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A growth curve model to estimate longitudinal effects of parental BMI on Indonesian children's growth patterns

Yoseph Leonardo Samodra and Ying-Chih Chuang

School of Public Health, Taipei Medical University, New Taipei City, Taiwan

Original Article

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Corresponding author:
Ying-Chih Chuang;
Email: yingchih@tmu.edu.tw.

Abstract

The global surge in childhood obesity is also evident in Indonesia. Parental body mass index (BMI) values were found to be one of the major determinants of the increasing prevalence of childhood obesity. It is uncertain if parental BMI during their offspring's childhood significantly affects their children's BMI trajectories into adulthood. We aimed to investigate the influence of parental BMI *Z*-scores on BMI trajectories of Indonesian school-aged children, with a focus on sex-specific effects. This study utilized data from the Indonesian Family Life Survey and tracked the same respondents over four time points, from wave 2 (1997–1998) to wave 5 (2014–2015). The sample of this study consisted of children aged 5–12 years in wave 2 for whom height and weight data were available. We utilized a two-level growth curve model to account for the hierarchical structure of the data, with time nested within individual children. Fathers' BMI Z-scores in wave 2 had a pronounced influence ($\beta = 0.31$) on female children's BMI *Z*-scores compared to the influence of mothers' BMI Z-scores ($\beta = 0.17$). Mothers' BMI *Z*-scores in wave 2 showed a stronger positive association with male children's BMI *Z*-scores ($\beta = 0.22$) than did the father's BMI *Z*-scores ($\beta = 0.19$). A significant interaction of fathers' BMI *Z*-scores and years of follow-up was found for male children. As male children's BMI *Z*-scores increased by year, this effect was stronger in those whose fathers' BMI *Z*-scores were at a higher level. In conclusion, we found that parental BMI values profoundly influenced their children's BMI trajectories.

Introduction

Childhood obesity is on the rise worldwide, and overweight children and adolescents are more likely to develop problems later in life.[1] Between 1993 and 2014, prevalences of overweight children aged 6–12 and 13–18 years in Indonesia, respectively, grew from 5.1% to 15.6% and 7.1% to 14.1%.[2] A full array of risk factors was identified for the rise in children's obesity.[3] One major risk factor is the parental body mass index (BMI).[4] Having one overweight parent raised the likelihood of being overweight by almost twice for boys and girls aged 3–8 years and for boys aged 11–16 years. However, only when both parents were overweight did the likelihood of being overweight increase over two times in girls aged 11–16 years.[5] In addition to the significant relationship between parents' BMI and their child's BMI assessed at a single point of time, an increasing number of studies focused on how parental BMI can affect children's BMI trajectories.[6] Parental BMI values were shown to be associated to increased children's BMI Z-score trajectories at the ages of 6–12 years.[7] Fathers and mothers seem to contribute differently to the development of a child's BMI trajectory. While an increase in the father's BMI was associated with an increase in BMI for both male and female children, an increase in the mother's BMI was associated with an increase in BMI for male children only.[8,9] From a longitudinal study, the risk of being overweight at 20 years old was influenced by parental BMI values during one's childhood.[10] Additionally, another study, utilizing self-reported parental weight and height, revealed that the impact of parental BMI values on childhood obesity became more pronounced as the child progressed into adolescence.[11]

Many hypotheses have been proposed to explain why parental BMI values in their offspring's childhood predict their children's BMI trajectories even until early adulthood.[12,13] Researchers indicated that genetic and fetal environments can increase the risk of children's obesity.[14] Many genetic loci were proven to be associated with BMI, but studies suggested that genetics alone cannot be the sole explanation for either the prevalence or the degree of obesity.[15] Another important influential factor explaining the parent–child BMI association is the family environment.[16] Studies have consistently shown that many family environmental factors can moderate expressions of obesogenic genetic tendencies.[17] Parenting styles, the parental socioeconomic status (SES), and parental health behaviors, such as parents' diet and physical activity patterns, all have strong impacts on the food and exercise choices of the child even until adulthood.[18,19] In addition to parental factors, other risk factors contributing to one's increased

