

Original Article

# Investigating Nutritional Deficiencies and Lifestyle Factors Contributing to Rising Incidence of Rickets among Children in Developing Regions

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## ABSTRACT

**Background:** Nutritional rickets persists in many low- and middle-income countries despite abundant sunlight, driven by a complex interplay of inadequate dietary intake, limited effective sunlight exposure, and evolving lifestyle patterns among children. **Objective:** To examine the association between dietary calcium and vitamin D intake, sunlight exposure, lifestyle behaviours, and rickets-related outcomes among children in urban and semi-urban settings in Pakistan. **Methods:** A cross-sectional study was conducted over four months among 410 children aged 1–12 years selected through multistage sampling from healthcare facilities and schools. Data were collected using an interviewer-administered questionnaire, semi-quantitative food frequency questionnaire, 24-hour dietary recall, and a Sunlight Exposure Index. Anthropometry and standardised clinical assessment for rickets were performed, and available biochemical data were extracted from records. Descriptive statistics, t-tests, ANOVA, Pearson's correlation, and multiple linear regression were applied using SPSS version 26. **Results:** Mean daily calcium and vitamin D intakes were  $528 \pm 211$  mg and  $3.8 \pm 1.9$  µg, respectively, with fewer than 21% of children meeting age-specific recommendations. Overall, 16.8% had clinical or radiographic features consistent with rickets. Lower calcium intake, reduced daily sunlight exposure, lower Sunlight Exposure Index scores, and shorter outdoor play time were significantly associated with rickets indicators. In multivariable models, inadequate calcium intake and limited sunlight exposure remained the strongest independent predictors. **Conclusion:** Inadequate intakes of calcium and vitamin D, combined with limited effective sunlight exposure and sedentary lifestyles, are major determinants of rickets-related outcomes among children in this setting, highlighting the need for integrated nutritional and behavioural interventions.

**Keywords:** nutritional rickets; vitamin D deficiency; calcium intake; sunlight exposure; child health; Pakistan.

## INTRODUCTION

Rickets remains a preventable yet persistent cause of skeletal morbidity among children in many low- and middle-income countries, including Pakistan, despite the availability of sunlight and advances in primary care and nutrition services (1–4). Recent evidence from South Asian cohorts has shown that a substantial proportion of school-aged children and

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adolescents have concurrent vitamin D deficiency and suboptimal calcium status, leading to impaired bone mineralization and increased risk of deformities and fractures (1–4). Nutritional rickets is now understood as a disorder arising from the combined effects of inadequate vitamin D, calcium, and, to a lesser extent, phosphorus, resulting in defective mineralization of the growth plate and osteoid tissue (5,6). Global and regional reviews consistently highlight that nutritional rickets continues to occur in settings where it should be largely preventable, underscoring gaps in dietary quality, sunlight exposure, and public health responses (5,6).

In Pakistan and neighbouring South Asian countries, multiple studies have documented high prevalence of vitamin D deficiency across different age groups, from infants to school-going children and adolescents, often in association with poor dietary calcium intake and limited diversity of micronutrient-rich foods (7–10). Hospital- and community-based surveys in under-five children with malnutrition and clinical bone deformities have reported a substantial burden of nutritional rickets, with many children presenting late in the course of disease (9,11,12). These findings mirror global observations that nutritional rickets persists in resource-constrained environments where diets are dominated by cereal-based staples and intake of animal-source foods, fortified products, and dairy is limited by affordability and access (5,11–13). At the same time, the epidemiology of rickets appears to be changing, with increasing recognition of rickets in older children and adolescents rather than exclusively in infants and toddlers (10–12).

The pathophysiology of nutritional rickets reflects a complex interplay between biological requirements for bone growth and modifiable environmental and behavioural determinants. Vitamin D is critical for intestinal calcium absorption and skeletal mineralization, and its primary source in most populations is endogenous synthesis in the skin following exposure to ultraviolet B (UVB) radiation (5,13,14). However, even in regions with ample sunshine, vitamin D deficiency is common because of cultural clothing practices, conservative dress, air pollution, indoor confinement, and parental concerns about outdoor safety and sun exposure (13–16). Studies from South Asia and the Middle East have shown that urban residence, high-rise living, atmospheric pollution, and limited outdoor play are associated with low serum 25-hydroxyvitamin D and increased risk of rickets and low-energy fractures in children and adolescents (8,13,16–19). Maternal vitamin D deficiency during pregnancy and lactation further compounds the risk, particularly in exclusively breastfed infants who receive inadequate vitamin D either from breast milk or supplementary sources (16,17).

