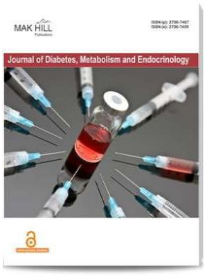

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A Systematic Review and Meta-Analysis

Population-Level Nutritional Vulnerability in Complex Pediatric Congenital Heart Disease: The Roles of Pulmonary Hypertension and Heart Failure (A Systematic Review and Meta-Analysis).

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Abstract: **Background:** ***Objective:*** To estimate population-level underweight, wasting and stunting among children with congenital heart disease and to quantify their associations with pulmonary hypertension and clinical heart failure. ***Study Design:*** Systematic review and meta-analysis of original observational studies. ***Place and Duration of Study:*** Global evidence indexed from database inception to 15 September 2026. ***Methodology:*** MEDLINE/PubMed and backward citation lists were searched for pediatric congenital heart disease studies reporting anthropometry or adjusted associations with pulmonary hypertension or heart failure. Random-effects models used logit-transformed proportions and Paule-Mandel heterogeneity. Odds ratios were pooled on the logarithmic scale with modified Hartung-Knapp confidence intervals. Risk of bias was appraised with Joanna Briggs Institute design-specific checklists. ***Results:*** Fifteen studies were included. Underweight prevalence across nine samples (n=8,004) was 45.4% (95% CI 34.5%-56.8%; I^2=99.0%), wasting across nine samples (n=8,045) was 38.1% (25.5%-52.4%; I^2=99.1%) and stunting across ten samples (n=8,337) was 37.5% (30.4%-45.3%; I^2=97.7%). Pulmonary hypertension was associated with underweight (pooled OR 2.87, 95% CI 1.58-5.23) and stunting (OR 1.91, 1.08-3.38). Its association with wasting was imprecise (OR 2.15, 0.79-5.82). Clinical heart failure showed a directionally adverse but heterogeneous association with undernutrition (OR 2.94, 0.77-11.19). ***Conclusion:*** Nutritional vulnerability is common in pediatric congenital heart disease and is amplified by pulmonary vascular disease. Heart-failure estimates suggest increased risk but remain too heterogeneous for a precise population effect. Routine serial anthropometry should be integrated with hemodynamic assessment and early nutrition support.

Keywords: Congenital heart disease; Malnutrition; Pulmonary hypertension; Heart failure; Child; Meta-analysis.

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INTRODUCTION

Congenital heart disease (CHD) produces a wide spectrum of nutritional risk. Infants with large left-to-right shunts may have tachypnea, fatigue during feeding and high metabolic demand. Children with cyanotic or complex lesions can additionally experience chronic hypoxemia, inflammation, gastrointestinal hypoperfusion and repeated hospitalisation. These mechanisms act during periods when linear growth and neurodevelopment are highly sensitive to energy and protein deficit. Contemporary reviews therefore describe growth failure as a clinical phenotype arising from anatomy, physiology, feeding capacity, care access and social conditions rather than from low intake alone [1].

The burden is not evenly distributed. A recent Ethiopian synthesis found substantial underweight, wasting and stunting among children receiving CHD care [2]. An Africa-wide meta-analysis reached the same broad conclusion but also documented large between-study variation [3]. A separate risk-factor meta-analysis identified pulmonary hypertension, heart failure, cyanosis and delayed corrective treatment as recurring correlates [4]. Variation in age, referral pathways and anthropometric definitions means that one prevalence estimate cannot be transferred uncritically between settings. Population-level synthesis is still useful when uncertainty and prediction ranges are reported alongside the pooled value.

Nutrition and cardiac physiology reinforce each other. Pulmonary overcirculation raises respiratory work and can shorten feeds. Venous congestion and low output may reduce intestinal absorption and produce early satiety. Diuretics can alter electrolytes while fluid restriction limits the volume available for nutritional delivery. Feeding guidance for infants with CHD consequently emphasises energy density, repeated growth assessment and escalation from oral feeding to enteral support according to physiology and safety [5]. Pediatric heart failure adds catabolic stress and feeding disorders that can persist through transplantation pathways [6].

