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Maternal Nutrient Intake and Birth Length: A Narrative Literature Review

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***Abstract*—*Birth length is a skeletal marker of linear fetal growth that carries information beyond birth weight and small-for-gestational-age classification, yet it is rarely the primary outcome in studies of maternal nutrition. This narrative review examines how maternal nutrient intake during pregnancy relates to length at birth, with attention to biological mechanisms, the consistency of findings across study designs, and the limits of the available evidence. Placental nutrient sensing, the insulin-like growth factor axis, and the supply of protein, calcium, phosphorus, vitamin D, and zinc to the growing skeleton provide plausible pathways, but most mechanistic evidence is indirect with respect to length specifically. Synthesis of systematic reviews, randomized trials, and cohort studies shows small and inconsistent associations. Pooled supplementation trials report mean differences from roughly 0.05 cm for multiple micronutrients to 0.3 cm for vitamin D, whereas balanced energy-protein supplementation and zinc supplementation show no clear benefit for length, despite effects on birth weight in some analyses. Observational data are discordant: dairy intake and, in one cohort, protein intake related positively to length, whereas in a large United States cohort with high protein intake, higher protein related to shorter length. Divergence is explained in part by baseline nutritional status, exposure timing and measurement, non-linear dose-response patterns, measurement error in newborn length, and residual confounding. Clinically, effects are modest at the individual level and are most plausible where deficiency is prevalent. Priorities include length-specific outcome reporting, standardized measurement, z-scores based on gestational age and sex standards, and trials that treat birth length as a prespecified endpoint.*

***Keywords*—*Birth length: Fetal growth: Linear growth: Maternal nutrition: Micronutrients: Narrative review: Pregnancy: Protein*.

I. Introduction

Maternal nutrition during pregnancy is widely regarded as a modifiable determinant of fetal growth and of later health, yet the evidence is unevenly distributed across newborn outcomes. Reviews of maternal diet have concentrated on perinatal endpoints such as preterm birth and birth weight [1], and low birth weight and small-for-gestational-age (SGA) status have dominated nutritional trials. Birth length, by contrast, is reported far less often. In a 2022 systematic review of milk and dairy intake during pregnancy that identified 42 eligible studies, only 11 reported birth length, and a dose-response meta-analysis for length could be performed on just three of them [2].

This imbalance matters because length and weight do not measure the same biology. Weight reflects the accretion of lean tissue, fat, and organ mass, whereas length is the skeletal dimension of fetal growth and the starting point of the linear growth trajectory that is later monitored as length-for-age or height-for-age. International standards now provide gestational age- and sex-specific centiles for newborn weight, length, and head circumference [3], which makes it possible to express length independently of weight. At the population level, fetal growth restriction and preterm birth form the leading risk factor cluster for childhood stunting in developing countries [4], and a recent meta-analysis found that the benefit of prenatal multiple micronutrient supplementation on length-for-age was visible at birth and persisted until 6 months of age, but not thereafter [5]. Whether and how maternal nutrient intake shapes length at birth specifically, rather than overall size, is therefore a question with both biological and public health relevance.

The purpose of this narrative review is to examine the relationship between maternal nutrient intake during pregnancy and birth length. We (i) clarify how birth length differs from birth weight and SGA as an outcome, (ii) outline biological mechanisms that could link maternal nutrient supply to skeletal growth, (iii) synthesize findings from systematic reviews, trials, and cohort studies by type of exposure, (iv) discuss why results differ, and (v) identify limitations of the evidence and priorities for research. We use maternal nutrient intake in a broad sense that includes dietary intake, food-based and micronutrient supplementation, and, where intake data are absent, nutritional biomarkers, and we state the type of exposure each time.

