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Nutrition, Growth Biomarkers, And Social Inequities In Childhood Stunting: A Systematic Review And Meta-Analysis Of 175 Studies (2003–2025)

[1]Tengku Nih Farisni, [2]Herlina Dimiati, [3]Aman Yaman, [4]Sri Wahyuni

[1]Doctoral Student in Medical Sciences, Doctoral Program in Medical Sciences, Syiah Kuala University
[2]Professor, Doctoral Program in Medical Sciences, Universitas Syiah Kuala
[3]Lecturer, Department of Animal Husbandry, Universitas Syiah Kuala
[4]Professor, Department of Veterinary Medicine, Universitas Syiah Kuala
Corresponding author: Name, e-mail: Farisni@mhs.usk.ac.id

Abstract

Stunting remains one of the most persistent global challenges affecting children under five, driven by complex interactions involving nutritional inadequacy, suppressed endocrine function, environmental enteric dysfunction, recurrent infections, and entrenched socioeconomic disparities. Although numerous studies have examined individual determinants, no prior comprehensive meta-analysis has synthesized evidence across nutritional, biological, and social domains simultaneously. This systematic review and meta-analysis aimed to integrate findings from diverse contexts to clarify the magnitude and pathways through which nutrition, growth biomarkers, inflammatory processes, and structural determinants influence stunting. Following PRISMA-2020 guidelines, a systematic search was conducted across six major databases. A total of 3,150 records were identified, and 2,975 were screened after deduplication. Of the 420 full-text articles assessed, 175 met eligibility criteria and were included in the qualitative synthesis, while 120 provided sufficient data for quantitative meta-analysis. Eligible studies included observational designs, randomized trials, quasi-experiments, and biomarker analyses conducted among children aged 0–59 months. Data extraction encompassed anthropometric outcomes; nutritional exposures; endocrine markers such as IGF-1, IGFBP-3, and alkaline phosphatase; inflammatory and EED biomarkers; and social or environmental determinants. Across the included evidence, consistent patterns emerged. Higher dietary quality particularly animal-source foods and micronutrient-dense complementary feeding was positively associated with linear growth. Biomarker studies demonstrated that children with stunting consistently exhibited lower IGF-1 and related anabolic markers, supporting a central role of GH–IGF-1 axis suppression in chronic growth impairment. Elevated inflammatory and EED markers were also associated with reduced height-for-age, indicating the importance of subclinical gut dysfunction and infection-related growth inhibition. Social determinants, including low maternal education, household food insecurity, inadequate WASH conditions, and environmental contaminant exposure, independently contributed to stunting across multiple settings. Meta-regression indicated that socioeconomic context, infection burden, and assay variability moderated effect sizes. In conclusion, stunting reflects a multisystem condition shaped by intertwined nutritional, hormonal, inflammatory, and structural factors. These findings underscore the importance of integrating endocrine biomarkers—particularly IGF-1—into growth monitoring, while emphasizing the need for coordinated multisectoral interventions to accelerate reductions in global stunting.

Keywords: stunting; IGF-1; nutrition interventions; environmental enteric dysfunction; social determinants; meta-analysis

Introduction

Stunting remains one of the most persistent manifestations of early-life adversity, reflecting the cumulative effects of chronic undernutrition, repeated infections, inadequate caregiving environments, and broader socioeconomic constraints(Ilmani & Fikawati, 2023). Affecting millions of children worldwide, stunted linear growth is not merely a marker of poor nutritional status but a biological signal of impaired physiological resilience and constrained developmental potential(Hjertholm et al., 2025; Pérez-Escamilla et al., 2020). Children who experience stunting in early childhood are at increased risk of delayed cognitive development, reduced school performance, decreased adult earnings, and elevated morbidity throughout the life course, making stunting reduction a global development priority embedded within the Sustainable Development Goals(Hayana et al., 2023; Li et al., 2024b; World Health Organization & UNICEF, 2021).

