

Underlying Contributors to Childhood Stunting: What Evidence Exists on Nutrition-Sensitive Risk Factors?

RESULTS AND LESSONS LEARNED FROM SYSTEMATIC REVIEWS AND META-ANALYSES

Globally, an estimated 155 million children under the age of five have stunted linear growth.¹ This form of malnutrition is concentrated in low- and middle-income settings, with children in Africa and Asia disproportionately affected. Linear growth faltering, which results in stunting, is a dynamic process that begins in utero and continues into childhood. Children are most vulnerable during the first 1,000 days of life, from conception through age two, when growth is of highest velocity.² This critical period requires adequate nutrition, feeding, care, and stimulation to support optimal physical and brain development.

Stunting is a significant global health concern and a critical indicator for progress on the post-2015 United Nations Sustainable Development Goals agenda.³ The World Health Assembly has targeted a 40 percent global reduction in the prevalence of stunting from 162 million in 2012 to 100 million by 2025.⁴

What is stunting?

A child is “stunted” if he or she is too short for his or her age, having a length/height-for-age Z-score more than two standard deviations below the World Health Organization Child Growth Standards median.⁵

WHAT IS AT RISK

Stunting is a well-established population-level indicator of overall well-being and social inequity.^{6,7} During the early years of life, growth faltering due to undernutrition has both immediate and lifelong consequences for human health and development that are largely irreversible.

In the short-term, child growth restriction impedes brain, muscle, and organ development. Such restriction is also associated with an increased risk of morbidity and mortality.⁸ In the longer-term, childhood stunting is also associated with decreased adult stature, an increased risk of noncommunicable diseases, and an increased risk of adverse

Facts and figures: stunting at a glance

- Globally, 155 million children are stunted.
- Stunting affects 23 percent of children under five worldwide.
- Africa and Asia bear the greatest burdens of stunting:
 - More than one-third (38 percent) of all stunted children under five live in Africa.
 - More than half (56 percent) of all stunted children under five live in Asia.
 - Two out of five (40 percent) of stunted children in the world live in Southern Asia

pregnancy outcomes.^{9,10} More broadly, deficits in linear growth are associated with delayed cognitive development, which can result in lower school performance, decreased learning capacity, and reduced work and earning potential. This hampers not only the productivity and potential of individuals but of entire communities and nations.^{11,12}

As an intergenerational condition, the consequences of stunting create a harmful cycle of malnutrition and poverty that is challenging to disrupt.^{8,11,13} The severity, persistence, and intergenerational nature of stunting and its many ramifications for human capital point to the urgent need for global action.

WHAT CONTRIBUTES TO STUNTING?

Childhood stunting results from a complex array of interrelated factors. These contributors range from immediate drivers—such as dietary diversity, infant and young child feeding and care practices, and micronutrient status—to more distal, underlying socioeconomic, environmental, and other contextual factors.^{8,14} The diversity of the many factors that contribute to stunting are reflected in the World Health Organization’s conceptual framework, adapted in Figure 1.^{12,15}

Definitions

Nutrition-specific: A term that refers to interventions or programs that address immediate determinants of malnutrition, which include dietary intake, caregiving and feeding practices, and micronutrient deficiencies.¹⁷ The most impactful nutrition-specific interventions are identified in the Lancet series on maternal and child undernutrition.¹⁶

Nutrition-sensitive: A term that refers to interventions or programs that address the underlying causes of malnutrition, which include food security; access to health services; social safety nets; maternal health, education, social status, and empowerment; and healthy environments. Nutrition-sensitive interventions and programs can also serve as platforms for the delivery of nutrition-specific interventions.¹⁷

Despite the global scale and severity of stunting and its many consequences, there is still much that we do not know about the many contributors to linear growth faltering, how these factors interact with one another and differ across contexts, and how interventions can most effectively combat this complex and multifaceted nutrition and health outcome.

HOW DO WE PREVENT STUNTING?

Given this complexity, there is no “one-size-fits-all” solution for childhood stunting. In many ways, the prevention and reduction of stunting as a nutrition outcome has proven to be a policy and programmatic enigma.