II. Review Approach

This article is a narrative literature review. It was designed to interpret and critically integrate evidence rather than to provide an exhaustive, protocol-driven systematic review, and the authors did not undertake a formal risk-of-bias assessment or quantitative pooling. Sources were peer-reviewed systematic reviews, meta-analyses, randomized trials, and

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cohort studies that addressed maternal dietary intake, food-based or micronutrient supplementation, or nutritional biomarkers in relation to newborn length or closely related linear growth outcomes. Publications from 2021 to 2026 were prioritized; earlier studies were retained when they supplied the only length-specific data, defined measurement standards, or described mechanisms. Literature was identified in October 2026 through targeted searches using a general web search engine, which returned records hosted by PubMed Central, publisher websites, the Cochrane Library, and institutional repositories, rather than through direct queries of individual bibliographic databases. Search terms combined maternal diet or supplementation (for example, maternal dietary intake, protein, balanced energy-protein, multiple micronutrient, zinc, vitamin D, calcium, dairy) with birth length or newborn length, and separate searches addressed placental nutrient sensing and the precision of newborn length measurement. The reference lists of key reviews, notably the 2022 dose-response review of dairy intake [2], were screened to locate additional primary studies. Only English-language sources were considered, titles and abstracts were screened by a single reviewer, and the bibliographic details of each included source were checked against the retrieved record. Effect estimates are reported as stated by the original authors and are labeled by design, because estimates from supplementation trials, observational intake studies, and biomarker cohorts are not interchangeable. Findings for birth weight and SGA are reported only to contrast them with length.

III. Birth Length as a Distinct Outcome

A. Definition and Measurement

Birth length is conventionally the recumbent crown-heel length measured on an infantometer. Compared with weight, it is harder to measure precisely. Crown-heel length depends on technique, training, experience, and infant cooperation; in newborns, measurements taken with only one leg extended are systematically longer than those taken with both legs extended, and the two-leg technique reduces inter-observer variability [7]. Length recorded in routine clinical records, as in a recent Chinese cohort in which birth length was taken from birth records [8], is therefore likely to contain more heterogeneous error than length measured by trained research staff. Because gestational age and sex strongly influence newborn size, interpretation requires reference data. The INTERGROWTH-21st newborn standards provide sex-specific 3rd, 10th, 50th, 90th, and 97th centiles for length by gestational age, derived from eight geographically defined urban populations [3]. Studies that report raw centimeters, z-scores, or categories of short length cannot be pooled directly without careful harmonization.

B. Birth Length, Birth Weight, and SGA

Birth weight, low birth weight (LBW), and SGA are all weight-based. Birth weight integrates skeletal size with soft tissue and fat, so an intervention can change weight without changing length, and the reverse. SGA is a categorical classification of weight for gestational age, and LBW additionally mixes preterm birth with growth restriction. Neither describes the skeleton directly.

Several findings in the nutrition literature illustrate the dissociation. In trials from low- and middle-income countries, balanced energy-protein supplementation increased birth weight by 107.28 g and reduced SGA (risk ratio 0.71), yet the pooled difference in birth length was 0.28 cm with a 95% confidence interval that spanned zero (-0.36 to 0.92) [9]. Dietary and lifestyle interventions to prevent maternal obesity reduced birth weight by 195.57 g across two small trials, with no detectable difference in birth length in the one trial that reported it [9]. In the dairy literature, higher intake was associated with lower SGA risk but also with higher risk of large-for-gestational-age (LGA) births at the upper end of intake, whereas the dose-response effect on length was small [2]. These observations do not show that length is insensitive to nutrition. They show that weight-based outcomes cannot stand in for length, and that conclusions about fetal growth drawn from weight should not be extended to skeletal growth without length-specific data.

C. Why Length Matters

Length at birth is the first point on the linear growth curve. A pooled analysis of 19 trials reported that infants of mothers given multiple micronutrients were longer at birth than those of mothers given iron and folic acid (mean difference 0.05 cm) and had higher length-for-age z-scores at birth, 3 months, and 6 months, but not later [5]. In a United States cohort, higher maternal protein intake was associated with shorter birth length and also with slower length growth from birth to under 6 months and from 6 months to mid-childhood [6]. Together, these reports suggest that length at birth is a marker of, and possibly a contributor to, early linear growth trajectories that prenatal exposures can influence for some months after birth. Although fetal growth restriction and preterm birth are major contributors to childhood stunting [4], those categories are usually operationalized through weight and gestational age, and how much of the prenatal contribution is mediated by length itself has not been clearly quantified in the sources reviewed here.

IV. Biological Mechanisms

The mechanisms below are plausible but, with few exceptions, were established for fetal growth in general rather than for length. We note where human length-specific evidence exists. A conceptual summary of the proposed pathways is shown in Fig. 1.