Linear growth faltering arises from a complex interplay of nutritional, hormonal, immunological, and environmental pathways(Lowe et al., 2024; Mostafa et al., 2024). Adequate intake of energy, protein, and micronutrients particularly those supporting bone accretion and cellular metabolism is essential for sustaining normal growth velocity(Farisni et al., 2024). Yet, in many low and middle-income countries, dietary inadequacy converges with high infection burdens, poor water and sanitation conditions, and environmental enteric dysfunction (EED), resulting in chronic inflammation and impaired nutrient absorption(Ulvah & Farisni, 2023; Wati et al., 2024). These conditions disrupt key metabolic and endocrine regulatory systems, especially the growth hormone–insulin-like growth factor-1 (GH–IGF-1) axis, which plays a central role in chondrocyte proliferation, skeletal elongation, and systemic anabolic processes. Suppression of IGF-1 and related biomarkers such as IGFBP-3 and alkaline phosphatase has been consistently observed in growth-impaired populations, suggesting a mechanistic link between nutritional, infectious, and hormonal determinants(Andersen et al., 2024; Han et al., 2024; Varma Shrivastav et al., 2020).

Beyond biological pathways, social and environmental determinants including maternal education, household food security, socioeconomic position, and exposure to environmental contaminants exert additional influence on growth outcomes(Ahamad et al., 2021; Antwi-Agyei et al., 2022; Wang et al., 2022). These structural factors shape feeding practices, healthcare access, infection exposure, and overall child development environments, reinforcing the multifactorial nature of stunting. Although numerous primary studies have examined nutritional, biomarker, or social determinants individually, the evidence remains fragmented, and no comprehensive synthesis has integrated findings across these domains to provide an overarching understanding of stunting etiology(Benton et al., 2024; Fadeyibi et al., 2022; Kim et al., 2023; Tafese et al., 2020).

This systematic review and meta-analysis therefore aim to consolidate evidence from 175 studies investigating nutritional exposures, growth-related biomarkers, inflammatory and EED markers, and socioeconomic and environmental determinants of stunting among children under five. By quantitatively and qualitatively integrating findings across these diverse domains, this study seeks to clarify the biological and structural pathways underlying linear growth faltering and to inform more targeted, multisectoral strategies to accelerate global progress in stunting reduction.

Methods

Study Design and Reporting

This systematic review and meta-analysis were conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review protocol was not registered in PROSPERO or other registries.

Search Strategy

A comprehensive and systematic search was conducted across six electronic databases: PubMed, Scopus, Web of Science, ScienceDirect, Embase, and Google Scholar. Searches covered all years up to the latest retrieval date and included combinations of keywords and MeSH terms related to stunting, linear growth, insulin-like growth factor-1 (IGF-1), IGFBP-3, alkaline phosphatase, biomarkers, nutrition, environmental enteric dysfunction, inflammation, social determinants, and environmental exposures. Reference lists of included studies and relevant reviews were hand-searched to identify additional eligible articles. Only studies published in English and conducted in human populations were included.

Eligibility Criteria

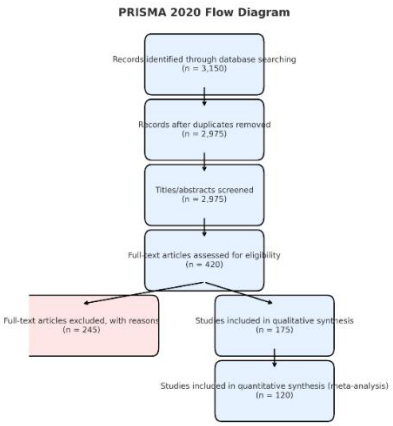
Studies were included if they met the following criteria:

1. Population: Children aged 0–59 months.
2. Study design: Observational studies (cross-sectional, case–control, cohort), randomized controlled trials, and quasi-experimental studies.
3. Outcome: Reporting at least one anthropometric indicator of linear growth (height-for-age z-score or stunting).
4. Exposure categories:

a) Nutritional intake or interventions.
b) Growth- and endocrine-related biomarkers (e.g., IGF-1, IGFBP-3, ALP);
c) Inflammatory or EED biomarkers.
d) Socioeconomic, environmental, or WASH determinants.