BMI trajectory include poor sleep quality, smoking, disadvantaged community settings, and urbanicity.[20–22]

BMI Z-scores have consistently been used as an indicator of children's nutritional status.[23] They can indicate longitudinal changes in children and are comparable across different ages.[24] Although BMI *Z*-scores are commonly used to assess children's overweight and obese status, the number of studies analyzing risk factors for BMI *Z*-score trajectories has been relatively small compared to the studies using raw BMI values.[25,26] Additionally, previous studies on the influence of parental BMI on children's long-term BMI using BMI *Z*-scores as measures showed conflicting findings.[27,28] The present study aimed to examine the influence of Indonesian parental BMI *Z*-scores on the BMI *Z*-score trajectories of their children from ages 5–12 to 21–30 years. We further examined the effects separately by sex of the child. The hypothesis is that Indonesian parental BMI during their offspring's childhood significantly influences their children's BMI trajectories into adulthood, with differential effects by child's sex.

Methods

Study population

The Indonesian Family Life Survey (IFLS) is an extensive longitudinal survey that investigates social, economic, and health-related behaviors at individual, household, and community levels. The inaugural IFLS, conducted in 1993 and 1994, covered 83% of the Indonesian population. The first wave encompassed 13 of 27 provinces, and surveyed 22,000 individuals from 7244 households, using a stratified random sampling technique. The sample represented population heterogeneity and covered the population that resided on four of the five largest Indonesian islands (viz., Sumatra, Java, Kalimantan, and Sulawesi). There have been five waves of data collection in the IFLS: wave 1 (1993–1994), wave 2 (1997–1998), wave 3 (2000), wave 4 (2007–2008), and wave 5 (2014–2015). Face-to-face interviews, were conducted with one or two household members aged 12 years and older utilizing a structured questionnaire. For children under the age of 12 years, parents or caregivers were interviewed. In wave 5, the data collection method transitioned from pen-and-paper personal interviews to computer-assisted personal interviews. Further details on the sampling frame and data collection procedures were discussed elsewhere.[29]

This panel study followed the same respondents across four time points from waves 2 to 5. The main target sample was children aged 5–12 years in wave 2 for whom height and weight data were available. When a family had multiple children, only the eldest was chosen in order to avoid a clustering effect within a family.[30] Both children and parents with extreme BMI Z-scores (below -5 or above 5) were excluded. In wave 2, there were 2031 children aged 5–12 years whose parents' heights and weights were measured. Sample sizes in waves 3, 4, and 5 were 1959, 1461, and 1083 children, respectively (see Fig. 1). The IFLS underwent institutional review board reviews, gaining approval from the RAND Corporation and Indonesian institutions, that thoroughly assessed the study's human subject concerns and adhered to ethical standards.[29,31] Ethical approval for this study was obtained from the Research Ethic Committee of Universitas Ahmad Dahlan, Indonesia (no.: 022302023).

Measures

Dependent variables

The dependent outcome variable was children's BMI *Z*-scores based on their height and weight measurements. Regarding the measures of height and weight, heights were measured using a Seca plastic height board, model 213, which measures to the nearest millimeter. Weights were recorded using a Camry model EB1003 scale, accurate to the nearest tenth of a kilogram.[29,32] The anthropometric measurements for both children and parents were collected by nurses or doctors in waves 2–4 and trained interviewers in wave 5.[29,31] The BMI *Z*-score, which served as an indicator of general adiposity, was calculated in accordance with the World Health Organization (WHO) growth standards, using the age- and sex-specific WHO Anthro plugin.[33]

