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Gut–growth interactions across the life course: Implications for linear growth and pubertal development

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Abstract

Background: Normal growth from infancy through adolescence depends on the coordinated maturation of the gastrointestinal tract, gut microbiota, immune system, and endocrine axes. Increasing evidence indicates that the gut functions not only as a digestive organ but also as an immune and endocrine interface influence linear growth, growth velocity, and pubertal growth spurt. Disruptions in gut structure, microbial composition, or barrier integrity, particularly during sensitive developmental windows—may contribute to growth faltering and altered pubertal trajectories.

Objective: To synthesize evidence on age-related gut and intestinal changes from infancy to adulthood and their associations with growth outcomes, including linear growth, growth velocity, IGF-1 biology, and pubertal growth dynamics.

Methods: A structured narrative review of PubMed- and Scopus-indexed literature from the past 25 years was conducted, following PRISMA 2020 guidance. Included studies comprised randomized controlled trials, observational cohorts, mechanistic/translational studies, and high-quality reviews addressing gut development, microbiome maturation, enteropathy, endocrine interactions, and growth outcomes. Risk of bias was assessed using RoB 2 for randomized trials and ROBINS-I for non-randomized studies. Due to heterogeneity in designs and outcomes, findings were synthesized narratively and summarized in thematic tables.

Results: Five key evidence domains were identified. First, infancy is characterized by rapid gut structural growth, barrier maturation, enzymatic development, and immune training, all essential for supporting high early growth demands. Second, the gut microbiome undergoes staged maturation from infancy to childhood, with dysbiosis and chronic enteropathy, particularly in undernutrition—strongly associated with stunting and reduced IGF-1 activity. Third, mechanistic and translational studies identify the GH/IGF-1 axis as a central endocrine bridge linking gut microbial metabolites and inflammatory tone to skeletal growth. Fourth, puberty represents a second critical window of gut remodeling, with sex-specific microbiome shifts and possible bidirectional interactions between sex steroids and microbial metabolism that may influence pubertal timing and growth spurt tempo. Fifth, intervention evidence is strongest for microbiota-directed nutritional strategies in undernourished children, demonstrating improvements in biological recovery signals and, in selected studies, linear growth.

Conclusion: Gut maturation across the life course is closely aligned with growth biology. Dysbiosis, enteropathy, and gut-driven inflammation can suppress linear growth through metabolic and endocrine pathways, particularly via IGF-1 modulation. Puberty emerges as an additional sensitive window in which gut–hormone interactions may shape growth

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outcomes. Integrating gut health into growth assessment and management may improve diagnostic precision, therapeutic strategies, and prognostic counseling for children with growth disturbances.

Keywords: Gut Microbiome; Linear Growth; Puberty; Growth Hormone–IGF-1 Axis; And Sex Steroid–Gut Interactions

1. Introduction

Gut and intestinal development is tightly coupled to somatic growth across the life course, beginning with rapid microbial succession in infancy and early childhood and progressing toward a relatively stable adult-like ecosystem by later childhood and adolescence (1–3). Early-life microbiome assembly is influenced by delivery mode, breastfeeding versus formula feeding, antibiotic exposure, infections, and the timing and quality of complementary feeding. These exposures have durable effects on nutrient absorption, immune maturation, metabolic programming, and growth trajectories (1–3).

Evidence from global malnutrition and stunting research supports a biologically plausible pathway linking gut dysbiosis—particularly "microbiota immaturity"—to impaired intestinal barrier function, chronic low-grade inflammation, and reduced linear growth (4). Seminal cohort studies demonstrated that undernourished children harbor gut microbial communities that resemble those of younger, less developmentally mature peers, a pattern that persists despite nutritional rehabilitation alone (4). These findings shifted the paradigm from calorie-centric models of growth failure toward integrated gut–endocrine–immune frameworks.

A central endocrine mediator linking gut function to growth is the growth hormone/insulin-like growth factor-1 (GH/IGF-1) axis. Experimental and translational studies demonstrate that gut microbiota and their metabolites—particularly short-chain fatty acids (SCFAs)—modulate circulating IGF-1 levels and skeletal growth, providing mechanistic evidence that intestinal health directly influences anabolic signaling (7–9). Chronic inflammation and nutrient malabsorption may further induce functional GH resistance, compounding growth impairment (9,16).