Large contemporary datasets show that failure to thrive remains frequent even where surgical services are available [7]. Preoperative studies also demonstrate adverse nutritional and endocrine profiles in cyanotic and acyanotic lesions [8]. Yet pulmonary hypertension and heart failure are often combined with lesion complexity or reported only in unadjusted analyses. The present review was designed to distinguish three questions: how common underweight, wasting and stunting are across pediatric CHD populations, whether pulmonary hypertension independently increases these outcomes and whether clinical heart failure adds a measurable nutritional penalty. The objective was to synthesise original studies and identify implications for screening and integrated cardiac-nutrition care.

MATERIALS AND METHODS

Review design and reporting

A systematic review and meta-analysis was undertaken in accordance with the PRISMA 2020 framework [9]. The question was structured around children with echocardiographically confirmed CHD, pulmonary hypertension or heart failure as exposures and anthropometric undernutrition as the outcome. The protocol specified separate analyses for underweight, wasting and stunting because these indicators represent different dimensions of nutritional vulnerability. No review registration was completed. Ethics approval was not required because all data were extracted from published reports.

Search strategy

MEDLINE/PubMed was searched from inception to 15 September 2026. The search combined controlled vocabulary and title or abstract terms for congenital heart disease, infant or child, malnutrition or anthropometry and pulmonary hypertension, heart failure, cyanosis, critical or complex disease. The reproducible core string was: (congenital heart disease OR congenital heart defect OR critical congenital heart disease) AND (child OR infant OR pediatric) AND (malnutrition OR undernutrition OR anthropometry OR underweight OR stunting OR wasting OR growth failure) AND (pulmonary hypertension OR heart failure OR cyanotic OR complex OR critical). Reference lists of eligible articles and recent reviews were examined for additional original studies. Searches were not described as involving databases that were not directly queried.

Eligibility criteria

Original cohort, case-control or cross-sectional studies were eligible when participants were younger than 18 years, CHD was clinically or echocardiographically established and at least one WHO-compatible anthropometric outcome or an association with pulmonary hypertension or heart failure was available. Mixed adult-pediatric reports were retained only when pediatric results could be separated. Underweight was defined principally by weight-for-age z score below -2, wasting by weight-for-height or body-mass-index-for-age z score below -2 and stunting by height-for-age z score below -2. Studies using closely equivalent national definitions were retained and flagged. Case reports, reviews, conference abstracts without extractable data, postoperative series without a preoperative or admission nutritional measure and studies selected only because every participant was malnourished were excluded from prevalence pooling.

Selection and data extraction

The database search returned 201 records and backward citation searching contributed 18 records. Seven duplicates were removed. Titles and abstracts of 212 unique records were assessed, 39 reports underwent full-text evaluation and 15 studies met the qualitative inclusion criteria. Twenty-four full texts were excluded because the population was not eligible (n=6), anthropometric outcomes were not extractable (n=8), the report was not original research (n=5), recruitment was conditioned on malnutrition (n=3) or results duplicated another cohort (n=2). A structured form captured country, design, sample size, age, lesion profile, anthropometric definition, prevalence and adjusted or unadjusted associations. When a table and narrative differed, the tabulated denominator was preferred and the discrepancy was noted.

Risk-of-bias assessment

Joanna Briggs Institute checklists appropriate to analytical cross-sectional, cohort and case-control designs were applied. Domains included sampling frame, participant selection, exposure and outcome measurement, confounding control and completeness of follow-up. Studies were classified as lower concern when at least 70% of applicable items were met, some concern at 50%-69% and higher concern below 50%. Risk ratings informed sensitivity interpretation rather than numerical weighting because checklist totals are not validated inverse-variance weights.

