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Growth Trajectories Following Early Nutritional Intervention in Small-for-Gestational-Age Infants

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Abstract

Infants born small for gestational age (SGA) present clinicians with two competing priorities: promoting sufficient catch-up growth to support neurodevelopment and adult stature, while avoiding the rapid, fat-predominant weight gain that developmental-origins research links to later insulin resistance and cardiometabolic risk. This article synthesizes the literature on postnatal nutritional intervention and growth trajectories in SGA infants, tracing the question from the definition and placental etiology of SGA birth, through the foundational developmental-origins and thrifty-phenotype evidence that first raised concern about the composition and tempo of catch-up growth, to the direct randomized-trial evidence on nutrient-enriched feeding in term SGA and postdischarge preterm infants. We give particular weight to cohort data linking catch-up growth patterns to measurable differences in body composition and metabolic markers in childhood, and to the neurodevelopmental and endocrine literature, including growth hormone therapy, bearing on children whose catch-up growth fails to materialize. Read together, the evidence supports nutrient-enriched feeding strategies that favor linear and head-circumference growth without disproportionate weight gain as a reasonable approach to threading this needle, while leaving an up-to-date, etiology-stratified, body-composition-informed trial base as a genuine and consequential gap in an evidence base still substantially anchored in trials from the 1990s and 2000s.

Keywords: small for gestational age; catch-up growth; postnatal nutrition; neonatal growth trajectory; developmental origins of disease

1. Introduction

A baby born small for gestational age (SGA) — conventionally defined as a birth weight below the 10th percentile for gestational age and sex on a population-appropriate growth standard — sits at the intersection of two clinical questions that are often treated separately but are, in practice, inseparable. The first is short-term: how do you feed this infant so that it grows adequately without exposing it to the immediate metabolic risks, chiefly hypoglycemia, that accompany a body with unusually small energy reserves. The second is long-term: what growth trajectory, over the following months and years, best serves that child's neurodevelopment, final height, and cardiometabolic health across the rest of its life. Nutritional intervention in the SGA infant sits squarely at the hinge between these two questions, because the feeding decisions made in the first weeks and months of life are also, whether or not clinicians frame them this way, decisions about the shape of the growth trajectory that follows.

The scale of the problem is not small. Roughly one in ten newborns worldwide meets a population-based SGA definition, and a meaningful fraction of these are SGA because of pathological intrauterine growth restriction (IUGR) rather than simple constitutional smallness — most commonly the consequence of placental insufficiency that limits the transfer of oxygen and nutrients across the second half of gestation [1]. The distinction matters clinically: a constitutionally small but proportionately grown infant behaves very differently, nutritionally and metabolically, from an infant whose placenta failed to keep pace with fetal demand in the third trimester. Both groups, however, are captured by the same birth-weight-percentile definition, and both are, in different ways, at the center of the growth-trajectory question this article addresses.

That question was given its modern shape by an observation that at first seems almost paradoxical: infants who are small at birth and then grow rapidly to "catch up" toward the population mean are, on average, at higher long-term risk for the same cluster of cardiometabolic disease — insulin resistance, central adiposity, elevated blood pressure — that a naive reading of growth science would predict rapid catch-up growth to protect against [2,3]. This is the developmental-origins or "thrifty phenotype" literature, and it has reframed how pediatric endocrinologists and

neonatologists think about postnatal growth in the SGA infant: not simply as a deficit to be corrected as quickly and completely as possible, but as a trajectory whose velocity, timing, and composition (lean mass versus fat mass, linear growth versus weight gain) all carry independent prognostic weight [4,5]. At the same time, an entirely separate and equally well-established literature shows that inadequate postnatal growth in the SGA infant, particularly failure to achieve catch-up linear growth in the first two years, is associated with impaired neurodevelopment and, if height catch-up never occurs, adult short stature that may itself carry psychosocial and quality-of-life costs [6,7]. The clinician managing an SGA infant's nutrition is, in other words, navigating between two real and well-documented risks that point in partially opposite directions.

