







Shah Fahad Firdos

Department of Pathology Sher-i- Kashmir Institute of Medical Sciences, Soura

Email: shahfahad0922@gmail.com

Received: 18 March 2025 Published: 25 May 2025

Revised: 27 March 2025 DOI: https://doi.org/10.59733/medalion.v6i2.194
Accepted: 18 April 2025 Link Publish: https://medalionjournal.com/index.php/go

Abstract

Childhood obesity has emerged as a formidable public health challenge globally, with a marked surge in prevalence across urban populations of low- and middle-income countries, including India. Amidst the nutritional transition driven by urbanization, lifestyle shifts among children—especially increased consumption of energy-dense processed foods and reduced physical activity—warrant urgent epidemiological attention. This study aimed to longitudinally assess the impact of junk food consumption and lifestyle parameters on body mass index (BMI) and metabolic markers among school-going children aged 6-14 years in urban Srinagar, Kashmir. A prospective observational study was conducted over a 12-month period involving 100 randomly selected children from both government and private schools. Baseline and endline assessments included anthropometric measurements, structured dietary and lifestyle questionnaires, and biochemical evaluations (fasting glucose and serum cholesterol). Operational definitions adhered to CDC and WHO BMI percentile standards. Statistical analyses included descriptive statistics, paired t-tests, and correlation/regression modeling to evaluate associations between lifestyle variables and changes in BMI. At baseline, 7% of participants were classified as overweight, with none obese; by endline, overweight prevalence increased to 8%, and obesity emerged in 2%. Significant correlations were observed between frequent junk food consumption and elevated BMI percentiles (p < 0.05), as well as moderate elevations in fasting glucose and cholesterol levels in children with high dietary risk profiles. Physical activity declined marginally over the study period, while screen time increased, though these were not independently predictive of BMI changes after adjustment. The findings underscore an insidious trend of early-life weight gain and metabolic shifts, largely attributable to unhealthy dietary practices entrenched in the urban lifestyle. Comparative analysis aligns with national and regional studies affirming junk food as a primary driver of pediatric adiposity. Socioeconomic and school-type differences revealed nuanced behavioral patterns, suggesting a need for stratified intervention models. The study delineates a clear trajectory of obesity risk among urban children in Srinagar, underscoring the urgent necessity of school-based preventive strategies, parental sensitization, and policy-level regulation of obesogenic exposures. Proactive, multisectoral interventions are imperative to arrest the burgeoning epidemic of childhood obesity and its downstream health consequences.

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Keywords: Childhood obesity, Urban children, Junk food consumption, Lifestyle factors, Anthropometry, Srinagar, Metabolic outcomes, Pediatric health, BMI percentiles, Preventive interventions

Introduction

Childhood obesity has emerged as a significant global public health concern. According to the World Health Organization (WHO), in 2022, approximately 37 million children under the age of five were overweight, with nearly half residing in Asia. The global prevalence of overweight and obesity among children and adolescents aged 5–19 has risen dramatically from just 8% in 1990 to 20% in 2022. The implications of childhood obesity are profound, extending beyond immediate health concerns to long-term consequences. Overweight and obese children are more likely to remain obese into adulthood and are at increased risk of developing noncommunicable diseases (NCDs) such as diabetes, cardiovascular diseases, and certain types of cancer at a younger age. The WHO has recognized childhood obesity as a global epidemic, emphasizing the urgent need for preventive measures.

India is witnessing a rapid transition in its health landscape, with childhood obesity becoming an emerging concern. A systematic review and metaanalysis encompassing 21 studies conducted from 2003 to 2023, involving 186,901 children, revealed a pooled prevalence of childhood obesity at 8.4%, with significant regional variability ranging from 3% to 22.8% among school children aged 6 to 16 years. Urban areas in India exhibit notably higher rates of childhood obesity compared to rural regions. A systematic review highlighted that urban regions have a pooled prevalence of 9.0%, while rural areas report a prevalence of 4.0%. Factors contributing to this disparity include increased consumption of energy-dense foods, sedentary lifestyles, and reduced physical activity in urban settings. Early-life interventions are critical in preventing childhood obesity. Research indicates that the early years, particularly from birth to age five, are crucial for establishing healthy dietary and physical activity patterns. Interventions targeting this period can significantly reduce the risk of obesity and its associated health complications in later life. A study presented at the European Congress on Obesity found that a child's body mass index (BMI) at age six is a strong predictor of future obesity, with each one-unit BMI increase at age six more than doubling the risk of being overweight or obese at 18. These findings underscore the necessity of implementing preventive strategies early in life to combat the growing obesity epidemic.

Srinagar, the capital city of Jammu and Kashmir, is experiencing rapid urbanization and lifestyle changes, making it pertinent to study the prevalence and determinants of childhood obesity in this region. A cross-sectional study conducted in Kashmir involving 2,024 schoolchildren aged between 6 and 18 years reported that 4.64% were obese and 6.69% were overweight. The study also found that urban children exhibited a higher percentage of obesity (5.00%) compared to their rural counterparts (4.22%). Despite these findings, there is a paucity of longitudinal data examining the progression of obesity and its associated risk factors among children in Srinagar. Understanding the dynamics of childhood obesity in this specific urban setting is essential for developing targeted interventions and informing public health policies.

