

STRUCTURAL EQUATION MODEL OF DETERMINANTS OF ADOLESCENT BODY MASS INDEX

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ABSTRACT

Adolescence is a critical developmental stage in which Body Mass Index (BMI) is a key indicator of nutritional and health status. In Nigeria, limited research has examined the behavioral, psychosocial, dietary, lifestyle, and socioeconomic factors influencing adolescent BMI within an integrated framework. This study applied Partial Least Squares Structural Equation Modeling (PLS-SEM) using secondary data from the 2018 Nigeria Demographic and Health Survey (NDHS) to investigate both direct and indirect determinants of adolescent BMI. Findings revealed that lifestyle factors were the only construct exerting a significant direct effect on BMI ($\beta = 0.164$, $p < 0.001$), underscoring the critical role of modifiable daily behaviors. Socioeconomic status (SES), while not directly associated with BMI, demonstrated significant indirect effects through its influence on behavioral patterns ($\beta = -0.273$, $p = 0.006$) and lifestyle ($\beta = -0.289$, $p = 0.011$). Other constructs, including dietary, psychosocial, physical activity, and health factors, did not yield significant direct effects. Despite these associations, the model's explanatory power was only moderate ($R^2 = 49\%$), suggesting that unmeasured determinants, such as genetic, biological, and environmental influences, may account for more of the observed variation. The study concludes that adolescent BMI is multidimensional, with lifestyle factors playing a central role, while SES influences outcomes indirectly. It recommended refining measurement tools, expanding research frameworks to include biological and environmental markers, adopting longitudinal study designs, and strengthening methodological capacity in SEM to improve public health research and intervention strategies.

Keywords: Partial Least Squares, Body Mass Index, Coefficient of Determination, Structural Equation Model.

INTRODUCTION

Body Mass Index (BMI) is a practical anthropometric indicator of overall body composition, particularly during adolescence, a period marked by significant physiological and psychosocial development. Unlike its common use as a binary marker for identifying underweight or overweight status, BMI in adolescents reflects a broader array of influences, including lifestyle, environmental, social, and economic contexts. Recognizing these influences is essential for understanding variability in BMI and for guiding health promotion strategies that target optimal adolescent growth and well-being (Vale *et al.*, 2022).

Previous studies have demonstrated that adolescents' BMI is shaped by multiple interacting determinants. For instance, Aynehchi *et al.* (2023) reported that self-efficacy and coping mechanisms influenced BMI indirectly through diet quality and eating behaviour,

highlighting the role of psychological and behavioural determinants. Similarly, Lemes *et al.* (2021) found that physical fitness mediated the relationship between personal and social lifestyle factors and BMI in adolescents, underscoring the importance of lifestyle behaviours in regulating body mass. These findings reflect a broader consensus that BMI outcomes are rarely the result of single influences but rather emerge from interacting pathways involving behavioural, social, and psychological mechanisms.

Socioeconomic and environmental conditions also play a crucial role. In a Brazilian study, Vale *et al.* (2017) demonstrated that food insecurity, parental education, and sleep duration were statistically associated with BMI, with the association mediated by dietary intake and physical activity. Likewise, Mchiza *et al.* (2019) identified physical activity and socioeconomic status as mediators in the relationship between environment and adolescent BMI in a South African cohort. These findings emphasized the importance of studying indirect effects and context-specific variables when analyzing general BMI outcomes. Psychological determinants such as self-efficacy, coping strategies, and emotional well-being are also increasingly recognized in the literature. Aynehchi *et al.* (2023) showed that adolescents with low self-efficacy were more likely to adopt unhealthy dietary patterns, indirectly raising their BMI. This aligns with the work of Chaiton *et al.* (2009), who demonstrated that body image dissatisfaction and depressive symptoms could lead to greater adiposity through psychological and behavioural mechanisms.