A. Placental Supply and Nutrient Sensing

Fetal growth is constrained by the supply of substrates that cross the placenta. Trophoblast nutrient sensing has been proposed as a link between maternal nutrient availability and fetal growth. Mechanistic target of rapamycin complex 1 (mTORC1) integrates signals from amino acids, glucose, oxygen, and insulin or insulin-like growth factor signaling, acts as a positive regulator of placental amino acid transporters, and is altered in pregnancies with abnormal fetal growth and in animal models in which maternal nutrient

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availability was changed experimentally [10]. More broadly, mTOR functions as a central hub that couples nutrient availability to cell growth [11]. These pathways would make placental transport capacity a modulator of how much dietary protein, amino acids, and other nutrients reach fetal tissues, including growing bone. They do not, by themselves, explain why length rather than adiposity would respond.

B. Endocrine Mediators

The insulin-like growth factor (IGF) axis is a candidate mediator between maternal diet and fetal size, and the relationship of fetal growth with maternal and fetal IGF axes has been examined in a systematic review and meta-analysis [12]. Authors of the dairy review proposed that milk-related IGF-1 signaling and mTORC1 activation could explain positive associations between milk intake and birth size, while stressing that clearer evidence is needed [2]. In the Project Viva cohort, the unexpected inverse relationship between protein intake and birth length led the investigators to suggest that high protein intake might reduce early length growth through hormonal effects [6]. Whether these hormonal pathways act on length, weight, or both, and in which direction at which intake levels, remains unresolved.

C. Skeletal Mineral Supply: Calcium, Phosphorus, and Vitamin D

Bone mineral accrual is the most direct nutritional requirement of the fetal skeleton. Maternal adaptations in mineral metabolism during pregnancy, including changes in intestinal absorption, supply the calcium and phosphorus required, with the largest fetal demand late in gestation, and experimental evidence indicates that these adaptations can occur largely without vitamin D [13]. Two implications follow. First, in women with adequate baseline intake, additional dietary calcium may add little to fetal skeletal supply. Consistent with this, crown-heel length in an Australian cohort correlated weakly with maternal intake of most nutrients but not with calcium [14]. Second, the effect of dietary calcium or dairy intake may depend on how closely maternal adaptation meets demand, which could differ among adolescents and women with chronically low intake. Fetal femur length was reported to be influenced by maternal dairy intake in African American adolescent mothers [15], and in a randomized trial of 72 adolescent mothers, a dairy-based calcium intervention was followed by heavier infants with higher total body calcium, as summarized in the dairy review [2], [16]. Vitamin D supplementation has been associated with greater birth length in pooled trials [17], but because intestinal calcium handling during pregnancy can adapt independently of vitamin D [13], the pathway for such an effect remains uncertain.

D. Protein and Amino Acids

Amino acids supply the building blocks for cell proliferation and matrix synthesis, so protein adequacy is a plausible requirement for linear growth. Yet the human data point to a non-monotonic relationship. In 1,961 mother-child pairs in Project Viva, in which mean second-trimester protein intake was 1.4 g/kg/day, each standard deviation (0.36 g/kg/day) of higher second-trimester intake was associated with a 0.10 lower birth length z-score (95% CI -0.18 to -0.03) [6]. The same authors cited an early report of 216 mother-infant pairs from 1943 that found a strong positive correlation between maternal protein intake and birth length (r of 0.8), while other studies found no direct association [6]. The pattern is compatible with a threshold or U-shaped relationship in which protein is limiting in undernourished women but not in replete women, although, to our knowledge, formal tests of such curves in pregnancy are rare. The dairy dose-response analysis likewise suggested that the effect on length rose and then declined, with a predicted maximum at about one serving per day [2].

