6.3)	22(13.3)	
Wave 8 (n = 2520)						
Underweight	14(4.5)	88(6.0)	30(7.0)	10(6.5)	4(2.7)	< 0.001
Normal weight	201(64.2)	1057(71.7)	245(57.1)	96(62.8)	91(60.7)	
Overweight	67(21.4)	265(18.0)	108(25.2)	34(22.2)	35(23.3)	
Obesity	31(10.0)	65(4.4)	46(10.7)	13(8.5)	20(6.9)	

* p-value is obtained from Chi-Square test

the prevalence of these weight categories across clusters, demonstrating variations in health outcomes and risks.

Cluster 5 exhibits the highest levels of vulnerability regarding weight status across the observed waves. This cluster consistently reports the highest obesity rates, reaching 13.2% in Wave 7, and substantial proportions of overweight children, peaking at 26.5% in the same wave. The persistently high rates of obesity and overweight in Cluster 5 highlight it as the most health-compromised group, facing severe challenges related to excess weight. Additionally, Cluster 5 shows the lowest proportion of normal weight children, underscoring its pronounced health risks.

Cluster 3, while not as extreme as Cluster 5, still demonstrates significant vulnerabilities, particularly with underweight children. It has the highest rates of underweight children across most waves, such as 7.4% in Wave 3 and 7.4% in Wave 7. This cluster also faces notable concerns with overweight (21.34% in wave 6) and obesity (10.7% in wave 8), although not to the same extent as Cluster 5. The underweight prevalence in Cluster 3 suggests it is at significant risk for weight-related health issues, though less severe compared to Cluster 5.

Cluster 2 is the most favourable in terms of weight status outcomes. It consistently has the highest proportions of children with normal weight, reaching 78.4% in Wave 4 and maintaining high percentages across other waves. Cluster 2 also shows the lowest rates of overweight, and obesity compared to other clusters, with its highest obesity rate at 14.5% in Wave 5. The low prevalence of overweight and obesity indicates that Cluster 2 has the most stable and healthiest weight distribution among the clusters.

Cluster 4 displays a moderate level of vulnerability in weight status distribution. Despite having lower rates of obesity and overweight compared to Clusters 3 and 5, it faces health challenges, particularly due to a higher proportion of dietary exclusions among mothers during pregnancy. Notably, while Cluster 4's obesity rate peaks at 17.9% in Wave 5 highest among clusters for that wave—the proportion of normal weight children is the highest at 73.9% in Wave 3. In comparison to Cluster 2, which has more favourable outcomes, Cluster 4 shows moderate rates of normal weight and lower proportions of obesity and overweight. Although its normal weight rates are steady, they do not reach the levels seen in Cluster 2, and its obesity rates are lower than those of the more vulnerable clusters.

Cluster 1, though less favourable than Cluster 2, shows reasonably balanced weight outcomes. It has moderate rates of normal weight children and does not exhibit the extreme levels of obesity seen in Cluster 5. However, its rates of overweight and obesity are higher than those in

Cluster 2, positioning it as less favourable but still healthier than the more vulnerable clusters.

Regression model

Table 5 presents the findings of multinomial logistic models that examined the association between the selected cluster membership and obesity across different waves of observations. This study investigated the influence of selected clusters on obesity across different waves from two perspectives: (1) without adjusting for these covariates. [11] adjusted for some covariates such as maternal factor (weekly family income, mother's education and mother's employment) and, child physical activity, diet and energy drink. The corresponding results are more clearly explained as follows: According to Table 5, Cluster 5 shows the highest vulnerability to obesity and overweight in both the unadjusted and adjusted models. In the unadjusted model, children in Cluster 5 have significantly higher odds of developing obesity across all waves, with odds ratios approximately four times greater in Wave 5 (OR: 3.9, 95% CI: 2.5–6.3) and Wave 6 (OR: 4.1, 95% CI: 2.5–6.9), and 4.8 times (OR: 4.8, 95% CI: 2.8–8.1) and 3.6 times (OR: 3.6, 95% CI: 2.1–6.2) higher in Waves 7 and 8, respectively, compared to children in Cluster 2. After adjusting for socio-economic factors and child factors, the odds ratios for Cluster 5 remain significant, particularly in Waves 5 through 8, when the children were aged 7–8 to 14–15 years though slightly lower. A similar trend was observed for obesity in this cluster, with an odds ratio of 2.7 (AOR: 2.7, 95% CI: 1.2–6.1) in Wave 8, when the children were aged 14–15 years. This indicates that Cluster 5's elevated vulnerability to obesity and overweight persists even after accounting for confounding variables.