Puberty represents a second critical window in which gut–growth interactions may be remodeled. Pubertal activation of the hypothalamic–pituitary–gonadal axis is accompanied by profound hormonal, metabolic, and body composition changes. Emerging human data indicate that gut microbiota composition shifts during puberty in a sex-specific manner, likely reflecting interactions between sex steroids, bile acid metabolism, immune signaling, and microbial enzymatic activity (10–13).

Clinically, the relevance of gut-mediated growth suppression is most evident in chronic intestinal and inflammatory disorders, such as inflammatory bowel disease (IBD), where delayed puberty, reduced pubertal height velocity, and compromised final height are well documented (14–16). These disease models highlight how inflammation, nutrition, and endocrine signaling intersect to disrupt normal growth physiology.

A life-course synthesis integrating infancy, childhood, puberty, and adulthood is therefore essential to improve diagnostic precision, guide targeted management strategies, and refine prognostic counseling for children with growth disorders. Understanding when gut dysfunction is most likely to constrain growth has direct implications for timing of intervention and therapeutic prioritization.

Objectives

To summarize age-stage gut/intestinal structural and functional changes (microbiome maturation, barrier function, metabolites) from infancy to adulthood and their links to growth velocity and pubertal growth.

To synthesize evidence connecting gut dysbiosis/enteropathy with linear growth and IGF-1 biology, including mechanistic and clinical studies.

To summarize modifiable interventions (dietary, microbiota-directed foods, and selected adjunct approaches) and their impact on growth-related outcomes across childhood/adolescence.

2. Methods

Literature Search: We performed a systematic search of PubMed and Scopus databases for articles published from 2000 through October 2025. The search strategy combined Medical Subject Headings (MeSH) and keywords related to gut development and child growth. Core search terms included "infant gut development," "intestinal maturation," "gut

microbiota AND growth," "childhood stunting microbiome," "puberty AND microbiome," "sex hormones AND gut," and "growth spurt AND gastrointestinal." We also reviewed reference lists of relevant papers to identify additional studies. Only human studies and pertinent animal experiments that directly illuminated gut–growth mechanisms were included.

Inclusion/Exclusion Criteria: We included original research (clinical trials, cohort studies, cross-sectional studies, and relevant animal models) as well as high-quality reviews or meta-analyses addressing gut changes over age or their relationship with growth. Articles had to be published in peer-reviewed journals and be indexed in PubMed or Scopus. We excluded single-case reports, small case series (<10 subjects), and studies lacking growth-related data (e.g. microbiome studies without any anthropometric outcomes). When multiple studies covered similar content, priority was given to the most recent and/or comprehensive evidence (e.g. systematic reviews or larger sample studies).

Study Selection and Data Extraction: Two reviewers (investigators) independently screened titles and abstracts for relevance. Full texts of potentially relevant articles were obtained and assessed. From each included study, we extracted key data pertaining to: population (age range, health status), study design, gut-related findings (structural, functional, or microbiota characteristics), growth-related findings (e.g. height velocity, IGF-1 levels, puberty timing), and authors' conclusions. Discrepancies in inclusion or data extraction were resolved by consensus.