The 2013 *Lancet Series on Maternal and Child Nutrition* discusses ten nutrition-specific interventions with a strong evidence-base for positive impact on maternal and child nutrition outcomes.¹⁶ These interventions target immediate determinants of malnutrition, such as dietary intake, infant and young child feeding and care practices, and micronutrient intake of both mother and child. However, scaling up even these highly effective interventions to 90 percent coverage in 34 of the highest burden countries is estimated to only alleviate a very limited portion of the total global burden of stunting.¹⁶

These findings underscore the urgent need to better understand and address the underlying risk factors for stunting in addition to the immediate risk factors. In order to make further strides, we must promote the most effective nutrition-sensitive interventions as well as those supporting women, families, communities, and our shared environment. Stunting is a crosscutting challenge that demands a coordinated, multisectoral response.

ABOUT THIS BRIEF

In this brief, we examine 11 systematic reviews and meta-analyses published from January 2000 to February 2018 that investigate the impacts of selected underlying, nutrition-sensitive risk factors on stunting. To assemble this list, we

searched PubMed and the Cochrane Library using search terms related to selected, nutrition-sensitive risk factors from the World Health Organization framework (Figure 1, highlighted in purple) each in combination with “stunting,” “growth,” and/or “child growth.” Due to the existing, robust body of literature on the immediate drivers of stunting targeted by nutrition-specific interventions, we did not search for systematic reviews on these risk factors (Figure 1, highlighted in orange).

Systematic reviews and meta-analyses were included if they investigated the relationship between a selected risk factor and stunting. Studies were excluded if they (a) included fewer than two studies with child growth outcomes, (b) did not examine the isolated effect of the selected risk factor on child growth outcomes, or (c) investigated the effects of interventions on stunting. We aimed to identify studies that clearly quantify the impact of the risk factor on stunting and not allow the efficacy or effectiveness of one or more interventions to cloud the relationship.

We acknowledge that this brief does not comprehensively represent all of the available evidence on the many risk factors that contribute to stunting. Rather, our aim is to demonstrate the *range* of underlying, nutrition-sensitive factors that contribute to child growth, highlight the results of rigorous studies examining relevant interlinkages, and call attention to the need for further research.

Why systematic reviews and meta-analyses?

Systematic reviews are summaries of the results of multiple available studies on a given topic. Through a systematic search process, individual studies are identified, screened, and reviewed to generate combined knowledge about a particular relationship. Meta-analyses go a step further and employ statistical methods to synthesize data from multiple studies into a quantitative estimate of effect size.¹⁸

Due to the rigor of these types of studies and their ability to synthesize a vast amount of accumulating evidence, we have chosen to highlight the results of only systematic reviews and meta-analyses in this brief, as opposed to individual studies. However, it is important to note that systematic reviews have not been conducted for every relevant risk factor. As such, this brief does not reflect a comprehensive view of all available evidence on these varied topics.

