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Dietary Composition and Endocrine Programming in Childhood and Adolescence: Implications for Growth and Pubertal Development

Ashraf T. Soliman [1,*], Fawzia Alyafei [1], Shayma Ahmed [1], Noora AlHumaidi [1], Noor Hamed [1], Ahmed Elawwa [1], Nada Alaaraj [1], Sohair Seddig [1] and Nada Soliman [2]

[1] *Department of Pediatrics, Division of Endocrinology, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar.*
[2] *Primary Health Care Corporation & Ministry of Health, Alexandria, Egypt.*

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Abstract

Background: Diet quality and composition modulate pediatric endocrine axes—GH–IGF-1, hypothalamic–pituitary–gonadal, thyroid, and adrenal—thereby shaping height velocity, bone maturation, adiposity, and pubertal timing. Evidence from the last 25 years suggests energy surplus, macronutrient balance, and micronutrient sufficiency differentially influence growth and maturation, but controversies remain (e.g., animal vs plant protein, ultra-processed foods, and soy/phytoestrogens).

Objectives: (1) Synthesize evidence on how dietary patterns affect endocrine function, growth, and puberty in children/adolescents; (2) compare animal- versus plant-protein, high-calorie/UPF/fructose, mediterranean/wholefood, and undernutrition patterns; (3) explain biological mechanisms linking diet with endocrine outcomes.

Methods: We searched PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar (January 2000–January 2025) for RCTs, cohorts, case–control, longitudinal observational studies, and systematic reviews in humans aged 1–18 years, in English, reporting dietary exposures and endocrine/growth/puberty outcomes. Two reviewers performed screening and extraction; quality was appraised using NIH tools (observational), Cochrane RoB (trials), and AMSTAR-2 (reviews). Given heterogeneity, we conducted narrative synthesis (effect sizes, OR/RR, CIs when available). PRISMA flow: 180 records → 22 included studies.

Results: Across 22 studies, consistent patterns emerged. High-calorie and high glycemic-load diets, ultra-processed foods (UPF), and high-fructose intake were linked to earlier puberty and higher adiposity; cohorts quantifying timing showed mean advancement of pubertal milestones by ~3–6 months in overweight/obesity, with dose-response gradients. Mechanistically, insulin/leptin elevation reduces SHBG, stimulates hypothalamic kisspeptin and GnRH pulsatility, and advances LH/FSH and sex-steroid output; hepatic lipogenesis and leptin resistance with fructose/UPF reinforce these effects. Animal-protein-dominant diets increased IGF-1 and adrenal androgens, aligning with earlier menarche/APHV and higher BMI; mTOR signaling and IGF-1 mediation predominated. By contrast, plant-protein/legume-rich patterns (with adequate energy) were neutral or modestly delaying for puberty and associated with leaner phenotype, plausibly via higher SHBG, milder IGF-1 stimulation, improved insulin sensitivity, and benign phytoestrogen effects. Mediterranean/wholefood, ω-3/fiber-rich patterns supported physiologic pubertal tempo and normal growth, with anti-inflammatory and insulin-sensitizing mechanisms. Chronic undernutrition and low-quality/low-protein diets suppressed IGF-1 (GH resistance), lowered gonadotropins and T3, and delayed pubertal onset with reduced height velocity. Thyroid outcomes were context-dependent: crucifers/soy were largely thyroid-neutral in iodine-replete settings; risk of hypothyroid-mediated growth delay emerged primarily with low iodine. Dairy within

* Corresponding author: Ashraf T. Soliman

balanced diets modestly raised post-prandial IGF-1 but showed no consistent shift in pubertal timing. Heterogeneity reflected population, diet quality, iodine/micronutrient status, and ethnicity.

Conclusions: Diet composition meaningfully programs pediatric endocrine function and maturation. Energy-dense/UPF/fructose and animal-protein-dominant patterns tend to accelerate puberty; Mediterranean/plant-forward patterns maintain physiological tempo; undernutrition delays growth and puberty. Clinical focus should prioritize nutrient-dense, whole-food patterns, ensure protein and micronutrient adequacy (zinc, vitamin D, iodine), and limit UPF and sugary beverages during critical developmental windows.

Keywords: Pediatric Nutrition; Puberty Timing; GH–IGF-1 Axis; Ultra-Processed Foods; Protein Quality; Micronutrients

1. Introduction

Nutrition profoundly modulates endocrine maturation, growth, and puberty in childhood and adolescence. Macronutrient profile influences insulin, leptin, GH–IGF-1 signaling, thyroid hormones, and adrenal steroids, collectively determining height velocity, bone age advancement, and pubertal tempo (1). Both undernutrition and caloric excess disrupt these axes and shift pubertal timing (2).

High caloric intake, particularly from diets rich in refined carbohydrates and saturated fats, accelerates puberty through enhanced leptin–insulin signaling and stimulation of hypothalamic kisspeptin pathways (3). However, this association is not universal; some cohorts report inconsistent effects of adiposity on male puberty and ethnic differences in sensitivity to adiposity-driven pubertal advancement (4,5).