Biological and genetic predispositions further interact with lifestyle and psychosocial factors in shaping BMI. For example, Kaainen *et al.* (2010) examined the fat mass and obesity-associated FTO gene variants in Finnish adolescents. They found that genetic risk factors influenced BMI indirectly through behaviours such as smoking and physical inactivity. This integration of genetic and behavioural data highlights how predisposition amplifies the effects of modifiable risk factors. Environmental influences also remain critical. (2017) modelled how neighbourhood safety and access to recreational facilities impacted BMI across age and gender, mediated by physical activity. Their findings underscore the importance of designing interventions that are sensitive to age, gender, and neighbourhood context. Overall, these studies demonstrated that adolescent BMI is shaped by a complex network of biological, psychological, social, and environmental factors rather than isolated variables. However, there is a lack of integrated frameworks that simultaneously capture these multiple domains and quantify both their direct and indirect effects, particularly among Nigerian adolescents. To address this gap, an advanced analytical approach, such as Structural Equation Modelling (SEM), is used to model BMI among Nigerian adolescents. Structural Equation modelling (SEM) is a robust statistical technique that enables

examination of complex relationships among variables, including both direct and indirect pathways, providing insights into the underlying mechanisms influencing BMI outcomes.

MATERIALS AND METHODS

This section presents the methodology adopted to analyze the determinants of adolescents' Body Mass Index (BMI) in Nigeria using Structural Equation Modelling. It outlines the study design, data source, analytical framework, and modelling strategy.

Study Design

The study adopts a quantitative, cross-sectional design. Secondary data are analysed at a single time point, allowing for the exploration

of associations and predictive pathways.

Source of Data

The data for this study were extracted from the 2018 Nigeria Demographic and Health Survey (NDHS), conducted by the National Population Commission (NPC) in collaboration with International Classification of Functioning, Disability and Health (ICF) under the DHS Program. The survey includes a wide range of variables relevant to demographic, health, nutrition, and socioeconomic indicators. For this study, adolescents aged from 10 to 19 years were extracted from the NDHS.

Variable Description

Category	Latent Construct	Indicators (Observed Variables) = Type
Dependent Variable	BMI	Body Mass Index = Continuous
Independent Variables	Demographics	Age = Continuous
		Sex = Discrete
	Socioeconomic Status (SES)	Class = Discrete
		Religion = Discrete
		Ethnic = Discrete
		Address = Discrete
		Housing = Discrete
		Household = Discrete
		EducationStatusF = Discrete
		EducationStatusM = Discrete
Mediating Variables	Behavioural Factors	OccupationF = Discrete
		OccupationM = Discrete
		SECgroup = Discrete
		Location = Discrete
		Meals = Discrete
		Eating = Discrete
		Snacks = Discrete
		SnackType = Discrete
		Juice = Discrete
		Volume = Discrete
Dietary Factors	FoodType = Discrete	
	Fruits = Discrete	
Lifestyle/Sedentary	Sleeping = Discrete	
	Television = Discrete	
	Computer = Discrete	
	Transportation = Discrete	
Physical Activity	Exercise = Discrete	
	ExerciseType = Discrete	
	ExerciseFrequency = Discrete	
Psychosocial Factors	ExerciseDuration = Discrete	
	Perception = Discrete	
	Knowledge = Discrete	
	Attitude = Discrete	
	Habit = Discrete	
	Consciousness = Discrete	
Health Factors	Maintenance = Discrete	
	Medication = Discrete	
	Illness = Discrete	
		Systolic = Continuous

Diastolic = Continuous
 Circumference = Continuous
 BMI Percentile Range = Discrete
 WCP Percentile Range = Discrete
 SD Score = Continuous

Model Specification

Structural Equation modelling is a multivariate statistical technique that tests hypothesized relationships between observed and latent variables. The general mathematical expression for SEM comprises two sub-models: the measurement model, which relates observed variables to latent constructs, and the structural model. The basic equations are:

Measurement Model

The measurement model links latent constructs (ξ, η) with their observed indicators (x, y). It is expressed as according to Kline (2015) formulation:

$$x = \Lambda_x \xi + \delta \quad (1)$$

$$y = \Lambda_y \eta + \epsilon \quad (2)$$

Where;

x = vector of observed indicators for exogenous constructs (e.g., Wealth_Index, fruits, exercise).

y = vector of observed indicators for endogenous constructs BMI

Λ_x, Λ_y = matrices of loading for x and y , respectively

ξ, η = latent exogenous and endogenous variables (ξ_1 = SES, ξ_2 = Dietary Behaviour, ξ_3 = Physical Activity, ξ_4 = Psychosocial Well-being).

δ, ϵ = measurement errors

Structural Model

The structural model specifies the hypothesized causal relationships among latent constructs:

$$\eta = B\eta + \Gamma\xi + \zeta \quad (3)$$

Where:

B = coefficients among endogenous latent variables

Γ = coefficients from exogenous to endogenous variables

ζ = residual errors

For this study, BMI (η) is modelled as a function of physical activity, dietary behaviour, socioeconomic status, and psychosocial well-being (ξ).

An example model for BMI determination may be expressed as:

$$BMI = \gamma_1 \xi_1 + \gamma_2 \xi_2 + \gamma_3 \xi_3 + \gamma_4 \xi_4 + \zeta$$

Where:

η_1 = Body Mass Index (BMI) (the dependent latent variable).

ξ_1 = Socioeconomic Status (SES).

ξ_2 = Dietary Behaviour.

ξ_3 = Physical Activity.

ξ_4 = Psychosocial Well-being.

γ_1 to γ_4 = path coefficients representing the direct effects of each exogenous latent variable on BMI.

ζ = disturbance term for the endogenous latent variable (η_1), representing the unexplained variance in BMI.

SEM Estimation Technique

Partial Least Squares SEM (PLS-SEM) was employed for model estimation in this study. PLS-SEM, as a variance-based technique, is optimal for predictions and complex models. It does not require

normally distributed data and is much more effective with smaller sample sizes than Co-Variance Based SEM (CB-SEM). The estimation is done in a systematic and iterative process. The first step is the measurement model (outer model) estimation, where each latent variable is calculated as a weighted sum of its indicators:

$$\xi_j = \sum_i w_{ij} x_i$$

j : index for latent construct

These weights are iteratively updated using the rule:

$$w^{(t+1)} = \text{normalize}(X^T Y)$$

until convergence is achieved. Afterward, the structural model (inner model) is estimated by regressing the endogenous variable ' η ' (Body Mass Index) on the exogenous latent variables (ξ_1 to ξ_5) to derive the path coefficients. Since PLS-SEM is non-parametric, bootstrapping was effectively used to assess the statistical significance of the path coefficients and loadings. This process (bootstrapping) generates thousands of subsamples to construct empirical confidence intervals, enabling hypothesis testing without assuming the data follow any specific distribution.

Model-Implied Covariance and Estimation

The covariance structure underlying SEM is defined as based on Kline (2015) definition:

$$\Sigma(\theta) = \Lambda_y(I - B)^{-1}\Gamma\Phi\Gamma^T(I - B)^{-T}\Lambda_y^T + \Lambda_y(I - B)^{-1}\Psi(I - B)^{-T}\Lambda_y^T\Theta_\epsilon \quad (4)$$

Where;

Φ : covariance of exogenous constructs (ξ)

Ψ : covariance of structural errors (ζ)

Θ_ϵ : measurement error covariance of y

$\Sigma(\theta)$: model-implied covariance matrix, compared with the sample covariance S for estimation.