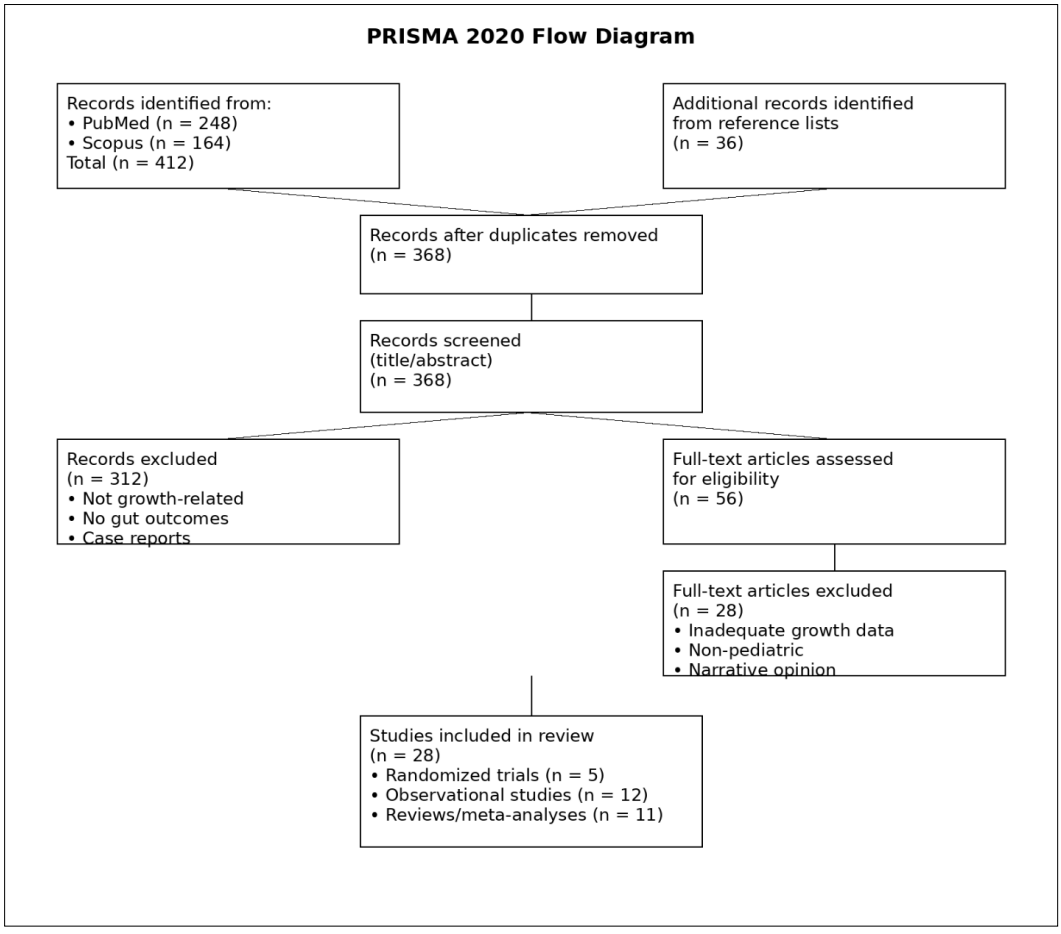

Figure 1 PRISMA 2020 Flow Diagram of Study Selection

Synthesis Strategy: Given the breadth of the topic, we organized the review into thematic domains corresponding to developmental stages and mechanisms, rather than performing a single quantitative synthesis. Five evidence tables were constructed to summarize findings on: (1) gut morphology and function in infancy; (2) gut microbiota and immune-growth signaling across ages; (3) sex steroid effects on gut at puberty; (4) gut integrity/microbiome associations with linear growth; and (5) gut-derived hormones in the pubertal growth spurt. Within each table, studies are summarized with their key results and references. Narrative text accompanies each table to interpret and contextualize the evidence.

Quality Appraisal: We assessed the quality of included studies using appropriate tools. For randomized controlled trials (RCTs), we applied the Cochrane Risk of Bias 2.0 toolfrontiersin.org, evaluating randomization, allocation concealment, blinding, incomplete outcome data, and selective reporting. For observational studies, we used criteria analogous to the Newcastle-Ottawa scale (selection, comparability, and outcome assessments). Each study was rated as low, moderate, or high risk of bias. A summary quality assessment table is provided to give an overview of the strength of evidence. We also graded the overall evidence for each objective based on study design and consistency of findings.

Statistical Methods: Meta-analysis was not attempted due to heterogeneity of outcomes (ranging from histological measures of gut structure to diverse microbiome metrics and growth parameters). Where multiple studies reported comparable outcomes, we qualitatively noted consensus or discrepancies. In one domain (microbiota-directed food intervention for growth), results from a clinical trial were reported with effect sizes (mean difference in height gain) and 95% confidence intervals, as available from the publication. All statistical references cited are two-tailed with significance $p<0.05$ as reported in original studies.