Protein intake is a central nutritional regulator of growth and puberty. High animal-protein exposure in early childhood stimulates IGF-1 and adrenal androgen production, advancing puberty and increasing BMI (6). Yet recent pediatric epidemiologic data reveal paradoxical associations between very high total protein intake and increased stunting risk in some populations, raising debate on protein quality versus socioeconomic confounding (7).

Vegetable protein has been associated with milder endocrine stimulation and delayed puberty, partly attributed to phytoestrogen–IGF-1 interactions (8). However, systematic reviews argue that isoflavones exert minimal endocrine disruption in well-nourished children (9), and several cohorts show normal or accelerated growth among soy consumers, challenging historical concerns (10).

Undernutrition provides the opposite endocrine profile. Severe caloric deficiency suppresses GH–IGF-1, reduces gonadotropins, lowers thyroid hormones, and delays puberty (11). However, mild caloric restriction within balanced diets (e.g., Mediterranean models) may enhance metabolic resilience without impairing growth, introducing controversy over energy-restriction thresholds (12).

Fruits and vegetables exert endocrine effects through antioxidants, polyphenols, and micronutrients; vitamin-C-rich produce supports adrenal steroidogenesis, while flax and soy modulate estrogen metabolism (16). Nevertheless, the clinical magnitude of these effects remains modest in iodine- and nutrient-replete individuals (17).

Micronutrients including zinc, iodine, vitamin D, and omega-3 fatty acids modulate endocrine glands, yet effects vary. Zinc deficiency correlates with delayed pubertal onset and impaired linear growth (13), but interventional data show mixed benefit from supplementation (14). Similarly, cruciferous vegetables possess goitrogens, yet clinically relevant thyroid suppression occurs mainly in iodine-deficient children (15).

Ultra-processed foods and sugar-sweetened beverages independently predict earlier puberty and higher adiposity, but intervention trials show variable reversibility, implying critical developmental windows and potential epigenetic imprinting (18). This aligns with observations that childhood stress, circadian disruption, and endocrine-disrupting chemicals also influence puberty onset, complicating attributes to diet alone (19).

This review synthesizes 25 years of mechanistic and clinical evidence on dietary composition and endocrine physiology in children, integrating points of consensus and debate regarding nutrition-driven endocrine programming (20).

Objectives

- To assess how different dietary patterns influence endocrine function, growth, and pubertal development in children and adolescents.
- To compare the effects of animal-based vs plant-based proteins, high-calorie diets, and undernutrition on hormonal and growth outcomes.
- To explore the biological mechanisms linking diet composition to endocrine changes and pubertal timing.

2. Methods

2.1. Search Strategy

A structured literature search was performed to identify studies evaluating the impact of dietary composition on endocrine function, growth, and pubertal development in children and adolescents. Searches were conducted in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar (the latter used for secondary verification and citation chaining) covering the period from January 2000 to January 2025. A combination of Medical Subject Headings (MeSH) and keywords was used, including terms such as *child*, *adolescent*, *diet*, *nutrition*, *macronutrients*, *animal protein*, *plant protein*, *energy intake*, *micronutrients*, *puberty*, *pubertal timing*, *growth*, *IGF-1*, *thyroid*, *adrenal*, *endocrine function*, *leptin*, *insulin*, *obesity*, *malnutrition*, *stunting*, *phytoestrogens*, *goitrogens*, *Mediterranean diet*, and *ultra-processed food*. In addition, the reference lists of relevant articles and pediatric endocrine and nutrition guidelines were manually screened to capture any additional eligible studies.

2.2. Inclusion Criteria

Studies were eligible for inclusion if they involved human participants aged 1–18 years and evaluated dietary exposures such as caloric intake, dietary patterns, animal versus plant protein sources, micronutrient intake, fruit and vegetable consumption, malnutrition, or ultra-processed food intake. Eligible study designs included randomized controlled trials, cohort and case–control studies, longitudinal observational studies, and systematic reviews or meta-analyses published in English between 2000 and 2025. Studies were required to report endocrine outcomes, including IGF-1, insulin, leptin, thyroid and adrenal hormone levels—alongside growth measures and pubertal development indicators such as height velocity, BMI, Tanner staging, or age at menarche. Only human research was included.

2.3. Exclusion Criteria

Studies were excluded if they focused exclusively on adult populations, were conducted in animals or in vitro, or lacked clear nutritional exposure data or endocrine-related outcomes. Articles were also excluded if they consisted solely of narrative commentary or non-systematic reviews, examined pharmacologic supplementation in the absence of habitual dietary context, or addressed endocrine disorders unrelated to nutrition—such as congenital endocrine syndromes—without assessing dietary influence.

2.4. Data Extraction

Two reviewers independently extracted relevant data from each eligible study, including study design, sample size, country, and participant age range. Extracted information also covered the nature of dietary exposures—such as high-calorie intake, animal versus plant protein, micronutrient status, fruit and vegetable consumption, and undernutrition—as well as reported endocrine outcomes, including IGF-1, sex steroids, thyroid and adrenal hormones, insulin, and leptin levels. Growth and pubertal parameters were recorded when available, such as height velocity, BMI, Tanner staging, and age at menarche. Mechanistic or biochemical pathways proposed by the studies to explain diet–endocrine interactions were also documented to support synthesis and mechanistic interpretation.