Parameters are estimated by minimizing the discrepancy between S and $\Sigma(\theta)$. The most common estimation method is Maximum Likelihood (ML), which minimizes the following fitting function under the assumption of multivariate normality:

$$F_{ML} = \log | \Sigma(\theta) | + \text{tr}(S\Sigma(\theta)^{-1}) - \log | S | - p \quad (5)$$

Where:

$\Sigma(\theta)$ = model-implied covariance matrix

S = sample covariance matrix

p = number of observed variables

This estimation process provides estimates of the following parameters from equation(4):

Factor Loadings (λ)

Structural Path Coefficients (β)

Error Variances (δ, ϵ)

The model is then evaluated using goodness-of-fit indices as described earlier to determine how well the theoretical model fits the observed data.

PLS-SEM Algorithm

PLS-SEM estimation proceeds in the following steps:

i. Outer model (measurement): latent variable scores are approximated by linear combinations of indicators:

$$\xi_j = \sum_i w_{ij} x_i$$

$$\eta_k = \sum_{i=1}^{qk} w_{ki} x_{ki} \quad (6)$$

ii. Inner model (structural): latent endogenous constructs are regressed on exogenous constructs.

$$\eta_j = \beta_{j0} + \sum_{k \rightarrow j} \beta_{jk} \eta_k + \sum_{m \rightarrow j} \gamma_{jm} \xi_m + \zeta_j \quad (8)$$

This estimates the path coefficients between the latent variables.

iii. Inference: bootstrapping provides standard errors and significance for path coefficients and factor loadings.

iv. Iterative weight updates: weights are refined until convergence using correlations between indicators and latent variables.

$$w_{ji}^{(t+1)} \propto \text{Cor}(x_{ji}, \xi_j^{(t)}) \quad (9)$$

$$w_{ji}^{(t+1)} = \text{Cor}(x_{ji}, \xi_j^{(t)}) / \sqrt{\sum_i [\text{Cor}(x_{ji}, \xi_j^{(t)})]^2} \quad (9a)$$

w_{ji} : weight of indicator i for latent variable j

x_{ji} : observed indicator i of construct j

$\xi_j^{(t)}$: estimated latent variable score at iteration t

t : iteration number

$\text{Cov}(\cdot)$: covariance

$\text{Cor}(\cdot)$: correlation

Model Evaluation

Measurement model: reliability (Cronbach's alpha, Composite Reliability), validity (Average Variance Extracted, Fornell-Larcker criterion).

Composite Reliability (ρ^c): Assesses internal consistency reliability.

$$\rho^c = \frac{(\sum \lambda_i)^2}{(\sum \lambda_i)^2 + \sum \text{Var}(\delta_i)} \quad (10)$$

Acceptable value: $\rho^c > 0.7$

Average Variance Extracted (AVE): Measures convergent validity.

$$\text{AVE} = \frac{\sum \lambda_i^2}{\sum \lambda_i^2 + \sum \text{Var}(\delta_i)} \quad (11)$$

Acceptable value: $\text{AVE} \geq 0.5$

Fornell-Larcker Criterion: Assesses discriminant validity.

$$\sqrt{\text{AVE}_A} > |r_{AB}| \quad (12)$$

Structural model: path coefficients, R^2 (explained variance), Q^2 (predictive relevance), effect sizes (f^2).

Effect Size (f^2): Measures the contribution of an exogenous construct.

$$f^2 = \frac{R_{included}^2 - R_{excluded}^2}{1 - R_{included}^2} \quad (13)$$

Interpreted as 0.02 (small), 0.15 (medium), or 0.35 (large).

Predictive Relevance (Q^2): Assesses out-of-sample predictive power.

$$Q^2 = \frac{SSE}{SSO} \quad (14)$$

Where SSE is the sum of squares of prediction errors, and SSO is the sum of squares of observations. $Q > 0$ indicates predictive relevance.