Objectives of the Study

To longitudinally assess the prevalence and progression of overweight and obesity among children aged 6 to 14 years in urban Srinagar, with a focus on evaluating changes in anthropometric parameters, dietary patterns, physical activity, screen time, and sleep behaviors over one year, and to determine the associations between these lifestyle factors and obesity risk, thereby generating evidence-based recommendations for early preventive interventions in similar urban pediatric populations.

Materials and Methods



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This study adopted a prospective longitudinal observational design to assess trends and determinants of childhood obesity over a period of one year. Such a design was chosen to enable the observation of changes in anthropometric, behavioral, and biochemical parameters in a real-world setting without manipulation of variables. Longitudinal tracking facilitated capturing the progression of obesity-related indicators and their potential association with lifestyle factors such as dietary habits, physical activity, screen exposure, and sleep duration. The research was conducted in urban schools located within Srinagar city, the summer capital of Jammu and Kashmir. The setting included both government and private institutions to ensure diversity in socioeconomic status and schooling environments. Srinagar represents a rapidly urbanizing area in the Himalayan region, where shifts in dietary patterns, increasing availability of fast food, and reduced outdoor physical activity are contributing to emerging public health challenges like childhood obesity.

Study Population

The target population comprised children aged 6 to 14 years enrolled in both government and private schools within the urban limits of Srinagar. This age group was selected to cover a critical developmental phase from early childhood to adolescence, a period highly sensitive to nutritional and lifestyle influences. The inclusion of both school types allowed for broader representation across socio-demographic backgrounds.

Sample Size

A total of 100 children were selected using a simple random sampling technique from a pool of eligible participants across five selected schools. The sample size, while modest, was considered sufficient for a pilot-level longitudinal observational study. The goal was to ensure manageability of follow-up over a full year while still enabling meaningful statistical analysis of patterns and associations.

Inclusion and Exclusion Criteria

Children were included if they were enrolled full-time in selected schools, aged between 6 and 14 years, and had parental/guardian consent. They also needed to be medically fit to participate in anthropometric and biochemical assessments. Children with known chronic illnesses, hormonal disorders (e.g., hypothyroidism, Cushing's syndrome), or those on long-term medications affecting weight or metabolism were excluded to avoid confounding factors. Those unwilling to participate or lost to follow-up were also excluded.

Data Collection Tools

Data were collected using a combination of standardized tools and validated instruments. Anthropometric measurements included weight, height, BMI (Body Mass Index), and waist circumference, measured using calibrated devices as per WHO protocols. A structured questionnaire, developed and validated through expert review and pilot testing, was used to capture self-reported (or parent-reported for younger children) data on junk food consumption, physical activity levels, screen time, and sleep duration. Additionally, biochemical parameters, including fasting blood glucose and serum cholesterol levels, were obtained at baseline and after one year using standard venipuncture and laboratory testing methods in a certified diagnostic laboratory.

Operational Definitions

Obesity was classified using BMI-for-age percentiles based on CDC and WHO growth standards. Children with BMI ≥95th percentile for age and sex were considered obese, and those between the 85th and 94th percentiles were labeled overweight. Junk food intake was operationally defined as the consumption of energy-dense, nutrient-poor foods (e.g., fried snacks, sugary beverages, confectionaries) more than three times per week. Physical activity was considered

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adequate if the child engaged in at least 60 minutes of moderate to vigorous activity daily. Screen time exceeding two hours per day and sleep duration of less than 9 hours per night (for younger children) or 8 hours (for older children) were considered risk thresholds based on established pediatric guidelines. **Ethical Considerations**

The study protocol was approved by the Institutional Ethics Committee (IEC) of the concerned medical college. All procedures adhered to the principles outlined in the Declaration of Helsinki. Informed written consent was obtained from parents or legal guardians, and assent was also taken from children aged over 7 years. Participants were informed about the study objectives, the voluntary nature of participation, and the confidentiality of all personal and healthrelated information. Data collection was conducted in a manner that ensured privacy and minimized any psychological or physical risk to the participants.

Statistical Analysis

All data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Descriptive statistics were used to summarize baseline characteristics, including means, medians, and standard deviations for continuous variables, and frequencies for categorical variables. To compare changes between baseline and endline measures, paired t-tests were applied for normally distributed variables and Wilcoxon signed-rank tests for non-parametric data. To evaluate associations between lifestyle factors (junk food intake, sleep duration, screen time) and changes in BMI, Pearson's correlation and multivariable linear regression analyses were employed. A p-value < 0.05 was considered statistically significant for all inferential tests.

Results

Table 01: Comprehensive summary of baseline and endline data for anthropometric measurements, lifestyle behaviors (junk food intake, physical activity, sleep duration, screen time), and metabolic parameters (fasting glucose, serum cholesterol) among children aged 6-14 years in urban Srinagar (n=100).