This article synthesizes the literature bearing on that navigation problem. We are not reporting a new trial or a new cohort; we are pulling together what randomized nutritional intervention trials, large observational cohorts, and consensus guidelines have actually found about growth trajectories in SGA infants who receive different postnatal nutritional exposures, and about how those trajectories relate to later neurodevelopmental, anthropometric, and metabolic outcomes. We organize the discussion around five themes: the definition and etiology of SGA birth as the starting condition that nutritional intervention has to work with; the developmental-origins evidence that motivates concern about the pace and composition of catch-up growth; the direct evidence from randomized nutritional intervention trials in SGA and related preterm populations; the neurodevelopmental and endocrine consequences of different growth patterns, including the specific evidence on growth hormone therapy for SGA children who fail to catch up; and the open questions that remain despite several decades of accumulated evidence. We close with a discussion of what we see as the field's most consequential unresolved tensions.

It is worth being explicit about scope from the outset. The randomized-trial evidence on postnatal nutrition specifically in SGA infants is smaller and older than one might expect for a condition this common; much of what is known blends trials conducted in SGA term infants with a larger and more recent literature on preterm infants (who are frequently, but not always, also SGA), and with observational cohort data that can

describe association but not intervention effect. We have tried to be careful throughout about which claim rests on which kind of evidence, and we flag directly where the literature's confidence outruns its evidence base.

2. Defining and Diagnosing Small-for-Gestational-Age Birth

The birth-weight-percentile definition of SGA is simple to state and surprisingly consequential to apply well, because the percentile a given infant occupies depends entirely on which reference population and which growth standard is used to compute it. For most of the twentieth century, national and regional birth-weight charts, often derived from a single hospital's delivery population decades earlier, were used to classify newborns, producing SGA rates that varied for reasons having nothing to do with the babies themselves. The INTERGROWTH-21st Project addressed this directly by prospectively recruiting a large, multi-country, health-optimized population across eight countries and deriving international standards for newborn weight, length, and head circumference by gestational age and sex, intended to describe how human fetuses and newborns grow when maternal health, nutrition, and antenatal care are not limiting factors, in the same way the WHO Child Growth Standards describe optimal postnatal growth for infants and young children [8,9]. Both standards share a philosophy that matters directly for this article's subject: they are prescriptive standards of how growth should proceed under good conditions, not merely descriptive references of how a particular population happened to grow, and using a prescriptive rather than a descriptive standard changes which infants get labeled SGA and, downstream, which infants get flagged for the kind of nutritional attention this article is about.

Etiologically, SGA is not one condition but a common final pathway for a heterogeneous set of causes, and the distinction between them shapes both the nutritional approach and the growth trajectory that can realistically be expected. Constitutional smallness — a small but healthy, proportionately grown infant, often reflecting parental size or ethnic growth norms — carries a much better prognosis, nutritionally and developmentally, than pathological intrauterine growth restriction driven by placental insufficiency, maternal vascular disease, fetal genetic or chromosomal abnormality, congenital infection, or maternal substance exposure. Burton and

Jauniaux's review of the pathophysiology of placental-derived fetal growth restriction lays out the mechanistic core of the most common pathological route: inadequate remodeling of the maternal spiral arteries during placentation leads to reduced and increasingly turbulent uteroplacental blood flow, progressive placental oxidative and nutrient stress, and, in the most severe cases, a redistribution of fetal cardiac output ("brain-sparing") that preferentially protects the fetal brain at the expense of somatic, hepatic, and adipose growth [1]. This mechanistic detail is not academic for the nutrition question: an infant whose growth restriction reflects late, severe placental insufficiency is typically born with disproportionately depleted fat and glycogen stores relative to its length and head circumference, which is precisely the phenotype at highest immediate risk of neonatal hypoglycemia and precisely the phenotype in which the composition, not just the quantity, of postnatal nutrition is likely to matter most.

The clinical challenge this creates is that birth-weight percentile alone cannot reliably distinguish a constitutionally small, well-nourished infant from a pathologically growth-restricted one; additional information — the fetal growth trajectory across pregnancy (a fetus that crossed percentiles downward is a different case than one that was always at the 5th percentile), placental Doppler findings, and postnatal signs such as disproportion between weight and length — is needed to make that distinction with any confidence. The International Consensus Guideline on the management of children born SGA, coordinated across the pediatric endocrinology and growth hormone research societies, makes this point centrally: it recommends a structured evaluation for the underlying cause of SGA birth, not simply a growth-percentile label, precisely because etiology carries prognostic information that a percentile score on its own does not [7]. For the purposes of this article, we note that essentially the entire evidence base on postnatal nutritional intervention that follows was generated in cohorts that mixed constitutional and pathological SGA to varying and often incompletely reported degrees, which is itself one of the evidentiary limitations we return to in the closing section.