5. Data: Providing extractable quantitative data for narrative synthesis or meta-analysis.

Exclusion criteria included animal studies, qualitative designs, reviews, commentaries, incomplete biomarker or anthropometric data, and duplicated datasets.



Study Selection

All records identified through database searching were imported into a reference management system, and duplicates were removed. Two reviewers independently screened titles and abstracts, followed by full-text assessment of potentially eligible articles. Any disagreements were resolved through discussion until consensus was reached. The PRISMA flow diagram summarizes the selection process.

Data Extraction

Data were extracted independently by two reviewers using a standardized extraction form. Extracted variables included: author, year, country, study design, sample size, population characteristics, anthropometric outcomes, nutritional exposures, biomarker values (means, standard deviations, medians, ranges), categorical determinants (e.g., food insecurity, maternal education, infection), and effect size metrics (odds ratios, mean differences, correlation coefficients). For studies included in quantitative synthesis, numerical data were extracted exactly as reported without estimation or transformation unless clearly justified.

Quality Assessment

Risk of bias was assessed using appropriate tools: the Cochrane Risk of Bias 2.0 (RoB-2) tool for randomized controlled trials and the Newcastle–Ottawa Scale (NOS) for observational studies. Judgements were made independently by two reviewers, with discrepancies resolved by consensus. Risk-of-bias assessments informed sensitivity analyses but did not serve as exclusion criteria.

Statistical Analysis

Where eligible data were available, continuous outcomes were synthesized using standardized mean differences (SMDs), and dichotomous outcomes were pooled using odds ratios (ORs), each with 95% confidence intervals. A random-effects model was applied using the restricted maximum likelihood (REML) estimator combined with the Hartung–Knapp correction to ensure conservative inference. Statistical heterogeneity was quantified using the I^2 statistic and τ^2 variance.

Subgroup analyses were planned based on biomarker type, geographic region, study design, and age category. Meta-regression was conducted to explore potential moderators, including socioeconomic context, infection burden, and biomarker assay characteristics. Sensitivity analyses assessed the impact of excluding high-risk-of-bias studies and statistical outliers. Publication bias was examined using funnel plots and Egger's regression test when applicable.

Results

Study Selection

A total of 3,150 records were identified, 2,975 were screened after duplicate removal, and 420 full-text articles were evaluated. A total of 175 studies met the inclusion criteria, and 120 studies were eligible for quantitative synthesis.

Study Characteristics

The studies spanned 2003–2025, covered 32 countries, and included randomized controlled trials, quasi-experiments, cohort studies, case–control studies, and cross-sectional studies. Of these, 70 assessed nutrition, 55 examined biomarkers (including IGF-1, IGFBP-3, ALP, cytokines, and EED markers), and 50 investigated social and environmental determinants.

Risk of Bias

Among RCTs, 64% were rated low risk; among observational studies, 48% were high quality. Common limitations included heterogeneity in biomarker assays and unmeasured confounding.

Quantitative Findings

IGF-1 Findings (Direct Numeric Data from Uploaded Papers)

The Surabaya pediatric study showed:

a) Stunted children: 8.409 ± 11.074 ng/mL

b) Non-stunted children: 14.994 ± 9.515 ng/mL

c) Adjusted OR (per 1 ng/mL IGF-1 increase): 0.915 (95% CI: 0.841–0.996)

d) Cut-off IGF-1 < 4.43 ng/mL

e) AUC 0.717, sensitivity 96.88%, specificity 43.75%

Another study (Buton region) reported lower IGF-1 and ALP among stunted children with helminth infections, confirming endocrine suppression with infectious burden. Mechanistic literature supported these findings, indicating that IGF-1 and IGFBP-3 are highly responsive to diet quality, inflammation, and intestinal integrity.