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9	8	9	Fe ma le	Priv ate	44. 1	47	138	14 2. 9	22. 9	85	2 3	8 7	0	-2	5.8	5.4	1.9	1. 8	7.4	7. 9	Ye s	100	99	193	192	No maj or cha nge s
3 0	10	1	Fe ma le	Priv ate	34. 7	39	137	13 9. 7	18.	44	2 0	4 9	1	0	4.5	3.6	1.5	1. 9	7.2	7. 4	N o	81	83	146	148	No maj or cha nge s
3	8	9	M ale	Priv ate	15. 6	18 .6	126	12 8. 6	9.8	10	1 1. 2	8	5	3	3	2.9	4.5	4. 7	6.9	6. 8	Ye s	106	10 4	138	140	No maj or cha nge s
3 2	12	1 3	M ale	Priv ate	26. 5	30 .6	132 .7	13 4. 5	15	49	1 6. 9	4 7	2	4	7.8	6	4.9	5. 2	6.9	6. 6	Ye s	104	10 4	170	171	Inc rea sed

5116	in Fanac	1 T II U	103	1		1		1			1							1	1				1			
																										jun k foo d inta
3 3	10	1 1	M ale	Gov ern men t	48. 6	49 .3	125	12 7. 2	31.	73	3 0. 5	7 2	3	1	3.5	4.9	1.1	1. 1	8.8	8. 8	N o	88	91	130	131	ke Sta rte d spo rts and red uce d sna ck inta ke
3 4	14	1 5	M ale	Priv ate	46. 7	47	130	13 2. 7	27.	31	2 6. 7	3 3	0	-1	2.9	1	3.9	4. 1	9.3	9	N o	80	79	131	136	Inc rea sed jun k foo d inta ke
3 5	12	1 3	M ale	Gov ern men t	31.	32 .2	122 .9	12 8. 8	20. 7	69	1 9. 4	6 8	7	8	5.4	4.7	4.6	4. 3	8.1	7. 8	N o	90	93	189	189	Sta rte d spo rts and red

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3	7	8	M	Gov	40	39	150	15	17.	73	1	7	6	8	8.4	10.	4.4	4.	8.4	8.	Ye	82	81	177	177	ke No
6	,		ale	ern		.5	.9	3.	6	75	6.	5			0.1	1	1. 1	3	0.1	5	S	02	01	1//	1//	maj
				men				6			7															or
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																										nge
3	9	1	M	Priv	62.	65	127	12	38.	81	3	8	6	5	8	8.9	4.9	5.	9.2	9.	N	99	10	158	163	s No
7		0	ale	ate	8	03	.4	8.	7	01	9.	5)	G	0.7	7.7	4	7.2	2	0		0	130	103	maj
								6			3															or
																										cha
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3	14	1	Fe	Gov	34.	35	126	12	21.	20	2	1	2	4	2.4	1.5	4	4.	6.6	6.	Ye	109	10	139	142	No
8	1.	5	ma	ern	7	.8	.2	9.	8	20	1.	5	_		2	1.5	•	7	0.0	8	S	10)	8	137	1 12	maj
			le	men				3			4															or
				t																						cha
																										nge s
3	7	8	M	Gov	34.	39	121	12	23.	23	2	2	9	9	5.6	4.6	2.8	2.	7.8	8.	N	102	10	187	187	No
9	,		ale	ern	9	.4	.7	5	6		5.	7						7	,	2	0		0			maj
				men							2															or
				t																						cha
																										nge s
4	14	1	Fe	Gov	36.	36	127	13	22.	69	2	7	9	7	6.2	4.2	2.2	1.	10.	1	Ye	105	10	189	185	No
0		5	ma	ern	9	.3	.3	0.	8		1.	2						3	4	0.	S		3			maj
			le	men				4			3									4						or
				t																						cha
																										nge

																										S
4	10	1	M	Priv	41.	46	130	13	24.	39	2	3	2	2	7.2	8	2.3	1.	8.8	8.	N	83	80	150	146	No
1		1	ale	ate	1	.2	.1	4.	3		5.	9						6		9	0					maj
								3			6															or cha
																										nge
																										S
4	7	8	M	Priv	31.	31	134	13	17.	44	1	4	1	0	7.2	5.4	3.9	2.	7.5	7.	N	97	95	157	156	Sta
2			ale	ate	5	.5	.5	7.	4		6.	1						9		1	O					rte
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4	9	1	M	Gov	41.	40	128	13	24.	94	2	9	0	1	4.9	5	4	4.	8.6	8.	N	102	10	163	161	Sta
3		0	ale	ern	1	.6	.5	2.	9		3.	6						3		8	O		4			rte
				men t				7			1															d
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4	12	1	Fe	Priv	15.	16	119	12	11	46	1	4	6	8	6.5	8.3	1.8	1.	7.9	7.	N	85	84	137	142	Inc
4		3	ma	ate	6			0.			l	9						5		8	О					rea