Independent variables

Parental BMI *Z*-score data from waves 2 and 5, calculated from heights and weights,[29] were used as time-invariant predictors in the models. Parental BMI *Z*-scores in wave 2 served as the main independent variables, while parental BMI *Z*-scores from wave 5 served as control variables. Other predictors of children's BMI trajectories included child sociodemographic characteristics and behaviors that were previously suggested to be related to children's BMI.[2,34–36] These time-invariant predictors were the child's sex (wave 2) and child's health behavioral variables (wave 5) including smoking behavior, self-rated health, physical activity, sleep quality, and green vegetable consumption. This study also included random effects of years of follow-up to address differences in children's BMI trajectories throughout the four waves.

The measure for smoking was based on a binary variable of smoking status, by asking the respondents, "Do you have the habit of smoking self-rolled cigarettes, commercial cigarettes, or cigars?" Self-rated health was assessed by asking the respondents, "What is your current general health condition?" Answers were categorized into "very unhealthy," "sometimes unhealthy," "sometimes healthy," and "very healthy." Respondents were considered to have good self-perceived health if they responded with "sometimes healthy" or "very healthy," while they were considered to have poor self-perceived health if they responded with "sometimes unhealthy" or "very unhealthy." Respondents were asked about the type, duration, and frequency of physical activities they had engaged in over the past week. The cumulative duration of activities across various aspects of life – work, home, and exercise – was converted into metabolic equivalents of task (MET)-minutes and aggregated. This aggregation provided an overall assessment of physical activity in the past week, which was subsequently categorized as "low physical activity" for < 600 MET-min.[37] Sleep quality was assessed by asking respondents "During the past week, how would you assess your sleep quality?" Response categories were "very bad," "bad," "moderate," "good," and "very good." Respondents were considered to have adequate sleep quality if they responded "moderate," "good," or "very good."[38] For dietary habits, participants were asked about their food intake in the previous week. Specifically, they were asked whether they had eaten certain types of food and the intake frequency. For this study, we focused on the daily consumption of green leafy vegetables.[39] The SES level was measured by asking respondents to subjectively rate their economic situation using the question, "If you visualize a six-step ladder, with the first step representing the poorest and the sixth step representing the richest, which step do you think you are currently on?" Respondents perceiving themselves as being on the fourth, fifth, or sixth step were classified as having a high SES level.[40] Geographical setting was measured by asking respondents if they resided in Java-Bali Provinces or elsewhere and whether they lived in an urban or rural area.