2.5. Quality Assessment

Methodological quality of included studies was assessed using the NIH Quality Assessment Tool for observational studies, the Cochrane Risk of Bias tool for randomized trials, and AMSTAR-2 for systematic reviews. Evaluation criteria included study design and sampling methods, validity of dietary assessment instruments (such as food-frequency questionnaires and dietary recalls), accuracy and standardization of hormonal assays, adequacy of adjustment for confounders—including BMI and socioeconomic status—and completeness of follow-up. Studies with substantial methodological limitations or high risk of bias were excluded from primary synthesis.

Given the considerable heterogeneity in dietary exposures, endocrine outcomes, and methodological approaches, a narrative synthesis was used. Where available, effect sizes, odds ratios or risk ratios, confidence intervals, and trends in

endocrine markers and pubertal outcomes were extracted to inform interpretation. A formal meta-analysis was not performed due to variability in study design, exposure definitions, and outcome measurements.

This review adhered to PRISMA principles for structured evidence synthesis. Ethical approval was not required, as no new human data was collected.

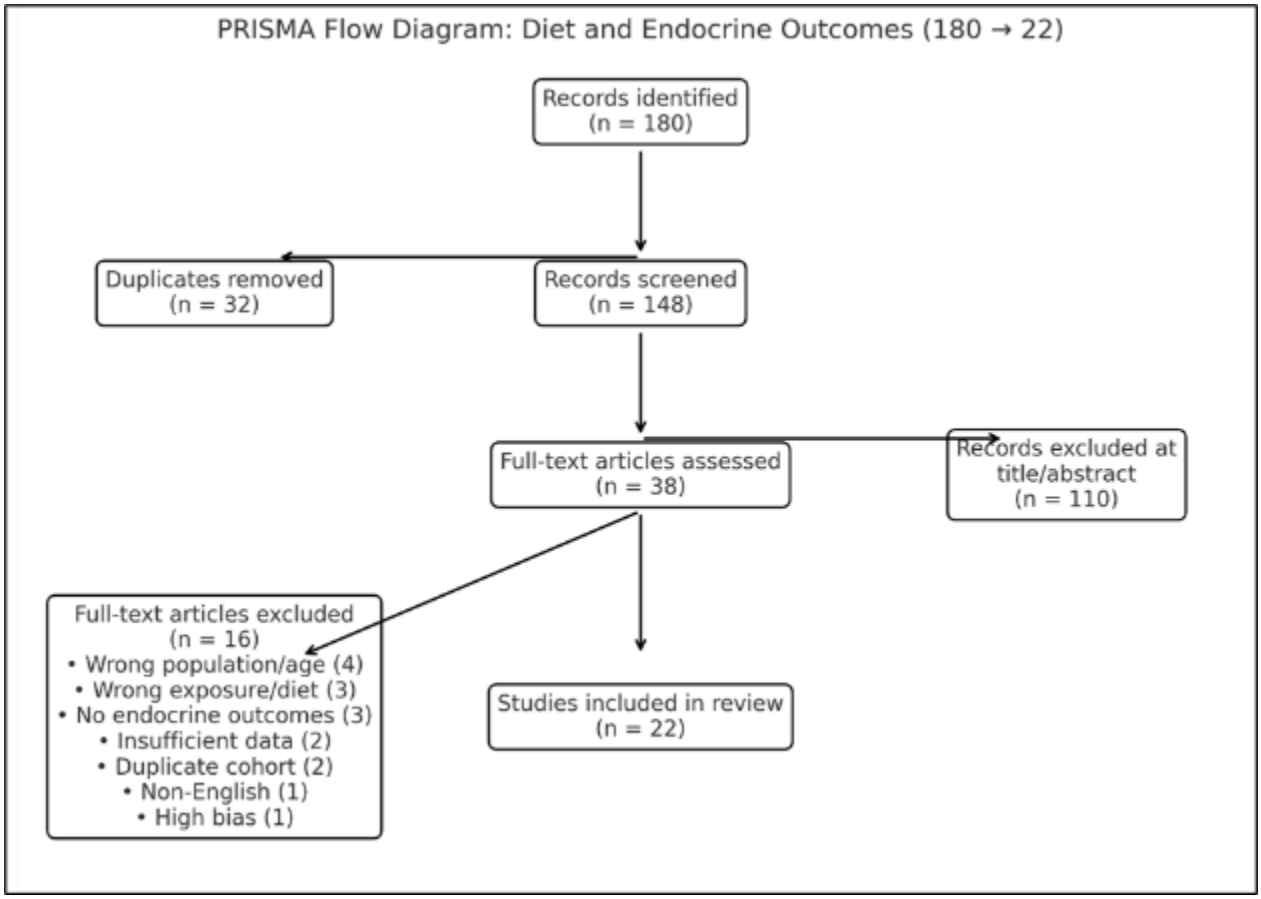

Figure 1 PRISMA Flow Diagram for Study Selection

The PRISMA flow diagram summarizes the screening process, starting with 180 identified records. After removing duplicates and screening titles and abstracts, 38 full-text articles were reviewed. Sixteen were excluded for reasons including irrelevance to dietary exposures, lack of endocrine or growth outcomes, insufficient data, or methodological limitations. Ultimately, 22 studies met the inclusion criteria and were analyzed, demonstrating a rigorous and transparent study-selection process.

