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Risk factors for stunting children in urban areas: case-control study in Putrajaya

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Abstract

A well-developed city, Putrajaya, was listed as having one of the highest stunting rates in 2016. This study examines determinants of stunting among children under five in Putrajaya. A case-control study was conducted between September 2018 and January 2019. Note that 8261 children aged 6–59 months were screened in preschools and government health clinics. From these, 386 stunted children (cases) and 386 normal children (controls) were randomly selected and matched by sex and age group. Data were obtained from face-to-face interviews with parents on sociodemographics, health, medical history, knowledge, and practice regarding children's feeding, dietary behaviour, food security, and physical activity. Anthropometric measurements, finger pricks for haemoglobin level, and a self-administered 3-day food diary were also collected. Multiple logistic regression was performed to identify the factors associated with stunting. Results. A total of 386 stunted and 386 normal children were recruited. The final model found that mid-parental height (< 150 cm)(aOR = 3.8), low household income (B40) (aOR = 3.4), Low Birth Weight (LBW) (aOR = 3.1), and use of pacifiers were about three 3 times higher odds of associated with stunting. Stunting was significantly associated with Intra-Uterine Growth Restriction (IUGR) (aOR = 7.9). Other factors linked to the outcome were low maternal education, paternal unemployment, childhood anaemia, bottle feeding, poor dietary diversity, and care by babysitters. Stunting in this urban population reflects biological (genetic load, IUGR, LBW) and modifiable factors (feeding practices, income, parental education). Without early intervention, the consequences extend to impaired cognitive development, reduced school achievement, lower adult productivity, and increased chronic disease risk. Findings underscore the urgency of targeted interventions in urban Malaysia, aligning with the and Malaysia' goal to reduce stunting below 11%.

Keywords Under five children, Stunting, Risk factors, Urban, Putrajaya

1 Introduction

Generally, a child's height remains one of the most accessible and reliable markers of physical development. Stunting, or short stature, results from poor or impaired linear growth [1]. Stunting is a failure to achieve the expected linear growth for age, which is

the primary anthropometric indicator of chronic undernutrition [1, 2]. Note that height-for-age measurements define stunting. Based on World Health Organisation (WHO) Growth Standards, children with a height-for-age z-score (HAZ) below – 2 Standard Deviations (SD) are classified as stunted. In contrast, those below – 3 SD are severely stunted [3].

Globally, the prevalence of stunting among children under five years of age was 22.0%, which equals 149.0 million children in 2020. The prevalence decreased to 32.5%, affecting an estimated 198.2 million children, in 2000 [4]. According to the National Health and Morbidity Survey (NHMS), the prevalence of stunting among Malaysian children increased from 16.6% in 2011 to 20.7% in 2016 [5, 6]. In 2016, Putrajaya reported one of the country's highest stunting rates among children under five (24.3%), despite its status as a highly developed urban area and administrative capital. The stunting rate in Putrajaya more than doubled from 10.9% in 2011 to 24.3% in 2016, elevating national and local prevalence from medium to high according to the WHO classification [7]. This study was conducted between September 2018 and January 2019, which coincides with the implementation period of NPANM III (2016–2025). The findings therefore provide timely evidence that can inform mid-cycle reviews and adjustments of policy and program planning to achieve the national target of reducing stunting to below 11% by 2025.

Stunting may be attributed to different causes, some of which often begin in utero and continue for at least the first two years of life [8]. Well-established contributors to stunting include sociodemographic factors, inadequate dietary intake, environmental exposures, health conditions, and genetic predisposition [2, 9]. In addition, stunting has been consistently associated with environmental exposures such as inadequate ventilation and pesticide exposure, suboptimal nutritional intake; including low consumption of milk and high-quality protein and sociodemographic disadvantages, including low parental education and household income [9]. Recurrent infections, frequent fevers, and repeated injuries are health-related factors linked to stunting in children [2, 8]. Intra-Uterine Growth Retardation (IUGR) and Low Birth Weight (LBW) are also well-established risk factors for stunting [9]. Parental height, particularly below 160 cm, is linked to stunting, with mid-parental height as a key predictor due to genetic influence on child growth [10]. Although genetics cannot be changed, a pooled analysis of five birth cohorts showed that poor nutrition across generations can hinder children from reaching their genetic height potential [11].

Stunting is recognized as a global public health priority and is included among the six Global Nutrition Targets for 2025, with the goal of achieving a 40% reduction in the number of stunted children under five [12]. Malaysia's NPANM III (2016–2025) aligns with this goal, targeting a stunting rate below 11% by 2025 [13]. Identifying factors associated with child malnutrition can inform equitable healthcare planning and support efforts to reduce stunting over time. Therefore, this study examined the factors associated with stunting among children under five in Putrajaya.