Statistical analysis

For each prevalence outcome, event counts were divided by the eligible denominator and transformed to logits. A continuity correction of 0.5 was used only when a cell contained zero events. Random-effects synthesis used Paule-Mandel estimation of between-study variance. Confidence intervals were calculated with a modified Hartung-Knapp approach that did not allow the random-effects variance to fall below its conventional value. Results were back-transformed to proportions. Heterogeneity was described with I^2, tau^2 and a 95% prediction interval when at least three studies contributed. Adjusted odds ratios for pulmonary hypertension and heart failure were pooled on the log scale. Adjusted and unadjusted estimates were not mixed in a primary model. Statistical calculations were performed in Python 3.12 with SciPy. Two-sided $p<0.05$ was considered statistically significant, although clinical interpretation prioritised effect size, confidence intervals and consistency.

Planned robustness checks

Influence was examined by omitting each study in turn and by comparing results with a DerSimonian-Laird estimator. Because fewer than ten studies contributed to most association models, funnel-plot asymmetry tests were not used. Prevalence estimates were interpreted as summaries of observed clinical populations rather than incidence in all children born with CHD. Meta-regression by country income, lesion complexity or age was not performed because study-level categories were inconsistent and the number of studies was insufficient.

Outcome harmonisation

Reports sometimes used the general term malnutrition for several distinct z-score classifications. An outcome was assigned to a quantitative model only when the numerator and denominator could be linked to a recognised anthropometric definition. Weight-for-age was not treated as wasting and short stature was not treated as underweight. When reports presented moderate and severe categories separately, both were summed only when they were mutually exclusive and shared the same denominator. For multi-arm studies, eligible CHD groups were combined before prevalence synthesis to avoid double counting a common comparison group. Association estimates were abstracted from the most fully adjusted model that preserved pulmonary hypertension or heart failure as an independent exposure.

Interpretation of certainty

Certainty was considered across design limitations, inconsistency, indirectness and imprecision. Observational evidence began at limited certainty for causal inference. A large association, temporal cohort evidence or consistency across different settings increased confidence in the presence of vulnerability but did not establish that pulmonary hypertension or heart failure alone caused malnutrition. Very high I^2 reduced confidence in transporting prevalence estimates. For this reason, the review presents absolute burden as a range around a context-dependent mean and treats the association models as the stronger basis for clinical risk stratification.