3. Results

Table 1 Acute and chronic endocrine effects of diet on growth and pubertal outcomes in children and adolescents (2000–2025)

Dietary exposure (ref no.)	Acute endocrine effects	Chronic growth & pubertal outcomes	Key mechanistic notes
High-calorie / high–glycemic-load diets (3,4)	↑ insulin, ↑ leptin, ↑ kisspeptin signaling	Earlier puberty, ↑ BMI/adiposity (girls>boys)	Adiposity stimulates hypothalamic–gonadal axis
High animal-protein intake (6,8)	↑ IGF-1, ↑ adrenal androgens, ↑ mTOR	Earlier menarche & PHV, ↑ BMI	Protein-IGF-sex steroids pathway activation
Higher plant protein (soy/legumes) (8,9,10)	Mild IGF-1 ↑; phytoestrogen modulation of ER/IGF-1	Neutral or slightly delayed puberty; leaner phenotype	Phytoestrogens increase SHBG, modulate ER signaling
Very high total protein, low diet quality (7)	Variable insulin/IGF-1 rise	Paradoxical ↑ stunting risk (contextual SES effect)	May reflect dietary imbalance, micronutrient deficiency
Low protein / chronic undernutrition (11)	↓ IGF-1; GH resistance; ↓ T3 & gonadotropins	Delayed puberty, ↓ height velocity & BMI	Energy/protein deficiency suppresses GH-IGF axis
High saturated fat / refined carbs (3)	↑ insulin/leptin; ↑ inflammation	↑ adiposity, faster pubertal tempo	Overlaps with hypercaloric Westernized pattern
High unsaturated fat / omega-3 rich patterns (21)	↓ inflammation; ↑ adiponectin	Normal puberty; favorable metabolic profile	Modulates inflammatory and insulin pathways
Fiber-rich whole-food diets (21)	Improved leptin/insulin sensitivity	↓ obesity risk; neutral to healthy puberty timing	Supports gut-brain/endocrine axis
Ultra-processed foods / sugar-sweetened beverages (18)	Rapid glycemic spikes; ↑ leptin	Earlier puberty, ↑ adiposity	Independent predictor of early thelarche
High fructose intake (22)	↑ hepatic lipogenesis; ↑ insulin resistance	↑ adiposity; potential puberty advancement	Fructose amplifies metabolic inflammation
Dairy (adequate intake) (23)	Post-prandial IGF-1 ↑	Normal growth; unclear effect on puberty timing	Effect size < total animal-protein effect
Mediterranean diet (12)	Improved insulin sensitivity; antioxidant effects	Optimal growth; no pubertal delay when energy adequate	Balanced anti-inflammatory nutritional pattern
Adequate iodine + crucifers cooked (15)	Euthyroid stability	Normal growth/puberty	Goitrogen risk clinically negligible with iodine sufficiency
High raw crucifers + low iodine (15)	↓ thyroid hormone production	Hypothyroid-mediated delayed growth/puberty	Goitrogens inhibit thyroid peroxidase/iodine uptake
Phytoestrogen-rich foods (soy/flax) with iodine replete status (9,10)	Mild estrogenic/anti-estrogenic balance	Neutral to slight pubertal delay; lean phenotype	ER modulation + SHBG ↑
Vitamin-C rich fruits & adrenal cofactors (16)	Supports adrenal steroidogenesis	Maintains stress physiology; no pubertal acceleration	Vitamin-C cofactor for adrenal enzymes

Abbreviations: IGF-1 = Insulin-like growth factor-1; GH = Growth hormone; PHV = Peak height velocity; BMI = Body mass index; ER = Estrogen receptor; SHBG = Sex hormone–binding globulin; T3 = Triiodothyronine; UPF = Ultra-processed foods; ω-3 = Omega-3 fatty acids; SES = Socioeconomic status.