Cluster 3 exhibits moderate vulnerability across both models. In the unadjusted model, this cluster has elevated rates of obesity across all waves and overweight in most waves compared to children in Cluster 2, with significant unadjusted odds ratios indicating higher risks. Specifically, children in Cluster 3 are 2.6 times (OR: 2.6, 95% CI: 1.7–3.8) and 3.1 times (OR: 3.1, 95% CI: 2.0–4.6) more likely to become obesity in waves 6 and 8, respectively, compared to those in Cluster 2. A similar trend was observed for overweight in Cluster 3. After adjustment, the only significant effect was in wave 8, when children were 14–15 years old, with children in this cluster being 2.4 times (AOR: 2.4, 95% CI: 1.3–4.8) more likely to become obesity compared to Cluster 2. The likelihood of overweight is also higher in this cluster, particularly as the children grow older in waves 7 (OR: 1.6, 95% CI: 1.0–2.4) and 8 (OR: 1.9, 95% CI: 1.3–2.9) at ages 12–13 and 14–15 years. This suggests that while Cluster 3 remains

Table 5 Associations between cluster membership and weight status across different waves using Multinomial logistic regression models

Waves	Unadjusted R: Normal weight		Adjusted Ref: Normal weight			
	Underweight	Overweight	Obesity	Underweight	Overweight	Obesity
Wave 2	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Cluster 2	RC	1.0(0.7–1.3)	1.7(1.1–2.8)*	0.5(0.3–1.2)	1.1(0.8–1.5)	1.3(0.7–2.6)
Cluster 1	0.8(0.5–1.4)					
Cluster 3	1.33(0.9–1.9)	1.3(1.0–1.6)*	2.4(1.6–3.5)**	1.1(0.6–1.9)	1.1(0.8–1.6)	1.6(0.9–3.1)
Cluster 4	1.18(0.7–2.1)	0.9(0.6–1.4)	1.2(0.6–2.6)	1.3(0.7–2.6)	1.1(0.7–1.8)	1.6(0.7–3.9)
Cluster 5	0.61(0.3–1.3)	1.3(0.9–1.8)	3.2(1.9–5.3)**	0.7(0.3–1.7)	1.0(0.6–1.6)	2.0(0.9–4.3)
Wave 3						
Cluster 2	RC					
Cluster 1	1.2(0.8–1.7)	1.0(0.77–1.35)	1.2(0.7–1.9)	1.0(0.5–1.8)	1.0(0.7–1.4)	0.8(0.4–1.7)
Cluster 3	1.3(0.9–1.8)	1.2(0.9–1.4)	1.5(1.0–2.2)*	1.1(0.6–1.9)	1.2(0.9–1.7)	0.8(0.4–1.6)
Cluster 4	0.7(0.4–1.4)	0.9(0.6–1.3)	1.5(0.8–2.7)	0.6(0.2–1.5)	0.9(0.5–1.5)	1.9(1.0–3.8)
Cluster 5	0.9(0.5–1.7)	1.4(1.0–2.0)	2.7(1.6–4.4)**	0.7(0.3–1.7)	1.1(0.7–1.8)	1.3(0.6–2.8)
Wave 4						
Cluster 2	RC					
Cluster 1	0.9(0.6–1.5)	1.2(0.9–1.7)	1.7(1.1–2.7)*	0.6(0.3–1.2)	1.2(0.8–1.8)	1.4(0.8–2.7)