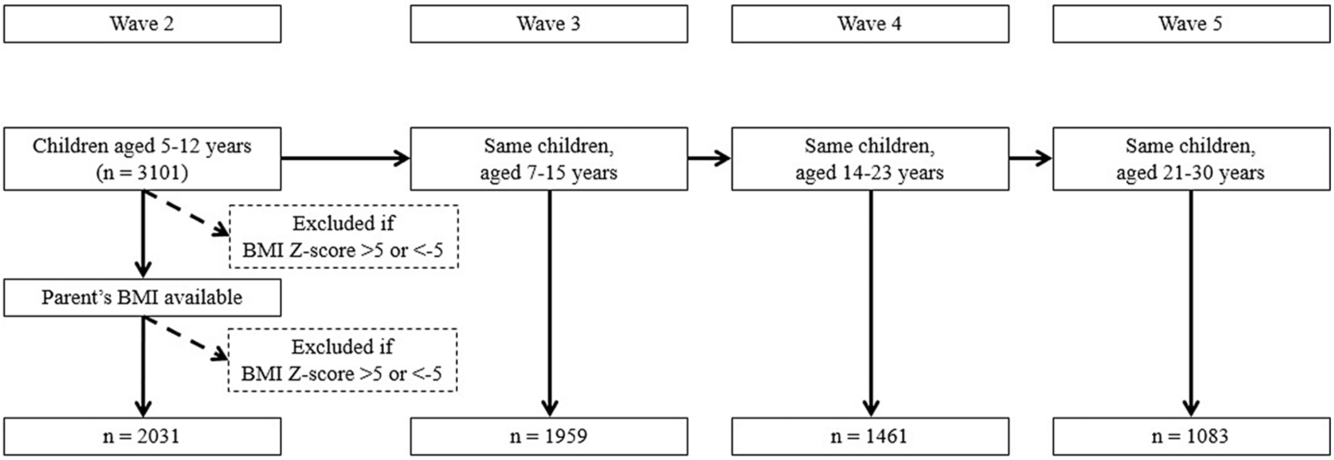

Figure 1. Flow diagram of sample selection by data wave.

Statistical analyses

A two-level growth curve model with an unrestricted covariance matrix for dependence across time was employed to analyze the data. This approach accounted for the hierarchical nature of the dataset, where time (level 1) was nested within children (level 2). This method allowed us to understand the longitudinal progression of children's BMI *Z*-scores from waves 2 through 5. At level 1, the model estimated individual growth trajectories based on BMI *Z*-scores over time. At level 2, the model captured the variability in initial BMI *Z*-scores between children and variations in growth rates among children. In this model, a time indicator called "year" was utilized, which respectively indicated 0, 3, 10, and 17 years of follow-up from waves 2 to 5. Selection of the appropriate functional form for incorporating the personal-level predictor was based on the Akaike information criterion (AIC). Specifically, linear, quadratic, and cubic models were ranked based on their AIC values. The linear model was finally selected because it had the smallest AIC value. Conditional growth curve models were conducted by adding parental BMI Z-scores, family environmental covariates, personal health behavioral factors, year, and parental BMI Z-scores × year interaction terms as covariates. To ensure accurate interpretation of child growth trajectories, BMI z-scores, which are age- and sex-specific according to WHO standards, were employed to allow consistent comparisons over time.[41]

The growth curve model was tested using the following equations.

Level 1:

$$Y_{ti} = \pi_{0i} + \pi_{1i}(\text{year})_{ti} + eti$$

Level 2:

$$\pi_{0i} = \beta_{00} + \sum_{k=0}^{17} \beta_{0k}(\text{covariates})_i + r_{0i};$$

$$\pi_{1i} = \beta 10 + \sum_{k=0}^{17} \beta_{1k}(\text{covariates})_i + r_{1i}.$$

The level 1 equation specifies the level 1 model, where Y_{ti} is the BMI Z-score at time t for child i, π_{0i} is the BMI Z-score of child i at the baseline (wave 2), π_{1i} is the linear growth rate of the BMI Z-score over time for child i, and e_{ti} is the unexplained level-1 residual. The first level-2 equation specifies initial BMI Z-scores across children (π_{0ij}), which were modeled as a function of intercept (β_{00}), covariates (β_{0k}), and a residual (r_{0i}) for the random intercept. The second level-2 equation specifies that linear growth rates (π_{1i}) were modeled as a function of intercepts (β_{10}), covariates (β_{1k}), and a residual for linear growth (r_{1i}).

Before growth curve modeling, we conducted univariate and bivariate analyses. In the univariate analyses, continuous variables were displayed as the mean ± standard deviation, while categorical variables were shown as frequencies along with their respective percentages. Bivariate analyses were conducted using either a chi-squared or Kruskal–Wallis test, with only significant variables later being included in growth curve modeling. All analyses were executed in Stata 14.2 (Stata Corp., College Station, TX, USA). A *p* value of < 0.05 was considered statistically significant.