FINDINGS

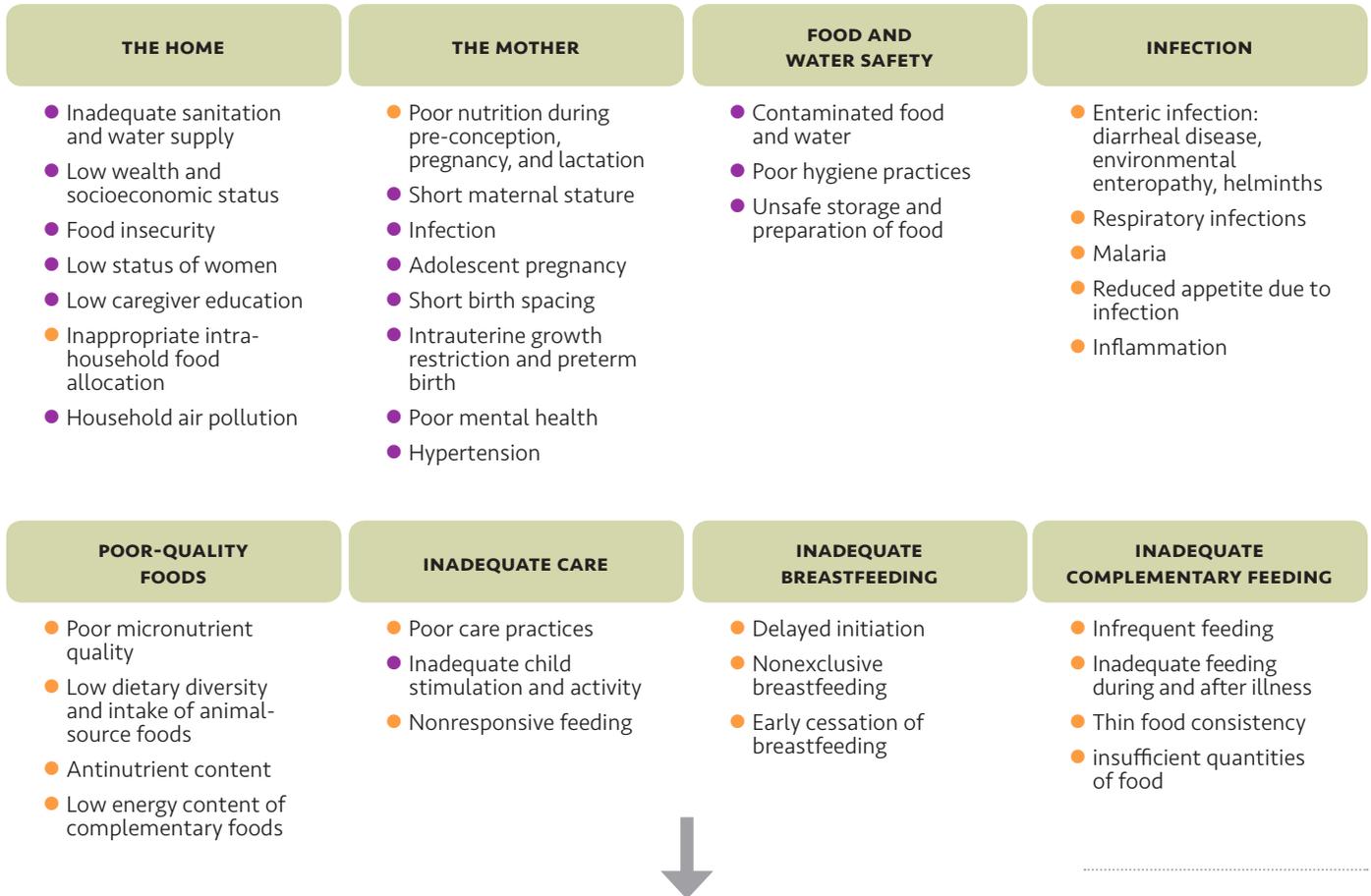
We identified systematic reviews on nutrition-sensitive risk factors that fell within the following three categories: the home (household factors), the mother (maternal factors), and infection-related factors. No systematic reviews were identified for risk factors within the categories of food and water safety or inadequate care. A summary of our search findings can be found in Tables 1, 2, and 3. Risk factors have been grouped in alignment with the World Health Organization framework shown in Figure 1.

FIGURE 1. Conceptual framework on context, causes, and consequences of stunting.

CONTEXT

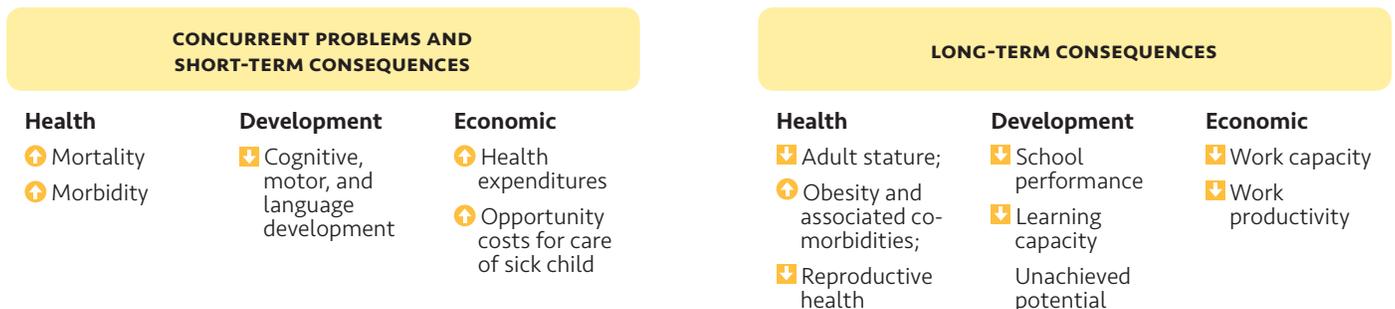


CAUSES



KEY
 ● Purple factors indicate underlying, nutrition-sensitive risk factors.
 ● Orange factors indicate immediate, nutrition-specific risk factors.
 ⬆ Increase ⬇ Decrease

CONSEQUENCES



Adapted from: World Health Organization, 2017, and Stewart et al., 2013.