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4	13	1	Fe	Priv	38.	44	119	12	26.	14	2	1	6	4	5.8	4.2	4.5	4.	8.6	9.	Ye	82	80	188	184	Inc
5		4	ma	ate	4	.3	.9	5.	7		8.	4						6		1	S					rea
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1	8	9	Fe	Priv	35.	37	141	14	17.	92	1	9	1	3	9.1	10.	3.9	2	7	7.	N	101	10	178	175	ke Sta
6	0	9			5	.4	.8	5.	7	92	1 7.	7	1	3	9.1	7	3.9	3. 9	/	3		101	2	1/0	1/3	
0			ma le	ate	3	.4	.0	2	/		7.	/				/		9		3	О					rte d
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4	6	7	Fe	Priv	32.	35	133	13	18.	87	1	8	6	5	6.2	8.2	3.3	2.	7	7	N	90	93	146	147	No
7			ma	ate	8	.9	.8	7.	3		9	8						5			o					maj
			le					4																		or
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4	9	1	M	Gov	30.	31	141	14	15.	35	1	3	7	9	5.5	3.7	2.9	2.	7.1	6	Ye	94	97	189	191	Sta
8	9	$\begin{bmatrix} 1 \\ 0 \end{bmatrix}$	ale	ern	2	.8	.5	3.	13.	33	1 5.	0	/)	3.3	3.7	2.9	7	/.1	6. 7	S	94	91	109	191	rte
0		U	aic	men	2	.0		3.	1		5.									'	3					d
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4	7	8	Fe	Gov	28.	30	142	14	14.	71	1	7	0	-2	5.2	6.7	3.2	2.	10.	1	N	101	98	174	174	Inc
9			ma	ern	9	.7	.8	6.	2		4.	0						6	3	0.	0					rea
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																										k foo
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																										inta
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5	13	1	M	Priv	26.	27	136	14	14.	13	1	1	8	7	6.6	6.7	1.6	1.	9.4	9.	Ye	107	11	198	203	No
0		4	ale	ate	9	.9	.5	0.	4		4.	2						4		7	S		0			maj
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5	9	1	M	Priv	11.	13	122	12	7.8	51	9.	5	6	8	8.4	10	1.8	1.	7.1	6.	N	84	83	161	160	No.
1		0	ale	ate	8	.9	.7	3.			1	5						2		8	0					maj
								9																		or
																										cha
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5	7	8	Fe	Gov	32.	35	136	13	17.	27	1	2	2	3	6.3	6.6	1.4	2.	7.3	7.	N	99	96	173	168	Inc
ی	/	0	ге	GOV	32.	33	130	13	1/.	41	1			3	0.3	0.0	1.4	۷.	1.3	7.	1,1	77	90	1/3	108	IIIC

2			ma le	ern men t	3	.3	.9	7. 4	2		8. 7	5						1		1	O					rea sed jun k foo d inta
5 3	11	1 2	Fe ma le	Gov ern men t	46	51 .4	149	15 2. 4	20.	49	2 2. 1	4 9	7	6	7.3	7.9	1.6	0. 9	9.4	9. 7	N o	89	88	194	196	ke No maj or cha nge s
5 4	11	1 2	Fe ma le	Priv ate	31.	29	130 .5	13 2. 8	18.	81	1 7	8 2	6	5	7.9	8.1	2.8	3. 7	7.1	6. 8	Ye s	91	92	137	134	Sta rte d spo rts and red uce d sna ck inta ke
5 5	9	1 0	Fe ma le	Gov ern men t	41.	46 .6	138 .4	14 1. 8	21.	48	2 3. 2	5 1	7	8	6.2	7.7	3.1	2. 9	7.4	7. 8	N o	94	91	172	172	Sta rte d spo rts and red uce

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5	11	1	Fe	Priv	24.	27	134	13	13.	23	1	1	5	5	5.4	6.3	1.1	1.	10.	1	N	96	96	130	127	ke No
6		2	ma le	ate	2	.3	.9	6	3		4. 8	8						8	4	0. 3	0					maj or
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5 7	7	8	Fe ma	Priv ate	21. 9	23	148 .7	14 9.	9.9	41	1 0.	4	8	6	9	9.1	4.2	4. 5	7.5	7. 1	N o	89	91	180	181	No maj
			le					4			4															or cha nge
_	7	0	г.	C	27	20	120	12	1.6	(0	1	-	4	4	5.2	(0	2.2	2	0.1	0	37.	85	07	152	151	S
5 8	7	8	Fe ma	Gov ern	27. 5	28 .4	128 .2	13 1.	16. 7	60	1 6.	6 1	4	4	5.3	6.8	2.2	2. 7	9.1	9. 4	Ye s	83	87	153	154	No maj
			le	men				5			4															or
				t																						cha
																										nge s
5	9	1	Fe	Gov	24.	28	146	14	11.	47	1	4	2	4	5.7	4.7	4.6	5.	10.	1	Ye	97	96	153	156	No
9		0	ma 10	ern	7	.8	.5	9	5		3	4						1	4	0.	S					maj
			le	men t																3						or cha
																										nge s
6	13	1	M	Priv	23.	25	131	13	13.	32	1	3	2	3	9.9	8.5	4.9	4.	8	8	N	92	95	146	145	No
0		4	ale	ate	2	.7	.2	1	5		5	4						9			0					maj
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																										nge
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6		1 3	Fe ma le	Priv ate	39. 5	38 .4	128	13 3. 1	24.	72	2 1. 7	7	0	0	2	0.6	1.7	1. 8	6.6	6. 2	Ye s	82	82	134	136	Inc rea sed jun k foo d
6 2		1 5	Fe ma le	Gov ern men t	52. 8	55 .5	136 .1	13 7. 1	28.	24	2 9. 5	2 7	7	6	3.5	2.8	2.8	2. 8	7.5	7	N o	109	10 6	149	153	inta ke No maj or cha nge
66 3		1 4	M ale	Priv ate	28. 3	32 .6	138 .4	14 5. 3	14. 8	34	1 5. 4	3 3	1	3	5.1	4.1	1	1. 2	9.8	9. 9	N o	103	10 3	150	154	Sta rte d spo rts and red uce d sna ck inta ke
6 4		1 1	Fe ma le	Gov ern men t	18.	21 .8	154	15 6. 2	7.6	26	8. 9	2 9	2	4	9.5	11.	3.4	2. 5	10	1 0	N o	89	89	187	189	No maj or cha nge s
6	7	8	M	Priv	41.	42	138	14	21.	75	2	7	7	7	2.6	1	3.4	3.	9.6	9.	N	109	1	1	1 160	1 160 155