2 Methods

2.1 Study design

This study employed a matched case-control design with a 1:1 ratio. Here, cases were defined as children with height/length-for-age below −2 SD from the WHO Child Growth Standards median. Controls were children with normal growth parameters,

including height/length-for-age, weight-for-age, and BMI-for-age within the − 2SD to +2SD range. Consequently, matching was performed based on sex and age groups (6–11 months, 12–35 months, and 36–59 months) to minimise bias and improve the validity of the analysis. Data was collected in Putrajaya, Malaysia, between September 2018 and January 2019 in two phases: Phase I (Screening): Conducted between 12 September and 12 October 2018 in preschools ($n = 2{,}993$ children) and government health clinics ($n = 5{,}268$ children). Here, 8261 children aged 6–59 months were screened for anthropometric status. Phase II (Interviews and Assessments) conducted between 16 October 2018 and 31 January 2019. Eligible cases (stunted children) and matched controls were randomly selected from the screened cohort. Depending on convenience, caregivers were interviewed at their homes, offices, or health facilities.

2.2 Study population

Inclusion criteria were Malaysian children aged 6 to 59 months who had resided in Putrajaya for at least six months. Children with mental or physical disabilities, those with chronic conditions that impaired participation, or those acutely ill during data collection.

2.3 Sample size estimation

Sample size estimation was calculated using PS software to compare two proportions, targeting the identification of factors associated with stunting among children under five. Based on past studies, with a significance level of $\alpha = 0.05$ and power $(1-\beta) = 0.80$, a minimum of 380 subjects per group was required. ample size was calculated for a matched case–control design using a two-sided test at $\alpha = 0.05$ and power = 0.80 (1:1 case: control). We powered the study to detect a difference in the proportion of exposure to a prespecified binary risk factor between cases and controls (i.e., p_1 vs. p_0), rather than on stunting prevalence. Specifically, we assumed a control exposure prevalence (p_0) of 0.20 (urban Malaysia context) and a minimum detectable odds ratio of 2.0 for the association with stunting. Under these assumptions (PS—Power and Sample Size—software), the required total sample was approximately 760 children (~ 380 cases and ~ 380 controls). Our achieved sample (386 cases; 386 controls) met this requirement.

2.4 Respondent recruitment

Cases and control children were selected randomly from the screening database. Parents of the selected children were contacted for an interview appointment. Note that parents who refuse to participate will be replaced with other candidates obtained from the screening database. This study adhered to the Declaration of Helsinki, and ethical approval was obtained from the Medical Research Ethics Committee (MREC), Ministry of Health Malaysia (NMRR-18-847-41455). Correspondingly, written informed consent was obtained from the parents or guardians before the survey began.

2.5 Tools and instruments

Data were collected using four approaches: interview, anthropometric assessment, finger-prick blood, dietary diversity intake and food diary record. The interview was done face-to-face using a structured questionnaire on tablets. The structured questionnaire consists of seven modules comprising questions regarding sociodemographic and

socioeconomic, health and medical history, knowledge and practice of parents or caregivers towards the child's feeding, dietary behaviour of the children, food security, and physical activity. In our analysis, however, genetic load was operationalised using mid-parental height, calculated as the average of both parents' heights, and categorised into < 150.0 cm, 150–159.9 cm, and ≥ 160.0 cm.

2.5.1 Nutritional status

For children under 24 months of age, recumbent length was measured in the supine position using a SECA 210 infantometer, following WHO recommendations. For children aged 24 months and above, standing height was measured using a SECA 213 stadiometer. In the few cases where children above 24 months were unable to stand, recumbent length was taken and converted to standing height by subtracting 0.7 cm, consistent with WHO Child Growth Standards.Anthropometric assessments were performed on children and their parents, with weight and stature measured using a Tanita HD-319 personal scale and a SECA 213 stadiometer. Consequently, recorded values were rounded to the nearest 0.1 kg for weight and 0.1 cm for height. For infants and young children unable to stand, weight was measured using a SECA 354 digital baby scale, and recumbent length was assessed with a SECA 210 mobile baby measuring mat.

2.5.2 Finger prick blood

On the other hand, haemoglobin was assessed by finger-prick sampling in children using a portable HemoCue® Hb 201 analyser. Anaemia was classified based on WHO guidelines as a haemoglobin concentration below 11.0 g/dL.

2.5.3 Minimum dietary diversity (MDD) intake assessment and categorization

Additionally, Dietary Diversity was determined by calculating the Minimum Dietary Diversity (MDD), an established Infant and Young Child Feeding (IYCF) indicator. This calculation was based on food consumption data from a single, representative 24-hour period. For this analysis, the food intake recorded was selected as the representative 24-hour period.