Cluster 3	1.3(0.9–1.9)	1.3(1.0–1.7)	2.2(1.5–3.2)**	1.1(0.6–1.9)	1.3(0.8–1.9)	1.3(0.7–2.5)
Cluster 4	1.0(0.5–2.0)	1.3(0.8–1.9)	1.6(0.9–3.0)	1.5(0.7–3.0)	1.4(0.8–2.3)	2.5(1.2–5.1)*
Cluster 5	0.9(0.5–1.9)	1.4(1.0–2.1)	2.7(1.6–4.5)**	0.9(0.4–2.1)	1.0(0.6–1.8)	1.8(0.9–3.9)
Wave 5						
Cluster 2	RC					
Cluster 1	0.7(0.4–1.2)	1.27(0.10–1.7)	1.5(1.0–2.4)	0.4(0.2–1.0)	1.2(0.8–1.7)	1.2(0.6–2.4)
Cluster 3	0.8(0.5–1.3)	1.3(1.0–1.7)*	2.5(1.6–3.4)**	0.9(0.5–1.7)	1.2(0.8–1.8)	1.7(1.0–3.1)
Cluster 4	0.5(0.2–1.3)	1.2(0.9–1.9)	1.6(0.9–2.9)	0.7(0.3–1.7)	1.3(0.8–2.1)	2.3(1.1–4.7)*
Cluster 5	1.2(0.6–2.2)	1.7(1.2–2.4)**	3.9(2.5–6.3)**	1.1(0.5–2.6)	1.5(0.9–2.4)	2.9(1.5–5.4)**
Wave 6						
Cluster 2	RC					
Cluster 1	0.6(0.4–1.1)	1.3(1.0–1.7)	1.5(0.9–2.5)	0.3(0.1–0.8)	1.1(0.7–1.6)	1.3(0.6–2.3)
Cluster 3	0.8(0.5–1.1)	1.5(1.2–1.9)*	2.6(1.7–3.8)**	0.7(0.4–1.4)	1.4(0.9–2.0)	1.5(0.8–2.8)
Cluster 4	0.6(0.3–1.2)	1.2(0.8–1.8)	1.9(1.0–3.6)	0.9(0.4–1.9)	1.3(0.8–2.1)	1.9(0.8–4.1)
Cluster 5	1.0(0.5–1.9)	1.6(1.1–2.3)*	4.1(2.5–6.9)**	0.5(0.2–1.3)	1.1(0.6–1.8)	2.3(1.1–4.5)*
Wave 7						
Cluster 2	RC					
Cluster 1	0.6(0.3–1.0)	1.2(0.9–1.6)	1.9(1.2–3.2)*	0.3(0.1–0.8)	1.2(0.8–1.8)	1.5(0.7–3.1)
Cluster 3	1.2(0.8–1.8)	1.6(1.2–2.1)**	2.2(1.4–3.5)**	1.3(0.7–2.4)	1.6(1.0–2.4)*	1.2(0.5–2.4)
Cluster 4	1.0(0.5–1.9)	1.2(0.8–1.8)	1.9(0.9–3.7)	0.8(0.3–1.9)	1.1(0.7–2.0)	1.6(0.6–4.0)
Cluster 5	0.9(0.4–1.8)	2.1(1.4–3.0)**	4.8(2.8–8.1)**	0.4(0.1–1.4)	1.4(0.8–2.4)	2.8(1.3–6.3)*
Wave 8						
Cluster 2	RC					
Cluster 1	0.8(0.5–1.5)	1.3 (1.0–1.8)	2.5(1.6–3.9)**	0.8(0.4–1.8)	1.4(1.0–2.2)	1.6(0.8–3.4)
Cluster 3	1.47(1.0–2.3)	1.8(1.4–2.3)**	3.1(2.0–4.6)**	1.9(1.0–3.5)	1.9(1.3–2.9)**	2.4(1.3–4.8)**
Cluster 4	1.3(0.6–2.5)	1.4(0.9–2.1)	2.2(1.2–4.1)	1.2(0.5–3.0)	1.5(0.9–2.5)	2.7(1.2–6.1)*
Cluster 5	0.5 (0.2–1.5)	1.5 (1.0–2.3)*	3.6(2.1–6.2)**	0.4(0.1–1.8)	1.3(0.8–2.2)	2.4(1.1–5.1)*

RC Reference Category, OR Odds Ratio, AOR Adjusted Odds Ratio, CI Confidence Interval

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. Control variables "Maternal factors (weekly family income, mother's education and mother's employment) and Child factors (diet, energy drinks and physical activity)"

vulnerable, controlling for socio-economic and child factors slightly reduces the observed risks.