3. Catch-Up Growth, Body Composition, and the Developmental-Origins Literature

Table 1: Body composition and metabolic risk markers at a mean age of 6.1 years in children born small for gestational age, comparing those with catch-up growth in weight to those without, from Marcovecchio and colleagues' UK cohort of 259 children born SGA (150 assessed in detail) [12].

Marker	Weight catch-up group	No weight catch-up	P value
Proportion of cohort with catch-up growth in weight	122/150 (81%)	28/150 (19%)	--
Fat mass index (kg/m ²)	3.13 (SD 1.36)	2.56 (SD 0.91)	0.04
Trunk-to-limb fat ratio	0.63 (SD 0.14)	0.56 (SD 0.08)	0.002
Fasting glucose (mmol/L)	4.5 (SD 0.5)	4.3 (SD 0.5)	0.03
Systolic blood pressure SD score	0.09 (SD 0.71)	-0.32 (SD 0.63)	0.008

The single most influential idea shaping how clinicians think about SGA nutrition over the past three decades did not originate in a neonatal nursery at all. David Barker and colleagues' analysis of historical birth and infant-weight records linked to adult mortality outcomes in a cohort of English men found that men who had been small at birth and remained thin at one year of age had substantially higher rates of death from ischaemic heart disease than men who had been larger at both time points, an association that held after adjusting for adult risk factors [3]. Hales and Barker's subsequent "thrifty phenotype hypothesis" proposed a mechanistic account for why: a fetus exposed to poor intrauterine nutrition adapts by permanently reprogramming its metabolism toward glucose conservation and insulin resistance, an adaptation that is protective in a nutrient-scarce environment but becomes maladaptive, predisposing to type 2 diabetes and cardiovascular disease, if the postnatal environment turns out to be nutrient-abundant [2]. This is the conceptual seed of what is now a large developmental-origins-of-health-and-disease literature, and its specific relevance to this article is the corollary that followed: if fetal undernutrition programs a thrifty metabolic phenotype, then rapid postnatal catch-up growth — precisely the growth pattern nutritional intervention in SGA infants is often designed to promote — could be the trigger that converts a latent thrifty adaptation into overt metabolic disease.

Three decades of subsequent work have refined rather than overturned this concern, and have been particularly important in showing that it is not catch-up growth per se but its composition and tempo that carry the risk. Dullo, Jacquet, Seydoux, and Montani's review

of the "thrifty catch-up fat" phenotype synthesizes evidence that catch-up growth after any period of growth restriction is disproportionately composed of fat rather than lean tissue, reflecting a preferential replenishment of depleted energy stores, and that this catch-up fat, rather than catch-up growth in stature or lean mass, is the component most consistently linked to later insulin resistance and metabolic syndrome risk across animal and human studies [4]. Ibáñez, Ong, Dunger, and de Zegher's prospective study of SGA children followed from infancy found that those who showed the most pronounced catch-up weight gain in the first two years went on to develop measurably higher fasting insulin and reduced insulin sensitivity in early childhood compared with SGA children whose catch-up was more gradual, directly linking the tempo of early catch-up, not merely its eventual magnitude, to a detectable insulin-resistance phenotype years before any clinical metabolic disease would be apparent [10]. Mericq and colleagues' broader review of long-term metabolic risk in children born premature or SGA situates these infancy-specific findings within the full trajectory to adulthood, concluding that early catch-up growth is one of several converging risk markers, alongside prematurity itself and persistent postnatal growth failure, that together identify a subset of children who warrant longer-term cardiometabolic surveillance well beyond the neonatal and infancy period during which nutritional decisions are actually made [11]. Morrison and colleagues' review of fetal growth restriction and catch-up growth makes a complementary point about mechanism and timing: the developmental window during which rapid postnatal growth is metabolically consequential appears to be concentrated in infancy and early childhood, when adipose tissue, pancreatic beta-cell mass, and hypothalamic appetite-regulatory circuits are still undergoing substantial postnatal maturation and are therefore more susceptible to being permanently reset by an early nutrient-rich environment than they would be later in childhood [5].