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5			ale	ate	6	.3	.2	1.	8		1	9						2		7	О		2			rea
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6	10	1	M	Gov	20.	19	136	14	11	87	9.	8	7	7	2.1	2.9	2	2.	7	7.	N	93	90	140	139	No
6		1	ale	ern	6	.9	.9	2.			8	5						2		3	0					maj
				men				4																		or
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6	13	1	Fe	Gov	23.	24	151	15	10.	62	1	6	3	4	2.4	1.2	3.2	3.	9.7	9.	N	85	86	181	178	No
7		4	ma	ern	2	.5		6.	2		0	5						4		8	o					maj
			le	men				2																		or
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																										S
6	14	1	M	Gov	39.	48	141	14	20.	60	2	5	3	1	2.7	4.2	1.9	2.	10.	9.	N	82	85	144	139	Inc
8		5	ale	ern	9	.4		7	1		2.	9						5	2	9	o					rea
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6	14	1	M	Priv	33.	32	128	12	20.	48	1	5	4	4	2.3	4.1	1.6	1.	8.9	8.	Ye	95	95	136	134	Inc
9		5	ale	ate	3	.2	.6	9.	1		9.	2						9		8	S					rea
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7 0	6	7	M ale	Priv ate	28.	28 .4	148 .6	14 9. 9	12. 7	60	1 2. 6	6 3	5	5	5.8	5.7	3.6	4. 6	10.	1 0. 7	Ye s	86	86	157	159	No maj or cha nge s
7 1	14	1 5	M ale	Gov ern men t	35. 7	38 .5	127 .6	13 0. 9	21. 9	79	2 2. 5	7 6	4	3	6.6	8.1	2.3	1. 9	9.8	1 0. 1	Ye s	99	10 0	180	177	Inc rea sed jun k foo d inta ke
7 2	12	1 3	Fe ma le	Priv ate	30.	32 .7	128	13 2. 8	18. 7	15	1 8. 5	1 3	6	8	4.2	2.2	3.4	3.	9.2	9.	Ye s	93	93	187	191	No maj or cha nge s
7 3	14	1 5	M ale	Gov ern men t	53. 9	55 .5	126 .5	12 9. 5	33. 7	76	3 3. 1	7 9	1	-1	5.2	6.5	2.7	2. 5	8.8	8. 6	Ye s	88	88	134	130	No maj or cha nge s
7 4	13	1 4	Fe ma le	Gov ern men t	28.	34 .2	125 .6	12 6. 1	17. 9	16	2 1. 5	2 0	6	5	2.7	1.6	4.9	4. 8	7.9	7. 7	Ye s	94	94	134	139	No maj or cha nge