Cluster 4 shows moderate vulnerability to obesity only in the adjusted models, with no significant differences in overweight compared to Cluster 2 in both the adjusted and unadjusted models. After adjusting for family weekly income, mothers' education, and employment and child factors, children in this cluster were 2.5 times (AOR: 2.5, 95% CI: 1.2–5.1), 2.3 times (AOR: 2.3, 95% CI: 1.1–4.7) and 2.7 times (AOR: 2.7, 95% CI: 1.2–6.1) more likely to become obesity in waves 4, 5, and 8, when they were 6–7 years, 8–9 years and 14–15 years old, compared to children in Cluster 2. The adjusted model provides a more nuanced perspective, indicating that although the health outcomes in this cluster slightly improve after accounting for control variables, it still faces moderate challenges compared to Cluster 2.

Cluster 1 appears to be relatively better off compared to Clusters 3, 4, and 5 in respect of the obesity status of children. The likelihood of being overweight in this cluster is quite similar to that of Cluster 2 in both the unadjusted and adjusted models. In the adjusted model, the likelihood of being obesity for children in Cluster 1 is also comparable to Cluster 2. However, in the unadjusted model, the odds of obesity are higher in Waves 2, 4, 7, and 8 compared to Cluster 2. Specifically, the unadjusted model reveals that children in Cluster 1 were almost two to three times more likely to be obesity at ages 2–3 (OR: 1.7, 95% CI: 1.1–2.8), 6–7 (OR: 1.7, 95% CI: 1.1–2.7), 12–13 (OR: 1.9, 95% CI: 1.2–3.2) and 14–15 years (OR: 2.5, 95% CI: 1.6–3.9), respectively, compared to children in Cluster 2. This suggests that while the health outcomes in Cluster 1 are comparatively better in the adjusted model, it still faces some challenges relative to Cluster 2, particularly in the unadjusted model.

Discussions

This study provides novel insights into the maternal determinants of childhood obesity by identifying distinct clusters based on maternal health, lifestyle, dietary habits, and socio-demographic characteristics during pregnancy. The findings reinforce the pivotal role of maternal health factors in shaping long-term obesity risks for children and underscore the complexity of these interrelations.

The high prevalence of maternal obesity, gestational diabetes mellitus (GDM), hypertension, and mental health issues in Cluster 5 highlights a group with compounded vulnerabilities. The elevated childhood obesity rates in this cluster are consistent with prior studies linking maternal obesity and GDM to offspring obesity and metabolic risks, independent of other confounding factors [9, 13, 37, 38]. These findings suggest that tailored interventions targeting high-risk mothers during

pregnancy could mitigate intergenerational transmission of obesity.

The analysis of Cluster 3 reveals the detrimental impact of maternal smoking on childhood weight outcomes, aligning with evidence that in utero exposure to tobacco increases offspring's obesity risk [39]. Public health campaigns to reduce smoking during pregnancy could therefore significantly impact childhood obesity prevalence. Conversely, Cluster 1 offers an intriguing counterpoint: despite high maternal mental health issues, children displayed lower obesity risk. This deviation suggests potential protective factors, such as healthier dietary practices, that merit further investigation to inform nuanced intervention strategies.

Cluster 2 emerges as the most favourable group, characterized by healthier maternal profiles and lower childhood obesity rates. This aligns with studies demonstrating the protective effects of optimal maternal nutrition, particularly adequate protein intake during pregnancy, on reducing childhood obesity risks [19, 40]. These results emphasize the importance of promoting balanced maternal diets as a cornerstone of prenatal care.