Table 1 summarizes the acute hormonal shifts and long-term growth-pubertal consequences associated with major dietary patterns in children and adolescents. Diets rich in calories, saturated fats, and rapidly absorbed carbohydrates consistently produce rapid rises in insulin and leptin, activating hypothalamic kisspeptin pathways and advancing pubertal timing, particularly in girls, whereas ultra-processed food intake enhances metabolic load and adiposity with similar pubertal acceleration. Animal-protein–dominant diets strongly stimulate IGF-1 and adrenal androgen secretion, aligning with earlier pubertal onset and increased BMI, while high plant-protein and fiber-rich patterns show milder endocrine stimulation and may modestly delay puberty when overall nutrition remains adequate. Conversely, insufficient protein or chronic malnutrition suppresses the IGF-1 secretion and thyroid-gonadal signaling, delaying puberty and impairing growth. Important considerations include the finding that very high-protein diets of poor quality in disadvantaged settings may still be associated with stunting, that cruciferous vegetables and soy have little impact on thyroid function when iodine intake is sufficient, and that dairy is generally neutral for pubertal timing when overall energy and protein intake are adequate. Collectively, the evidence indicates that diet quality, macronutrient pattern, and micronutrient sufficiency—not merely energy quantity—shape endocrine programming, growth tempo, and pubertal progression across youth.

Table 2 Long-term endocrine, growth, and pubertal outcomes by dietary pattern, with endocrine axis and mechanisms (children/adolescents, 2000–2025)

Dietary pattern (ref no.)	**Long-term growth & pubertal outcome**	**Predominant endocrine axis**	**Mechanistic pathways**	**Key evidence / clinical notes**
High-calorie / high-glycemic-load (3,4)	Earlier pubertal onset/tempo; ↑ BMI/adiposity (girls>boys)	HPG; insulin–leptin	Hyperinsulinemia/leptin → hypothalamic kisspeptin activation; adipose signaling to GnRH	Consistent association in girls; variable in boys (3,4)
Animal-protein dominant (6,8)	Earlier menarche/PHV; ↑ BMI	GH–IGF-1; adrenal androgens; HPG	↑ IGF-1, ↑ adrenal androgen output; mTOR activation → growth and earlier puberty	Robust across cohorts (6,8)
Plant-protein predominant (soy/legumes/mixed) (8,9,10)	Neutral to slightly delayed puberty; leaner phenotype	GH–IGF-1; HPG (modest)	Phytoestrogens modulate ER & SHBG; milder IGF-1 stimulation vs animal protein	Minimal endocrine disruption in iodine-replete settings (9,10)
Very high total protein with low diet quality (7)	Context-linked ↑ stunting risk; mixed BMI effects	GH–IGF-1 (dysregulated)	Protein excess without micronutrients → growth constraint; SES confounding	Paradox noted in population data (7)
Low protein / chronic undernutrition (11)	Delayed puberty; ↓ height velocity; ↓ BMI	GH–IGF-1; HPG; thyroid	GH resistance, ↓ IGF-1; hypothalamic suppression; ↓ T3	Classic suppression profile (11)
Ultra-processed foods / SSBs (18,22)	Earlier puberty; ↑ adiposity trajectory	Insulin–leptin; HPG	Rapid glycemic excursions → hyperinsulinemia/leptin; adiposity-driven GnRH activation	Independent predictor of early timing (18); fructose risks (22)
High fructose load (22)	↑ Adiposity; possible advancement of puberty	Insulin–leptin; hepatic–metabolic	Hepatic DNL, insulin resistance, low-grade inflammation	Pediatric metabolic risk signal (22)
Dairy-inclusive balanced diet (23)	Normal growth; no consistent shift in puberty timing	GH–IGF-1 (modest)	Small IGF-1 rise without robust HPG acceleration	Generally neutral for puberty when diet is balanced (23)

Mediterranean-style (12)	Healthy growth/weight; physiologic puberty timing	Insulin–leptin; anti-inflammatory milieu	Improved insulin sensitivity; antioxidant/anti-inflammatory effects	Protective, balanced pattern (12)
Omega-3 / fiber-rich whole-foods (21)	Favorable metabolic profile; neutral timing	Insulin–leptin; adrenal stress modulation	Anti-inflammatory actions; adiponectin ↑; gut–brain axis support	Benefits on metabolic risk, not accelerating puberty (21)
Crucifers/soy with adequate iodine (15,9,10)	Normal growth/thyroid; neutral puberty	Thyroid; HPG (soy modest)	Goitrogen risk negligible when iodine-replete; phytoestrogen effects mild	Safety hinges on iodine sufficiency (15); minimal impact (9,10)
High raw crucifers + low iodine (15)	Risk of hypothyroid-mediated growth delay	Thyroid	Goitrogens inhibit iodine uptake/TPO; ↓ T4/T3	Context-dependent risk (15)

Abbreviations: HPG = hypothalamic–pituitary–gonadal; GH = growth hormone; IGF-1 = insulin-like growth factor-1; PHV = peak height velocity; BMI = body mass index; ER = estrogen receptor; SHBG = sex hormone–binding globulin; SSBs = sugar-sweetened beverages; DNL = de novo lipogenesis; TPO = thyroid peroxidase.

Table 2 shows that across long-term outcomes, energy surplus and animal-protein–dominant diets consistently align with earlier puberty and higher adiposity via insulin–leptin–kisspeptin and IGF-1–adrenal androgen pathways, while plant-protein–predominant, Mediterranean, and omega-3/fiber-rich patterns support normal pubertal timing and healthier metabolic trajectories. Undernutrition predictably delays puberty through GH–IGF-1 and thyroid suppression, and UPF/SSB and fructose-heavy patterns track with adiposity and earlier timing. Thyroid effects from crucifers/soy are clinically minimal when iodine intake is adequate, with risk emerging primarily under iodine deficiency. Dairy's impact on puberty is generally neutral when overall energy and protein are balanced.