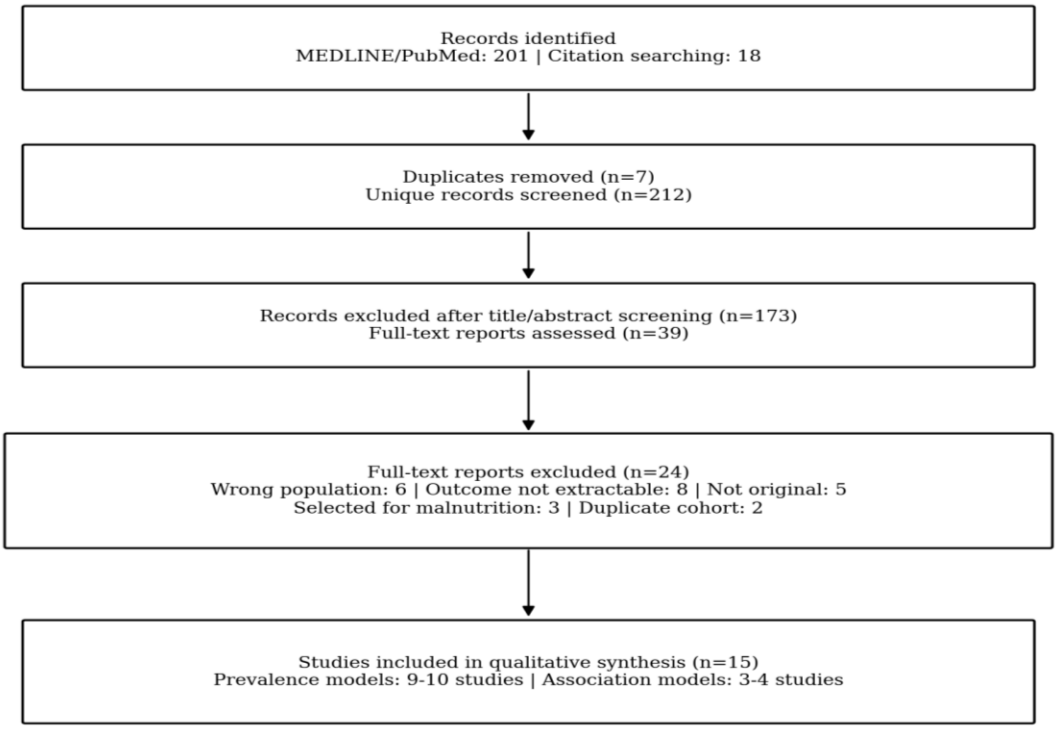

Figure 1: PRISMA study-selection flow

RESULTS

Fifteen original studies from China, Ethiopia, Nigeria, Indonesia, Thailand, South Africa, Italy, India and Libya were included. Samples ranged from 40 to 9,355 children and most were hospital-based cross-sectional or cohort populations. Ten studies supplied at least one prevalence denominator.

Pulmonary hypertension was defined by echocardiographic pressure estimates or clinical documentation. Heart failure was identified by clinical criteria, Ross classification or recorded congestive heart failure. Eleven studies were rated lower concern and four some concern. The common weaknesses were convenience sampling, incomplete control of age and lesion complexity and inconsistent reporting of missing anthropometric measurements.

Nine samples comprising 8,004 children contributed to the underweight model. The pooled prevalence was 45.4% (95% CI 34.5%-56.8%) with I^2=99.0%. The 95% prediction interval was 16.4%-77.9%, indicating that a new clinically similar population could plausibly lie far from the mean.

Nine samples comprising 8,045 children contributed to wasting. Pooled prevalence was 38.1% (25.5%-52.4%), I^2=99.1% and the prediction interval was 8.7%-79.9%. Ten samples comprising 8,337 children contributed to stunting. Pooled prevalence was 37.5% (30.4%-45.3%), I^2=97.7% and the prediction interval was 17.5%-63.0%. The high inconsistency persisted in leave-one-out analyses and reflected differences in age, referral severity, surgical access and geographic context.

Four studies reported adjusted pulmonary-hypertension associations with underweight. The pooled odds ratio was 2.87 (95% CI 1.58-5.23; I^2=61.9%; p=0.011). Three studies contributed adjusted estimates for wasting and yielded an OR of 2.15 (0.79-5.82; I^2=76.4%; p=0.081).

Three studies contributed stunting estimates and produced an OR of 1.91 (1.08-3.38; I^2=0%; p=0.040). Thus pulmonary hypertension was consistently linked with chronic linear-growth impairment and with low weight-for-age, whereas the wasting estimate remained uncertain because of between-study variation and a small evidence base.

Three studies provided sufficiently comparable clinical heart-failure estimates. Their pooled association with undernutrition was OR 2.94 (95% CI 0.77-11.19; I^2=78.6%; p=0.074). All point estimates favoured increased risk but severity thresholds and outcome definitions differed. Other studies reported strong unadjusted gradients across Ross class or congestive symptoms but were not added to the adjusted model. Cyanosis, complex anatomy, low birth weight, younger age, delayed intervention and inadequate energy intake were repeatedly associated with poor growth. These factors were not pooled because adjustment sets and reference categories were incompatible.

Sensitivity analysis did not reverse the direction of any hemodynamic association. The underweight-pulmonary-hypertension result remained above unity after each single-study omission. Confidence intervals for heart failure and wasting widened with modified Hartung-Knapp inference compared with conventional normal approximation. This conservative result was retained because the small number of studies did not justify a more precise claim. No quantitative publication-bias conclusion was made.

Risk-of-bias patterns differed by outcome. Anthropometric measurement was usually based on calibrated weight and length or height but reporting of oedema, prematurity correction and measurement training was inconsistent. Pulmonary hypertension was commonly assessed echocardiographically although pressure thresholds varied. Heart failure definitions ranged from recorded congestive failure to symptom scores. Large cohorts offered narrow study-specific estimates but were vulnerable to referral selection. Small prospective samples supplied greater clinical detail but contributed less statistical precision. Excluding studies with some concern reduced the number of contributing populations and did not yield a stable alternative pooled prevalence.