The role of socio-economic and child factors as highlighted in the adjusted models, is critical. The reduced obesity risks in Cluster 4 after accounting for socio-economic and child disparities emphasize the mediating role of these variables. This suggests that improving socio-economic conditions, such as maternal education and access to resources, child factor (physical activity, diet) could buffer against adverse outcomes even in clusters with suboptimal maternal health profiles.

The findings also contribute to ongoing debates on maternal alcohol consumption. While some clusters with higher alcohol use did not show increased obesity risks, the lack of a consistent association warrants further investigation [41, 42]. These results highlight the need for nuanced guidelines that consider potential confounding factors when assessing alcohol's impact on offspring health.

The study's findings have critical implications for public health policy. Identifying high-risk maternal clusters provides a framework for targeted interventions aimed at reducing childhood obesity rates. Strategies such as early identification of at-risk mothers through antenatal screening, providing tailored nutritional and psychological support, and addressing socio-economic disparities can have a transformative impact. Moreover, incorporating cluster-specific insights into public health campaigns and prenatal care guidelines could enhance their efficacy.

Future studies should explore the interplay between maternal factors and contextual variables, such as environmental exposures and access to healthcare, to further elucidate the pathways linking maternal health to

childhood obesity. Longitudinal analyses with a focus on potential protective factors in low-risk clusters, such as dietary behaviours in Cluster 1, could provide actionable insights for designing preventive interventions.

Strengths and limitations

The strength of this study lies in its use of a large sample size from the LSAC data, which includes information on childhood obesity. It is the first study to identify cluster patterns based on maternal health, dietary habits, and socio-demographic behaviours during pregnancy using an advanced statistical Latent Class Analysis (LCA) approach. This study also examines their association with obesity. The advantage of using LCA is its ability to assess relationships among multiple maternal health, dietary, and socio-demographic factors simultaneously, as well as to analyse various combinations of unobserved data patterns before forming clusters [43]. Our study has several limitations. Maternal health, dietary, and socio-demographic behaviour patterns were self-reported by mothers during pregnancy. Self-reporting can introduce recall bias and impact data accuracy, as mothers may not remember or report certain behaviours accurately, particularly for behaviours that took place in early pregnancy or before conceiving. Retrospective data limits the study's ability to capture real-time behaviour changes and could influence the validity of the identified clusters. An additional limitation of our study is the missing BMI data for approximately 20% of mothers, which prevented these individuals from being included in the cluster analysis focused on BMI-related characteristics. While we provided a comparative analysis of baseline characteristics, the exclusion of this group may introduce potential biases that could influence the generalizability of our findings. The LCA based approach was used to form clusters based on maternal characteristics. Although LCA is advantageous in identifying unobserved subgroups, it involves probabilistic classification, meaning that individuals are assigned to classes based on probabilities rather than definitive groupings. As a result, there is a potential for misclassification, where some individuals might be assigned to clusters that do not fully represent their actual behaviours. Moreover, to determine the optimal number of clusters depends on the selection criteria, including BIC, AIC, likelihood ratio, and log-likelihood values. Since these metrics offer statistical guidance rather than absolute criteria, slightly different choices in model selection could lead to alternative clustering outcomes, affecting the study's reproducibility. Additionally, the properties of the identified clusters are inherently complex, with clusters being named according to the most prominent indicators. This naming may oversimplify the nuanced patterns within each cluster and does

not account for the dynamic interplay among less prominent factors. Future studies could benefit from methods that further explore the interactions between maternal health, dietary, and socio-demographic characteristics, allowing for a more refined understanding of how each factor uniquely contributes to childhood obesity. Parental, Biological and Genetics factor that might play an important role but that were not considered although the paper focusses on maternal factors, paternal factors might also be important.

We also acknowledge the significant impact of attrition and reduced sample sizes across waves on our study. While imputation methods were not applied in the current analysis, we have discussed their potential in future research to mitigate attrition bias. Characteristics of dropouts compared to retained participants suggest possible selection bias, which may affect generalizability. Additionally, small sample sizes in certain categories likely reduced the statistical power of regression models, limiting precision and reliability. Finally, comparisons across waves are exploratory, given differing sample compositions, and should be interpreted cautiously.