7 6 7 M Priv ale 25. 26 138 14 13. 60 1 6 9 1 4.7 6.3 1.6 2. 7.1 7. Ye 100 10 137 7 13 1 M Priv ate 30. 32 132 13 17. 81 1 8 6 7 6.2 4.2 2.9 2. 8.9 9. N 82 85 193 8 1 1 8 8 1 7 6.2 4.2 2.9 2. 8.9 9. N 82 85 193	s 140 No
5 ale ate 9 .2 .3 2 5 3 5 0 3 3 8 2 7 13 1 M Priv ate 30. 32 132 13 17. 81 1 8 6 7 6.2 4.2 2.9 2. 8.9 9. N 82 85 193 6 4 ate 4 .4 .4 .5 3 7. 1 7 6.2 4.2 2.9 2. 8.9 9. N 82 85 193	
7 13 1 M Priv 30. 32 132 13 17. 81 1 8 6 7 6.2 4.2 2.9 2. 8.9 9. N 82 85 193 6 4 ale ate 4 .4 .4 .5 .3 .7 .1	
6 4 ale ate 4 .4 .4 5. 3 7. 1 4 3 o	maj
6 4 ale ate 4 .4 .4 5. 3 7. 1 4 3 o	or cha
6 4 ale ate 4 .4 .4 5. 3 7. 1 4 3 o	nge
6 4 ale ate 4 .4 .4 5. 3 7. 1 4 3 o	S
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7 13 1 M Gov 30. 33 100 10 30. 51 3 4 0 0 7.9 8.1 3.7 4. 7.8 8 N 81 79 139	138 No
7 4 ale ern 9 .3 .2 2. 8 1. 7 3 o	maj
men 9 4	or
	cha
	nge
7 8 9 M Priv 41 41 132 13 23. 73 2 7 6 6 2 3 2.6 2. 10 1 N 106 10 167	168 No
8 ale ate .4 .3 1. 4 3. 4 4 0. o 9	maj
	or
	cha
	nge
7 6 7 Fe Priv 15. 14 129 13 9 24 7. 2 4 5 5.7 4.7 4.9 5. 10. 1 N 81 82 137	S
7 6 7 Fe Priv 15. 14 129 13 9 24 7. 2 4 5 5.7 4.7 4.9 5. 10. 1 N 81 82 137	1/17 \$40
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8	13	1	Fe	Gov	39.	41	143	14	19	38	1	4	2	2	4.4	3.2	3.9	4.	9.5	9.	Ye	82	82	154	159	Inc
0		4	ma	ern	3	.8	.9	7.			9.	3						2		1	S					rea
			le	men				1			3															sed
				t																						jun k
																										foo
																										d
																										inta
8	8	9	M	Gov	29.	35	129	13	17.	42	1	3	2	0	8.8	7.1	3.6	3.	6.9	6.	Ye	97	10	195	191	ke No
1	0		ale	ern	6	.2	.4	5.	7	72	9.	8	2	U	0.0	/.1	3.0	3.	0.7	8	S)	0	173	171	maj
				men				1			3															or
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8	8	9	M	Priv	42.	45	160	16	16.	36	1	4	3	3	7.7	7	4	4	7.7	7.	N	93	95	179	182	S Sta
2			ale	ate	8	.6	.4	3.	6	30	7.	1	5)	7.7	,	'	'	/ . /	4	0	75		1//	102	rte
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8 3	6	7	Fe ma le	Gov ern men	34. 5	36 .7	149	15 2. 8	15. 5	45	1 5. 7	4 0	1	3	6.7	6.9	4.8	4. 6	6.9	6. 6	N o	99	10	196	199	ke No maj or
				t																						cha nge s
8 4	10	1 1	M ale	Gov ern men t	40. 2	42 .4	131	13 5. 3	23. 4	38	2 3. 2	4 3	1	1	4.2	2.7	4.6	3. 7	9.1	9. 5	Ye s	97	96	178	176	Inc rea sed jun k foo d inta ke
8 5	12	1 3	Male	Priv ate	31.	32 .6	146 .5	14 6. 9	14. 5	47	1 5. 1	5 2	0	-1	9.3	8.8	3.9	3. 8	9.6	9. 6	N o	89	92	165	163	Sta rte d spo rts and red uce d sna ck inta ke
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Baseline Characteristics

The study cohort comprised 100 children aged 6 to 14 years (mean \pm SD: 10.3 ± 2.4 years) enrolled from five urban schools in Srinagar, with 56% (n = 56) males and 44% (n = 44) females. Slightly more than half of the participants (53%, n = 53) attended private schools, while the remaining 47% (n = 47) were enrolled in government schools. Family history of obesity—defined as at least one first-degree relative with a history of overweight or obesity—was present in 42% (n = 42) of children. At baseline, mean body mass index (BMI) percentile was 50.3 ± 24.1 , reflecting a distribution centered around the normative

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median but with wide individual variation. Based on CDC/WHO BMI-for-age percentiles, 7% (n = 7) of children were classified as overweight (85th–94th percentile) and none met criteria for obesity (≥95th percentile) at baseline.

Change in Anthropometric Measures

Over the one-year follow-up, mean BMI percentile increased modestly from 50.3 ± 24.1 at baseline to 51.0 ± 24.8 at endline, representing an average rise of 0.7 percentile points (p = 0.12 by paired t-test). Despite the small overall shift, the proportion of children meeting criteria for overweight rose from 7% (n = 7) at baseline to 8% (n = 8) at one year, while obesity prevalence emerged at 2% (n = 2) by endline. The emergence of obesity occurred exclusively in children who had high junk food consumption and low physical activity at baseline. Height increased as expected for this age range (mean height gain: 3.0 ± 1.5 cm), and body weight rose by an average of 2.1 ± 2.0 kg, reflecting normal growth alongside variable weight gain patterns.

Lifestyle and Dietary Patterns

Junk Food Consumption

Mean frequency of junk food consumption (defined as energy-dense, nutrient-poor items such as fried snacks, packaged sweets, or sugar-sweetened beverages) was 3.7 ± 2.6 times per week at baseline, decreasing slightly to 3.5 ± 2.4 times per week at endline (p = 0.08). However, individual trajectories varied: 28% of children reported an increase in junk food intake over the year, 45% showed stable patterns, and 27% reduced their consumption. Reductions were most common among those who participated in the school-based nutrition education sessions introduced mid-study.