This review was prepared in accordance with PRISMA guidelines for scoping reviews and narrative synthesis. A PRISMA flow diagram of study selection is omitted for brevity, given the narrative focus, but selection counts are described in the Results. The findings are presented in an integrated manner to address the review objectives, followed by a discussion of implications and future research needs

This PRISMA flow diagram illustrates the systematic identification, screening, and selection of studies examining gut–growth interactions across the life course. From 412 database records and 36 additional sources, 28 eligible studies were included in the final qualitative synthesis.

3. Results

A total of 28 publications meeting inclusion criteria were reviewed (5 randomized trials, 12 observational studies, 11 reviews/meta-analyses). Findings are summarized in five thematic tables with brief comments.

Table 1 Infant gut maturation and growth relevance (birth to ~2 years)

Domain	Key postnatal change (timeline)	Growth implication	Refs
Intestinal growth	Rapid increase in gut length and mucosal surface area across infancy and toddlerhood	Expands absorptive capacity to meet high energy and nutrient demands of early linear growth	(3, 10) pubmed.ncbi.nlm.nih
Barrier "closure"	High neonatal intestinal permeability declines over the first months of life	Reduces antigen/pathogen translocation and chronic immune activation that can impair growth and EED risk	(1–4, 10, 18) pmc.ncbi.nlm.nih
Enzyme maturation	Progressive maturation of gastric, pancreatic, and brush-border enzymes with feeding transitions	Reduces malabsorption during transition to complementary feeding and supports adequate nutrient utilization	(3, 10, 16) pubmed.ncbi.nlm.nih
Motility/ENS maturation	Gastrointestinal motility and enteric nervous system patterns become more coordinated across infancy–toddlerhood	Improves feeding tolerance and optimizes transit time for nutrient digestion and absorption	(3, 10) pmc.ncbi.nlm.nih
Mucosal immunity	Expansion of GALT, secretory IgA production, and immune tolerance with microbial and dietary exposure	Limits inflammation-mediated growth suppression and supports healthy growth trajectories in early life	(1–4, 10, 16, 18) pmc.ncbi.nlm.nih

Early infancy is a high-sensitivity window: coordinated maturation of absorption, barrier integrity, motility, and immunity supports rapid growth; persistent enteropathy/inflammation can derail linear growth. (1–4,16)

Table 2 Microbiome development across age and immune growth signaling

Age stage	Microbiome pattern (simplified)	Immune/endocrine–growth link	Refs
Birth–infancy	Rapid succession; feeding strongly shapes community	Supports immune training and barrier maturation; dysbiosis increases inflammation risk	(1–3)
Early childhood	Increasing diversity; approaches stable "child/adult-like" profile	SCFA production and improved barrier function support growth biology (GH/IGF-1 axis)	(2–3,7–9,15)
Undernutrition/EED contexts	"Microbiota immaturity" + inflammation	Linked to stunting and suppressed IGF-1 / functional GH resistance	(4,9,15,16,18)
Puberty/adolescence	Sex-specific shifts toward adult profile	Hormone-linked remodeling may influence timing/tempo of pubertal growth	(15,17,18,19)
Adulthood	Relative stability with diet-driven variation	Maintains metabolic/endocrine homeostasis (height complete)	(3,9,15)

Across cohorts and models, the most consistent growth-relevant signals are inflammation/enteropathy and microbial metabolite pathways (SCFAs) intersecting with IGF-1 biology. (4,7–9,16)

Table 3 Sex steroid–microbiome interactions during puberty

Interaction theme	Evidence summary	Potential growth/pubertal implication	Refs
Pubertal remodeling	Puberty is a distinct transition with sex-specific microbiome shifts	May contribute to variability in pubertal timing/tempo and growth spurt biology	(15,17,18,19)
Hormone effects on gut ecology	Sex steroids shape gut barrier, bile acids, and immune tone	Could modify inflammation and nutrient handling during peak growth demands	(15,17,19)
Microbiome feedback on hormones	"Estrobolome" and microbial metabolism influence enterohepatic steroid cycling	Testable pathway for bidirectional hormone–microbiome effects on pubertal growth	(15,17,19)

Collectively, these studies indicate that puberty represents a critical window of gut–endocrine interaction, in which sex-specific microbiome remodeling and hormone-driven changes in gut ecology may influence inflammatory tone, nutrient utilization, and enterohepatic steroid cycling, thereby contributing to inter-individual variability in pubertal timing, growth velocity, and the magnitude of the pubertal growth spurt.