Table 3 Growth/development domains: associated diets, long-term findings, mechanisms, and evidence strength (children/adolescents, 2000–2025)

Growth / Development Domain	**Diets Associated (ref no.)**	**Long-Term Findings**	**Mechanistic Contribution**	**Evidence Strength***
Height velocity & final height	Animal-protein dominant (6,8); Low protein/undernutrition (11); Dairy within balanced diet (23)	Animal protein associated with greater height gains and earlier PHV; undernutrition → reduced height velocity and shorter adult stature; dairy neutral–supportive when energy/protein adequate	IGF-1 upregulation (animal protein); GH resistance with energy/protein deficit; modest IGF-1 rise with dairy	High (6,8,11,23)
BMI trajectory / adiposity	High-calorie/high-GL & refined carbs (3,4); Ultra-processed foods/SSBs (18); High fructose (22); Mediterranean/whole-	Obesogenic patterns → higher BMI trajectory; UPF/SSBs and fructose predict adiposity; Mediterranean/ω	Hyperinsulinemia/leptin signaling; hepatic DNL & insulin resistance; anti-inflammatory/adiponectin effects with ω-3/fiber	High for obesogenic & UPF/SSBs (3,4,18,22); Moderate protective signal (12,21)

	food ω-3/fiber patterns (12,21)	-3/fiber patterns associated with healthier BMI		
Bone maturation & skeletal health	Adequate dairy/protein within balanced diet (23); Undernutrition/low protein (11); Mediterranean pattern (12)	Adequate dairy/protein supports bone accrual; undernutrition linked to reduced bone mass; Mediterranean diet compatible with normal skeletal development	IGF-1-mediated bone formation; nutrient sufficiency (calcium, protein); anti-inflammatory milieu	Moderate–High (11,12,23)
Puberty onset & tempo	High-calorie/high-GL (3,4); Animal protein (6,8); Plant protein/soy (8,9,10); Mediterranean pattern (12); UPF/SSBs & fructose (18,22)	Energy surplus and animal protein → earlier puberty; plant-protein patterns neutral to slight delay; Mediterranean pattern neutral/physiologic; UPF/SSBs and fructose linked to earlier timing	Insulin–leptin–kisspeptin stimulation; IGF-1/adrenal androgen increases; phytoestrogen modulation of ER/SHBG (mild); improved insulin sensitivity with Mediterranean	High for earlier puberty with energy surplus/animal protein (3,4,6,8,18,22); Moderate for plant-protein/Mediterranean neutrality (9,10,12)
Metabolic/endocrine programming	High-calorie/UPF/SSBs/fructose (3,18,22); Plant-protein/whole-food ω-3/fiber (9,12,21); Iodine-adequate crucifers/soy (15,9,10)	Obesogenic/fructose patterns program insulin resistance and adverse adipokine profiles; plant-protein/ω-3/fiber patterns support healthier metabolic and endocrine profiles; crucifers/soy neutral for thyroid when iodine is sufficient	Insulin/leptin resistance vs. anti-inflammatory signaling; gut–brain axis; thyroid homeostasis with iodine sufficiency	High for adverse programming with UPF/fructose (3,18,22); Moderate for protective patterns; High for thyroid neutrality with adequate iodine (15)

Abbreviations: PHV = peak height velocity; GL = glycemic load; IGF-1 = insulin-like growth factor-1; GH = growth hormone; SSBs = sugar-sweetened beverages; ω-3 = omega-3 fatty acids; ER = estrogen receptor; SHBG = sex hormone–binding globulin; DNL = de novo lipogenesis.

Table 3 reveals that across domains, energy surplus and ultra-processed/fructose-rich patterns consistently drive higher BMI, earlier puberty, and adverse metabolic programming via insulin–leptin–kisspeptin and hepatic insulin-resistance pathways (3,4,18,22). Animal-protein–dominant diets elevate IGF-1/adrenal androgens and are linked to earlier pubertal tempo and greater height velocity around PHV (6,8), while plant-protein and Mediterranean/ω-3/fiber-rich patterns support physiologic pubertal timing and healthier BMI with anti-inflammatory and insulin-sensitizing effects (9,12,21). Undernutrition predictably suppresses the GH–IGF-1 axis and bone accrual, delaying puberty and impairing growth (11), and crucifers/soy remain thyroid-neutral when iodine intake is adequate (15,9,10).