Population-based and hospital-based data from Pakistan indicate that vitamin D deficiency is widespread across different demographic strata, including children, adolescents, women of reproductive age, and adults, often coexisting with poor dietary calcium intake and low awareness of vitamin D sources (7,14,18). Several reports have highlighted the high prevalence of low vitamin D status in healthy adults and special populations such as mothers, alongside elevated bone turnover markers and biochemical abnormalities that may precede overt skeletal deformities (14,17,18). Similar patterns have been documented in South Asian immigrant and refugee populations, as well as in primary school children from Afghanistan and East African countries, where low vitamin D and calcium intakes, limited sun exposure, and acute malnutrition intersect to create a high-risk environment for nutritional rickets (20–22). Although these studies have advanced understanding of biochemical deficiency and clinical burden, many have been conducted in narrowly defined hospital samples, specific age groups, or single geographic locations, limiting their generalizability to broader community settings (9–12,20–22).

Taken together, the emerging evidence suggests that nutritional rickets in developing regions is no longer solely a problem of extreme deprivation but is increasingly driven by a combination of inadequate diets, constrained outdoor activity, and lifestyle transitions that reduce effective sunlight exposure (5,10,13–16). Yet, there remains a critical knowledge gap regarding how dietary patterns, sunlight exposure, and everyday lifestyle behaviours interact at the community level to influence rickets risk in school-aged children in Pakistan. Existing studies often examine isolated components—biochemical vitamin D status, clinical rickets diagnosis, or dietary intake—without simultaneously quantifying the relative contribution of nutrition, sun exposure, and sedentary or indoor lifestyles within the same population (7–12,20–22). Furthermore, there is a paucity of multivariable analyses that adjust for sociodemographic and environmental factors when assessing predictors of rickets-related outcomes in children.

In this context, the present cross-sectional study was designed to characterise the nutritional intake of vitamin D and calcium, patterns of sunlight exposure, and lifestyle behaviours among children aged 1–12 years living in urban and semi-urban communities in Pakistan, and to examine their association with clinical and biochemical indicators of rickets. The underlying hypothesis was that children with lower calcium and vitamin D intake, shorter and less effective sunlight exposure, and predominantly indoor, screen-based lifestyles would have a higher prevalence of clinical signs of rickets and abnormal bone-related biochemical markers than their peers with more favourable nutritional and behavioural profiles. The specific objective was to analyse the independent contribution of dietary intake, sunlight exposure, and lifestyle factors to rickets-related outcomes in this population, after accounting for age, sex, and socioeconomic status.

## MATERIALS AND METHODS

This cross-sectional observational study was conducted over a four-month period to investigate the association between nutritional intake, sunlight exposure, lifestyle behaviours, and rickets-related outcomes among children aged 1–12 years in Pakistan. The study design was chosen to provide a snapshot of current dietary and behavioural patterns in a community-based paediatric population and to estimate the prevalence of clinical signs consistent with rickets while exploring potential predictors. Data were collected from both urban and semi-urban settings to reflect variability in environmental exposure and living conditions and to improve external validity.

A multistage sampling strategy was employed. In the first stage, three districts representing different provinces and metropolitan profiles were randomly selected from an official list. In the second stage, primary healthcare facilities and primary schools within each district were sampled to capture both clinic-attending and school-going children. In the final stage, children within the target age range were selected using systematic random sampling from outpatient paediatric clinics and pre-identified school health sessions. The sampling frame and interval were calculated separately for each site based on anticipated attendance and enrolment numbers to minimise selection bias.

Children were eligible if they were between 1 and 12 completed years of age, resident in the catchment area for at least six months, and accompanied by a parent or legal guardian able to provide informed consent. Exclusion criteria were a history of chronic conditions known to affect bone metabolism (such as chronic kidney disease, chronic liver disease, or endocrine disorders), current or recent (within the preceding three months) use of vitamin D or calcium supplements, long-term use of medications affecting bone metabolism (e.g.,

systemic glucocorticoids or anticonvulsants), and physical disabilities limiting routine outdoor activity.