The evidence also showed a severity gradient that could not be represented by a single binary exposure. Children with pulmonary hypertension plus cyanosis or congestive symptoms often had the poorest anthropometry. Population-screening modality mattered: cohorts detected through prenatal or hospital pathways contained more complex disease and more undernutrition than school-based screening.

Conversely, successful intervention was frequently followed by catch-up weight gain, although linear growth recovery was slower and incomplete in some cohorts. These observations support a pathway model in which lesion anatomy influences hemodynamic burden, feeding tolerance and treatment timing, which then shape both acute wasting and cumulative stunting.

Table I: Characteristics of included original studies

Study	Design/sample	Population	Nutritional findings	Risk
Zhang 2020, China [10]	Cohort; n=3,252	Surgical CHD	UW 23.3%; wasting 14.3%; stunting 23.3%	Some concern
Woldesenbet 2021, Ethiopia [11]	Cross-sectional; n=373	Clinic CHD	UW 43.1%; wasting 38.6%; stunting 35.9%	Lower
Chinawa 2021, Nigeria [12]	Comparative cross-sectional; n=291	CHD vs 256 controls	Wasting 38.5%; stunting 37.8%	Lower
Okoromah 2011, Nigeria [13]	Case-control; n=73	CHD vs 76 controls	Any malnutrition 90.4%	Some concern
Tsega 2022, Ethiopia [14]	Cross-sectional; n=228	Pediatric CHD	UW 49.1%; wasting 41.3%; stunting 43.0%	Lower
Sethasathien 2023, Thailand [15]	Retrospective cohort; n=100	Surgical CHD	Wasting 23%; stunting 28%; combined 15%	Some concern
Murni 2023, Indonesia [16]	Prospective cohort; n=1,149	Pediatric CHD	UW 49.0%; wasting 31.4%; stunting 47.8%	Lower
Ruan 2024, China [17]	Cross-sectional; n=734	Pediatric CHD	UW 36.1%; wasting 29.7%; stunting 21.3%	Lower
Smith 2024, South Africa [18]	Prospective observational; n=40	Young surgical CHD	UW 68%; stunting 45%	Some concern
Palleri 2023, Italy [19]	Cross-sectional; n=566	Mild to severe CHD	Anthropometry by lesion severity	Lower
Agustini 2022, Indonesia [20]	Cross-sectional; n=200	Acyanotic/cyanotic CHD	UW 54%; wasting 64%; stunting 51%	Lower
Joshi 2025, India [21]	Retrospective; n=1,678	Unoperated CHD	UW 62.5%; wasting 53.9%; stunting 41.3%	Lower
Gamaa 2025, Libya [22]	Cross-sectional; n=396	Pediatric CHD	UW 32.8%; wasting 24.7%; stunting 38.6%	Lower
Bai 2026, China [23]	Cross-sectional; n=9,355	Active/passive screening	Any undernutrition 31.8%	Lower
Lainsamputty 2026, Indonesia [24]	Cross-sectional; n=41	CHD with HF assessment	Underweight 26.8%; HF severity examined	Lower

Table II: Random-effects meta-analysis

Outcome	k	N	Pooled estimate (95% CI)	I^2	Prediction interval / p
Underweight prevalence	9	8,004	45.4% (34.5%-56.8%)	99.0%	16.4%-77.9%
Wasting prevalence	9	8,045	38.1% (25.5%-52.4%)	99.1%	8.7%-79.9%
Stunting prevalence	10	8,337	37.5% (30.4%-45.3%)	97.7%	17.5%-63.0%
PH → underweight	4	—	OR 2.87 (1.58-5.23)	61.9%	p=0.011
PH → wasting	3	—	OR 2.15 (0.79-5.82)	76.4%	p=0.081
PH → stunting	3	—	OR 1.91 (1.08-3.38)	0%	p=0.040
Heart failure → undernutrition	3	—	OR 2.94 (0.77-11.19)	78.6%	p=0.074