Results

Table 1 shows descriptive statistics of the sample by wave. Both males and females had a nearly equal representation across all waves. The average age of participants increased in each wave, beginning at 9.33 years in wave 2 and reaching 26.33 years in wave 5. Around 40.93% of participants in wave 2 had a high SES, which had increased to 60.67% in wave 5. Regarding health behaviors, about 36% of participants reported having smoking behaviors across all waves. Around 84% rated their health as healthy, and this pattern remained consistent across waves. Approximately 36% of participants had a low level of physical activity, and around 85% of participants rated their sleep quality as adequate across waves. Nearly 40% of participants had daily green leafy vegetable consumption. In terms of residence, around 43% of participants in wave 2 lived in urban areas, and this had increased to 51.62% in wave 5. A majority of participants (62%) were from the Java-Bali region. Fathers had slightly negative BMI *Z*-scores in wave 2 ($Z = -0.31$), which hovered around zero by wave 5. Mothers, on the other hand, had slightly positive BMI Z-scores in wave 2 ($Z = 0.30$), which had increased to 0.93 in wave 5.

Table 2 provides an overview of the progression of total and sex-specific children's BMI Z-scores over time. Regarding BMI *Z*-scores for the total sample, the *Z*-score began at −0.66 in wave 2 and increased to 0.15 in wave 5. Males had a *Z*-score of −0.71 in wave 2, which had increased to −0.22 in wave 5. In contrast,

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Table 1. Characteristics of the sample population by data wave

	Wave 2	Wave 3	Wave 4	Wave 5
n	2031	1959	1461	1083
Male	1031 (50.76)[a]	991 (50.59)	717 (49.08)	523 (48.29)
Age (years)*	9.33 ± 2.22[b]	12.08 ± 2.23	19.37 ± 2.30	26.33 ± 2.32
High SES level*	826 (40.93)	719 (36.76)	628 (43.07)	654 (60.67)
Smoking habit	548 (36.34)	520 (35.94)	413 (34.97)	379 (35.09)
Healthy self-rated health	1268 (84.08)	1217 (84.11)	985 (83.40)	910 (84.26)
Low physical activity	528 (36.36)	512 (36.78)	426 (37.27)	389 (36.42)
Adequate sleep quality	1237 (85.49)	1185 (85.44)	982 (86.14)	914 (85.74)
Daily green leafy vegetable consumption	577 (39.88)	545 (39.29)	440 (38.60)	437 (40.99)
Urban residence*	884 (43.53)	846 (43.19)	710 (48.60)	559 (51.62)
Java-Bali region	1273 (62.68)	1229 (62.74)	947 (64.82)	672 (62.05)
Fathers' BMI *Z*-scores in wave 2	−0.31 ± 1.04	−0.31 ± 1.05	−0.30 ± 1.04	−0.37 ± 0.99
Mothers' BMI *Z*-scores in wave 2	0.30 ± 1.03	0.30 ± 1.03	0.32 ± 1.02	0.26 ± 0.99
Fathers' BMI *Z*-scores in wave 5	0.03 ± 1.28	0.03 ± 1.27	0.05 ± 1.28	0.03 ± 1.26
Mothers' BMI *Z*-scores in wave 5	0.90 ± 1.22	0.90 ± 1.23	0.96 ± 1.19	0.93 ± 1.19

SES, socioeconomic status; BMI, body mass index.
[a]*n* (%).
[b] mean ± standard deviation.
*$p < 0.05$.

Table 2. Body mass index (BMI) *Z*-scores of children by sex and data wave

	Total				Males				Females			
	Wave 2	Wave 3	Wave 4	Wave 5	Wave 2	Wave 3	Wave 4	Wave 5	Wave 2	Wave 3	Wave 4	Wave 5
n	2031	1959	1461	1083	1031	991	717	523	1000	968	744	560
BMI *Z*-score	−0.66 ± 1.15	−0.66 ± 1.13	−0.46 ± 1.08	0.15 ± 1.25	−0.71 ± 1.15	−0.75 ± 1.11	−0.77 ± 1.04	−0.22 ± 1.24	−0.61 ± 1.14	−0.56 ± 1.14	−0.17 ± 1.03	0.50 ± 1.15
p value for trend	<0.001				<0.001				<0.001			

Data are the mean ± standard deviation.

females began with a *Z*-score of −0.61 in wave 2 and had a more significant increase of BMI *Z*-scores to 0.50 in wave 5.