Recruitment was performed on site by trained research assistants who approached caregivers in clinic waiting areas and school health session venues. After explaining the study objectives and procedures in simple language, written informed consent was obtained from parents or legal guardians; assent was sought from older children wherever appropriate. To reduce non-response and participation bias, recruitment was spread across different days of the week and times of day, and the research team avoided selective enrolment based on visible nutritional or health status. The final sample included 410 children, which satisfied the minimum sample size estimated from an expected rickets prevalence of 20%, a 95% confidence level, 5% absolute precision, and allowance for clustering and potential non-response.

Data collection followed a standardised protocol. Information was obtained using an interviewer-administered questionnaire that had been developed from existing literature and adapted to the local context. The instrument was first drafted in English, translated into the local language, and back-translated to ensure conceptual equivalence. It was piloted in a separate group of 20 children and their caregivers; minor wording and ordering changes were made to improve clarity. The final questionnaire captured sociodemographic data (age, sex, parental education, household income, residence), lifestyle behaviours (time spent in outdoor play, time spent in screen-based activities per day), clothing practices during outdoor time (proportion of body surface area covered), and parental awareness regarding vitamin D and calcium sources.

Dietary intake was assessed using a semi-quantitative food frequency questionnaire (FFQ) focusing on commonly consumed calcium- and vitamin D-rich foods, including milk and dairy products, eggs, fish, and fortified cereals. Caregivers were asked to report the usual frequency and portion size of each food item over the preceding month, aided by food models and standard household measures to improve recall. To complement the FFQ and capture day-to-day variability, a single 24-hour dietary recall was conducted for each child, documenting all foods and beverages consumed in the preceding day. Nutrient intakes of calcium and vitamin D were estimated using locally appropriate food composition tables and compared with age- and sex-specific dietary reference intakes. For analytic purposes, children were classified as having adequate or inadequate intake based on achieving at least the recommended daily intake thresholds for their age group.

Sunlight exposure was quantified using a structured Sunlight Exposure Index (SEI). Caregivers reported the approximate average duration of outdoor exposure per day, typical timing of exposure (morning, midday, afternoon), and the proportion of body surface area usually exposed (face and hands only, up to forearms and lower legs, or greater than 50% of body surface). Use of sunscreen or parasols and predominant clothing style (minimal, partial, extensive coverage) were recorded. The SEI score was derived by combining duration, time-of-day weighting for UVB intensity, and body surface exposure, with higher scores indicating more effective cutaneous UVB exposure. Based on the distribution of SEI scores, children were categorised into low, moderate, or high sunlight exposure groups.

Anthropometric measurements were obtained by trained staff using calibrated digital weighing scales and portable stadiometers. Weight was measured to the nearest 0.1 kg with the child in light clothing and without shoes, and height to the nearest 0.1 cm with the child standing upright. Body mass index (BMI) was calculated and converted to BMI-for-age percentiles using World Health Organization child growth standards. Underweight and overweight were defined according to standard percentile cut-offs for age and sex. Clinical assessment for rickets was performed by physicians trained in paediatric musculoskeletal

examination. Standardised inspection and palpation were conducted for frontal bossing, widening of wrists and ankles, bowing of long bones, knock-knees, rachitic rosary, and delayed closure of fontanelles in younger children. Where available, recent radiographs of wrists or knees were reviewed for metaphyseal cupping, fraying, or widening. Biochemical test results for serum calcium, phosphorus, and alkaline phosphatase (ALP) were retrieved from medical records for children in whom clinicians had requested such investigations as part of routine care.

Primary variables of interest included dietary calcium and vitamin D intake (continuous and categorised as adequate/inadequate), SEI score and daily minutes of outdoor exposure (continuous and categorised), screen time and outdoor play time per day, and presence or absence of clinical signs consistent with rickets. Secondary variables included BMI-for-age percentile and ALP levels where available.

Potential confounders considered in the analysis were age, sex, and socioeconomic status, the latter operationalised from parental education and household income and grouped into low, middle, and high categories. To minimise information bias, all interviewers were trained to follow the same questioning and probing script, and measurement protocols were standardised. Data were entered into a dedicated database with range and logic checks; 10% of questionnaires were double-entered to detect and resolve discrepancies.

Statistical analyses were performed using IBM SPSS Statistics version 26. Descriptive statistics were used to summarise sample characteristics, with means and standard deviations for continuous variables and frequencies and percentages for categorical variables. Data distribution for key continuous variables (age, nutrient intakes, SEI scores, ALP) was examined using histograms, Q-Q plots, and the Shapiro–Wilk test. For normally distributed variables, independent samples t-tests or one-way analysis of variance (ANOVA) were used to compare means across groups defined by presence or absence of clinical signs of rickets, exposure categories, or socioeconomic strata.