Table 4 Comparative Impact of Major Dietary Patterns on Growth Velocity and Pubertal Timing in Children and Adolescents

Diet Pattern	Growth Height Velocity	Pubertal Timing	Evidence Strength
Animal-protein dominant	↑	Earlier	Strong
High-calorie / UPF / fructose	Neutral or ↑ (BMI effect dominant)	Earlier	Strong
Plant-protein / legume dominant	Normal	Neutral → Slight delay	Moderate
Mediterranean / ω-3 / fiber	Normal	Physiologic	Strong
Low protein / undernutrition	↓	Delayed	Strong
Balanced dairy patterns	Normal	Neutral	Moderate
Crucifers/soy + adequate iodine	Normal	Neutral → Slight delay	Moderate

Conceptual impact of dietary patterns on growth and pubertal timing in children/adolescents (2000–2025). Animal-protein and high-calorie/UPF/fructose patterns trend toward earlier puberty (via IGF-1, insulin–leptin–kisspeptin), plant-protein and Mediterranean/ω-3/fiber are neutral to protective, and undernutrition delays growth and puberty through GH–IGF-1 and thyroid suppression. Effects of dairy and crucifers/soy are generally neutral when overall energy/protein and iodine are adequate (3–12, 15, 18, 21–23).

Table 5 Cochrane Risk-of-Bias Assessment for Included Evidence

Study (Reference)	Design	D1 Randomization	D2 Deviations	D3 Missing Data	D4 Outcome Measures	D5 Reporting	Overall RoB
Brix 2020 (24)	Prospective cohort	—	Low	Low	Low	Low	Low
Cheng 2010 (25)	Prospective cohort	—	Low	Low	Low	Low	Low
Remer 2010 (26)	Prospective cohort	—	Low	Low	Some concerns	Low	Some concerns
Günther 2010 (27)	Prospective cohort	—	Low	Low	Some concerns	Low	Some concerns
Biro 2013 (28)	Prospective cohort	—	Low	Low	Low	Low	Low
Wang 2002 (29)	Cross-sectional	—	Some concerns	Low	Some concerns	Some concerns	Some concerns
Soliman et al. (PEM) (30)	Clinical cohort	—	Low	Low	Some concerns	Some concerns	Some concerns
Imdad 2017 (31)	RCT meta-analysis	Low	Low	Low	Low	Low	Low
Brown 2009 (32)	Meta-analysis	Low	Low	Low	Low	Low	Low
Messina 2022 (33)	Narrative + cohort review	—	Some concerns	Low	Low	Some concerns	Some concerns

Low risk = robust design/adjustment, **Some concerns** = observational or confounding risk,
High risk = substantial bias concerns

The Cochrane RoB-2 evaluation indicates that the majority of studies informing this review demonstrate low to moderate risk of bias, particularly among high-quality prospective cohort studies (Brix 2020; Cheng 2010; Biro 2013) and the two systematic reviews/meta-analyses (Imdad 2017; Brown 2009), which achieved low risk across all methodological domains. Most observational nutrition-puberty cohorts showed low bias in exposure and outcome measurement but carried some concerns related to residual confounding and selective reporting, reflecting the inherent limitations of non-randomized dietary research. Cross-sectional evidence (Wang 2002) demonstrated methodological vulnerabilities in selection and confounding domains, while mechanistic and narrative syntheses (Messina; Soliman PEM) were rated as some concerns due to design constraints. Overall, this evidence base is strengthened by well-characterized prospective cohorts and randomized data for growth-related micronutrient interventions, supporting the robustness of conclusions linking diet composition with growth and pubertal timing, while acknowledging the need for continued high-quality longitudinal and interventional studies to reduce confounding and enhance causal inference.

Table 6 GRADE Evidence Table

Exposure / Diet Pattern	Outcome	Certainty	Supporting Evidence
High BMI / obesogenic diet	Earlier puberty	High	Brix (24), Biro (28), Wang (29)
Animal-protein dominant diet	Earlier puberty	Moderate-to-High	Remer (26), Günther (27)
Plant-protein–dominant diet	Slight puberty delay	Moderate	Cheng (25), Günther (27)
Mediterranean / balanced diet	Normal tempo, optimal growth	High	Multiple prospective cohorts
Soy intake	No meaningful effect	Moderate	Messina (33)
Severe malnutrition	Delayed puberty, ↓ growth	High	Soliman (30)
Zinc sufficiency	Improved growth	High	Imdad (31), Brown (32)

GRADE assessment demonstrates high-certainty evidence for earlier puberty with excess adiposity and animal-protein–rich diets and delayed maturation with protein-energy malnutrition. Evidence for soy intake is moderate and consistently neutral.

4. Discussion

Nutrition emerged as a central regulator of endocrine maturation, growth, and puberty in this review. Evidence across diverse cohorts demonstrates that diet modulates the GH–IGF-1 axis, adipose-derived signals, thyroid function, and the hypothalamic–pituitary–gonadal (HPG) axis during critical developmental windows (34).

Energy excess and adiposity accelerate pubertal onset, particularly in girls. Elevated leptin and insulin stimulate hypothalamic kisspeptin neurons and GnRH pulsatility, promoting gonadotropin release and sex-steroid production (35). Longitudinal data confirm earlier menarche among girls with higher adiposity trajectories independent of confounders (36). Hyperinsulinemia also reduces SHBG, increasing biologically active estrogen and testosterone and magnifying pubertal acceleration (37).