Using two-level growth curve modeling, we examined whether parental BMI Z-scores in wave 2 influenced children's BMI *Z*-score trajectories (Table 3). In the total sample, fathers' ($\beta = 0.25$) and mothers' ($\beta = 0.19$) BMI Z-scores in wave 2 were significantly and positively associated with children's BMI *Z*-scores across waves, indicating a potential genetic or environmental influence from parents during early life stages. As the years of follow-up increased, there was a notable rise in BMI *Z*-scores for all participants. Other factors, such as urban residence and a high SES level, showed no associations with the children's BMI *Z*-score trajectories when taking into account parental BMI *Z*-scores.

Study results showed different patterns of influence from parents to children by sex. Specifically, fathers' BMI *Z*-scores in wave 2 had a pronounced influence ($\beta = 0.31$) on female children's BMI *Z*-scores compared to the influence of mothers' BMI *Z*-scores ($\beta = 0.17$). Conversely, mothers' BMI *Z*-scores in wave 2 showed a stronger positive association with male children's BMI *Z*-scores ($\beta = 0.22$) than did fathers' BMI Z-scores ($\beta = 0.19$). Interestingly, in wave 5, mothers' BMI *Z*-scores were positively associated with children's BMI *Z*-scores only for females ($\beta = 0.1$), suggesting that mothers still had an influence on their daughters even when they had become adults.

Table 3 shows the results of significant interaction effects of parental BMI and years of follow-up. A significant interaction effect of fathers' BMI Z-scores and years of follow-up was found for male children. To demonstrate the interaction effect, in Fig. 2, we divided fathers' BMI Z-scores into tertiles. The figure shows that the slope of the trajectory increased when fathers' BMI Z-scores increased from the third to the first tertile. This suggests that as BMI Z-scores of male children increased by year, this effect was more pronounced when their fathers had higher BMI Z-scores.

Discussion

This study examined the impacts of parental BMI values during childhood on BMI *Z*-score trajectories of Indonesian school-aged children and separately estimated the effect by sex using a 17-year longitudinal dataset. We utilized a two-level growth curve model to

Table 3. Growth curve modeling of children's body mass index (BMI) *Z*-score trajectories on parental BMI *Z*-scores and sociodemographic characteristics

Characteristic	Total		Males		Females	
	β	SE	β	SE	β	SE
Fathers' BMI *Z*-scores in wave 2	0.25*	0.04	0.19*	0.05	0.31*	0.05
Mothers' BMI *Z*-scores in wave 2	0.19*	0.04	0.22*	0.05	0.17*	0.05
Fathers' BMI *Z*-scores in wave 5	−0.03	0.03	−0.01	0.04	−0.04	0.04
Mothers' BMI *Z*-scores in wave 5	0.04	0.03	−0.02	0.04	0.10*	0.04
Male	−0.37*	0.05				
Age	−0.00	0.01	−0.03*	0.01	0.02	0.01
High SES level	0.04	0.03	0.05	0.04	0.03	0.04
Urban residence	−0.02	0.04	0.03	0.06	−0.08	0.06
Years of follow-up	0.05*	0.01	0.06*	0.01	0.04*	0.01
Fathers' BMI *Z*-scores in wave 2 × Years of follow-up	0.00	0.00	0.01*	0.00	−0.01	0.00
Mothers' BMI *Z*-scores in wave 2 × Years of follow-up	0.00	0.00	0.00	0.00	0.00	0.00

SES, socioeconomic status.
*$p < 0.05$.