Pearson's correlation coefficients were calculated to assess linear relationships between nutrient intakes, sunlight exposure measures, and rickets-related indicators. Where appropriate, effect sizes such as Cohen's *d* were computed for differences in means, and 95% confidence intervals were derived. Multiple linear regression models were fitted to examine independent associations between dietary calcium intake, dietary vitamin D intake, daily sunlight exposure, outdoor play time, socioeconomic status, and rickets-related outcomes, adjusting for age and sex. Predictor variables were checked for multicollinearity using variance inflation factors, and model residuals were examined to assess normality and homoscedasticity. Missing data were minimal and handled by complete case analysis; no imputation was performed.

Ethical approval for the study was obtained from the relevant institutional ethics committee, and all procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from parents or legal guardians prior to enrolment, and confidentiality was maintained by assigning unique study identifiers and storing data in password-protected files. Children identified as having possible nutritional deficiencies or clinical signs suggestive of rickets were referred to paediatric services for further assessment and management.

## RESULTS

A total of 410 children aged 1–12 years were enrolled, with a response rate of 92.6%. Slightly more than half of the participants were male (52.0%), and the mean age was  $6.9 \pm 3.1$  years.



Almost two thirds of the children resided in urban areas (61.5%), and the distribution of socioeconomic status was broadly balanced, with 32.9% from low-, 43.2% from middle-, and 23.9% from high-income households. The mean BMI-for-age percentile was  $47.3 \pm 16.5$ ; 18.2% of children were classified as underweight and 11.5% as overweight according to reference standards. These characteristics are summarised in Table 1.

**Table 1. Sociodemographic and anthropometric characteristics of study participants (n = 410)**

Variable	Category	n (%)	Mean $\pm$ SD
Age (years)	—	—	6.9 $\pm$ 3.1
Gender	Male	213 (52.0)	—
	Female	197 (48.0)	—
Residence	Urban	252 (61.5)	—
	Semi-urban	158 (38.5)	—
Socioeconomic status	Low	135 (32.9)	—
	Middle	177 (43.2)	—
	High	98 (23.9)	—
BMI-for-age percentile	—	—	47.3 $\pm$ 16.5
Nutritional status	Underweight	75 (18.2)	—
	Overweight	47 (11.5)	—

Dietary analysis demonstrated marked inadequacy of both calcium and vitamin D intake in the sample. Mean daily calcium intake was  $528 \pm 211$  mg, well below the recommended 800–1000 mg/day for the age range studied, and only 20.7% of children achieved age-appropriate calcium intake thresholds. Mean dietary vitamin D intake was  $3.8 \pm 1.9$   $\mu$ g/day compared with a recommended 10  $\mu$ g/day, with 19.0% achieving adequate intake.

Milk consumption averaged  $4.3 \pm 2.7$  servings per week, and less than one-third of children consumed milk daily. Fish intake was particularly low, with a mean of  $0.7 \pm 0.5$  servings per week and only 14.6% consuming fish at least once weekly. Egg intake averaged  $2.1 \pm 1.2$  eggs per week, and fewer than one quarter met the threshold of three or more servings per week. In bivariate analyses, lower calcium and vitamin D intake, as well as lower consumption of milk, fish, and eggs, were significantly associated with the presence of clinical signs of rickets. These findings are presented in Table 2.

**Table 2. Nutrient intake and dietary adequacy in relation to clinical rickets (n = 410)**

Variable	Mean $\pm$ SD	Recommended intake	Adequate intake n (%)	p-value
Calcium (mg/day)	528 $\pm$ 211	800–1000	85 (20.7)	0.002
Vitamin D ( $\mu$ g/day)	3.8 $\pm$ 1.9	10	78 (19.0)	0.004
Milk consumption (servings/week)	4.3 $\pm$ 2.7	$\geq 7$	111 (27.1)	0.008
Fish consumption (servings/week)	0.7 $\pm$ 0.5	$\geq 1$	60 (14.6)	0.015
Egg consumption (servings/week)	2.1 $\pm$ 1.2	$\geq 3$	94 (22.9)	0.027

Sunlight exposure patterns showed that the mean duration of daily outdoor exposure was  $38.6 \pm 21.7$  minutes. Only 23.4% of children had high exposure ( $>60$  minutes/day), whereas 35.4% had moderate exposure (30–60 minutes/day) and 41.2% had low exposure ( $<30$  minutes/day). The mean SEI score was  $10.3 \pm 4.7$ , reflecting relatively limited effective UVB exposure when timing and clothing were considered. Nearly half of the children (46.5%) wore clothing that covered more than 80% of body surface area while outdoors, and only 9.2% reported regular use of sunscreen.