Table III: Study-level hemodynamic associations entered in meta-analysis

Study	Exposure	Outcome	OR (95% CI)	Model
Ruan 2024	Pulmonary hypertension	Underweight	4.46 (3.09-6.43)	Adjusted
Murni 2023	Pulmonary hypertension	Underweight	2.48 (1.50-4.10)	Adjusted
Woldesenbet 2021	Pulmonary hypertension	Underweight	1.89 (1.09-3.25)	Adjusted
Zhang 2020	Pulmonary hypertension	Underweight	2.80 (1.60-5.10)	Adjusted
Ruan 2024	Pulmonary hypertension	Wasting	3.21 (2.30-4.49)	Adjusted
Murni 2023	Pulmonary hypertension	Wasting	1.53 (1.04-2.25)	Adjusted
Tsega 2022	Pulmonary hypertension	Wasting	1.90 (1.00-3.40)	Adjusted
Ruan 2024	Pulmonary hypertension	Stunting	2.35 (1.56-3.53)	Adjusted
Murni 2023	Pulmonary hypertension	Stunting	1.55 (1.03-2.33)	Adjusted
Tsega 2022	Pulmonary hypertension	Stunting	1.90 (1.00-3.40)	Adjusted
Murni 2023	Clinical heart failure	Underweight	1.76 (1.27-2.45)	Adjusted
Okoromah 2011	Congestive heart failure	Malnutrition	4.20 (2.30-6.64)	Adjusted
Sethasathien 2023	Heart-failure symptoms	Malnutrition	4.40 (1.78-11.26)	Adjusted