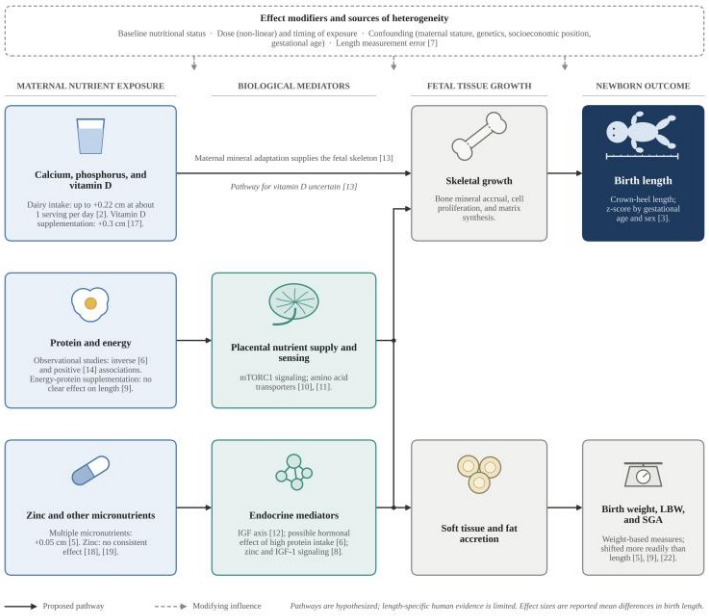


Fig. 1. Proposed pathways linking maternal nutrient exposure to birth length and to weight-based outcomes. Pathways are hypothesized from physiological and experimental evidence; length-specific human evidence is limited. Numbers in brackets refer to the reference list. LBW, low birth weight; SGA, small for gestational age

E. Zinc and Other Micronutrients

Zinc has catalytic, structural, and regulatory roles in DNA synthesis, cell division, and hormone regulation, and it has been linked experimentally to IGF-1 and growth hormone pathways, which motivates its study in fetal growth [8]. Human evidence for benefit, however, is weak. A systematic review of 20 supplementation trials found no effect on length at birth and noted that most studies tested zinc against a background of other micronutrient supplements, with only five placebo-controlled trials of zinc alone [18]. The most recent Cochrane review, covering 25 trials and more than 18,000 women, concluded that there is not enough evidence that zinc supplementation improves maternal or neonatal outcomes, and it recommended research on overall nutritional status rather than zinc in isolation [19]. In a 2026 cohort, maternal blood zinc status in the third trimester was not associated with birth length, and the authors speculated that bone growth may be less sensitive than weight to short-term variation in trace

elements [8]. This speculation is reasonable but untested, and it applies to a biomarker measured once late in pregnancy rather than to dietary intake.

Fig. 1 integrates these mediators and relates them to the length- and weight-based outcomes discussed in this review.

V. Synthesis of Evidence

Table I summarizes the exposures and the length-specific findings. Fig. 2 plots the pooled mean differences, in centimeters, from the four reviews that reported them in that form.

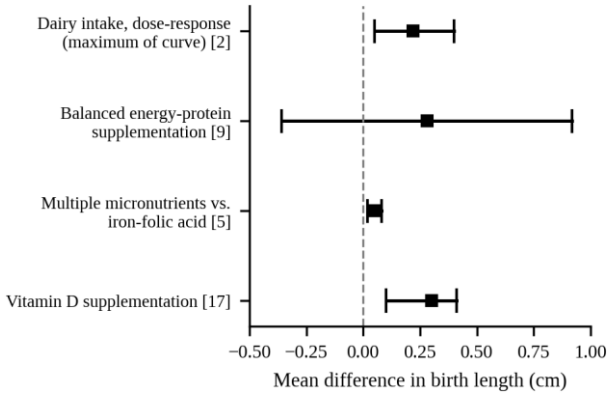


Fig. 2. Pooled mean differences in birth length (cm) with 95% confidence intervals reported by selected reviews [2], [5], [9], [17]. Estimates are not directly comparable because exposures, comparators, populations, and metrics differ. The dairy estimate is the maximum of a non-linear dose-response curve, not a pooled mean difference.

A. Food-Based Exposures: Dairy

The most developed food-based literature concerns milk and dairy products. The 2022 dose-response meta-analysis found a non-linear association in which birth length rose with intake up to a predicted maximum of 0.22 cm (95% CI 0.05 to 0.40) at about 1.07 servings per day, with marginal statistical significance (P of 0.0474), based on only three studies [2]. The same review reported that higher dairy intake was associated with lower SGA risk and higher LGA risk, and that the relationship with LBW was not significant [2]. An earlier systematic review of studies published up to May 2018 described a trend toward positive associations between maternal milk intake and infant birth weight and length, but it judged that the number and types of studies prevented definite conclusions [20]. An earlier meta-analysis had reported lower LBW risk with higher dairy intake [21], a result that was not reproduced in the dose-response analysis [2]. Individual cohorts summarized in the 2022 review included a Danish cohort in which milk intake of at least 150 ml per day was associated with a 0.34 higher birth length z-score [2].