There was a strong negative correlation between SEI scores and rickets indicators ( $r = -0.42$ ,  $p < 0.001$ ) and between average daily outdoor exposure and rickets indicators ( $r = -0.39$ ,  $p < 0.001$ ), indicating that children with lower effective sunlight exposure were more likely to have clinical or biochemical evidence of rickets. Biochemical data available for a subset of participants showed that serum ALP levels were substantially higher in children with clinical or radiographic features of rickets ( $513 \pm 104$  IU/L) compared with those without such features ( $287 \pm 92$  IU/L). The mean difference in ALP was 226 IU/L (95% CI 201.7 to 250.3), corresponding to a large effect size (Cohen's  $d = 2.40$ ), with  $p < 0.001$ . These results are summarised in Table 3.

**Table 3. Sunlight exposure patterns and biochemical indicators in relation to rickets**

Variable	Category / Statistic	Value	Effect estimate	p-value
Outdoor exposure (minutes/day)	Mean $\pm$ SD	38.6 $\pm$ 21.7	0.39	<0.001
SEI score	Mean $\pm$ SD	10.3 $\pm$ 4.7	-0.42	<0.001
Sunlight exposure category	High (>60 min/day)	96 (23.4%)	—	—
	Moderate (30–60 min/day)	145 (35.4%)	—	—
	Low (<30 min/day)	169 (41.2%)	—	—
Clothing coverage	>80% body surface area	191 (46.5%)	—	—
Sunscreen use	Regular use	38 (9.2%)	—	—
Serum ALP (IU/L)	Rickets present	513 $\pm$ 104	2.40	<0.001
	Rickets absent	287 $\pm$ 92	Reference	—

Overall, 16.8% (n = 69) of the children demonstrated clinical or radiographic features consistent with rickets. Among children with rickets, 72.5% had inadequate dietary calcium intake and 83.6% had low sunlight exposure, compared with substantially lower proportions among children without rickets. A majority of participants (58.0%) spent more than two hours per day engaged in screen-based activities, while only 34.9% reported at least one hour of outdoor physical play daily. Parental awareness was limited; only 29.5% of caregivers correctly identified sunlight as the main source of vitamin D, and 24.1% reported having ever received nutritional counselling regarding vitamin D or calcium from a health professional.

To identify independent predictors of rickets-related outcomes, multiple linear regression models were fitted with rickets indicators as dependent variables and dietary calcium intake, sunlight exposure, vitamin D intake, outdoor play time, and socioeconomic status as predictors, adjusted for age and sex. Lower dietary calcium intake and shorter daily sunlight exposure emerged as the strongest independent predictors, with negative  $\beta$  coefficients indicating that higher intake or exposure was associated with more favourable rickets-related profiles. Vitamin D intake and outdoor play time also showed significant, albeit smaller, associations, while socioeconomic status demonstrated a weaker and borderline association. The regression coefficients, standard errors, 95% confidence intervals, and p-values are presented in Table 4.

**Table 4. Multivariable linear regression of predictors of rickets-related indicators**

Predictor variable	$\beta$ coefficient	Standard error	95% CI for $\beta$	p-value
Dietary calcium intake	-0.32	0.07	-0.46 to -0.18	<0.001
Sunlight exposure (minutes/day)	-0.29	0.09	-0.47 to -0.11	0.002
Vitamin D intake	-0.18	0.08	-0.34 to -0.02	0.031
Outdoor play time (hours/day)	-0.15	0.06	-0.27 to -0.03	0.047
Socioeconomic status	-0.11	0.05	-0.21 to -0.01	0.062

Collectively, these findings indicate that inadequate calcium and vitamin D intake, limited and ineffective sunlight exposure, and low levels of outdoor physical activity are major determinants of rickets-related outcomes among children in this setting, even after accounting for age, sex, and socioeconomic status.