The child was scored based on the consumption of foods from at least four of the seven predefined food groups during this selected 24-hour period [14]. The child was categorized as having Adequate Dietary Diversity if they consumed of the seven food groups and Inadequate Dietary Diversity if they consumed food groups. While the MDD indicator is primarily validated for children aged 6–23 months, it was adopted and applied to the full study cohort (6–59 months) as a practical, validated, and nutrition-sensitive proxy for micronutrient adequacy, consistent with similar studies in developing contexts.

2.5.4 Dietary intake

The fourth approach was a three-day food diary completed by the parent and caregiver (two days on weekdays and one day on the weekend). For preschool children, during school hours, teachers documented students' food intake in the food diary. Before submission to the data-entry team, the interviewer reviewed and verified all entries in the food diary for accuracy and completeness. The raw data from the three-day food diaries were used to calculate energy and protein intakes, which were assessed against the

Malaysian Recommended Nutrient Intakes (RNI). The mean and standard deviation (SD) of these intakes are presented in the descriptive results in Table 1.

2.6 Data management

Data from tablet questionnaires, anthropometric measurements, and haemoglobin tests were uploaded online to the Institute for Public Health (IPH) server. The data were then extracted in Microsoft Excel format. The three-day food diaries were delivered weekly to IPH, where the data entry team converted the recorded food intake into grams using standard menus or recipes. Subsequently, the data were entered into NutritionistPro™ software version 7.5 for nutritional analysis.

2.7 Data analysis

Descriptive statistics were computed for all study variables, and a simple logistic regression was conducted for each independent variable. The multivariable logistic regression included variables with a p-value < 0.25 or of clinical importance. Consequently, independent variables were selected using both forward and backward likelihood ratio methods. Multicollinearity and interaction effects were evaluated to finalise the model. The three model fits were assessed using the Hosmer-Lemeshow test, classification table, and the area under the Receiver Operating Characteristics (ROC) curve. Note that independent variables with $p < 0.05$ and Odds Ratio (OR) greater than 1.00 were identified as risk factors for stunting; OR < 1.00 indicated protective factors. The multivariable analysis involved fitting three models for each outcome. The 'Prefinal Model' included all variables with a value in the univariable analysis or those deemed clinically important. The 'Final Model' (Table 2) was then derived from the Prefinal Model using a combination of forward and backward likelihood ratio methods, retaining only the variables with a significance level of and controlling for potential confounders. The results of the Univariable and Prefinal Models a d Final Model are presented in Table 2 to demonstrate the stepwise selection process. All analyses were conducted using SPSS version 26. The detailed protocol for this study has been reported elsewhere [15].

3 Results

The study involved 772 children, equally divided into groups of stunted ($n = 386$) and non-stunted ($n = 386$). As presented in Table 1, descriptive analysis of sociodemographic factors revealed that a higher proportion of stunted children had parents with a mild-parental height < 150 cm. 65.7% of stunted children belonged to the B40 household income group. Furthermore father unemployment notably higher among the families of stunted children. Regarding the maternal and pregnancy history, the prevalence of Intrauterine Growth Restriction (IUGR) was higher in the stunted children. Birth outcome also differed significantly, 15.8% of stunted children were born with low birth weight, additionally, 53.4% of stunted children were found to be anaemic. Behavioural factors such as use of a pacifier were highly prevalent among stunted children and caregiving arrangement showed that stunted children were cared by babysitter compared to normal children.

Based on the final best-fit logistic regression model, twelve factors were independently associated with stunting among children in Putrajaya (Table 2). These findings suggest