Conclusion

This study identified five distinct clusters of mothers based on maternal health, lifestyle, dietary habits, and socio-demographic characteristics, and their association with childhood and adolescent obesity. Cluster 5 was identified as the most vulnerable with highest odds of overweight and obesity in children, while Cluster 2 was revealed as the most favourable with high normal weight rates and lower obesity levels. The notable influence of specific groups on obesity across different waves can help governmental and non-governmental agencies, as well as practitioners, in distinguishing vulnerable populations from more privileged ones when formulating health-related policies and strategies. These findings have significant health implications and may enable policymakers to identify potential at-risk groups and implement appropriate interventions to promote maternal health and preventive measures to reduce obesity.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12889-025-21889-z>.

Additional file 1.

Acknowledgements

The authors thank the Australian Institute of Family Studies for providing the Longitudinal Study of Australian Children (LSAC) data. They also acknowledge the assistance of the UniSQ Women in STEMM Research program for awarding the SAGE Athena Swan Scholarship, as well as the contribution of the

Australian Commonwealth Government through the RTP fees offset scheme to the lead author.

Authors' contributions

The authors acknowledge the following contributions to this manuscript: N. B designed and implemented the study, wrote the initial draft, and conducted initial data analysis and curation. K. A assist in data analysis and curation. E.K and R. K. assist in designing the study, critically reviewed the manuscript and contributed to subsequent editing. All co authors accepted the final version of the manuscript.

Funding

The research did not receive any pacific grant from funding agencies in the public, commercial or not-for-profit sector. No author received payment for writing the article.

Data availability

This study utilised secondary data from the Longitudinal Study of Australian Children (LSAC); no new data were collected.

Declarations

Consent for publication

The manuscript used secondary data and did not contain any identifiable data of the participants. Hence, consent for publication was not required.

Competing interests

The authors declare no competing interests.

Author details

¹Ministry of Information, Bangladesh Betar, Rajshahi, People's Republic of Bangladesh. ²School of Mathematics, Physics, and Computing, University of Southern Queensland, Toowoomba, QLD, Australia. ³School of Mathematics, Physics, and Computing, and, Centre for Health Research, University of Southern Queensland, Toowoomba, QLD, Australia. ⁴School of Business, and Centre for Health Research, University of Southern Queensland, Toowoomba, QLD, Australia. ⁵Research Development Unit (RDU), Brown Health, Geelong VIC 3220, Australia.