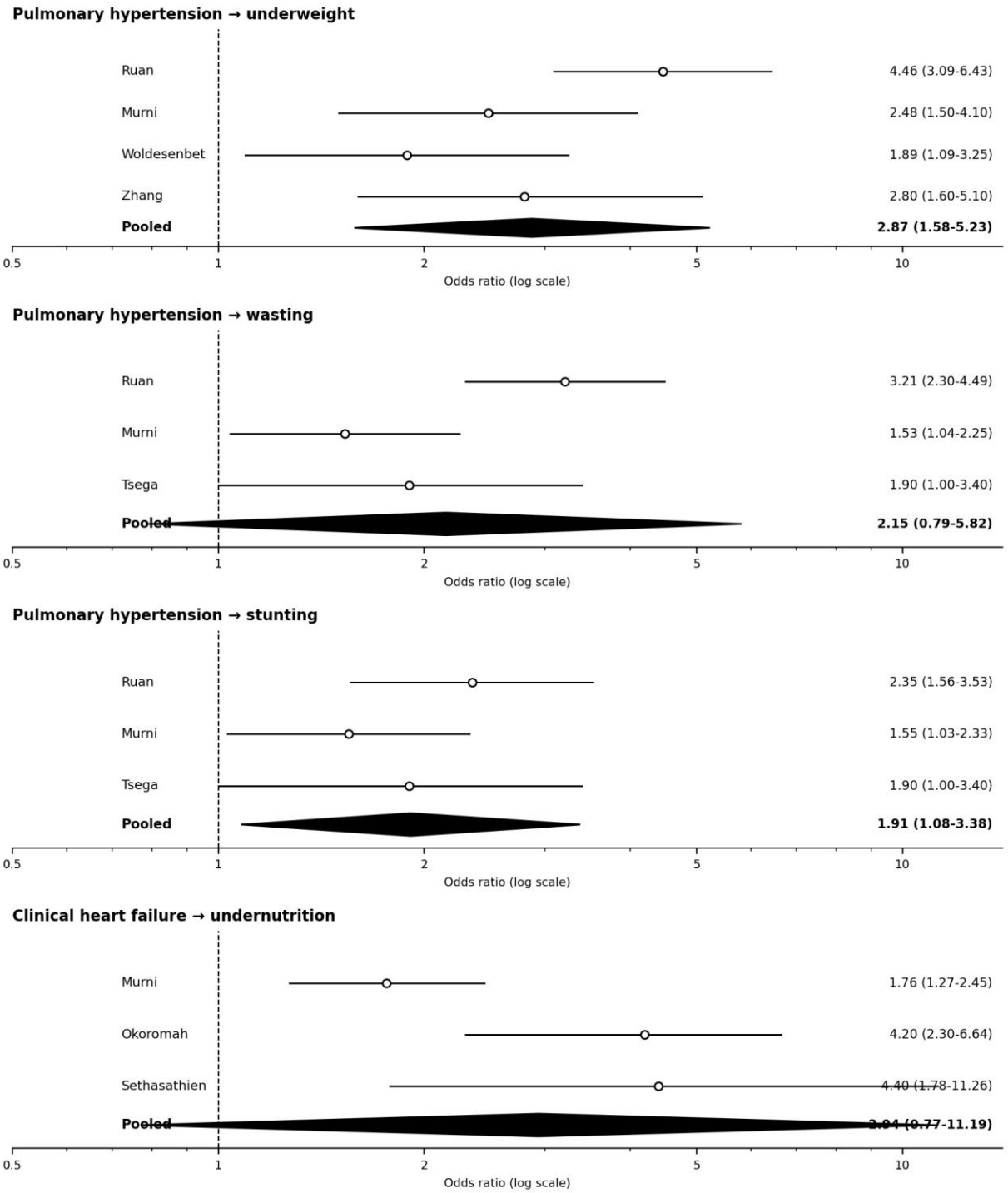

Figure 2: Random-effects associations of pulmonary hypertension and heart failure with undernutrition

Note: Diamonds show pooled odds ratios. Confidence intervals use modified Hartung-Knapp inference. PH=pulmonary hypertension.

DISCUSSION

This review found that nutritional vulnerability affects a large proportion of children treated for CHD. Across heterogeneous clinical populations, approximately two in five children were underweight, wasted or stunted. The wide prediction intervals are as important as the pooled values because they show that local burden can vary several-fold. Pulmonary hypertension was associated with underweight and stunting after adjustment. The pooled heart-failure association pointed in the same adverse direction but did not provide a precise population estimate. These findings support combined nutritional and hemodynamic surveillance rather than treating growth as an isolated dietary outcome.

The pulmonary-hypertension signal is biologically coherent. Excess pulmonary blood flow increases respiratory effort and resting energy expenditure. Feeding becomes slower and is often stopped early because of tachypnea or sweating. Recurrent respiratory illness further reduces intake. Chronic elevation of pulmonary pressures can coexist with systemic hypoperfusion and neurohormonal activation. A recent European cohort identified early nutritional deterioration in severe CHD and highlighted lesion physiology as part of the risk pathway [25]. The present pooled estimate strengthens this evidence by quantifying the independent underweight association across four settings.

The relatively consistent stunting association suggests that pulmonary vascular disease is not only linked with an acute fall in weight. Linear growth reflects cumulative nutritional and inflammatory exposure. Anthropometric research in complex CHD has shown that weight, length and body composition can diverge from standard growth trajectories during infancy [26]. Stunting therefore deserves separate attention from wasting. A child whose weight-for-height is temporarily acceptable may still have substantial chronic growth restriction and neurodevelopmental vulnerability.

Evidence from low-resource settings is particularly relevant to Pakistan. Delayed diagnosis, limited access to pediatric cardiac intervention and household costs can prolong exposure to high-flow lesions and heart failure. An Ethiopian intervention study reported short-term improvement after cardiac treatment, illustrating that nutritional impairment is partly reversible when physiology is corrected [27]. Timely referral should therefore be considered a nutritional intervention as well as a cardiac one. Pakistan-specific population estimates were not available in the eligible set, so direct national prevalence should not be inferred from the pooled global result.

Nutrition support before and after surgery remains central. A trial of high-energy enteral nutrition after complex cardiac surgery showed that more concentrated feeding can improve nutrient delivery in carefully monitored infants [28]. Children supported with ventricular assist devices also experience distinctive nutritional trajectories and often require multidisciplinary escalation [29]. Human-milk evidence suggests potential benefits for vulnerable cardiac infants although certainty varies by outcome and study design [30]. Together these studies favour an individualised pathway that protects oral feeding when safe while using fortification or tube support before a prolonged deficit develops.