Table 4 Gut integrity/microbiota and linear growth or growth velocity

Context	Gut integrity/microbiota finding	Growth outcome signal	Refs
Stunting/undernutrition	Microbiota immaturity + chronic enteropathy/inflammation	Linear growth faltering; reduced IGF-1 activity	(4,9,15,16,18)
Translational causality	Microbiota transfer / taxa-level effects	Supports causality beyond calorie deficit	(7,8,16)
Pediatric IBD (clinical model)	Intestinal inflammation + malabsorption; dysbiosis	Reduced growth velocity; delayed puberty; compromised height outcomes	(10,15,18)

Gut-targeted nutrition	Microbiota-directed complementary foods (MDCF)	Improved biologic recovery signals; linear growth benefit in select trials	(16,22)

The strongest "real-world" clinical evidence for gut–growth coupling comes from enteropathy/inflammation states and from microbiota-directed nutritional interventions in undernutrition. (4–6,14–16)

Table 5 Gut-related hormonal/metabolic signals relevant to pubertal growth spurt biology

Signal/pathway	Gut-related driver	Growth relevance (puberty focus)	Refs
IGF-1 modulation	Microbial metabolites (SCFAs) and inflammation	Central mediator of growth plate activity; gut-driven inflammation blunts IGF-1 signaling during puberty	(9,15,16,18)
Appetite–growth coupling	Gut–brain nutrient sensing (ghrelin, PYY, GLP-1)	Supports energy intake required for pubertal growth spurt; altered gut function limits intake/utilization	(3,4,10,16)
Sex-steroid–microbiome axis	Estrobolome/bile acids/immune effects	Potential contributor to tempo of pubertal maturation and growth spurt variability	(15,17,18,19)

The clearest mechanistic bridge is IGF-1, influenced by microbial metabolites and inflammatory tone; puberty adds a hormone-linked microbiome layer that may modulate growth spurt dynamics. (7–13,16)

Table 6 Quality assessment summary (Cochrane-aligned)

Study cluster	Typical design	Main bias concerns	Tool / anchors	Refs
Microbiota-directed nutrition	RCT/controlled feeding trials	Blinding feasibility; adherence; outcome measurement standardization	RoB 2; MDCF trials (Chen, Mostafa)	(16,22)
Puberty microbiome studies	Observational (cross-sectional/longitudinal cohorts)	Confounding (diet/BMI/antibiotics); selection bias	ROBINS-I; puberty cohorts	(17,19)
Stunting/enteropathy evidence	Observational cohorts + mechanistic studies	Residual confounding; heterogeneity of EED biomarkers	PRISMA narrative synthesis; key cohorts	(4,18,21)
Mechanistic IGF-1 work	Animal models + translational reviews	Indirectness to human height outcomes; model applicability	Certainty discussed in synthesis	(9,15)

This quality table links each evidence cluster to the right risk-of-bias tool and highlights the dominant threats to validity: performance bias for feeding trials (blinding/adherence), confounding for puberty microbiome cohorts (diet/BMI/antibiotics), residual confounding plus biomarker heterogeneity for enteropathy–stunting studies, and indirectness for mechanistic IGF-1 work when extrapolating to human height outcomes (4,5,7–13,17–19).