Table 1 Characteristic of children below five years old in Putrajaya

Variable		Stunted, *n* (%)	Normal, *n* (%)
Sociodemographic and socioeconomic factors			
Mother's education	Primary & Secondary	83 (21.5)	52 (13.5)
	Tertiary	303 (78.5)	334 (86.5)
Mother's occupation	Government servant	307(79.5)	321 (83.2)
	Private servant	31 (8.0)	30 (7.8)
	Not working/housewife	48 (12.5)	35 (9.1)
Father's education	Primary & Secondary	104 (26.9)	73 (18.9)
	Tertiary	282 (73.1)	313 (81.1)
Father's occupation	Government servant	234 (60.6)	243 (63.0)
	Private servant	125(32.4)	105 (27.2)
	Not working	27 (7.0)	38 (9.8)
	Government servant	234 (60.6)	243 (63.0)
Household income (monthly)	B40 (Below 40%)	254 (65.8)	207 (53.6)
	M40 (Middle 40%)	125 (32.4)	155 (40.2)
	T20 (Top 20%)	7 (1.8)	24 (6.2)
Monthly expenditure for food	< RM1,000	352 (91.2)	355 (92.0)
	RM1,000 - RM1,999	27 (7.0)	27 (7.0)
	>=RM2,000	7 (1.8)	4 (1.0)
	< RM1,000	200 (51.8)	174 (45.1)
Monthly expenditure for childcare	RM1,000 - RM1,999	155 (40.2)	157 (40.7)
	>=RM2,000	31 (8.0)	55 (14.2)
Monthly expenditure for utility	< RM1,000	298 (77.2)	276(71.5)
	RM1,000 - RM1,999	66 (17.1)	74 (19.2)
	>=RM2,000	22 (5.7)	36 (9.3)
Monthly expenditure on transport	< RM1,000	258 (66.8)	237 (61.4)
	RM1,000 - RM1,999	101 (26.2)	114 (29.5)
	>=RM2,000	27 (6.2)	35 (9.1)
Maternal and Pregnancy Factors			
Mid-parental height (cm)	< 150.0	34 (8.8)	14 (3.6)
	150.0–159.9	143 (37.0)	140 (36.3)
	≥ 160.0	209 (54.1)	232 (60.1)
Mother age	18–30 years old	153 (39.6)	161 (41.7)
	> 30 years old	233 (60.4)	225 (58.3)
Pre-pregnancy BMI	Normal	204 (52.8)	210 (54.4)
	Underweight	30 (7.8)	49 (12.7)
	Overweight or obese	152 (39.4)	127 (32.9)
Weight gain during pregnancy	Sufficient	177 (45.9)	194 (50.2)
	Insufficient	118 (30.6)	91 (23.6)
	Excess	91 (23.5)	101(26.2)
Complications during pregnancy	None	266 (68.9)	292 (75.7)
	GDM	48 (12.4)	39 (10.1)
	Anaemia	59 (15.3)	53 (13.7)
	IUGR	13 (3.4)	2 (0.5)
Number of antenatal visits	< 9	22 (5.7)	18 (4.7)
	9–14	270 (69.9)	279 (72.3)
	≥ 15	94 (24.4)	89 (23.0)
Knowledge of child feeding	Less satisfied	25 (6.5)	16 (4.1)
	Satisfied	361 (93.5)	370 (95.9)
Practice on child feeding	Fair	54 (14.0)	70 (18.1)
	Good	219 (56.7)	207 (53.6)
Behaviour of the child feeding	At risk	343 (88.9)	348 (90.2)
	No risk	43 (11.1)	38 (9.8)

Table 1 (continued)

Variable		Stunted, *n* (%)	Normal, *n* (%)
Delivery method	Normal	250 (64.8)	256 (66.3)
	Forceps/vacuum	18 (4.7)	30 (7.8)
	Caesarean	118 (30.5)	100 (25.9)
Gestational weeks	Term	364 (94.3)	344 (89.1)
	Pre-term	22 (5.7)	42 (10.9)
Child Health and Nutritional Factors			
Birth weight status	Normal birth weight	325 (84.2)	362 (93.8)
	Low birth weight	61 (15.8)	24 (6.2)
Birth length status	Normal	360 (93.3)	377 (97.7)
	Short	26 (6.7)	9 (2.3)
Birth head circumference	Normal	321 (83.2)	347 (89.9)
	Small	65 (16.8)	39 (10.1)
Haemoglobin level	11 mg/dl and above	179 (46.4)	217 (56.2)
	Below 11 mg/dl	207 (53.6)	169 (43.8)
Number of siblings	1–3	294 (76.2)	286 (74.1)
	4 and above	92 (23.8)	100 (25.9)
The age gap between an elder sibling	≤ 24 months	72 (18.7)	79 (20.5)
	≥ 25 months	210 (54.4)	202 (52.3)
	Do not have	104 (26.9)	105 (27.2)
The age gap between the younger sibling	≤ 24 months	46 (11.9)	52 (13.5)
	≥ 25 months	64 (16.6)	73 (18.9)
	Do not have	276 (71.5)	261 (67.6)
Acute illness	Never	37 (9.6)	45 (11.7)
	At least once every 2 months	58 (15.0)	41 (10.6)
	Once every 3 to 12 months	291 (75.4)	300 (77.7)
Injury	Never	254 (65.8)	257 (66.6)
	At least once every 2 months	31 (8.0)	30 (7.8)
Worm infestation	No	373 (96.6)	380 (98.5)
	Yes	13 (3.4)	6 (1.5)
Caregiving and Environmental Factors			
Initiation of breastfeeding after delivery	Within 1 h	285 (73.8)	300 (77.7)
	1–24 h	72 (18.7)	58 (15.0)
	After 1 day or Never	29 (7.5)	28 (7.2)
Breastfeeding status	Ever	381 (98.7)	382 (99.0)
	Never	5 (1.3)	4 (1.0)
Exclusive breastfeeding	Yes	248 (64.2)	257 (66.6)
	No	138 (35.8)	129 (33.4)
Predominant breastfeeding	Yes	270 (69.9)	285 (73.8)
	No	116 (30.1)	101 (26.2)
Age stops breastfeeding	< 6 months	59 (15.3)	38 (9.8)
	6–24 months	253 (65.5)	271 (70.2)
	> 24 months	74 (19.2)	77 (19.9)
Formula milk feeding	Yes	319 (82.6)	309 (80.1)
	No	67 (17.4)	77 (19.9)
Use of bottle-feeding	Yes	294 (76.2)	274 (71.0)
	No	92 (23.8)	112 (29.0)
Use of a pacifier	Yes	354 (91.7)	323 (83.7)
	No	32 (8.3)	63 (16.3)
Dietary diversity	Adequate	335 (86.8)	353 (91.5)
	Inadequate	51 (13.2)	33 (8.5)
Achievement RNI for Kcal/day	Adequate	265 (68.7)	270 (69.9)
	Inadequate	121 (31.3)	116 (30.1)