## DISCUSSION

This cross-sectional study explored how dietary intake, sunlight exposure, and lifestyle behaviours intersect to influence rickets-related outcomes among children aged 1–12 years in urban and semi-urban settings in Pakistan. The prevalence of clinical or radiographic features consistent with rickets in this sample (16.8%) is notable given that rickets is considered a preventable condition in the context of adequate nutrition and sunlight, and is broadly comparable to reports from similar low- and middle-income settings where vitamin D deficiency and low calcium intake coexist (1,5,9,10,13). The mean daily calcium and vitamin D intakes in our cohort were substantially below age-specific recommendations, and fewer than one in five children met adequacy thresholds for either nutrient, reinforcing prior findings from school-aged children in Lahore, Afghan primary school populations, and other

South Asian cohorts, where combined vitamin D and calcium insufficiency is highly prevalent (1,7,14,20). These patterns underscore that nutritional rickets in the region is not merely a rare residue of extreme deprivation but reflects widespread dietary inadequacy affecting large segments of the paediatric population (5,10,13).

The strong association observed between low calcium intake and rickets indicators is consistent with the broader literature suggesting that in many developing regions, calcium insufficiency is at least as important as vitamin D deficiency in the pathogenesis of nutritional rickets (5,15). Although vitamin D facilitates intestinal calcium absorption, when dietary calcium is chronically low, even adequate or moderately low vitamin D may not be sufficient to maintain normal bone mineralization (5,15). Previous work from Pakistani and regional settings has reported that diets dominated by cereal-based staples, with limited intake of dairy, eggs, fish, and fortified foods, contribute to both hypocalcemia and poor vitamin D status in children and adolescents (1,7,9,11,12,14). Our findings align with these observations, as children with rickets in this study were substantially more likely to have inadequate calcium intake and low consumption of calcium-rich foods compared with their unaffected peers. The relatively low proportion of children consuming fish at least once weekly and the modest frequency of egg consumption further highlight missed opportunities for improving both vitamin D and calcium intakes through culturally acceptable foods (7,8,11).

Sunlight exposure emerged as another key determinant in our analysis, with both raw minutes of outdoor exposure and composite SEI scores showing strong negative correlations with rickets-related indicators. These results are in line with epidemiological data from South Asia, the Middle East, and sub-Saharan Africa, which consistently demonstrate that limited effective UVB exposure—due to indoor confinement, conservative clothing, urban built environments, or atmospheric pollution—contributes significantly to vitamin D deficiency and rickets, even in regions with high ambient solar irradiance (6,10,13,16,18,19,22). Studies from Karachi, Bangladesh, and Gulf countries have reported that urban children with high indoor time and extensive clothing coverage have lower vitamin D levels and increased risk of low-energy fractures or clinical rickets compared with rural counterparts, who spend more time outdoors and often have greater skin exposure (8,16,19). Our findings that nearly half of children had clothing coverage exceeding 80% of body surface area while outdoors, and that over 40% had less than 30 minutes of daily outdoor exposure, are therefore highly relevant and likely to contribute to impaired cutaneous vitamin D synthesis. The strong association between low sunlight exposure and elevated ALP levels in our cohort further supports this mechanistic link.

Lifestyle changes, particularly increasing screen time and decreasing outdoor physical play, appear to be amplifying these risks. In our sample, a majority of children spent more than two hours per day in screen-based activities, while only about one third achieved at least one hour of outdoor play. These behavioural patterns mirror broader global trends in digital engagement and sedentary lifestyles among children, but in high-risk nutritional environments they carry a disproportionate burden, as reduced outdoor time simultaneously lowers opportunities for physical activity, sunlight exposure, and social play (10,13,16). The finding that outdoor play time remained an independent predictor of rickets-related indicators after adjustment for age, sex, and socioeconomic status suggests that promoting outdoor activity is not merely a behavioural nicety but a clinically relevant component of rickets prevention strategies in this context.

The multivariable analysis in this study provides a nuanced view of the relative contributions of nutritional and behavioural factors. After adjustment for potential confounders, low



dietary calcium intake and reduced sunlight exposure remained the strongest independent predictors of rickets-related indicators, while vitamin D intake and outdoor play time showed smaller but still significant effects. Socioeconomic status demonstrated only a borderline association once these proximal factors were included, suggesting that while poverty and material deprivation influence access to quality foods and safe outdoor environments, the immediate mediators of risk are dietary choices, exposure patterns, and daily routines. This interpretation is consistent with prior work showing that vitamin D deficiency and nutritional rickets are not confined to the poorest strata but also affect middle-income urban households where diets are energy-dense but nutrient-poor, and where children have limited safe outdoor spaces (7,10,14,18). Importantly, our findings are congruent with global reviews that emphasise the need to consider rickets as a multifactorial disorder driven by intersecting nutritional, environmental, and behavioural determinants rather than by any single deficiency alone (5,10,13).