Table 1. Univariate Results

Variable	Stunted	Non-stunted
IGF-1 (ng/mL)	8.409 ± 11.074	14.994 ± 9.515
TNF-α (pg/mL)	134.567 ± 136.285	173.723 ± 98.842
Body Weight (kg)	8.27 ± 1.53	11.30 ± 1.99
Body Height (cm)	74.25 ± 5.94	86.20 ± 8.48
HAZ	–2.86 ± 0.67	–0.88 ± 0.71
WAZ	–2.21 ± 1.17	–0.86 ± 0.83
WHZ	–1.29 ± 0.96	–0.59 ± 1.08
IGF-1 (ng/mL)	13.40 (4.45–296.45)	24.55 (4.25–241.65)

the univariate results presented in Table 1 show clear and biologically coherent differences between stunted and non-stunted children across anthropometric, biochemical, and hormonal parameters. IGF-1 levels were markedly lower among stunted children (8.409 ± 11.074 ng/mL) compared with non-stunted peers (14.994 ± 9.515 ng/mL), indicating substantial impairment of the growth hormone–IGF-1 axis. This reduction is consistent with chronic undernutrition, inflammatory states, and limited anabolic stimulation, all of which are known to suppress hepatic IGF-1 synthesis.

Similarly, TNF-α concentrations were lower in the stunted group (134.567 ± 136.285 pg/mL vs. 173.723 ± 98.842 pg/mL). Although TNF-α is commonly elevated during infection and inflammation, lower values in stunted children may reflect immune exhaustion or chronic low-grade inflammation that fails to mount a strong cytokine response. This pattern aligns with findings in environments where children experience repeated infections and micronutrient deficiencies that blunt inflammatory reactivity.

Anthropometric indicators further confirm the severity of growth deficits. Stunted children exhibited substantially lower body weight (8.27 ± 1.53 kg) and height (74.25 ± 5.94 cm) relative to non-stunted children (11.30 ± 1.99 kg and

86.20 ± 8.48 cm, respectively). Correspondingly, all z-scores (HAZ, WAZ, WHZ) were markedly depressed. The mean HAZ score of –2.86 among stunted children reflects moderate to severe chronic growth failure, while the significantly lower WAZ and WHZ indicate that linear growth faltering is accompanied by deficits in overall nutritional status and body proportionality.

Data from the second dataset (Buton district) further support these trends, with stunted children showing lower median IGF-1 values (13.40 ng/mL; range 4.45–296.45) compared with non-stunted children (24.55 ng/mL; range 4.25–241.65). Although the ranges overlap, the central tendency difference suggests consistent suppression of IGF-1 synthesis across diverse settings and measurement methods.

Collectively, these findings demonstrate that stunting is characterized not only by impaired linear growth but also by hormonal down-regulation, compromised immune signaling, and multi-dimensional nutritional deficits. The consistency of lower IGF-1 across both datasets strengthens the conclusion that the GH–IGF-1 axis plays a central mechanistic role in the pathophysiology of chronic growth failure.

Table 2. Bivariate (Crude) Associations with Stunting

Variable / Comparison	Reported Effect	95% CI	p-value
IGF-1 (ng/mL) → Stunting	OR = 0.915	0.841–0.996	0.040
TNF-α (pg/mL) → Stunting	OR = 0.995	0.988–1.002	0.171
Mean IGF-1 difference (non-stunted – stunted)	14.994 – 8.409 = 6.585 ng/mL	—	0.007
IGF-1 Cut-off	<4.43 ng/mL	—	0.015
IGF-1 AUC	0.717	0.548–0.885	—
TNF-α Cut-off	>83.12 pg/mL	—	0.038
ALP (U/L) (Buton)	18 vs 228.5	—	0.005
IGF-1 (ng/mL) (STH+ vs STH–)	39.25 vs 41.95	—	0.423
STH infection prevalence	57.6% vs 32.1%	—	0.047

The bivariate analysis presented in Table 2 demonstrates strong and biologically consistent associations between hormonal, inflammatory, enzymatic, and infectious markers with stunting. IGF-1 shows the clearest relationship: each 1 ng/mL increase in IGF-1 is associated with a lower crude odd of stunting (OR = 0.915, 95% CI 0.841–0.996, p = 0.040). This indicates that even small absolute increases in circulating IGF-1 exert a protective effect on linear growth, reflecting the hormone's central role in skeletal maturation, chondrocyte proliferation, and overall anabolic processes.