Human data bearing directly on this tempo-and-composition question come from cohort studies that follow SGA children with detailed body-composition and metabolic phenotyping rather than weight and height alone. Marcovecchio and colleagues' UK cohort study of 259 children born SGA, with detailed assessment in 150 of them at a mean age of

6.1 years, found that 122 children — 81% of those assessed — had shown catch-up growth in weight by that age, confirming that catch-up growth is the modal rather than the exceptional outcome after SGA birth [5,6]. Critically, within the catch-up-growth group, children were not metabolically homogeneous: those who had achieved weight catch-up without a proportionate gain in height had higher fat mass index (3.13 vs. 2.56 kg/m², $P = 0.04$) and a higher trunk-to-limb fat ratio (0.63 vs. 0.56, $P = 0.002$) than children whose catch-up growth in weight was matched by catch-up in height, and the weight-catch-up group as a whole had higher fasting glucose (4.5 vs. 4.3 mmol/L, $P = 0.03$) and higher systolic blood pressure SD score (0.09 vs. -0.32, $P = 0.008$) than children without weight catch-up [12]. Beltrand and colleagues, following infants with fetal growth restriction to one year of age, reported that catch-up growth in this cohort was accompanied by comparatively rapid restoration of fat mass relative to lean mass, though without detectable adverse metabolic consequences as early as one year — a finding the authors themselves frame as consistent with the catch-up-fat hypothesis while noting that one year of age may simply be too early in the developmental window for metabolic consequences to be measurable [13]. Read together, this body of work supports a specific, more nuanced version of the original catch-up-growth concern: it is not catch-up growth in general that appears hazardous, but catch-up growth that is disproportionately adipose, occurs without matched linear (height) catch-up, and occurs early, in infancy rather than later in childhood.

This nuance matters enormously for how one reads the nutritional intervention literature that follows, because a formula or feeding strategy that accelerates weight gain without also supporting proportionate linear growth is, on the logic just described, arguably promoting exactly the growth pattern the developmental-origins literature flags as concerning — even though "faster weight gain" is often the crude, easily measured endpoint such interventions are evaluated against.

4. Nutritional Intervention Trials and Growth Trajectories

The most directly relevant randomized evidence comes from trials that assigned SGA or growth-restricted infants to nutritionally different postnatal feeding regimens and measured resulting growth. Fewtrell and

colleagues randomized term-born SGA infants to a nutrient-enriched formula or a standard term formula from birth through nine months of age, with a breastfed reference group followed in parallel (nutrient-enriched formula group $n = 152$, standard formula group $n = 147$, breastfed reference $n = 175$), and measured weight, length, and head circumference through 18 months [14]. The result is instructive precisely because it did not confirm a simple "more nutrients, more catch-up" story. By nine months, the nutrient-enriched group had gained 1.1 cm more in length than the standard-formula group (95% CI 0.38–1.79 cm, $P \leq 0.01$), a difference that persisted at roughly the same magnitude, 1.0 cm, at 18 months (95% CI 0.25–1.83 cm, $P \leq 0.01$); head circumference showed a smaller but similarly persistent advantage, 0.5 cm at nine months and 0.6 cm at 18 months (both $P \leq 0.01$). Weight, however, did not differ significantly between the nutrient-enriched and standard-formula groups at either time point, and the length and head-circumference advantages of the enriched formula were substantially larger, and statistically significant only, in girls, with boys showing no significant diet-group differences on any growth measure [14]. In the framework the previous section laid out, this is in some ways a reassuring result: a nutritionally enriched feeding strategy produced measurable catch-up in linear growth and head growth — the dimensions of growth most tied to neurodevelopmental adequacy — without producing the excess weight (and, by inference, excess adiposity) gain that the developmental-origins literature would flag as concerning. It is also a result that resists easy generalization, given the pronounced and only partly explained sex difference in response.