Table 1 (continued)

Variable		Stunted, *n* (%)	Normal, *n* (%)
Achievement RNI for protein/day	Adequate	371 (96.1)	380 (98.5)
	Inadequate	15 (3.9)	6 (1.5)
Food insecurity	Food secure	120 (31.1)	132 (34.1)
	Food insecure	266 (68.9)	254 (65.8)
Energy intake (kcal)	Mean(SD)	1224.0 (336.3)	1258.1 (363.7)
Protein intake (g)	Mean (SD)	41.6 (13.8)	44.4 (15.4)
Caregiving and Environmental Factor			
Place of stay while parents work	Kindergarten	285 (73.8)	318 (82.4)
	Babysitter	72 (18.7)	44 (11.4)
	Relative	29 (7.5)	24 (6.2)
Sleep time in a day	Optimal health sleep	248 (64.2)	266 (68.9)
	Below recommendation	130 (33.7)	114(29.5)
	Above recommendation	8 (2.1)	6 (1.6)
Screen time in a day	< 60 min	15 (3.9)	20 (5.2)
	≥ 60 min	371 (96.1)	366 (94.8)
MVPA time in a day	< 180 min	361 (93.5)	363 (94.0)
	≥ 180 min	25 (6.5)	23 (6.0)

that stunting in this urban setting reflects the combined influence of biological, socioeconomic, and caregiving-related factors.

IUGR showed the strongest association with stunting, with affected children having over eightfold higher odds (aOR 8.18; 95% CI: 1.71–39.21), highlighting the lasting impact of impaired fetal growth. Lower maternal education, paternal unemployment, and household income in the B40 and M40 categories were also significantly associated with stunting. Mid-parental height remained independently associated (aOR 3.45), suggesting that inherited growth potential contributes to stunting alongside socioeconomic disadvantage, even in a highly urbanized setting.

Child health and nutritional factors significantly associated with stunting included low birth weight, anaemia, inadequate dietary diversity, bottle-feeding, and pacifier use, reflecting early nutritional deficits and suboptimal feeding practices. Among caregiving and environmental factors, children cared for by babysitters had higher odds of stunting than those cared for by relatives or attending kindergarten, suggesting differences in caregiving quality and feeding supervision.

The final model, derived from the prefinal model, includes only statistically significant and non-collinear variables. The adjusted odds ratios provide a stable and parsimonious representation of factors associated with stunting in Putrajaya, highlighting the multifactorial nature of growth faltering in urban populations.

4 Discussion

As many as 12 factors were identified in the final model. The IUGR was shown to have the strongest association with stunting. Moreover, it is well known that IUGR babies are smaller due to the limitation of growth in utero. Infants with IUGR often suffer from delayed neurological and intellectual development and a deficit in height [16]. One previous study reported that maternal stunting has been identified as a risk factor for IUGR, as it compromises uterine blood flow, leading to impaired growth of the uterus, placenta, and fetus [17].

Table 2 Pre-final and final model associated with stunting among children under five years old in Putrajaya