Table 1. THE HOME

Inadequate sanitation and water supply, low wealth and socioeconomic status, food insecurity, low status of women, low caregiver education, inappropriate intra-household food allocation, air pollution*

Risk factor	Citation	Definition of risk factor	Study type	Effect estimate	Findings	# of studies	Quality of studies included
INADEQUATE SANITATION AND WATER SUPPLY							
Access to community-level sanitation	[19]	Community-level sanitation was defined as the proportion of households in the sampling area that have any sanitation facility, unimproved or improved.	Systematic review and meta-analysis	Adjusted OR=0.97 (95% CI 0.94, 1.00) p<0.05	Among children living in households with any sanitation access, living in communities with 100% sanitation access was associated with lower odds of stunting in comparison to communities with <30% sanitation access.	301	Not reported
		Household-level sanitation was categorized into: (1) any type of sanitation facility (unimproved, improved, or shared) or (2) no access.		Adjusted OR=1.04 (95% CI 1.02, 1.06) p<0.01	Among children living in households without any sanitation access, living in communities with 0% sanitation access was associated with higher odds of stunting in comparison to communities with 1%–30% sanitation access.		Not reported
Water supply	[20]	Water supply was examined if (1) source of drinking water was unimproved or (2) no access to potable drinking water.	Systematic review	N/A	Unimproved sources of drinking water were consistently associated with stunting.	10	Medium [†]
LOW WEALTH AND SOCIOECONOMIC STATUS							
Socioeconomic status	[21]	Socioeconomic status quintiles were created from a principal component analysis (PCA) using asset ownership and dwelling structure materials. Regression factor scores were generated from the PCA, ranked, and categorized into quintiles from (1) poorest to (5) least poor.	Systematic review and meta-analysis	p-value for test of trend p<0.01	Higher stunting prevalence rates were systematically and inversely associated with asset index quintiles.	16	Not reported
	[20]	Household wealth quintiles (poorest, poor, middle, rich, and richest) or three categories (low, middle, high) were created from household assets for most studies included.	Systematic review	N/A	Poor household wealth (low socioeconomic status) was consistently associated with stunting.	16	Medium [†]
	[22]	Household wealth quintiles (poorest, poor, middle, rich, and richest) were created from home ownership and quality, household assets, and access to services.	Systematic review and meta-analysis	OR=1.71 (95% CI 1.61, 1.81) p<0.05	Low socioeconomic status was a significant risk factor for child undernutrition and stunting.	2	High [‡]

[†] According to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist.

[‡] According to adapted criteria from the Newcastle-Ottawa Scale.

Table 1. THE HOME *continued*

Risk factor	Citation	Definition of risk factor	Study type	Effect estimate	Findings	# of studies	Quality of studies included
LOW CAREGIVER EDUCATION							
Maternal education	[21]	Maternal education was categorized as follows: no education, primary education only, or secondary education.	Systematic review and meta-analysis	p-value for test of trend p<0.005	Higher stunting prevalence rates were inversely associated with lower maternal education.	16	Not reported
	[22]	Maternal education was categorized as follows: no education, primary education only, or secondary education.	Systematic review and meta-analysis	OR=0.84 (95% CI 0.75, 0.93) p=0.53	Higher maternal education was associated with lower odds of stunting.	3	High ⁱⁱ
	[20]	Maternal education was categorized as follows: no education, primary education only, or secondary education. Alternative categories: primary or less, secondary or higher.	Systematic review	N/A	Low maternal education was consistently associated with child stunting.	22	Medium ⁱ
AIR POLLUTION							
Household air pollution	[23]	Exposure to household air pollution was defined as use of solid fuel for cooking.	Systematic review and meta-analysis	Moderate stunting OR=1.27 (95% CI 1.12, 1.43) p<0.0001	Exposure to household air pollution increased the odds of moderate childhood stunting.	2	Low ⁱ
				Severe stunting OR=1.55 (95% CI 1.04, 2.30) p<0.05	Exposure to household air pollution increased the odds of severe childhood stunting.	2	Very low ⁱⁱ