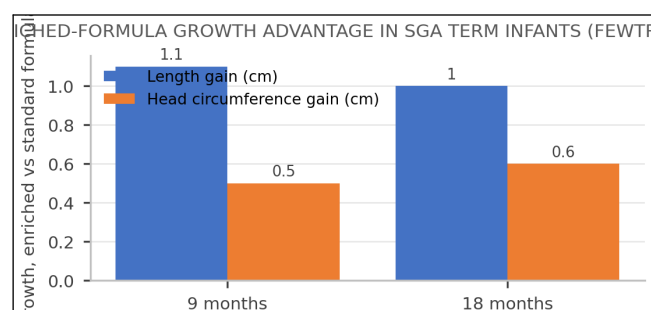


Figure 1: Additional linear and head-circumference growth (nutrient-enriched formula minus standard formula, cm) in small-for-gestational-age term infants at 9 and 18 months of age, reported in Fewtrell and colleagues' randomized trial. Weight did not differ significantly between groups at either time point [14].

A structurally similar but distinct body of evidence

comes from trials in preterm infants, a population that overlaps substantially with, but is not identical to, the SGA population, since a preterm infant can be appropriately grown for gestational age or SGA. Lucas and colleagues' randomized trial of nutrient-enriched versus standard formula for preterm infants following hospital discharge, one of the foundational trials in this area, similarly found that a nutritionally enriched postdischarge formula produced measurable anthropometric benefits in the postdischarge period, contributing to the evidence base that motivated the widespread clinical adoption of nutrient-enriched postdischarge formulas for preterm infants generally, including the SGA subset among them [15]. The Cochrane systematic review of nutrient-enriched versus standard formula for preterm infants following hospital discharge, however, is more circumspect in its overall synthesis: pooling across the available randomized trials (which vary considerably in the degree of nutrient enrichment tested, the duration of the intervention, and the age at outcome assessment), the review's authors concluded that the evidence for a durable effect of nutrient-enriched formula on long-term growth or neurodevelopmental outcome is limited and of generally low certainty, even though short-term anthropometric differences, similar to those Fewtrell and colleagues reported, are more consistently observed [16]. This gap between reasonably consistent short-term anthropometric signal and inconsistent long-term outcome signal recurs throughout this literature and is, in our reading, one of its most important honest limitations rather than a reason to dismiss the short-term findings.

Breastfeeding occupies a distinctive position in this evidence base that is worth naming explicitly, because it cannot be studied with the same randomized design used for formula comparisons. Observational and quasi-experimental evidence broadly associates breastfeeding of SGA infants with a growth trajectory that favors lean-mass accretion and slower, more gradual weight gain relative to formula feeding, a pattern that is, again by the logic of the developmental-origins literature, plausibly the more metabolically favorable trajectory even where the absolute rate of catch-up growth is slower [17]. Because mode of feeding is not randomly assigned in these studies, however, confounding by maternal socioeconomic status, health behavior, and the severity of the underlying cause of SGA

birth cannot be fully excluded, and the honest state of the evidence is an association that is directionally consistent across multiple observational cohorts but not established with the causal confidence a large randomized trial would provide.

Underlying all of these intervention trials is a monitoring question that is easy to overlook but is not trivial: growth trajectories can only be assessed against a growth standard, and the WHO Child Growth Standards that clinicians use to plot an SGA infant's catch-up trajectory were themselves explicitly constructed, using the attained-growth-curve methodology described by Borghi and colleagues, to represent prescriptive growth under optimal feeding (predominantly breastfeeding) and health conditions rather than a population-average reference [9]. Interpreting a given SGA infant's weight-for-age or length-for-age trajectory therefore implicitly compares that infant, whatever its feeding regimen, against a growth pattern anchored in exclusively or predominantly breastfed reference infants — a detail with real practical consequences for how "catch-up" is defined and flagged across different feeding regimens, and one that is rarely made explicit in clinical use of these charts.

5. Neurodevelopmental and Endocrine Consequences of Growth Patterns

The developmental-origins literature reviewed above establishes reasons for caution about rapid, fat-predominant catch-up growth. Set against this is an equally well-established literature showing that inadequate catch-up growth, particularly inadequate catch-up in linear growth and head circumference, carries its own well-documented costs, and the practical task facing neonatal and pediatric nutrition is to navigate between these two risks rather than to treat either one in isolation.