Interpretation is limited because dairy is a food group that delivers protein, calcium, and other nutrients together, so attribution to a single nutrient is not possible, and because residual confounding by overall diet quality and socioeconomic position is likely. Meta-analyses of maternal dietary patterns in cohort studies have focused on perinatal outcomes [1], which leaves whole-diet exposure in relation to length largely unexamined in the sources we reviewed.

B. Protein and Energy

Observational studies of protein intake are discordant. In Project Viva, higher second-trimester protein intake was associated with shorter birth length [6].

TABLE I. Maternal Nutritional Exposures and Reported Findings for Birth Length

Exposure	Evidence base	Finding for birth length	Main caveats
Dairy and milk intake (observational)	Systematic review of 42 studies; length reported in 11; dose-response meta-analysis of 3 studies [2]	Non-linear curve; maximum mean change 0.22 cm (95% CI 0.05 to 0.40) at about 1.07 servings per day; P = 0.0474	Three studies only; food group, not a nutrient; dietary self-report; confounding by diet quality and socioeconomic position
Dietary protein intake (observational)	Project Viva, 1,961 pairs [6]; Vitamin D in Pregnancy cohort, 346 pairs [14]	Project Viva: -0.10 z-score per 1-SD (0.36 g/kg/day) higher second-trimester intake. Australian cohort: weak positive correlation with crown-heel length (r 0.15 to 0.17 for most nutrients); protein association persisted after adjustment	Opposite directions; different intake ranges, assessment, and adjustment; observational
Balanced energy-protein supplementation (trials)	Eight trials, two reporting length [9]	Mean difference 0.28 cm (95% CI -0.36 to 0.92), 67 participants; birth weight +107.28 g	Very small sample for length; review authors uncertain about effects
Food distribution programs (trials and programs)	Three studies reporting length, 5,272 participants [9]	Mean difference 0.20 cm (95% CI 0.20 to 0.20)	Bundled services; zero-width interval; likely dominated by one large study (inference)
Multiple micronutrient supplements vs. iron-folic acid (trials)	19 trials [5]; 12 trials [22]	+0.05 cm (95% CI 0.02 to 0.08); length-for-age z-score +0.09 at birth [5]. No significant effect on birth length [22]	Statistically small; larger effects where LBW prevalence high; differences in trial sets and precision
Zinc supplementation or status	20 trials [18]; 25 trials [19]; cohort of 291 pairs [8]	No effect on length at birth [18]; no consistent benefit [19]; no association of third-trimester blood zinc with birth length [8]	Zinc often tested within multi-nutrient background; biomarker is not intake; single-center cohort
Vitamin D supplementation (trials)	13 trials in total, 2,299 newborns [17]	+0.3 cm (95% CI 0.10 to 0.41); birth weight +107.6 g	Search ended March 2014; heterogeneous doses, formulations, and timing
Dietary counseling to prevent maternal obesity (trials)	One trial reporting length, 90 participants [9]	Mean difference -0.36 cm (95% CI -1.12 to 0.40); birth weight -195.57 g across two trials	Single small trial; reverse direction of exposure

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In the Vitamin D in Pregnancy cohort in Australia, 346 mother-infant pairs with dietary intake measured at 28 to 32 weeks of gestation showed weak positive correlations between crown-heel length and maternal intake of all measured nutrients except calcium, fat, and carbohydrate (r of 0.15 to 0.17), and the associations with protein, phosphorus, and potassium persisted after adjustment for maternal and offspring characteristics [14]. The two studies differ in setting, dietary assessment, intake range, adjustment sets, and sample size, so the contrast cannot be resolved from the published reports, but it shows that the direction of the association is not stable across populations.

Trials of food-based macronutrient supplementation add little length-specific information. In a Campbell systematic review of low- and middle-income countries, eight trials of balanced energy-protein supplementation showed higher birth weight and lower SGA, but only two trials, with 67 participants in total, reported birth length, and the difference was not significant (0.28 cm; 95% CI -0.36 to 0.92) [9]. Food distribution programs, which in several trials were bundled with counseling or medical services, were associated with a 0.20 cm higher birth length across three studies and 5,272 participants, but the reported confidence interval had zero width [9]. An interval of zero width is implausible as a measure of uncertainty and suggests, as an inference, that the estimate was dominated by a very large study. The review authors concluded that, given the small number of studies and low quality of evidence, they were uncertain about the effects of these interventions [9].