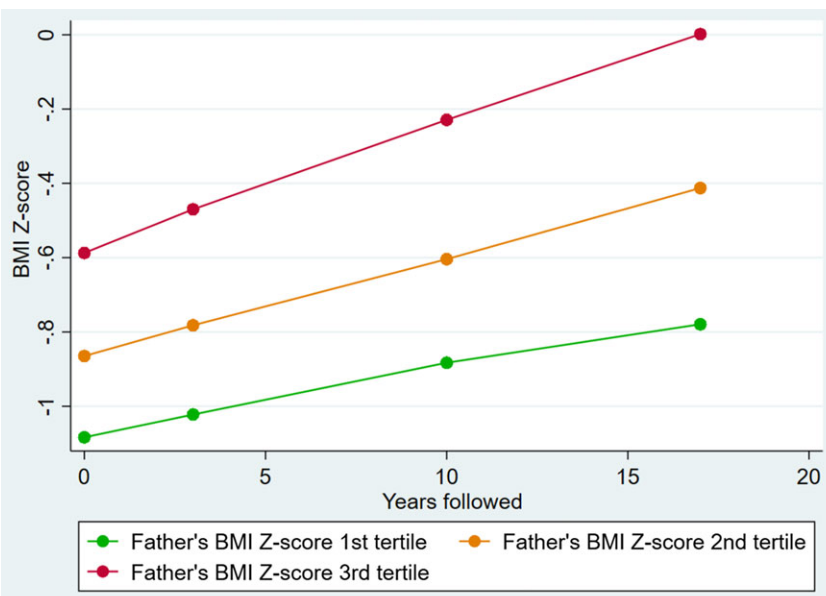

Figure 2. Trajectory of male children's body mass index (BMI) *Z*-scores by levels of fathers' BMI *Z*-scores.

account for the hierarchical structure of the data, with time nested within individual children. Results showed a consistent increase of children's BMI *Z*-scores throughout the survey period. This upward trend was evident in both boys and girls, although girls experienced a more pronounced rise in their BMI *Z*-scores compared to boys. Other studies also reported similar results of a more salient increase in BMI for girls over the years such as that in a longitudinal study conducted in the United States.[42] This study reported that while the overall cohort showed consistent BMI *Z*-scores, individuals with a BMI *Z*-score of < 0 in the first year had experienced significant increases by the fourth year, with girls having a more pronounced gain than boys.[42] The higher BMI Z-score gains in girls compared to boys might be in part influenced by hormonal effects of menarche.[43]

Our findings revealed positive associations between both parents' BMI Z-scores at the baseline and their children's BMI *Z*-scores throughout the study. This finding aligns with previous research which suggested that children's health behaviors and outcomes are often influenced by their parents, either through genetics, a shared environment, or a combination of these, especially in the early stages of life.[44,45] The association between parental and children's BMI values underscores the hereditary aspect of weight-related traits.[46] A stronger genetic predisposition to obesity was correlated with an earlier onset of adiposity rebound, which is associated with an increased risk of being overweight or obese in later life.[14]

In addition to hereditary influences, our study suggested that the family environment in the early stages of life may significantly shape one's BMI trajectory from childhood to adulthood. Families share common environments, including dietary habits, physical activity routines, and socioeconomic conditions.[47] Family dietary and physical activity habits play crucial roles in nutritional

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similarities between parents and their children.[48] Families often have shared eating behaviors influenced by shared preferences and the household food environment.[49] In addition, one's physical activity habits are influenced by social support from family and friends, and by the physical environment near the household, including the availability and accessibility of facilities.[50] Since children often model their behaviors after their parents, parents with unhealthy eating habits or a sedentary lifestyle can inadvertently influence their children to adopt similar behaviors[51–53]