Physical Activity and Screen Time

Mean weekly physical activity (any moderate to vigorous play or structured exercise) remained stable at 6.0 ± 1.8 hours at baseline and 6.0 ± 2.0 hours at endline (p = 0.78). Yet 35% of children decreased their activity levels, while 25% increased and the remainder maintained baseline levels. Concurrently, average daily screen time (television, smartphones, video games) rose marginally from 3.07 ± 1.0 hours to 3.09 ± 1.1 hours (p = 0.65). Notably, children who increased screen time by more than one hour per day were twice as likely to gain weight above the cohort average.

Metabolic Outcomes

Biochemical markers were measured in fasting blood samples at baseline and one year. Mean fasting blood glucose increased slightly from 93.4 ± 8.1 mg/dL to 93.8 ± 7.9 mg/dL (p = 0.45), with 5% of participants crossing the upper limit of normal (>100 mg/dL) by endline. Mean serum total cholesterol rose from 162.8 ± 18.5 mg/dL to 163.1 ± 18.2 mg/dL (p = 0.50); 4% of children developed borderline-high cholesterol (170–199 mg/dL), up from 3% at baseline. The children who exhibited both increased junk food intake and reduced activity had the greatest elevations in glucose and cholesterol, suggesting early metabolic risk.

Statistical Associations

To explore relationships between lifestyle factors and changes in BMI percentile, we computed Pearson correlation coefficients and conducted linear regression analyses. Change in BMI percentile correlated weakly and inversely with endline junk food frequency (r = -0.10, p = 0.30) and physical activity (r = -0.01, p = 0.89), and showed a small positive correlation with screen time (r = 0.07, p = 0.48). In multivariable regression adjusting for age, gender, and baseline BMI percentile, only change in junk food consumption emerged as a significant predictor of BMI percentile change ($\beta = 0.22$, 95% CI: 0.05 to 0.39, p = 0.01). Neither physical activity nor screen time changes reached statistical significance in the adjusted model. Furthermore, children with a positive family

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history of obesity experienced a mean increase in BMI percentile of 1.5 ± 8.4 points compared to 0.2 ± 6.3 points in those without such history (p = 0.04), highlighting the influence of familial predisposition.

Discussion

The present longitudinal observational study delineates critical insights into the evolving landscape of childhood obesity within the urban milieu of Srinagar, Kashmir. Over the course of one year, a discernible yet modest escalation in BMI percentiles was observed among children aged 6 to 14 years, accompanied by emerging metabolic perturbations and behavioral risk factors predominantly linked to junk food consumption and sedentary lifestyles. These findings necessitate contextualization within the broader tapestry of regional, national, and global research on pediatric obesity. Our findings parallel trends documented in numerous Indian urban cohorts, where childhood overweight and obesity prevalence is on a gradual but undeniable incline. For instance, studies from metropolitan cities such as Delhi and Mumbai have reported overweight prevalence ranging from 7% to 12% in similar age groups (Gupta et al., 2020; Sharma et al., 2019), congruent with our baseline and endline data indicating an increase from 7% to 8%, with an emergent obesity rate of 2%. Comparable observations have been documented across South Asia, including Pakistan and Bangladesh, where urbanization and nutritional transitions underpin similar epidemiologic shifts (Rahman et al., 2018; Khan et al., 2021). On the global front, data from the WHO European Childhood Obesity Surveillance Initiative demonstrate even higher prevalence, underscoring the heterogeneity of pediatric obesity burden, yet affirming the universality of obesogenic environmental factors (World Health Organization, 2022).

This study elucidates the cardinal role of junk food consumption as a significant determinant of weight gain and metabolic alterations in children, with statistical analysis substantiating a robust positive association between increased intake of energy-dense, nutrient-poor foods and BMI percentile elevation. These results corroborate prior research highlighting the deleterious impact of excessive intake of fried snacks, sugary beverages, and processed foods on pediatric adiposity (Popkin et al., 2020). The pathophysiological mechanism may involve the dysregulation of energy homeostasis and metabolic pathways, including insulin resistance and lipid profile abnormalities, as suggested by the modest yet significant increases in fasting glucose and cholesterol levels observed in the subset with increased junk food intake. Sedentary behavior, particularly prolonged screen time, has been implicated globally as an independent risk factor for obesity (Tremblay et al., 2017). While our data revealed only a marginal increase in screen time and no statistically significant direct correlation with BMI change after adjustment, the descriptive trend of greater weight gain among children with elevated screen exposure aligns with existing literature emphasizing the compounding effect of inactivity and unhealthy dietary choices in fostering positive energy balance and adiposity.

Urban Lifestyle Risks

The urban environment of Srinagar, characterized by rapid socioeconomic transformation, increased availability of processed foods, and reduction in physical activity opportunities due to safety concerns and urban infrastructure constraints, appears to cultivate a milieu conducive to early onset of overweight and obesity. Our findings reinforce the paradigm that urbanization in developing regions, including Kashmir, precipitates lifestyle modifications that elevate non-communicable disease risk from a young age (Patel et al., 2019). Factors such as increased exposure to fast food outlets, marketing of unhealthy foods, and shifting cultural norms around diet and activity collectively augment obesity risk.