Comparison with earlier studies from Pakistan and the wider region highlights both similarities and contributions of the present work. Previous investigations have focused on under-five children with acute malnutrition, fracture cohorts, or hospital-based samples, often emphasising biochemical markers and clinical outcomes (8,9,11,12,19,21). While these studies have been invaluable in characterising the burden of deficiency and its consequences, fewer community-based studies have simultaneously assessed dietary behaviours, quantified effective sunlight exposure, and examined lifestyle factors such as screen time and parental awareness in school-aged children. Our study adds to the literature by integrating these dimensions into a single analytic framework, providing evidence that in this population, behavioural and dietary patterns are tightly linked to clinical and biochemical indicators of rickets even in the absence of extreme undernutrition. The high ALP levels among children with rickets, alongside suboptimal intake and exposure, are broadly consistent with biochemical profiles reported in both local and international cohorts (6,10,18,22), reinforcing the validity of our clinical classification and analytic approach.

At the same time, several limitations should be acknowledged when interpreting these findings. The cross-sectional design precludes causal inference and does not allow us to determine temporal relationships between exposure variables and the development of rickets. Dietary intake and sunlight exposure were assessed through caregiver recall, which may be subject to misclassification and recall bias, although the use of a semi-quantitative FFQ, a 24-hour recall, and a structured SEI aimed to reduce this risk. Biochemical data were not available for all participants, as testing was restricted to those in whom clinicians deemed it clinically necessary; consequently, our ability to fully integrate biochemical markers with clinical and environmental data is constrained. The study was conducted in selected districts and may not fully capture rural populations or other regions with different cultural practices, dietary patterns, or environmental conditions. Nonetheless, the multicentre sampling across different provinces, inclusion of both urban and semi-urban sites, and relatively large sample size enhance the generalisability of the findings within similar settings in Pakistan.

Despite these limitations, the study has several strengths. It employed a rigorous multistage sampling strategy, used validated tools adapted to the local context, and integrated anthropometric, clinical, dietary, and sunlight exposure data to characterise rickets risk in a comprehensive manner. The use of multivariable regression allowed us to disentangle the relative impact of key modifiable factors while adjusting for important confounders. From a public health perspective, the findings provide actionable insights: they underscore the need for policies that address both nutritional quality and behavioural practices, including accessible food fortification strategies, targeted nutrition education for parents and caregivers, and community or school-based interventions that encourage safe, routine

outdoor activity and effective sunlight exposure in children. In addition, improving awareness among healthcare providers about the importance of early recognition of rickets risk factors, and integrating vitamin D and calcium counselling into routine child health and maternal services, could help reduce the burden of this largely preventable condition (7,10,14,18,21,22).

## CONCLUSION

Inadequate dietary intake of calcium and vitamin D, combined with limited and suboptimal sunlight exposure and increasingly sedentary, screen-based lifestyles, emerged as major determinants of rickets-related outcomes among children in urban and semi-urban settings in Pakistan, even after accounting for age, sex, and socioeconomic status. These findings highlight that nutritional rickets in this context reflects not only structural inequities in food access and housing but also modifiable behaviours and knowledge gaps at the household and community level, underscoring the need for integrated public health strategies that simultaneously improve diet quality, promote safe and regular outdoor activity, and enhance caregiver awareness to prevent the persistence of this avoidable childhood disorder.

## DECLARATIONS

### **Ethical Approval**

This study was approved by the Institutional Review Board of respective hospitals.

### **Informed Consent**

Written informed consent was obtained from all participants included in the study.

### **Conflict of Interest**

The authors declare no conflict of interest.

### **Funding**

This research received no external funding.

### **Authors' Contributions**

SA, MA; Design: SA, MA, TS; Data Collection: SE, RA, KM; Analysis: SA, FBAA; Drafting: SA, MA, TS, SE, BHAA, RA, FBAA, KM.

### **Data Availability**

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

### **Acknowledgments**

*Not applicable.*

### **Study Registration**

*Not applicable.*

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