C. Micronutrient Supplementation

Pooled trial evidence for multiple micronutrients is the most consistent, but the effect is very small. Gomes and colleagues combined 19 trials that compared multiple micronutrient supplements with iron and folic acid and found a mean difference in birth length of 0.05 cm (95% CI 0.02 to 0.08) and a higher length-for-age z-score at birth of 0.09 (95% CI 0.06 to 0.12) [5]. Effects were greater when supplementation continued after delivery and in settings with a higher prevalence of LBW [5]. An earlier meta-analysis of 12 trials in low-income countries found no significant effect on birth length, although it reported a 22.4 g higher birth weight and about 10 percent lower odds of LBW [22]. The two syntheses do not necessarily conflict. The later analysis had more trials and therefore more precision to detect a difference of half a millimeter, whose clinical meaning for an individual infant is negligible and which is likely small relative to the measurement variability of newborn length [7].

For zinc, supplementation trials show no consistent effect on length [18], [19]. In a Northeast China cohort of 291 mother-infant pairs, third-trimester maternal blood zinc status was not associated with the adjusted birth length-for-age z-score (coefficient 0.044; 95% CI -0.126 to 0.285), although infants of zinc-sufficient mothers had higher length-for-age z-scores at 6 months (coefficient 0.165) [8]. Some confidence intervals in that study are markedly asymmetric around their point estimates, which is atypical for linear regression coefficients. The study was also single-center, used a locally defined deficiency threshold, measured zinc at one time point, and lacked dietary intake data. The 6-month finding is therefore best treated as hypothesis-generating [8].

For vitamin D, a 2015 meta-analysis of 13 randomized trials (2,299 newborns) reported that supplementation increased birth length by 0.3 cm (95% CI 0.10 to 0.41) and birth weight by 107.6 g, while doses, formulations, timing of first administration, and outcomes were heterogeneous across trials [17]. Its search ended in March 2014, so more recent trials are not represented, and a current synthesis that addresses birth length explicitly is needed.

Across exposures, the point estimates for length are small (0.05 to 0.3 cm) (Fig. 2), confidence intervals either include zero or are narrow around very small values, and length is rarely the primary outcome.

VI. Discussion

A. Why Findings Differ

Five explanations, which are not mutually exclusive, can account for much of the discordance (see effect modifiers in Fig. 1).

First, baseline nutritional status and non-linearity. Effects on length were greater in settings with a higher prevalence of LBW [5], the effect of multiple micronutrients on birth weight was larger in mothers with higher body mass index [22], the inverse association between protein and length arose in a population with high protein intake [6], and the dairy curve was non-linear [2]. Pooling across such contexts can average opposing slopes.

Second, exposure definition and timing. Studies evaluate dietary intake, supplementation, or biomarkers (Table I), and they assess different windows: the first and second trimesters [6], 28 to 32 weeks of gestation [14], or the third trimester [8]. The critical window for length-relevant nutrients has not been established.

Third, collinearity and measurement of intake. Foods and nutrients co-vary, as with dairy, protein, phosphorus, and potassium [2], [14], and multi-nutrient supplements cannot separate their components [18]. Self-reported intake adds measurement error.

Fourth, outcome measurement and reporting. Technique-dependent differences in crown-heel length [7], and inconsistent use of gestational age- and sex-specific standards [3], mean that raw centimeters, z-scores, and categories are not interchangeable. Random error in the outcome reduces statistical power rather than biasing mean differences, but systematic differences between observers or sites add heterogeneity and may introduce bias.

Fifth, confounding by shared determinants. Maternal stature, parental genetic potential, socioeconomic position, and gestational age are plausible confounders whose handling varies across studies. For example, the Northeast China cohort adjusted for maternal age, body mass index, gestational weight gain, education, newborn sex, gestational age, parity, and delivery mode, but not for maternal height [8]. Only randomized comparisons avoid this problem, and few randomized trials have measured length.