On the neurodevelopmental side, Castanys-Muñoz and colleagues' systematic review of postnatal growth in term SGA infants found a consistent association across the reviewed studies between more favorable postnatal growth, particularly in length and head circumference, and better later neurocognitive outcomes, while postnatal growth that remained persistently poor was associated with the largest deficits [6]. This is consistent with, and gives added clinical weight to, the head-circumference findings in Fewtrell and colleagues' own trial, since head growth is the anthropometric

measure most directly, if imperfectly, linked to brain growth in infancy [14]. More recent large-cohort evidence continues to refine the picture of which infants are most at risk of failing to catch up in the first place: Choi and colleagues' nationwide, propensity-score-matched study of term SGA infants in Korea examined early postnatal growth predictors of subsequent catch-up growth and found that specific, identifiable early growth and feeding characteristics distinguish infants who go on to achieve catch-up growth from those who do not, supporting the case for early, individualized growth monitoring rather than a uniform feeding protocol applied to all SGA infants regardless of their early trajectory [18].

For the minority of SGA children who fail to achieve adequate catch-up in linear growth and remain short in later childhood, growth hormone (GH) therapy is an established intervention with a substantial trial evidence base of its own, distinct from but complementary to the nutritional literature discussed above. De Zegher and colleagues' meta-analysis of GH treatment trials in short children born SGA reported clinically meaningful gains in height over a multi-year treatment course along with an accompanying, better-tolerated increase in the rate of skeletal (bone) maturation relative to height gain, addressing an early theoretical concern that GH-accelerated growth in this population might advance bone age disproportionately and so fail to translate into greater adult height [19]. The International Consensus Guideline on the management of children born SGA, published more than two decades later, consolidates this and the subsequent GH trial literature into current practice recommendations: it specifies criteria, generally a combination of persistent short stature (typically height SD score below -2.5 , or below -2 with an additional risk factor) and documented failure of spontaneous catch-up growth by around two to four years of age, for considering GH therapy, and explicitly frames GH treatment as a tool reserved for children whose postnatal growth trajectory, nutritional and otherwise, has already failed to achieve adequate catch-up rather than as a substitute for optimizing nutrition in the first place [7]. This sequencing, nutritional optimization first, GH reserved for confirmed catch-up failure, is itself a synthesis of the tension this article has been describing: it treats the early nutritional window as the primary opportunity to shape a favorable trajectory, and GH as a corrective tool for the subset of

children for whom that window did not deliver on stature specifically, while nutrition's composition (not just its adequacy) continues to be managed with the metabolic risks of the developmental-origins literature in mind.

Body composition, rather than weight or height alone, is increasingly the outcome measure this literature converges on as the more informative one, precisely because it is the measure that best distinguishes the two competing risks this article has organized itself around. Lach and colleagues' study of body composition and "catch-up fat" growth specifically in healthy SGA preterm infants, incorporating neurodevelopmental outcome assessment alongside detailed body-composition measurement, exemplifies this shift in emphasis: rather than asking simply whether SGA infants catch up, the study asks what they catch up in, and how that composition relates to development, a framing that we think represents the current state of the art in how this question ought to be studied, even though, as the next section discusses, it is not yet how most of the field's evidence was generated [20].

6. Open Challenges

Several gaps recur across the literature synthesized here and deserve to be named plainly. First, and most fundamentally, there is a genuine tension in the goals themselves that the literature has not resolved so much as learned to hold in view simultaneously: sufficient catch-up growth, particularly in linear growth and head circumference, is necessary for good neurodevelopmental outcome and for avoiding persistent short stature [6,7], while excessive, fat-predominant, early catch-up growth carries measurable metabolic risk [2,4,12]. No trial in this literature has been designed with sufficient power and sufficiently long follow-up to identify the specific growth trajectory, in terms of velocity, timing, and body-composition mix, that optimizes both outcomes simultaneously in a given SGA infant; the field's working answer is closer to a set of qualitative heuristics, favor lean-mass over fat-mass catch-up, favor length and head-circumference catch-up specifically, avoid the most rapid tempo of weight catch-up in the first months of life, than a quantitative, individualized target.