Our study showed sex-specific trends of parental BMI influences. While both parents can affect their children's BMI trajectories, the extent of this influence varied based on the child's sex. Specifically, fathers' BMI *Z*-scores at the baseline had a greater impact on girls, while mothers' BMI Z-scores had more influence on boys. This possibly suggests specific sex-related roles and family dynamics in Indonesia regarding health behaviors and nutrition outcomes.[54] For instance, a mother might be more attentive to her son's dietary needs, while a father might encourage more physical activities from his daughter.[55] Findings of this study also possibly hint at potential genetic factors that differently influence weight trajectories for males and females.[56] In a genome-wide association meta-analysis, the genetic basis of obesity was explored, and 20 of 49 genetic loci that showed sex-specific differences play roles in regulating adipose tissues and insulin biology.[57]

Our study found a significant interaction effect of fathers' BMI values and years of follow-up on male children's BMI values. This result suggests that fathers' BMI during their offspring's childhood is important in determining an increasing BMI trajectory in male children. Lasting effects of fathers' BMI values, either because of genetic influences or early family environmental influences, were also found in prior studies. A longitudinal Canadian study revealed that paternal preconception BMI affected growth rates and mean BMI Z-scores in boys, but not in girls. Boys with a father with a higher BMI were more likely to be overweight or obese.[58]

Our study findings showed that the number of participants with a high SES tended to grow over different waves of data collection. While many studies have explored the relationship between SES and childhood obesity, the results were mixed.[59,60] Some research suggested that children from higher SES backgrounds are at a lower risk of obesity due to better access to health-promoting resources, while others argued the opposite.[61,62] In this study, a high SES level showed no associations with children's BMI *Z*-score trajectories when accounting for parental BMI Z-scores. This suggests that in the current Indonesian context, where rapid changes in lifestyles are taking place, other risky behavioral factors may play more significant roles in children's BMI and obesity, which should be explored in future studies. The increasing urbanization observed in the study, with more participants from waves 2 to 5 living in urban areas, mirrors global trends.[63,64] Previous research often linked urban living with higher obesity rates due to sedentary lifestyles and increased access to unhealthy foods.[65,66] However, this study did not find a direct association between urban residence and children's BMI Z-score trajectories when parental BMI Z-scores were considered.

The main strength of this study is the use of a 17-year longitudinal dataset, which allowed us to track BMI Z-score trajectories over an extended period providing insights into long-term trends and changes. Additionally, by employing a two-level growth curve model, we accounted for the hierarchical structure of the data (time nested within individuals), enhancing the robustness of our findings. The IFLS provided a comprehensive and representative sample of the Indonesian population, which ensures the generalizability of our findings.

The main limitation of the study is the reliance on self-reported data for certain variables, such as smoking behavior, self-rated health, physical activity, and sleep quality, which may introduce potential recall bias and social desirability bias.[67] Despite controlling for several confounding variables, there remains the possibility of unmeasured covariates influencing the observed associations. Future studies should consider additional factors such as cultural practices, local infrastructure for healthy living, and local nutrition and physical activity-related policies. Additionally, although we highlighted the significant role of parental BMI values, our study primarily focused on parents, which potentially overlooks the influences of siblings and other family members on a child's health behaviors and BMI.[68,69] Future research could explore the influence of siblings, especially in families with multiple children, to provide a more comprehensive understanding of family dynamics in children's growth patterns. Finally, while the study emphasized the influence of parental BMI values on children's BMI trajectories, the underlying mechanisms (i.e., genetic, environmental, behavioral, etc.) remain unclear and warrant further investigation.

Conclusions

The escalating trend of childhood obesity is a pressing concern worldwide, and Indonesia is no exception. This study has shed light on the intricate dynamics of childhood obesity within the Indonesian context, emphasizing the profound influence of parental BMI values on children's BMI trajectories. The findings underscore that the roots of childhood obesity are not just individual but are deeply intertwined with familial and environmental factors. Parental BMI values, as highlighted, serve as a significant predictor of a child's BMI trajectory. This association underscores the genetic, environmental, and behavioral influences that parents impart to their children. As children often mirror the habits, behaviors, and lifestyles of their parents, it is imperative to view the challenge of childhood obesity not just as an individual concern but as a familial one.

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