* The risk factors colored grey do not yet have systematic reviews published documenting their relationship with childhood stunting.
 CI= Confidence Interval
 OR= Odds Ratio
 N/A= Not Applicable

Table 2. THE MOTHER

Short maternal stature, short birth spacing, infection, adolescent pregnancy, intrauterine growth restriction and preterm birth, poor mental health, hypertension*

Risk factor	Citation	Definition of risk factor	Study type	Effect estimate	Findings	# of studies	Quality of studies included
SHORT MATERNAL STATURE							
Maternal height	[19]	Maternal height as measured in centimeters.	Systematic review and meta-analysis	OR= 0.99 (95% CI 0.98, 1.00) p<0.01	Maternal height is positively associated with child length and protective against stunting.	2	Medium to high ⁱⁱ

Table 2. THE MOTHER *continued*

Risk factor	Citation	Definition of risk factor	Study type	Effect estimate	Findings	# of studies	Quality of studies included
SHORT BIRTH SPACING							
Length of interpregnancy interval	[24]	<p>Birth interval and interpregnancy interval were used interchangeably.</p> <p>Interpregnancy interval: the time between birth of the previous child and conception of the index child.</p> <p>Previous birth interval: the time between birth of the previous child and birth of the index child.</p> <p>Subsequent birth interval: the time between birth of the index child and birth of the subsequent child.</p>	Systematic review	N/A	Longer birth intervals were inconsistently associated with a lower risk of malnutrition. A previous birth interval of ≥ 36 months was associated with 10%–50% reductions in stunting in countries where a significant relationship was observed.	44	Medium to high ⁱⁱⁱ
<p>ⁱⁱⁱ Quality scores were assigned on a scale of 1 (low quality) to 5 (high quality) based on the adequacy of control for the following key potentially confounding variables for child anthropometric outcomes: socioeconomic status and/or maternal education, parity, child age, maternal height, and breastfeeding of the index child.</p>							
INTRAUTERINE GROWTH RESTRICTION AND PRETERM BIRTH							
Small for gestational age	[25]	Size less than the 10th percentile for gestational age, compared with ≥ 10 th percentile for gestational age	Review and meta-analysis	<p>Adjusted OR=2.32 (95% CI 2.12, 2.54)</p> <p>RR= 1.76</p> <p>PAR= 0.16 (95% CI 0.12, 0.19)</p>	Small for gestational age was associated with overall increased odds of stunting in low- and middle-income countries.	19	Not reported
Preterm birth	[25]	Delivery at gestational age <37 weeks, compared with gestational age ≥ 37 weeks.	Review and meta-analysis	<p>Adjusted OR=1.69 (95% CI 1.48, 1.93)</p> <p>RR= 1.49</p> <p>PAR=0.04 (95% CI 0.03, 0.06)</p>	Preterm birth was associated with overall increased odds of stunting in low- and middle-income countries.	19	Not reported
POOR MENTAL HEALTH							
Maternal depression	[26]	Four studies used a diagnostic measure of depression based on either the Diagnostic and Statistical Manual of Mental Disorders or the International Classification of Diseases. Five studies used a measure of depressive symptoms (e.g., the Edinburgh Postnatal Depression Scale or the Center for Epidemiologic Studies Depression Scale). Eight studies used a measure of mental disorders (e.g., the World Health Organization Self-Reporting Questionnaire or the Adult Psychiatric Morbidity Questionnaire).	Systematic review and meta-analysis	<p>OR=1.4 (95% CI 1.2, 1.7)</p> <p>p<0.01</p>	There was a moderate, statistically significant relationship between maternal depression and stunting.	12	Varied ⁱⁱ

Maternal postpartum depression	[27]	Maternal depression was most frequently evaluated using the Edinburgh Postnatal Depression Scale, versions of the Center for Epidemiological Studies Depression Scale, and the Structured Clinical Interview for DSMIII.	Systematic review	N/A	Children <12 months	5	High ⁱⁱ
					All five studies reported a consistent, positive association between maternal depression and stunting in children at ages 3–12 months. Two studies found no effect at 9 and 12 months.		
					Children >12 months.	7	
					Maternal depression was associated with a higher chance of having a stunted child 2–5 years of age. Inconsistent results were observed for the association between maternal depression and standardized, continuous height-for-age.		