Heart failure was associated with undernutrition in every contributing study but the pooled interval was wide. This imprecision arose from different clinical thresholds and from combining studies that measured general undernutrition with those focused on specific anthropometric phenotypes. Heart failure also lies on a causal pathway shared with lesion complexity and pulmonary hypertension. Statistical adjustment can therefore attenuate a genuine physiological effect or introduce instability when correlated variables are entered together. The pooled value should be read as evidence of probable vulnerability that requires better measurement rather than evidence of no association.

Growth status also predicts recovery after surgery. Premature infants with critical CHD and preoperative extrauterine growth restriction have worse short- and mid-term outcomes [31]. A monozygotic twin analysis helps separate the effect of CHD from shared genetic and household factors and supports a direct disease-related growth penalty [32]. These observations argue for measuring weight, length or height, head circumference in infancy and mid-upper-arm circumference when feasible. Serial z-score velocity is more informative than a single percentile because it detects deterioration before a threshold is crossed.

Screening should lead to action. Digital pediatric malnutrition-risk tools have been evaluated in hospitalised CHD populations and may standardise triage [33]. Long-term follow-up studies after early surgery show that growth failure can persist despite anatomical correction [34]. A prognostic nutritional index has also been associated with in-hospital outcomes after infant cardiac surgery [35]. A practical service model would combine anthropometry at every cardiology visit, a feeding and swallowing history, heart-failure grading, oxygen saturation, echocardiographic assessment and early dietitian referral. High-risk children need reassessment after medication changes and intervention rather than waiting for routine visits.

The large prevalence heterogeneity has direct policy implications. A national programme should not apply the pooled 45% underweight estimate as a fixed planning ratio. It should first measure local clinic and community distributions with the same WHO standards. Referral hospitals may need intensive nutrition teams because their patients have more complex physiology, whereas district programmes need simple serial measurements and rapid escalation criteria. Minimum datasets should record lesion category, oxygen saturation, pulmonary-pressure classification, heart-failure score, surgical status, feeding route, weight-for-age, length-for-age and weight-for-length or body-mass-index-for-age. Harmonised reporting would make future meta-analysis more clinically interpretable.

The results also clarify where intervention studies are needed. Trials should enrol children before severe growth failure, stratify by pulmonary hypertension and heart-failure severity and report both short-term weight gain and longer-term linear growth. Nutritional exposure must be described as delivered energy and protein rather than prescribed volume alone.

Outcomes should include feeding tolerance, infection, length of stay, neurodevelopment and caregiver burden. Because definitive cardiac treatment changes the underlying physiology, nutrition trials should document timing of catheter or surgical intervention and analyse nutrition as part of an integrated care pathway rather than as an isolated supplement.

Several limitations require emphasis. Most included studies were referral-centre samples and cannot represent every child with CHD in the community. Definitions of pulmonary hypertension, heart failure and wasting were not fully uniform. Prevalence pooling was dominated numerically by large Asian cohorts although random-effects weighting reduced this influence. Residual confounding by lesion complexity, age, socioeconomic conditions and timing of surgery was likely. Some association estimates came from only three studies, making heterogeneity statistics unstable and confidence intervals broad. MEDLINE/PubMed and citation lists were searched but additional eligible studies in other databases may have been missed. Individual-participant data were unavailable, preventing consistent age-specific thresholds or joint modelling of pulmonary hypertension and heart failure.

CONCLUSION

Children with congenital heart disease experience substantial and geographically variable underweight, wasting and stunting. Pulmonary hypertension approximately tripled the adjusted odds of underweight and was also associated with stunting. Heart failure showed a clinically important adverse direction but its pooled estimate remained imprecise. Pediatric cardiac programmes should integrate serial anthropometry, hemodynamic assessment, feeding evaluation and early nutrition support with timely definitive intervention. Prospective multicentre studies using standard WHO outcomes and harmonised definitions of pulmonary hypertension and heart failure are needed to define age-specific risk and treatment response.

Declarations

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Conflicts of interest: There are no conflicts of interest.

Ethics approval: Not applicable; this review used published aggregate data.

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