Goodness-of-fit: Standardized Root Mean Square Residual (SRMR).

$$\text{SRMR} = \sqrt{\frac{2 \sum_{i=1}^p \sum_{j=1}^i (\theta_{ij} - \sigma_{ij})^2}{8_{ii} \theta_{jj}}}{p(p+1)}} \quad (15)$$

Acceptable fit: $\text{SRMR} < 0.08$

RESULTS

Table 4.1 shows that the respondents are predominantly adolescents with an average age of 14.55 years (SD = 1.89), indicating a uniform age distribution within the sample. The mean Body Mass Index (BMI) of 20.39 (SD = 11.51) suggests that most participants fall within the normal weight range, although the high variability implies the presence of underweight and overweight individuals. The mean systolic blood pressure of 108.41 mmHg (SD = 12.83) and diastolic pressure of 71.07 mmHg (SD = 10.57) are within normal limits for adolescents, indicating healthy cardiovascular profiles. The mean waist circumference of 70.28 cm (SD = 32.45) and waist circumference percentile range of 1.03 (SD = 0.18) reflect moderate to minimal variation in abdominal measurements among participants. Similarly, the mean height of 157.89 cm w (SD = 13.14cm) and weight of 50.49 kg (SD = 11.59kg) are consistent with expected growth patterns for this age group. The mean SD score was 18.45 (SD = 13.79), indicating some variability in standardized growth measures across respondents. Overall, these results indicated that the sample comprises a relatively healthy adolescent population with generally normal anthropometric and physiological characteristics, though some variation in body composition is evident.

Table 2: Descriptive statistics of BMI on adolescents

	Mean	Median	max	Standard deviation
SDscore	18.446	24	54	13.786
WCpercentilerange	1.033	1	2	0.178
Age	14.552	15	19	1.89
circumference	70.284	68.6	770.4	32.448
Diastolic	71.071	70	110	10.574
Systolic	108.411	110	160	12.829
BMI	20.391	19.4	35.1	11.514
Height	157.89	158.74	186.9	13.14
Weight	50.49	50	131	11.59

Correlation Analysis

Figure 2 shows the relationship between BMI and a set of hypothesized mediator variables (Behavioural, Psychosocial, Lifestyle, Physical Activity, Dietary, and Health). The strongest, albeit still weak-to-moderate, positive correlation is found between BMI and the Health variable ($r=0.27$), suggesting a positive association between BMI and the measure of 'Health'. Lifestyle also shows a weak positive correlation with BMI ($r = 0.11$). In contrast, Behavioural ($r = -0.07$) shows a very weak negative correlation. The remaining mediators, Psychosocial ($r=0.01$), Physical Activity ($r=0.02$), and Dietary ($r=0.02$), have correlations close to zero, showing essentially no linear link with BMI. Additionally, the mediator variables themselves exhibited some weak intercorrelations, such as Psychosocial and Health ($r=0.14$), and a weak negative correlation between Psychosocial and Dietary ($r=-0.16$). Figure 2 shows the linear relationships between SES (Socioeconomic Status) and the same set of mediator variables. This matrix shows that SES has very weak linear associations with all the mediator variables. The strongest correlation observed is a weak negative relationship between SES and Behavioural ($r = -0.11$), indicating a slight tendency for higher SES to be associated with lower Behavioural scores. All other mediator variables have correlations with SES that are very close to zero, ranging from -0.07 (Psychosocial) to 0.06 (Dietary), suggesting that SES, as measured, is not linearly related to these potential mediators. The correlation matrix among the mediator variables is identical to that of BMI and the mediating plot, reflecting the internal consistency of those variables, regardless of their relationship with BMI or SES.

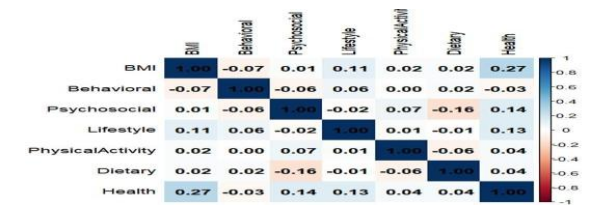


Figure 1: Partial correlation between BMI and the mediating variables

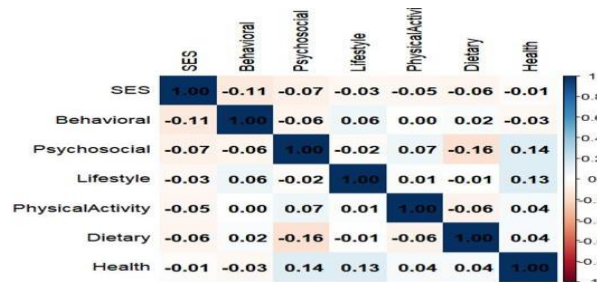


Figure 2: Partial correlation between the independent and the mediating variables