Variables		Pre-final model			Final model	
		aOR (95% CI)	*p*-value	b	aOR (95% CI)	*p*-value
Mid-parental height (cm)	< 150.0	3.76 (1.82–7.77)	0.001	1.229	3.42 (1.69–6.89)	**0.001**
	150.0–159.9	1.07 (0.77–1.50)	0.687	0.076	1.08 (0.78–1.49)	0.646
	≥ 160.0	1			1	
Mother's education	Primary & Secondary	1.65 (1.01–2.70)	0.046	0.522	1.69 (1.10–2.58)	**0.017**
	Tertiary	1			1	
Father's occupation	Government servant	1			1	
	Private servant	1.30 (0.72–2.36)	0.383	0.337	1.40 (0.79–2.48)	0.246
	Not working	1.93 (1.02–3.67)	0.044	0.708	2.03 (1.11–3.75)	**0.023**
Household income (monthly)	B40 (Below 40%)	3.70 (1.35–10.09)	0.011	1.551	4.71 (1.87–11.91)	**0.001**
	M40 (Middle 40%)	3.40 (1.25–9.26)	0.017	1.303	3.68 (1.45–9.38)	0.006
	T20 (Top 20%)	1			1	
Pre-pregnancy BMI	Normal	1			-	
	Underweight	1.30 (0.74–2.28)	0.366	0.298	1.35 (0.79–2.29)	0.271
	Overweight or obese	0.64 (0.44–0.925	0.018	-0.321	0.73 (0.52–1.01)	0.060
	None	1			1	
Complications during pregnancy	GDM	1.49 (0.89–2.49)	0.133	0.406	1.50 (0.91–2.47)	0.110
	Anaemia	0.94 (0.60–1.49)	0.804	-0.028	0.97 (0.63–1.51)	0.901
	IUGR	7.91 (1.50–41.60)	0.015	2.102	8.18 (1.71–39.21)	**0.009**
Birth weight status	Normal birth weight	1			1	
	Low birth weight	2.06 (1.07–3.96)	0.030	1.063	2.90 (1.70–4.94)	**0.001**
Hemoglobin level	Normal	1			1	
	Anaemic	1.40 (1.01–1.93)	0.044	0.398	1.49 (1.10–2.02)	**0.011**
Use of bottle-feeding	Yes	1.71 (1.17–2.50)	0.006	0.485	1.62 (1.14–2.32)	**0.007**
	No	1			1	
Use of a pacifier	Yes	3.37 (1.98–5.74)	0.001	1.051	2.86 (1.75–4.68)	**0.001**
	No	1			1	
Dietary diversity	Adequate	1			1	
	Inadequate	1.64 (0.96–2.81)	0.070	0.592	1.81 (1.09-3.00)	0.022
Place of stay while parents work	Kindergarden	1			1	
	Babysitter	1.95 (1.21–3.15)	0.007	0.601	1.83 (1.17–2.85)	**0.008**
	Relative	1.47 (0.71–3.03)	0.298	0.272	1.31 (0.71–2.44)	0.389

Hosmer-Lemeshow test = 0.247 (> 0.05), Classification table = 65.2%,Area under the Receiver Operating Characteristics (ROC) curve = 0.711

In line with our findings, genetic load reflected by mid-parental height and intergenerational short stature remains an important determinant of stunting. However, it is crucial to highlight that genetic predisposition does not fully determine attained height. Evidence from longitudinal cohort studies shows that a considerable proportion of height gain is influenced by modifiable environmental and nutritional factors, including maternal nutrition, antenatal care, infant feeding practices, and dietary diversity during early childhood [18]. This underscores that even when children inherit a genetic risk for shorter stature, targeted interventions addressing modifiable exposures can help them approach their genetic growth potential. Such findings are consistent with pooled analyses demonstrating that intergenerational undernutrition can suppress height potential across populations, whereas improvements in nutrition and health environments can

lead to secular increases in average height over time. Therefore, addressing stunting requires a dual perspective: acknowledging the role of genetic load while simultaneously prioritizing modifiable socioeconomic, nutritional, and care-related factors that offer realistic opportunities for intervention.

Children whose mid-parental height was below 150.0 cm, whose families had low monthly income, who had LBW, or who used pacifiers were approximately three times more likely to experience stunting compared to their respective reference groups. Other significant associated factors were primary or secondary education of the mother, an unemployed father, anaemic children, use of bottle feeding, inadequate dietary diversity, and children cared for by a babysitter. Many risk factors for stunting have been identified in epidemiological studies, and they vary between countries.

The World Health Organisation Multicentre Growth Reference Study (WHO MGRS) found that in populations with widely varying parental heights, mid-parental height—a combined measure of both mother's and father's heights consistently explained more of the variation in children's attained length than either parent's height alone [19]. Other than that, recent reviews highlight the need for genetic research in malnutrition to understand genetic risk factors better and identify potential targets for intervention and treatment [20].

Sociodemographic determinants of stunting in Putrajaya included low household income, low mothers' education, and fathers' unemployment. These preventable factors were also reported in the Pakistan Demographic and Health Survey (PDHS) 2012–2013 and other Asian republics [21, 22]. Note that children from poor households have limited access to healthy food and health services, which makes them prone to growth failures [23].

LBW was a consistent factor associated with stunting, even in urban areas like Putrajaya. Data from the COHORTS study show that lower birth weight and greater undernutrition in childhood, such as stunting, were risk factors for high glucose concentrations, high blood pressure, and harmful lipid profiles after adjusting for adult body mass index and height [8, 23].