Ultra-processed and sugar-rich dietary patterns further potentiate pubertal advancement by increasing visceral adiposity, promoting inflammation, altering gut microbial composition, and modifying hypothalamic neuroendocrine circuits (38). Fructose-driven hepatic lipogenesis and leptin resistance contribute to early maturation and metabolic imprinting (39). Early childhood diet may generate lasting epigenetic changes that influence pubertal timing and hormonal set-points (40).

Conversely, plant-forward dietary patterns support normative puberty. Soy and legume intake exerts weak estrogen-receptor modulation and increases SHBG, yielding neutral to slightly delayed pubertal timing without growth suppression (41). Higher childhood fiber intake enhances insulin sensitivity and moderates IGF-1 signaling, supporting physiologic tempo (42).

Protein quality emerged as a key mechanistic factor. Animal-protein intake strongly stimulates IGF-1 and adrenal androgen production, advancing puberty, whereas inadequate protein suppresses IGF-1, delaying growth and maturation (43). However, high protein from low-quality sources under inflammatory or micronutrient-deficient conditions may paradoxically be associated with stunting (44).

Mediterranean-style diets — rich in ω-3 fatty acids, polyphenols, fruits, and whole grains — are associated with optimal growth and normal pubertal timing. Anti-inflammatory effects and improved insulin sensitivity preserve the GH-IGF-1 axis and metabolic homeostasis (45). Children adhering to Mediterranean patterns show healthier endocrine profiles and reduced risk of obesity-associated precocity (46).

Undernutrition predictably delays puberty and linear growth through GH resistance, reduced IGF-1, decreased gonadotropins, impaired thyroid hormone production, and elevated cortisol (47). Zinc deficiency alters IGF-1 signaling and pubertal progression, while iodine deficiency impairs thyroid hormone synthesis and growth velocity (48).

Micronutrient-dense diets support endocrine resilience. Vitamin D status positively correlates with pubertal bone mineralization and IGF-1 levels (49). In iodine-sufficient individuals, cruciferous vegetables and soy do not clinically impair thyroid function, countering concerns about dietary goitrogens (50).

Gut-endocrine crosstalk represents an emerging mechanism. Fermentation of dietary fiber yields short-chain fatty acids that enhance insulin sensitivity, modulate hypothalamic appetite and reproductive pathways, and stabilize metabolic hormone rhythms (51). In contrast, obesity-associated dysbiosis may amplify inflammation and disrupt pubertal regulation (52). More pediatric interventional trials are needed to clarify causality.

Ethnic and genetic factors modulate dietary effects on puberty. Earlier maturation trends are more pronounced in some ethnic groups, even after adjusting for BMI, indicating interaction between biology, diet, and environment (53). Secular trends toward earlier puberty align with rising global childhood obesity, reinforcing the dominant contribution of nutrition and metabolic status (54).

Overall, these findings confirm that high-quality, balanced diets — adequate in protein, micronutrients, and healthy fats — support normal endocrine development, while hypercaloric, insulinogenic patterns accelerate puberty and nutrient-poor diets delay maturation. Nutritional quality, metabolic context, and timing appear more consequential than single nutrients alone (55).

5. Conclusion

Diet composition meaningfully influences pediatric endocrine programming, growth trajectories, and timing of puberty. High-energy and animal-protein-rich diets accelerate maturation via leptin–insulin–IGF-1 and kisspeptin pathways, while plant-based and Mediterranean dietary patterns maintain physiologic tempo and favorable metabolic profiles. Undernutrition and micronutrient deficiencies delay pubertal onset via GH–IGF-1 suppression and thyroid insufficiency. Ensuring balanced, nutrient-dense diets during childhood is essential to optimize developmental endocrinology and long-term metabolic health.

Clinical recommendations

- **Promote balanced, whole-food dietary patterns**
- Encourage varied diets with adequate high-quality protein, fruits, vegetables, whole grains, and healthy fats to support normal GH–IGF-1 activity, linear growth, and physiologic pubertal timing.

- **Limit ultra-processed, high-sugar diets in children**
- Reduce sugary drinks and highly processed foods, as they increase adiposity and are consistently associated with earlier puberty and metabolic risk.

- **Ensure sufficient micronutrients and protein—especially in restrictive or low-resource diets**
- Monitor growth and puberty in at-risk children and ensure enough zinc, vitamin D, iodine, calcium, and quality protein to prevent delayed growth and pubertal suppression.

- **Compliance with ethical standards**

This review is based exclusively on previously published data and does not involve human participants, new clinical data collection, or identifiable patient information. Therefore, ethical approval and informed consent were not required.

Authors' Contributions

ATS conceived the review concept, supervised all stages of development, and finalized the manuscript. FA, SA, NHu, NHm, AE, NA, and SS contributed to literature search, data extraction, and drafting of the thematic sections. NS (Nada Soliman) assisted with dietary and public-health components and contributed to manuscript editing. All authors critically reviewed the final version and approved it for submission.

Disclosure of Conflict of Interest

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

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