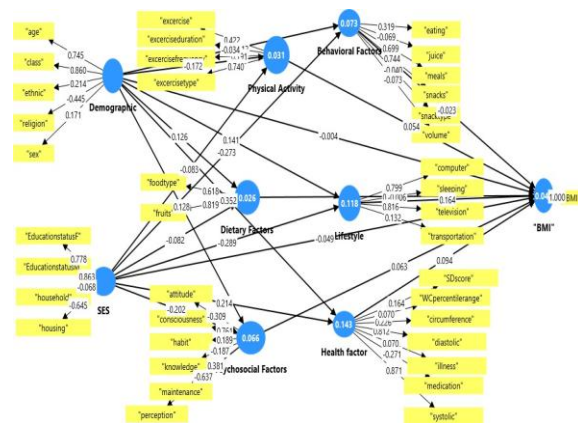


Figure 3: PLS SEM algorithm on the BMI of adolescents

Figure 3 presents the results of the Partial Least Squares Structural Equation modelling (PLS-SEM) algorithm, illustrating the structural relationships and measurement model for predicting Body Mass Index (BMI) in adolescents. The model revealed a complex web of influences, with Lifestyle emerging as the sole statistically significant direct predictor of BMI (Path Coefficient = 0.164), indicating a positive association where certain lifestyle factors are linked to an increase in adolescent BMI. In contrast, other hypothesized direct paths, including those from Behavioural, Dietary, Psychosocial Factors, and Physical Activity to BMI, demonstrated negligible and statistically nonsignificant path coefficients, suggesting they do not exert a substantial direct influence within this model. The explanatory power of the model, as indicated by the R-squared value of 0.49 for the BMI construct, is moderate, meaning that the collective variables included explain only 49% of the variance in adolescent BMI. This low predictive power is further underscored by the weak R-squared values for other endogenous constructs such as Health Factor ($R^2 = 0.143$) and Lifestyle ($R^2 = 0.118$). Importantly, the algorithm output also highlighted potential measurement model challenges, as indicated by several low factor loadings linking the latent constructs to their respective indicators, warranting caution in interpretation and suggesting avenues for future instrument refinement. Ultimately, Figure 3 showed that while lifestyle is a critical direct factor, the overall model structure explained only a small portion of the variation in BMI, suggesting the presence of other significant unmeasured determinants.

Model Assessment

Table 3 (measurement model assessment) shows significant reliability challenges across multiple constructs. Cronbach's Alpha values for all constructs fall substantially below the acceptable threshold of 0.70, indicating limited internal consistency. While Dietary Factors and SES demonstrated adequate convergent validity with AVE values above 0.50, the overall measurement model requires careful interpretation given these reliability limitations.

Table 3: Measurement Model Assessment (Reliability and Validity Metrics)

Construct	Cronbach's Alpha	Composite Reliability (rho_a)	Composite Reliability (rho_c)	AVE
Behavioural Factors	0.146	0.227	0.34	0.192
Demographic	0.122	0.536	0.41	0.313
Dietary Factors	0.103	0.109	0.685	0.526
Health factor	0.349	0.553	0.41	0.226
Lifestyle	0.185	0.475	0.56	0.333
Physical Activity	0.061	0.106	0.161	0.284
Psychosocial Factors	0.091	0.275	0.008	0.216
SES	0.125	0.667	0.278	0.442

Discriminant Validity Assessment

Discriminant validity assessment using the Fornell-Larcker criterion demonstrated mixed results. The square root of AVE (diagonal values) exceeded inter-construct correlations for most constructs,

supporting discriminant validity. However, the generally low AVE values across constructs suggest that the measurement model would benefit from refinement in future studies.