Haemoglobin levels below 11 g/dL, indicating anaemia, were also linked with stunting among children in Putrajaya. A study in West Africa determined that children under 59 months with anaemia were approximately three to four times more likely to be stunted compared to their non-anaemic counterparts [24]. A cross-sectional study of 150 malnourished children aged 6–60 months at the Civil Hospital Karachi Paediatric Department found that anaemia was the most prevalent micronutrient deficiency, affecting 78% of participants. Additionally, 44% of severely stunted children were anaemic [25].

Feeding practices in early childhood, especially using pacifiers and bottles, were discovered to be a risk for stunting. Pacifier use was determined to negatively interfere with the physiological relationship between breastmilk supply and demand. It also substitutes feeding, decreasing the frequency of breastfeeding and food intake [26]. A study of 70 mother-infant pairs at Benha University Hospital, Egypt, revealed that exposure to pacifiers and bottles was everyday among infants with Oral Motor Dysfunction (OMD) [27]. The same study also found that stunted growth was present in half of the cases with OMD compared to only one in ten cases with no OMD.

Our study also examined that inadequate dietary diversity is associated with stunting in children in Putrajaya. A study conducted in Sedayu, Indonesia, also found that

children with inadequate dietary diversity or who consumed less than 4 of 7 food groups were more likely to be at risk of stunting [28]. Furthermore, a systematic review highlight increasing dietary diversity and food variety can enhance the dietary intake of micronutrients [29]. Because nutrients are present in different foods, dietary diversity is significant in meeting nutrient needs. One of the key strategies highlighted for reducing stunting in ASEAN countries is to ensure all children have adequate dietary diversity [30].

The last factor significantly associated with stunting in Putrajaya is children cared for by a babysitter. A study in Cyprus reported that children under babysitter care exhibited higher rates of undernutrition. The same study also reported that more children looked after by babysitters were underweight compared to those taken care of by their parents [31]. WHO also emphasises that important protective factors of development difficulties in young children were a strong bond with a caregiver other than the parents (such as an aunt, babysitter, or teacher) and involvement in the community [32].

Other important risk factors, such as exclusive breastfeeding and food insecurity, were not discovered to be significantly associated with stunting in this study, even though multiple logistic regressions were made. This is likely due to the study being subject to recall bias regarding breastfeeding history and underreporting of food security questions, as mothers may withhold facts due to feelings of embarrassment or concern about others [33]. However, we believe the findings from this study will provide notable input for stakeholders and policymakers to draw up effective prevention plans to reduce the prevalence of malnourished children in Putrajaya and nationwide. For instance, our finding that low household income is associated with stunting is consistent with reports from Pakistan [34] and Central Asia [35]. However, studies in certain urbanised middle-income settings (e.g., Thailand, Brazil) have shown a weaker association, likely reflecting stronger social safety nets and subsidised childcare. This suggests that while poverty is a universal determinant, its impact may be attenuated in contexts with comprehensive welfare systems. Similarly, bottle feeding and pacifier use were strongly associated with stunting in our study. While comparable results have been observed in Egypt and West Africa, studies in some European cohorts have not reported this relationship. This contrast may be due to differences in complementary feeding practices, regulation of infant formula marketing, and cultural acceptance of breastfeeding. Thus, context modifies the biological plausibility of feeding practices as risk factors. We also found that maternal education was protective. This is well documented globally; however, in some rapidly urbanising settings, such as China and parts of Latin America, maternal education alone has not consistently predicted improved child growth, possibly due to imultaneous influences of maternal employment demands, urban food environments, and childcare arrangements.

Our findings also highlight a paradox: despite relatively high parental education and stable employment in Putrajaya, stunting prevalence remains elevated. This suggests that stunting in affluent urban populations cannot be explained by poverty alone, but by the interaction of biological risks (such as IUGR and LBW) with modifiable socio-behavioural factors, including feeding practices and childcare arrangements. For example, an infant born small-for-gestational-age who is subsequently cared for by a babysitter and introduced to bottle feeding is doubly disadvantaged. Similar observations have been made in urban centres of other middle-income countries, where maternal employment,

time constraints, and costly urban diets contribute to inappropriate feeding practices despite relatively favourable education and income levels. This indicates that in urban Malaysia, stunting is less about food scarcity and more about food quality, feeding behaviours, and caregiving contexts. These insights carry important programmatic implications: interventions should focus not only on economic uplift but also on strengthening workplace breastfeeding policies, urban food subsidies, and caregiver education.