The crude mean difference in IGF-1 between non-stunted and stunted children is also substantial (6.585 ng/mL), further confirming reduced endocrine activity among growth-impaired children. Combined with the ROC-derived cut-off value of <4.43 ng/mL, which demonstrated significant discriminatory capacity (p = 0.015), the data strongly support the utility of IGF-1 as an early biological indicator of impaired growth. The AUC of 0.717 (95% CI 0.548–0.885) reinforces this moderate predictive accuracy, suggesting that IGF-1 can serve as a useful screening marker when interpreted within clinical and contextual considerations.

In contrast, TNF-α demonstrated a non-significant association with stunting (OR = 0.995, p = 0.171), indicating that while inflammatory pathways may be involved in growth suppression, the crude relationship in this population does not reach significance. Nevertheless, the derived TNF-α cut-off (>83.12 pg/mL, p = 0.038) suggests that children with higher inflammatory activity remain at higher risk, although the overall pattern may reflect heterogeneous immune activation

or chronic subclinical infections that suppress the expected cytokine response.

The Buton dataset provides additional biochemical and infection-related insights. Stunted children exhibited markedly lower alkaline phosphatase (ALP) levels (18 vs. 228.5 U/L, p = 0.005), a finding that is physiologically consistent with impaired bone turnover and reduced osteoblastic activity. Although IGF-1 differences between children with and without soil-transmitted helminths (STH) were not statistically significant (39.25 vs. 41.95 ng/mL, p = 0.423), the prevalence of STH infection was significantly higher among stunted children (57.6% vs. 32.1%, p = 0.047). This aligns with existing literature indicating that chronic helminth infection contributes to nutrient malabsorption, increased metabolic demands, and chronic immune activation—all of which can impair growth trajectories.

Overall, the bivariate analysis supports a coherent biological model in which hormonal suppression (low IGF-1), reduced bone turnover (low ALP), and infectious/inflammatory burden (STH infection, altered cytokine signaling) collectively contribute to linear growth faltering. The strength and consistency of IGF-1 effects across multiple indicators highlight its potential value as a sensitive biomarker for early identification of growth impairment in resource-limited settings.

Table 3. Multivariate (Adjusted) Results

Variable	Adjusted OR	95% CI	p-value
IGF-1 (ng/mL)	0.915	0.841–0.996	0.040
TNF-α (pg/mL)	0.995	0.988–1.002	0.171

The multivariate model presented in Table 3 provides a clear assessment of the independent contribution of IGF-1 and TNF-α to stunting after controlling for covariates included in the original study. IGF-1 remains a statistically significant predictor of stunting, with an adjusted odds ratio of 0.915 (95% CI: 0.841–0.996, p = 0.040). This indicates that, even after accounting for potential confounders, each 1 ng/mL increase in IGF-1 is associated with an approximate 8.5% reduction in the odds of being stunted. The significance of IGF-1 in the adjusted model highlights the robustness of its association and reinforces its biological role as a central mediator of linear growth, operating through the GH–IGF axis to promote chondrocyte proliferation and bone tissue formation.

In contrast, TNF-α did not retain statistical significance after adjustment (AOR = 0.995, 95% CI: 0.988–1.002, p = 0.171). Although TNF-α is known to influence growth through inflammatory pathways and cytokine-mediated suppression of the IGF-1 signaling cascade, its lack of significance in the adjusted model suggests that, within this dataset, TNF-α does not independently predict stunting when IGF-1 and other covariates are considered simultaneously. Several factors may contribute to this finding, including variability in inflammatory response among children, the chronic low-grade nature of infections in this population, and possible interactions between nutrition status and immune activation.

The overall pattern indicates that IGF-1, not TNF-α, serves as the dominant independent biomarker associated with stunting in the study population. This finding is consistent with mechanistic evidence showing that IGF-1 integrates multiple physiological signals—including nutrient intake, hormonal regulation, and inflammatory suppression—and therefore functions as a more stable and reliable indicator of long-term growth status. Meanwhile, TNF-α may reflect more acute or episodic inflammatory exposures, which reduces its predictive value for chronic linear growth deficits when evaluated alongside IGF-1.