Second, the evidence base is older and thinner than the size of the clinical problem would suggest. The two most directly relevant randomized nutritional intervention

trials discussed in this article, Fewtrell and colleagues' term SGA trial and Lucas and colleagues' preterm postdischarge trial, are both now well over two decades old [14,15], and the Cochrane review synthesizing subsequent trials in preterm infants found the overall evidence for durable long-term benefit to be of limited certainty [16]. Modern SGA and preterm nutrition practice, including the composition of currently marketed enriched formulas, has moved on from what these trials tested, and a genuinely up-to-date randomized evidence base, ideally one with sufficiently detailed body-composition and metabolic outcome measurement to speak directly to the composition question raised above rather than weight and length alone, is a notable and, we think, somewhat surprising gap for a condition affecting roughly one in ten births worldwide.

Third, breastfeeding, plausibly the single most consequential feeding decision for a term SGA infant's growth trajectory, is also the one that is least amenable to the randomized designs that have generated the strongest evidence discussed in this article, leaving its apparent advantages resting on observational and necessarily confounded comparisons [17]. Fourth, sex-specific effects, visible clearly in Fewtrell and colleagues' trial, where growth benefits of nutrient enrichment were concentrated in girls with little effect in boys, are reported inconsistently and are rarely built into either trial design or clinical guidance as a stratification variable, despite a plausible biological basis for sex-differential nutritional programming [14]. Fifth, and more structural: almost none of the cohorts and trials synthesized here distinguish consistently between constitutionally small and pathologically growth-restricted SGA infants in their published growth and outcome analyses, despite the mechanistic case, laid out clearly in the placental-insufficiency literature, that these are different starting conditions likely to respond differently to the same nutritional intervention [1]. A nutritional intervention trial that pools these two populations without stratifying by etiology risks averaging over a real biological difference in a way that could obscure a benefit, or a risk, present in one subgroup but not the other.

We do not think these gaps undermine the core, reasonably well-supported conclusions this literature does support, namely that postnatal growth trajectory in

the SGA infant matters independently for both neurodevelopmental and metabolic outcomes, and that nutritional composition and tempo, not simply caloric adequacy, are the levers available to influence that trajectory. But the gaps are real, and a field this clinically important, affecting roughly one in ten newborns, would benefit from a modern, adequately powered, etiology-stratified, body-composition-informed trial base that the current literature, built substantially on trials from the 1990s and 2000s, does not yet provide.

7. Conclusion

The literature reviewed here supports a specific, more textured claim than either "catch-up growth is good" or "catch-up growth is dangerous": postnatal growth trajectory in the small-for-gestational-age infant is a genuinely double-edged clinical target, in which adequate catch-up in linear growth and head circumference supports neurodevelopment and avoids long-term short stature [6,18,7], while catch-up growth that is rapid, weight-predominant, and fat-rich, particularly when it occurs in the first months of life, carries measurable longer-term metabolic risk traceable to the developmental-origins and thrifty-phenotype literature [3,2,4,12]. Randomized nutritional intervention evidence, though older and thinner than the scale of the clinical problem warrants, is broadly consistent with the possibility of threading this needle: nutrient-enriched feeding regimens in both term SGA and preterm postdischarge populations have produced measurable gains in linear and head growth without producing the excess weight gain that would signal disproportionate fat accretion, even as the durability and long-term outcome benefit of these gains remains less certain than their short-term anthropometric signal [14,15,16].

Making this synthesis clinically actionable depends on treating growth-trajectory monitoring, not simply growth-adequacy monitoring, as the standard of care for SGA infants: distinguishing constitutional from pathological SGA where possible [1], tracking body composition and not only weight and length where feasible [20], using appropriately prescriptive growth standards to judge what "catch-up" should look like [9,8], and reserving growth hormone therapy, per current international consensus, for children whose catch-up genuinely fails to materialize despite adequate nutritional support [7]. None of these refinements are

reasons to abandon nutritional intervention as a tool in SGA care; they are, read together, a case for practicing it with more attention to the composition and tempo of the growth it produces, and less reliance on weight gain alone as the marker of success, than the field's earlier, foundational trials were designed to measure.

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Conflict of Interest

The authors declare that they have no competing interests relevant to the content of this article.

Data Availability Statement

This article is a literature synthesis and did not generate new data; all findings discussed derive from the cited publications, and readers are directed to those sources for underlying data and code availability.

Ethical Approval

Not applicable. As a synthesis of previously published literature, this article did not involve human participants, animal subjects, or the collection of primary clinical data.

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