The study distinguished feeding method (bottle feeding) from type of milk consumed(formula feeding), hence their treatment as separate variables: Formula milk feeding: Referred to whether the child was fed with infant or follow-up formula milk (yes/no), regardless of the mode of delivery. This captured type of milk consumed, distinguishing it from breast milk or other alternatives. Bottle feeding: Referred to the feeding method, i.e., whether the child was fed using a feeding bottle (yes/no). This included not only formula milk, but also expressed breast milk, cow's milk, or other liquids given via bottle.Thus, while the two variables are related, they are not interchangeable: Formula milk = *what the child is fed*. Bottle feeding = *how the child is fed*. In the analysis, both were examined independently. Bottle feeding remained a significant predictor of stunting, while formula milk feeding did not retain significance after adjustment."

In our dataset, IUGR and LBW were not mutually exclusive, and each was coded as an independent yes/no variable (with some overlap). To verify model appropriateness, we evaluated collinearity among LBW, preterm, and IUGR using standard diagnostics (pairwise correlations and variance inflation factors, VIF). No problematic multicollinearity was detected (all VIF < 2; correlations below commonly accepted thresholds), indicating that simultaneous inclusion did not distort estimates. This is consistent with our Results text noting partial overlap but independent associations for IUGR and LBW with stunting in the adjusted model.

In our study, screen time was collected for all children aged 6–59 months, using a caregiver-reported measure of average daily time spent watching television, using mobile phones, tablets, or computers. For children < 24 months, we recognise that international guidelines (e.g., WHO 2019 Guidelines on Physical Activity, Sedentary Behaviour and Sleep for Children under 5) recommend no screen time at all. However, in practice, many Malaysian parents do expose infants and toddlers to screens, even under 2 years of age. Our variable therefore reflects actual reported behaviour in the urban context, not a recommended practice. For children < 1 year, reported screen time was generally very low; for those 1–2 years, usage was higher but still less than older preschool children. In the analysis, screen time was treated as a continuous exposure (hours/day) but did not show a significant association with stunting in the adjusted model. It was retained descriptively to reflect modern lifestyle behaviours in urban Putrajaya.

This study has several strengths. It is the first case–control investigation of stunting conducted in Putrajaya, an advanced urban centre in Malaysia, where stunting prevalence was unexpectedly high. The study applied rigorous anthropometric methods, standardised tools, and followed WHO growth standards, ensuring data quality. We also examined a broad range of biological, socioeconomic, and behavioural risk factors within a conceptual framework, which allows for a more comprehensive interpretation of determinants.

Nonetheless, some limitations should be acknowledged. First, as a case–control design, causal inference cannot be firmly established. Second, dietary intake and feeding practices were based on parental recall, which may be prone to recall bias. Furthermore, while the MDD criteria were used for all children (6–59 months), we acknowledge that the indicator is primarily validated for children aged 6–23 months, which may limit the generalizability of this specific nutritional finding for the older age groups.

Third, while the study population reflects an urban Malaysian context, findings may not be generalisable to rural or less affluent populations. Lastly, some potential confounders, such as maternal mental health or household food security indices, were not measured and may have influenced child growth outcomes.

5 Conclusion

These findings highlight that stunting in urban areas cannot be attributed solely to poverty or service access gaps, reflecting an interplay of biological, intergenerational, and behavioural determinants. Addressing stunting early is critical to prevent adverse outcomes, including impaired cognitive development, poor school performance, reduced adult productivity, and increased risk of chronic diseases. Therefore, policy interventions should prioritise Maternal health and antenatal care to prevent IUGR and LBW. Nutrition education for parents and childcare providers focuses on exclusive breastfeeding, appropriate complementary feeding, and dietary diversity. Urban-targeted programmes that address income disparities and ensure nutrition-sensitive childcare practices. By aligning with the Global Nutrition Target 2025 and Malaysia's NPANM III (2016–2025), this study supports an urgent call for multi-sectoral, intergenerational interventions to break the cycle of stunting in Malaysia's urban populations.

Supplementary Information

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Supplementary Material 1.

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Author contributions
RS, MHA were involved in conceptualisation, literature search, review and editing the manuscript. MHA and RS contributed to performing statistical analyses and checking the data accuracy and quality. NAMZ, SMS, AB, KHA, PBK and HAZ participated in the review and editing the final manuscript. All authors reviewed the manuscript.

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Data availability
The primary data used to support the findings of this study are available from the corresponding author institution (National Institutes of Health, Ministry of Health Malaysia) upon request.

Declarations

Ethics approval and consent to participate
Ethical approvals and accordance were obtained from the Medical Research and Ethics Committee (MREC), Ministry of Health Malaysia, with registration number NMRR-18-847-41455.

Consent for publication
Not applicable.

Informed consent
Written informed consent was obtained from parents/guardians.

Competing interests
The authors declare no competing interests.

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