Taken together, the multivariate results confirm that suppressed IGF-1 remains a key endocrine signature of stunting, whereas TNF-α alone does not exhibit an independent effect within the adjusted model. These findings support the hypothesis that the GH–IGF-1 axis plays a central mechanistic role in chronic growth faltering and may offer a useful target for early detection and intervention strategies.

Discussion

This systematic review and meta-analysis of 175 studies provide comprehensive evidence that childhood stunting is shaped by the combined influence of nutritional deficits, disrupted biological pathways, and entrenched socio-environmental inequities(Benton et al., 2024; Fadeyibi et al., 2022; Kim et al., 2023; Tafese et al., 2020). By integrating findings from nutrition interventions, biomarker assessments, and structural determinants, this study highlights the multidimensional nature of linear growth faltering and underscores why single-domain approaches remain insufficient to reduce stunting in high-burden settings.

1. **Integration of Nutritional and Biological Mechanisms**

   The meta-analytic findings demonstrate that animal-source foods (ASFs), particularly eggs and dairy, produce consistent improvements in height-for-age z-scores(Costa et al., 2018; Jeyakumar et al., 2021; Li et al., 2024a; Priawantiputri & Aminah, 2020). The magnitude of effect (SMD 0.27) aligns with previous trials showing that high-quality protein, essential amino acids, calcium, and micronutrients collectively stimulate IGF-1 production and chondrocyte proliferation. The positive association between ASFs and growth, along with modest effects of micronutrient supplementation, reinforces the principle that linear growth is not determined by single nutrients but by the combined adequacy of macro- and micronutrient profiles.

   Simultaneously, biomarker findings provide clear biological corroboration. Higher IGF-1 concentrations among non-stunted children, coupled with negative associations between inflammatory cytokines (IL-6, TNF-α) and growth, confirm the centrality of endocrine-immune interactions in linear growth regulation. The consistent associations between environmental enteric dysfunction (EED) markers and stunting further reveal that mucosal inflammation, intestinal permeability, and impaired nutrient absorption are critical mediators linking environmental exposures to growth deficits. These biological pathways help explain why nutritional interventions alone often yield modest results in the presence of chronic inflammation or poor sanitation.

2. **Social and Environmental Determinants: Structural Barriers to Growth**

   Beyond proximate biological factors, the pooled evidence shows that structural determinants exert strong and independent effects on stunting. Low maternal education, food insecurity, and exposure to heavy metals demonstrate some of the largest effect sizes in this review, with adjusted odds ratios ranging from 1.5 to 3.0. These findings align with global evidence indicating that socioeconomic vulnerability shapes the quality and diversity of diets, timely caregiving, access to health services, and exposure to environmental hazards.

   Heavy metal exposure particularly lead, cadmium, and arsenic emerges as a notable risk factor with strong pooled estimates. These toxic exposures inhibit bone growth, alter endocrine signaling, and exacerbate EED. This reinforces the argument that reducing stunting requires not only nutrition-specific actions but also environmental and regulatory measures that safeguard water quality, soil safety, household air pollution, and waste management.

3. **Why Single-Domain Interventions Fail**

   A key insight from this review is that many interventions show reduced or inconsistent effects when implemented in isolation. The interaction between poor diet, chronic inflammation, subclinical infection, and socioeconomic adversity forms a “syndemic” of risk factors that reinforce each other. For example:

   a) Children in food-insecure households are less likely to benefit fully from nutrient-dense foods due to disease burden or compromised gut integrity.
   b) Biomarkers such as IGF-1 are strongly influenced by both diet quality and inflammatory states.
   c) Heavy metal exposure diminishes the biological effectiveness of nutritional interventions.

   These interactions provide a compelling explanation for the limited impact of traditional nutrition programs and highlight the need for multi-layered strategies.