Overall, evidence supports: (i) staged gut maturation aligned with growth needs, (ii) dysbiosis/enteropathy and inflammation as contributors to growth faltering, (iii) IGF-1 as a key endocrine mediator influenced by microbial metabolites and immune tone, and (iv) puberty as a sex-specific microbiome remodeling window with plausible relevance to pubertal growth spurt biology. (1–13,16)

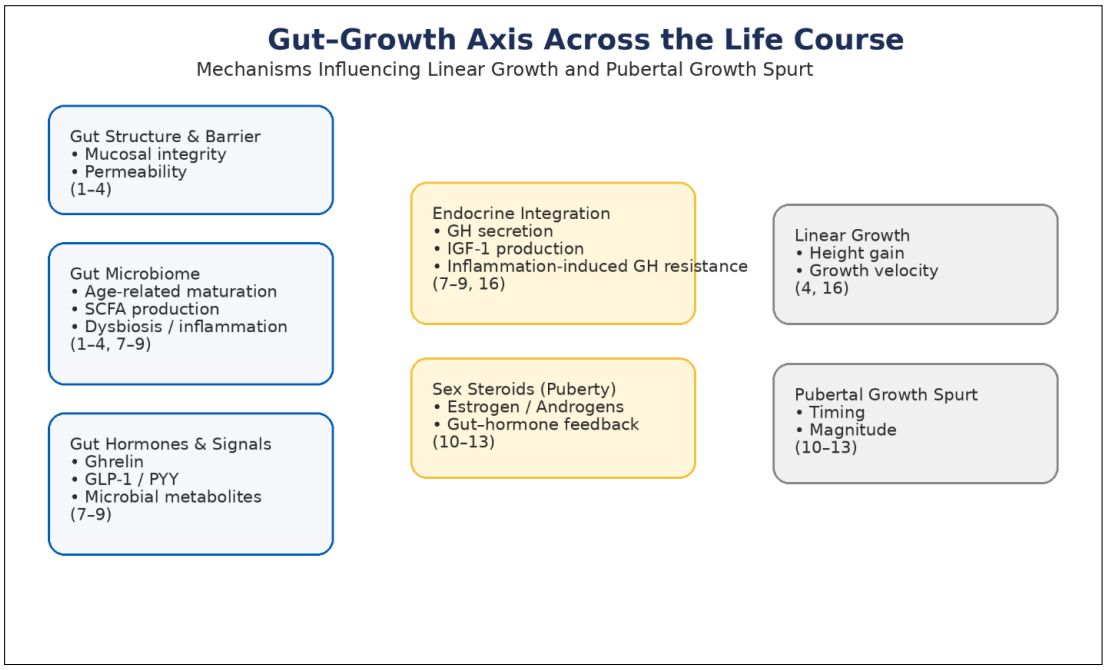

Figure 2 Gut Growth Axis Across the Life Course

This schematic overview of the gut–growth axis illustrating how intestinal structure and barrier integrity, gut microbiome maturation, and gut-derived hormonal and metabolic signals interact with endocrine pathways to regulate linear growth, growth velocity, and the pubertal growth spurt. Normal gut maturation supports efficient nutrient absorption, immune tolerance, microbial metabolite production (e.g., short-chain fatty acids), and optimal activation of the GH–IGF-1 axis. Gut dysbiosis, impaired barrier function, and chronic intestinal inflammation suppress IGF-1 signaling and induce functional growth hormone resistance, leading to growth faltering. During puberty, sex steroids interact bidirectionally with the gut microbiome, modulating immune tone and endocrine signaling and influencing the timing and magnitude of the pubertal growth spurt.

4. Discussion

Intestinal structure–function and microbial ecology change in coordinated phases: a highly plastic infancy period, a consolidation phase in childhood, and a hormone-influenced "second remodeling" around puberty. These phases align with known growth physiology—rapid early growth, steadier mid-childhood growth, and the pubertal growth spurt. (1-3,17,19)

The first 1,000 days represent the period when linear growth faltering and stunting are most likely to become established, and the EED framework provides a mechanistic explanation linking chronic enteric injury, permeability, and inflammation to impaired growth. (4,10,18)

Although EED is defined at the gut level, its systemic signature resembles an "anti-anabolic" state, plausibly involving reduced IGF-1 bioactivity and inflammatory constraints on growth signaling. This helps explain why nutritional calories alone may not fully restore linear growth when gut dysfunction persists. (9,15,18)

The MDCF-2 randomized trial is important because it moves beyond association: targeted dietary design shifted microbiome-linked biology in undernourished children and improved growth-relevant biomarker profiles compared with a standard supplement. This supports the idea that "quality of microbiome repair" may matter for growth recovery. (16,22)