Received: 29 September 2024 Accepted: 10 February 2025

Published online: 06 October 2025

References

- Abarca-Gómez L, Abdeen ZA, Hamid ZA, Abu-Rmeileh NM, Acosta-Cazares B, Acuin C, et al. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *The Lancet*. 2017;390(10113):2627–42.
- NHMRC. Summary Guide for the Management of Overweight and Obesity in Primary Care. Melbourne: National Health and Medical Research Council. <https://nla.gov.au/nla.obj-2454808641/view>. 2013.
- AIHW. Australian Burden of Disease Study: impact and causes of illness and death in Australia 2015. Australian Burden of Disease series no. 19. Cat. no. BOD 22. Canberra: AIHW. <https://www.aihw.gov.au/getmedia/c076f42f-61ea-4348-9c0a-d996353e838f/aihw-bod-22.pdf?v=20230605164109&inline=true>. 2019.
- AIHW. Overweight and obesity among Australian children and adolescents. Cat. no. PHE 274. Canberra: AIHW. <https://www.aihw.gov.au/getmedia/ac61b7d7-7991-4e15-8fa6-a7973479fa8b/aihw-phe-274.pdf>. 2020.
- Trost SG, Kerr LM, Ward DS, Pate RR. Physical activity and determinants of physical activity in obese and non-obese children. *Int J Obes*. 2001;25(6):822–9.
- Dennison BA, Erb TA, Jenkins PL. Television viewing and television in bedroom associated with overweight risk among low-income preschool children. *Pediatrics*. 2002;109(6):1028–35.
- Gibbs BG, Forste R. Socioeconomic status, infant feeding practices and early childhood obesity. *Pediatr Obes*. 2014;9(2):135–46.
- Heikkala E, Remes J, Paananen M, Taimela S, Auvinen J, Karppinen J. Accumulation of lifestyle and psychosocial problems and persistence of adverse lifestyle over two-year follow-up among Finnish adolescents. *BMC Public Health*. 2014;14:1–10.
- Rerkasem A, Lyons-Reid J, Namwongprom S, Wongsrithep S, Mangkla-bruks A, Phirom K, et al. Associations between maternal overweight/obesity during pregnancy and body composition in young adult offspring. *Front Public Health*. 2024;12:1346900.
- Ylöstalo T, Saha MT, Nummi T, Harjunmaa U, Salo MK, Vuorela N. Maternal weight, smoking, and diabetes provided early predictors of longitudinal body mass index growth patterns in childhood. *Acta Paediatrica*. 2024;113(5):1076–86.
- Heslehurst N, Vieira R, Akhter Z, Bailey H, Slack E, Ngongalah L, et al. The association between maternal body mass index and child obesity: A systematic review and meta-analysis. *PLoS Med*. 2019;16(6): e1002817.
- Audelo J, Kogut K, Harley KG, Rosas LG, Stein L, Eskenazi B. Maternal depression and childhood overweight in the CHAMACOS study of Mexican-American children. *Matern Child Health J*. 2016;20:1405–14.
- Kuciene R, Dulskiene V. Associations of maternal gestational hypertension with high blood pressure and overweight/obesity in their adolescent offspring: a retrospective cohort study. *Sci Rep*. 2022;12(1):3800.
- Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev*. 2007;29(1):6–28.
- Vazquez CE, Cubbin C. Socioeconomic Status and Childhood Obesity: a Review of Literature from the Past Decade to Inform Intervention Research. *Curr Obes Rep*. 2020;9(4):562–70.
- Barclay K, Myrskylä M. Maternal age and offspring health and health behaviours in late adolescence in Sweden. *SSM-population health*. 2016;2:68–76.
- Maessen SE, Ahlsson F, Lundgren M, Cutfield WS, Derraik JG. Maternal smoking early in pregnancy is associated with increased risk of short stature and obesity in adult daughters. *Sci Rep*. 2019;9(1):4290.
- Gustafson SL, Rhodes RE. Parental correlates of physical activity in children and early adolescents. *Sports Med*. 2006;36:79–97.
- Horne B, Kabir E, Alam K. Impact of prenatal maternal dietary exclusion on childhood obesity and overweight risk. *PLoS ONE*. 2024;19(3): e0297614.
- Barker DJ. Fetal origins of coronary heart disease. *BMJ*. 1995;311(6998):171–4.
- Gluckman PD, Hanson MA, Cooper C, Thornburg KL. Effect of in utero and early-life conditions on adult health and disease. *N Engl J Med*. 2008;359(1):61–73.
- Ahmad K, Keramat SA, Ormsby GM, Kabir E, Khanam R. Clustering of lifestyle and health behaviours in Australian adolescents and associations with obesity, self-rated health and quality of life. *BMC Public Health*. 2023;23(1):847.
- Anderson LN, Sandhu R, Keown-Stoneman CD, De Rubeis V, Borkhoff CM, Carsley S, et al. Latent class analysis of obesity-related characteristics and associations with body mass index among young children. *Obes Sci Pract*. 2020;6(4):390–400.
- Daw J, Margolis R, Wright L. Emerging adulthood, emergent health lifestyles: Sociodemographic determinants of trajectories of smoking, binge drinking, obesity, and sedentary behavior. *J Health Soc Behav*. 2017;58(2):181–97.
- Huh J, Riggs NR, Spruijt-Metz D, Chou CP, Huang Z, Pentz M. Identifying patterns of eating and physical activity in children: a latent class analysis of obesity risk. *Obesity*. 2011;19(3):652–8.
- Leech R, McNaughton S, Timperio A. Clustering of diet, physical activity and sedentary behaviour among Australian children: cross-sectional and longitudinal associations with overweight and obesity. *Int J Obes*. 2015;39(7):1079–85.
- Hu Z, Tylavsky FA, Kocak M, Fowke JH, Han JC, Davis RL, et al. Effects of maternal dietary patterns during pregnancy on early childhood growth trajectories and obesity risk: the CANDLE study. *Nutrients*. 2020;12(2):465.
- Soloff C, Lawrence D, Johnstone R. Sample design: Australian Institute of Family Studies Melbourne; 2005:1–30. <https://doi.org/10.13140/2.1.2587.8728>.
- Mantzorou M, Papandreou D, Pavlidou E, Papadopoulou SK, Tolia M, Mentzelou M, et al. Maternal gestational diabetes is associated with high