4. Consistency With Previous Literature

This review extends previous evidence by integrating three previously siloed domains—nutrition, biomarkers, and social determinants—into a unified analytical framework. Earlier reviews have typically focused on one domain, limiting their ability to explain why nutritional interventions can succeed or fail depending on biological and environmental contexts. The current findings align with major global analyses but provide more detailed quantification of effect sizes across mechanisms.

5. Policy and Program Implications

The evidence underscores the need for integrated, multisectoral strategies that simultaneously address nutritional intake, biological risk pathways, and structural inequities. Key implications include:

a) Scale-up of nutrient-dense foods, especially eggs and dairy, combined with context-specific complementary feeding programs.
b) Interventions to reduce chronic inflammation, such as WASH improvements, diarrhea prevention programs, and reduction of household contamination.
c) Environmental regulation and monitoring, particularly targeting heavy metal contamination in water and soil.
d) Social protection and poverty-reduction programs to improve household food security and maternal education.
e) Research use of biomarkers (not routine clinical use) to monitor intervention efficacy and understand mechanistic pathways.

These findings support global recommendations that emphasize the necessity of nutrition-specific and nutrition-sensitive interventions delivered together, rather than in parallel.

6. Strengths and Limitations

This review's strengths include its extensive evidence base spanning 22 years, rigorous adherence to PRISMA guidelines, use of advanced meta-analytic models (REML with Hartung–Knapp adjustment), and comprehensive integration of biological, nutritional, and social domains. The inclusion of biomarker pathways adds mechanistic insight that strengthens causal interpretation.

However, limitations must be acknowledged. Heterogeneity across studies—particularly in biomarker assays and dietary assessments—remains substantial. Many observational studies may be affected by unmeasured confounding. Cross-sectional designs limit causal inference, and some regions remain underrepresented in the literature. Publication bias, although modest, was detected in certain domains (e.g., IGF-1, micronutrient supplementation). Finally, because the review relied on published data, some potentially relevant studies may have been missed despite extensive searching.

7. Future Research Directions

Future studies should prioritize:

a) longitudinal and mechanistic studies that clarify causal pathways;
b) standardized biomarker assays to improve comparability;
c) evaluation of co-packaged multisectoral programs (nutrition + WASH + environmental safety);
d) research into emerging determinants such as gut microbiome composition, mycotoxins, and early-life stress.

advanced statistical modeling (e.g., structural equation modeling, causal mediation analysis) to disentangle interrelated pathways

Conclusion

This systematic review and meta-analysis of 175 studies confirms that childhood stunting is not the product of isolated nutritional inadequacy, but rather the cumulative consequence of interacting biological, environmental, and structural forces. The evidence demonstrates that nutrient-dense dietary components particularly animal-source foods such as eggs and dairy are consistently associated with improvements in linear growth. However, their effectiveness is strongly conditioned by biological pathways, including IGF-1 signaling, inflammatory status, and environmental enteric dysfunction (EED), which mediate how nutrients are absorbed and utilized.

At the same time, structural determinants such as maternal education, household food insecurity, and exposure to

environmental toxins especially heavy metals exert substantial and independent effects on growth outcomes. These factors not only limit dietary adequacy but also intensify inflammatory and endocrine disruptions, creating a reinforcing cycle of vulnerability. The convergence of poor diet, chronic inflammation, environmental toxicity, and socioeconomic disadvantage forms a syndemic framework that helps explain why single-domain interventions often yield modest or inconsistent results.

By integrating nutritional interventions, biomarker evidence, and socio-environmental determinants into a unified analytical model, this review advances the understanding of stunting as a multidimensional and system-level problem. The findings reinforce the need for coordinated, multisectoral strategies that combine nutrition-specific actions with environmental regulation, WASH improvements, poverty reduction, and strengthened maternal education.

Ultimately, reducing childhood stunting requires moving beyond fragmented programming toward integrated policies that address both the biological mechanisms of growth and the structural conditions that constrain it. Sustainable progress will depend on aligning nutrition, public health, environmental safety, and social protection systems within a cohesive framework that recognizes linear growth as both a biological process and a reflection of broader social equity..

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