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B. Interpreting the Weight-Length Divergence

A recurring theme is that supplementation shifts birth weight more than length. Balanced energy-protein supplementation increased weight by more than 100 g without a clear change in length [9], and multiple micronutrients changed weight by about 22 g and length by a fraction of a centimeter [5], [22]. One interpretation is that maternal nutrition modulates soft tissue and fat accretion more readily than skeletal growth (Fig. 1), which may be more tightly constrained by genetic potential and by placental and endocrine regulation, or may require earlier or longer exposure. The authors of the Northeast China cohort offered a similar speculation for zinc [8]. An alternative interpretation is that the length data are simply too sparse and noisy, since only two energy-protein trials reported length [9]. Neither interpretation can be tested with the present evidence, which is why length needs to be a prespecified outcome in future trials.

C. Clinical and Public Health Implications

The pooled differences of 0.05 to 0.3 cm are small (Fig. 2). In a cohort in which the standard deviation of birth length was 1.56 cm [8], 0.3 cm would correspond to about 0.2 standard deviations and 0.05 cm to about 0.03 standard deviations. Such shifts are unlikely to matter for an individual infant but could be relevant at the population level where deficiency is common, which is where trials show larger responses [5].

Three practical points follow. First, no evidence in this review supports single-nutrient supplementation for the purpose of lengthening newborns, and the zinc evidence is explicit on this point [19]; improving overall nutritional adequacy is better supported. Second, higher protein intake in already replete pregnant women has not been shown to increase length and, in one large cohort, was associated with shorter length [6]. This observational finding should not be read as a reason to restrict protein, but it argues against assuming that more is better. Third, programs that monitor newborn outcomes should measure length with a standardized two-leg technique and express it against gestational age- and sex-specific standards [3], [7], so that length can be tracked alongside weight.

D. Limitations of the Evidence

Few trials specified birth length as a primary outcome, and the reviews that reported it often pooled very few studies: three for dairy [2] and two for energy-protein supplementation [9]. Certainty of evidence was low or not graded for length. Observational studies rely on dietary self-report and differ in adjustment. Estimates come from different designs, comparators, and settings and cannot be compared on a common scale. Trial evidence derives mostly from low- and middle-income countries, whereas many cohorts come from higher-income settings, so context and design are partly confounded. Evidence on sex-specific effects, on diet quality indices in relation to length, and on dose and threshold effects is sparse.

E. Research Priorities

Priorities include: (1) randomized trials with birth length as a prespecified primary or key secondary outcome, measured with a standardized two-leg technique by trained staff; (2) reporting of length as gestational age- and sex-specific z-scores [3] alongside raw values, with weight-for-length or related indicators to distinguish skeletal from soft tissue effects; (3) individual participant data meta-analyses that can examine effect modification by baseline nutritional status, maternal stature, and sex; (4) designs that compare intake and biomarker exposures within the same cohorts; (5) explicit modeling of non-linear dose-response relationships and thresholds, as suggested by the dairy and protein findings [2], [6]; (6) trimester-specific and periconceptional exposure windows; (7) studies of nutrient combinations and diet quality rather than single nutrients [19]; (8) measurement of mediating biomarkers, including components of the IGF axis [12]; and (9) postnatal follow-up to determine whether small differences at birth translate into growth trajectories [5].

VII. Limitations of This Review

This review is narrative and was not conducted according to a registered protocol. The literature search was targeted rather than exhaustive, so relevant studies may have been missed, particularly very recent publications and those not indexed in the sources consulted. We relied on published summary estimates and did not re-analyze primary data or formally assess risk of bias. Where reviews pooled few studies, our conclusions inherit their limitations. Mechanistic statements are drawn largely from physiological and experimental literature and should be read as hypotheses with respect to length.

VIII. Conclusion

The evidence that maternal nutrient intake influences birth length is suggestive but weak. Plausible pathways exist through placental nutrient sensing, endocrine signaling, and skeletal mineral supply, yet length-specific human data are sparse, effect sizes are small, and results differ by exposure type, population, and outcome measurement. Supplementation appears to change birth weight more readily than length, which underlines that weight and SGA are not substitutes for length when skeletal growth is the question.

For practice, the most defensible message is to support overall dietary adequacy where deficiency is common rather than to rely on single nutrients, and to measure newborn length carefully. For research, the next step is not more weight-based studies but trials and cohorts that treat birth length as a primary, standardized outcome, with exposure windows, doses, and baseline status specified in advance.

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