- risk of childhood overweight and obesity: a cross-sectional study in pre-school children aged 2–5 years. *Medicina*. 2023;59(3):455.
30. Kelly R, Hatzikiakidis K, Kuswara K. Inequities in obesity: Indigenous, culturally and linguistically diverse, and disability perspectives. *Pub Health Res Pract*. 2022;32(3). <https://doi.org/10.17061/phrp3232225>.
31. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ*. 2007;335(7612):194.
32. Depaire B, Wets G, Vanhoof K. Traffic accident segmentation by means of latent class clustering. *Accid Anal Prev*. 2008;40(4):1257–66.
33. Vermunt JK. A general latent class approach to unobserved heterogeneity in the analysis of event history data. *Appl latent class anal*. 2002;383–407. <https://doi.org/10.1017/CBO9780511499531.015>.
34. Collins LM, Lanza ST. Latent class and latent transition analysis: With applications in the social, behavioral, and health sciences: John Wiley & Sons; 2009;718. <https://doi.org/10.1002/9780470567333>.
35. Vu X-BB, Biswas RK, Khanam R, Rahman M. Mental health service use in Australia: The role of family structure and socio-economic status. *Children Youth Serv Rev*. 2018;93:378–89.
36. Xiao Y, Romanelli M, Lindsey MA. A latent class analysis of health lifestyles and suicidal behaviors among US adolescents. *J Affect Disord*. 2019;255:116–26.
37. Razaz N, Villamor E, Muraca GM, Bonamy A-KE, Cnattingius S. Maternal obesity and risk of cardiovascular diseases in offspring: a population-based cohort and sibling-controlled study. *Lancet Diabet endocrinol*. 2020;8(7):572–81.
38. Janjua NZ, Mahmood B, Islam MA, Goldenberg RL. Maternal and early childhood risk factors for overweight and obesity among low-income predominantly black children at age five years: a prospective cohort study. *J Obes*. 2012;2012:457173–82. <https://doi.org/10.1155/2012/457173>.
39. Magriplis E, Farajian P, Panagiotakos DB, Risvas G, Zampelas A. Maternal smoking and risk of obesity in school children: Investigating early life theory from the GRECO study. *Preventive medicine reports*. 2017;8:177–82.
40. Malinowska AM, Młodzik-Czyżewska MA, Chmurzynska A. Dietary patterns associated with obesity and overweight: when should misreporters be included in analysis? *Nutrition*. 2020;70: 110605.
41. Shaikh RA, Siahpush M, Singh GK, Tibbits M. Socioeconomic status, smoking, alcohol use, physical activity, and dietary behavior as determinants of obesity and body mass index in the United States: findings from the National Health Interview Survey. *International Journal of MCH and AIDS*. 2015;4(1):22.
42. McDonald BW, Watson PE. Maternal alcohol intakes before and during pregnancy: Impact on the mother and infant outcome to 18 months. *Nordic Stud Alcohol Drugs*. 2020;37(2):153–71.
43. Jonsson KR, Corell M, Löfstedt P, Adjei NK. The clustering of multiple health and lifestyle behaviors among Swedish adolescents: a person-oriented analysis. *Front Public Health*. 2023;11:1178353.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.