Table 4: Discriminant Validity Assessment (Fornell-Larcker Criterion)

Construct	BMI	Behavioural	Demographic	Dietary	Health	Lifestyle	Physical Activity	Psychosocial	SES
BMI	1								
Behavioural	0.018	0.439							
Demographic	0.066	0.013	0.56						
Dietary	0.024	0.003	0.14	0.725					
Health	0.084	-0.049	0.314	0.039	0.475				
Lifestyle	0.177	0.202	0.191	0.112	-0.006	0.577			
Physical Activity	0.056	-0.062	-0.157	-0.032	-0.014	-0.017	0.533		
Psychosocial	0.086	0.055	0.163	0.083	0.001	0.081	0.02	0.465	
SES	-0.095	-0.267	-0.173	-0.103	0.153	-0.314	-0.053	-0.224	0.665

All Variance Inflation Factor (VIF) values are well below the conservative threshold of 3.0, indicating no concerning multicollinearity issues among the indicators. This confirms that the measurement model does not exhibit redundancy or excessive correlation among predictor variables, thereby strengthening the reliability of the structural parameter estimates.

direct effect on adolescent BMI ($\beta = 0.164, p < 0.001$). This positive relationship indicated that lifestyle factors are associated with increased BMI in the study population. None of the other hypothesized direct paths to BMI reached statistical significance at the 95% confidence level.

Table 5: Variance Inflation Factors (VIF)

Selected Indicators	VIF
Education Status (Father)	1.658
Education Status (Mother)	1.701
Age	1.63
Systolic Blood Pressure	1.51
Diastolic Blood Pressure	1.487
All other indicators	< 1.20

The structural model analysis revealed that among all factors examined, only Lifestyle demonstrated a statistically significant

Table 6: Direct Effects on BMI - Hypothesis Testing

Hypothesis	Path	β Coefficient	t-value	p-value
H1	Behavioural → BMI	-0.023	0.517	0.605
H2	Demographic → BMI	-0.004	0.037	0.97
H3	Dietary → BMI	-0.006	0.153	0.879

Hypothesis	Path	β	t-value	p-value
H4	Health → BMI	0.09	0.478	0.633
H5	Lifestyle → BMI	0.16	4.134	<0.001
H6	Physical Activity → BMI	0.05	1.284	0.199
H7	Psychosocial → BMI	0.06	1.172	0.241
H8	SES → BMI	-0.049	1.13	0.259

Table 7 shows that several significant indirect relationships emerged from the analysis. Socioeconomic status (SES) demonstrated substantial influence, showing significant negative effects on both Behavioural factors ($\beta = -0.273$, $p = 0.006$) and Lifestyle ($\beta = -0.289$, $p = 0.011$). Demographic factors strongly predict Health outcomes ($\beta = 0.352$, $p = 0.001$), while SES shows a marginal positive effect on Health ($\beta = 0.214$, $p = 0.084$).

Table 7: Significant Indirect Relationships

Relationship	β Coefficient	t-value	p-value
Demographic → Health	0.352	3.253	0.001
SES → Behavioural	-0.273	2.747	0.006
SES → Lifestyle	-0.289	2.539	0.011
SES → Health	0.214	1.729	0.084

The model demonstrated varying explanatory power across the endogenous constructs. The highest R^2 value is observed for BMI (49%), indicating a moderate level of variance explained by the predictors. This is followed by Health Factor (14.3%) and Lifestyle (11.8%), both of which show weak explanatory power. Other constructs, including Behavioral Factors (7.3%), Psychosocial Factors (6.6%), Physical Activity (3.1%), and Dietary Factors (2.6%), exhibited very low R^2 values, indicating limited predictive power of the exogenous variables. Overall, while the model moderately explained variations in BMI, it demonstrated weak predictive power for the mediating constructs, implying that additional factors may influence these relationships.