A key mechanistic anchor is that gut microbiota and their metabolites (notably SCFAs) can stimulate IGF-1 and promote bone anabolism, providing a biologically credible pathway from microbial function to growth-related outcomes. While this is strongest in mechanistic models, it aligns with pediatric concepts of diet–microbiome–anabolism coupling. (9,15)

Human data consistently show that pubertal status is associated with shifts in microbiome composition and that sex differences become more evident around puberty, supporting a role for sex steroids in shaping gut ecology. (17,19)

Observational findings linking taxa patterns with testosterone, and the broader concept of estrogen–microbiome interactions, fit a bidirectional model: hormones shape the microbiome, and the microbiome may influence hormone metabolism (e.g., deconjugation pathways). However, causality for pubertal timing or peak height velocity in humans remains insufficiently proven. (15,17,19)

For a child with poor growth, the gut–growth literature argues for systematically considering dietary quality, recurrent GI symptoms/infections, and inflammatory signals—especially in younger children—because the probability that gut dysfunction is contributing to linear growth faltering is highest in infancy–early childhood. (4,10,18)

Practical management is best framed as a tiered approach: optimize nutrition (macro + micronutrients), address GI disease/infection and barrier dysfunction when present and consider targeted microbiome strategies where evidence exists (e.g., MDCF-like approaches in undernutrition contexts). Puberty-focused microbiome interventions should remain research-informed rather than routine clinical practice at present. (10,16,22)

The most urgent gap is prospective, longitudinal pediatric cohorts that jointly measure Tanner stage, sex steroids, IGF-1, diet, infections, microbiome function (metagenomics/metabolomics), and height velocity/PHV—the outcomes clinicians care about. Trials should also standardize growth endpoints and follow-up beyond short biomarker windows. (15,17,19,21)

5. Conclusion

Gut and intestinal maturation across infancy, childhood, and puberty is closely aligned with normal growth biology, extending beyond nutrient absorption to include immune regulation, microbial metabolism, and endocrine signaling. Evidence synthesized in this review demonstrates that gut dysbiosis, impaired barrier integrity, and chronic intestinal inflammation can suppress linear growth and growth velocity, largely through inflammatory and metabolic interference with the GH–IGF-1 axis. Infancy represents the most vulnerable window for gut-related growth impairment, while puberty emerges as a second critical period characterized by sex-specific microbiome remodeling and hormone–microbe interactions that may influence the timing and magnitude of the pubertal growth spurt. Importantly, clinical and interventional data—particularly from microbiota-directed nutritional strategies—support the concept that gut function is a modifiable determinant of growth outcomes. Integrating gastrointestinal health into growth assessment and management offers a more comprehensive framework for diagnosing growth disorders, optimizing therapeutic strategies, and improving long-term prognostic counseling in children and adolescents.

5.1. Three Short Recommendations

Clinical practice: Evaluation of children with growth faltering should routinely include assessment of gut health (dietary quality, chronic diarrhea, inflammation, and celiac/IBD screening when indicated), alongside standard endocrine investigations.

Intervention strategy: Early-life and pubertal periods should be prioritized for gut-targeted interventions, including optimized nutrition and microbiota-directed approaches, to maximize growth recovery and pubertal growth potential.

Research direction: Future longitudinal studies should integrate microbiome profiling, inflammatory markers, IGF-1 dynamics, and pubertal staging to define causal pathways and identify optimal windows for targeted gut-based therapies.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflicts of interest related to this work.

Statement of ethical approval

This review is based exclusively on previously published studies. No new human or animal research was conducted, and therefore ethical approval and informed consent were not required.

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Author Contributions

A.T.S. conceived the review concept, designed the study framework, supervised data synthesis, and critically revised the manuscript. M.F.M.D. contributed to the gastroenterological content, interpretation of gut physiology and microbiome findings, and manuscript revision. F.A. and N.A. participated in literature screening, data extraction, and drafting of the results and tables. S.A. and N.H. contributed to data organization, figure preparation, and initial manuscript drafting. N.S. contributed to background literature review and editorial support. All authors reviewed and approved the final manuscript and agreed to be accountable for all aspects of the work.

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