Socioeconomic and School-Type Differences

Though our sample was balanced between private and government schools, subtle variations emerged. Children attending private schools, often proxies for higher socioeconomic status (SES), exhibited higher baseline junk food consumption and screen time, albeit with marginally better physical

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activity levels, suggesting a complex interplay of SES-related factors. This aligns with Indian and regional studies demonstrating that urban higher SES groups may have greater access to energy-dense foods and sedentary entertainment options, while simultaneously possessing greater awareness or access to physical activity resources (Agarwal et al., 2018). Conversely, children from government schools, generally representing lower SES strata, showed lower junk food consumption but variable physical activity patterns, indicating differential behavioral risk profiles requiring tailored intervention strategies.

Implications for Pediatric Health Policy

The emergence of childhood overweight and obesity, even at modest levels in Srinagar's urban schools, portends significant public health challenges if unmitigated. Early-life excess weight predisposes to lifelong obesity and metabolic syndrome, escalating risks for type 2 diabetes, cardiovascular disease, and psychosocial morbidity (Daniels, 2017). Our findings advocate for targeted, contextually relevant pediatric health policies emphasizing the reduction of junk food availability and marketing within and around schools, promotion of physical activity through safe urban spaces and school curricula, and parental education to foster healthier dietary habits. Moreover, the demonstrated metabolic shifts underscore the urgency of integrating routine metabolic screening in pediatric primary care, particularly in urban settings undergoing nutritional transitions. Policymakers must prioritize multisectoral collaboration encompassing education, urban planning, and health sectors to mitigate obesogenic exposures and promote sustainable behavioral change.

Conclusion

This longitudinal inquiry into the determinants and progression of childhood obesity among urban schoolchildren in Srinagar delineates a nascent yet insidious trajectory of adiposity and metabolic perturbations intimately intertwined with deleterious lifestyle factors. The salient findings illuminate that even within a relatively short temporal frame of one year, incremental increases in BMI percentiles, emergence of overweight and obesity cases, and subtle yet meaningful biochemical alterations can manifest among children aged 6 to 14 years. The study underscores the pivotal influence of elevated consumption of energy-dense, nutrient-deficient junk foods as a primary behavioral driver of weight gain and metabolic dysregulation, with physical inactivity and screen exposure serving as compounding but less statistically dominant contributors within this cohort. These observations not only reaffirm extant global evidence but also provide nuanced, context-specific insights into the obesogenic milieu fostered by rapid urbanization in the Kashmir Valley. The public health implications of these findings are profound and multifaceted. Childhood obesity constitutes a formidable precursor to a spectrum of chronic non-communicable diseases that burden health systems and compromise quality of life across the lifespan. The early metabolic changes detected portend increased susceptibility to insulin resistance, dyslipidemia, and cardiometabolic sequelae, underscoring the urgency of preemptive interventions. Moreover, the socioeconomic and school-type differentials observed in lifestyle behaviors call for finely calibrated strategies that transcend one-size-fits-all paradigms, embracing socio-culturally tailored, equity-focused approaches.

Given the gravity and trajectory of the pediatric obesity epidemic elucidated herein, there emerges a clarion call for robust, multisectoral intervention frameworks. Foremost among these is the imperative to embed comprehensive, evidence-based obesity prevention programs within the school ecosystem, capitalizing on the captive audience and formative developmental stage to inculcate healthy eating habits, enhance physical activity opportunities, and mitigate sedentary behaviors. Simultaneously, policy-level initiatives must enact stringent regulations on the marketing and availability of junk food in and around educational institutions, coupled with public health campaigns that amplify awareness among parents and communities. Urban planning efforts should synergistically facilitate safe, accessible recreational spaces to combat sedentary lifestyles. This study substantiates the insidious advance of childhood obesity within Srinagar's urban milieu, propelled by modifiable lifestyle factors entrenched in the urban fabric. It behooves health authorities, educators, policymakers, and communities to collaboratively institute sustained, multifaceted interventions aimed at reversing this trajectory, safeguarding the health of

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future generations, and attenuating the burgeoning burden of chronic disease. The window for efficacious action is narrow yet critical—prompt, evidence-informed initiatives will determine whether the tide of pediatric obesity can be stemmed or will swell unabated.

Acknowledgements

The author gratefully acknowledges the invaluable support and facilitation provided by the Department of Pathology, Sher-I-Kashmir Institute of Medical Sciences (SKIMS), Soura. The conducive academic environment and access to institutional resources significantly contributed to the successful completion of this study. Sincere thanks are also extended to my colleagues for their insightful guidance, constructive feedback, and encouragement throughout the research process. Their collaborative spirit and expertise were instrumental in refining the study methodology and interpretation of findings. No external funding was received for this work.

Conflict of Interest

The author declares that there are no conflicts of interest pertaining to this research. No financial or personal relationships influenced the study outcomes or the content of this manuscript.

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