Table 8: Model Explanatory Power

Dependent Construct	R-squared	R-squared Adjusted
BMI	0.49	0.43
Behavioural Factors	0.073	0.069
Dietary Factors	0.026	0.022
Health factor	0.143	0.14
Lifestyle	0.118	0.114
Physical Activity	0.031	0.027
Psychosocial Factors	0.066	0.062

The effect size analysis shows that, despite some statistically significant relationships, the practical effect sizes are predominantly

small to negligible. The strongest effects are observed for the Demographic → Health factor ($f^2 = 0.140$) and the SES → Lifestyle factor ($f^2 = 0.092$), but even these fall below the medium effect threshold. This indicated that while some relationships are statistically detectable, their practical significance in explaining variance is limited.

Table 9: Practical Significance (f^2 Effect Sizes)

Path Relationship	f^2 Value
Demographic → Health factor	0.14
SES → Lifestyle	0.092
SES → Behavioural Factors	0.078
SES → Health factor	0.052
Lifestyle → BMI	0.024
All other paths	< 0.020

DISCUSSION

This study reinforces the evidence presented in the literature review that a complex interplay of behavioural, psychosocial, and contextual factors shapes adolescent BMI. Consistent with prior findings (Mbada *et al.*, 2009; Micklesfield *et al.*, 2021), lifestyle emerged as the only significant direct predictor of BMI, emphasizing the importance of daily modifiable behaviours such as dietary habits, sleep patterns, screen time, and physical activity routines in determining adolescent weight status. However, the model's explanatory power was moderate ($R^2 = 0.49$), suggesting that although lifestyle significantly contributed to BMI, substantial variation remains unaccounted for, likely attributable to genetic, hormonal, and environmental influences, as noted by earlier scholars.

The indirect pathways identified align with previous research indicating that socioeconomic status (SES) affects BMI through behavioural and lifestyle mechanisms rather than direct influence (Yetubie *et al.*, 2010). This result highlights how socioeconomic conditions shape adolescents' access to nutritious foods, recreational opportunities, and health-related choices, which in turn influence body composition. Despite these relationships, the observed effect sizes were modest, consistent with prior studies reporting weak but meaningful associations among SES, lifestyle, and BMI in similar populations.

Additionally, as seen in several earlier studies, measurement reliability emerged as a methodological limitation; low indicator loadings may have attenuated the strength of observed relationships, thereby constraining the model's predictive capacity. Overall, the findings support existing literature emphasizing the central role of lifestyle and the contextual influence of SES (see Oguntade *et al.*, 2024), while reaffirming that adolescent BMI is a multifactorial outcome shaped by broader biological, social, and environmental determinants. This underscores the need for future studies to adopt more robust measurement instruments and integrative models that capture these diverse influences more comprehensively.

Conclusion

In conclusion, this study revealed that lifestyle factors were significantly and positively associated with BMI, whereas most other hypothesized relationships were not statistically significant and showed weak correlations. Regarding the quantification of direct effects, lifestyle emerged as the only construct with a statistically significant direct path to BMI ($\beta = 0.164$, $p < 0.001$). Behavioural, dietary, psychosocial, physical activity, and socioeconomic factors did not show significant direct effects, underscoring the predominant role of daily modifiable lifestyle behaviours in shaping adolescent body composition. The findings also indicated notable indirect pathways, particularly where socioeconomic status influenced BMI through behavioural and lifestyle mediators. However, the model's overall explanatory power was moderate ($R^2 = 0.49$), suggesting that although nearly half of the variance in BMI is accounted for by the included constructs, a substantial proportion of the variation remains unexplained, likely attributable to genetic, environmental, or biological factors beyond the model's scope.

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