* The risk factors colored grey do not yet have systematic reviews published documenting their relationship with childhood stunting.

CI= Confidence Interval
OR= Odds Ratio
RR= Relative Risk
PAR= Population Attributable Risk
N/A= Not Applicable

Table 3. INFECTION

Enteric infection, respiratory infections, malaria, reduced appetite due to infection, inflammation*

Risk factor	Citation	Definition of risk factor	Study type	Effect estimate	Findings	# of studies	Quality of studies included
ENTERIC INFECTION: DIARRHEAL DISEASE, ENVIRONMENTAL ENTEROPATHY, AND HELMINTHS							
Environmental enteric dysfunction	[28]	Microbial translocation as indicated by the presence of anti-lipopolysaccharide and anti-flagellin antibodies.	Systematic review	N/A	Insufficient evidence linking microbial translocation and stunting within the limits of current tests.	9	Not reported
		Intestinal inflammation as indicated by an environmental enteric dysfunction composite score composed of three fecal biomarkers: myeloperoxidase, neopterin, and alpha-1 antitrypsin.			Evidence linking intestinal inflammation to linear growth and stunting.	10	
		Intestinal damage and repair: circulating concentrations of citrulline, intestinal fatty acid binding protein, fecal-regenerating family proteins, and glucagon-like peptide 2.			Conflicting evidence of a link between the biomarkers of intestinal damage and repair and child stunting.	7	
Environmental enteric dysfunction	[28]	Permeability and absorption as indicated by a higher lactulose-mannitol or lactulose-rhamnose ratio and alpha-1 antitrypsin, claudin, and zonulin proteins.	Systematic review	N/A	No clear relationship between permeability/absorption and linear growth as the evidence conflicts.	17	Not reported

		Systemic inflammation as indicated by cytokines, acute-phase proteins, amino acids, and soluble CD14.			Evidence linking systemic inflammation and stunting.	10	
Exposure to animal feces	[29]	Four definitions were given: (1) observation of animal feces in the environment, (2) presence of animals or contact with animals, (3) poultry inside, and (4) animals inside.	Systematic review	N/A	The presence of animal feces within household compounds was negatively associated with child height-for-age Z score. Specifically, corralling chickens inside the home at night increased risk.	4	Not reported

*The risk factors colored grey do not yet have systematic reviews published documenting their relationship with childhood stunting.
N/A=Not Applicable

DISCUSSION

Systematic reviews and meta-analyses are powerful tools to synthesize the body of evidence on stunting risk factors. More of these rigorous research efforts are needed to fill critical gaps in the literature, especially with respect to infection (e.g., diarrheal disease, respiratory infections, malaria, reduced appetite due to infection, and inflammation) and inadequate care (e.g., inadequate child stimulation and activity). Understanding the most significant determinants of linear growth failure and stunting will help to set priorities in intervention and policy design and implementation.

Household risk factors

The systematic reviews on household risk factors indicate that a clean, stable home environment with an educated, empowered caregiver is essential for a child's health and well-being. Universal access to sanitation may protect children from the fecal-oral transmission of pathogens, but this costly intervention yields only a small reduction in stunted growth.¹⁹ Further evidence from the water, sanitation, and hygiene (WASH) sector has recently confirmed that while nutrition interventions are capable of modestly improving growth, WASH interventions do not provide an added benefit to linear growth.^{30,31}

Low socioeconomic status is a significant risk factor for child undernutrition and stunting.²⁰⁻²² Social protection strategies have become increasingly popular to address poverty and social vulnerability.¹⁷ Today, more than 700 million people in low- and middle-income countries (LMICs) are estimated to have access to cash transfer (CT) programs. While these programs may positively impact many of the outcomes along the pathway to improved nutrition (e.g., reduction in poverty, increase in household food consumption and dietary diversity), they have limited ability to impact linear growth.³³⁻³⁵ Therefore, CT programs must be combined with additional nutrition and behavior change components.³⁶ The exact pathway by which CT programs impact nutritional status remains unclear.

There is still a need for evidence-based poverty alleviation and economic development interventions that can impact child growth.

Reviews by Wamani et al.,²¹ Abdulahi et al.,²² and Akombi et al.²⁰ all found an association between higher maternal education and lower risk of childhood stunting. To improve maternal education levels, sustained policy and programmatic investments should focus on educating adolescent girls and organizing community education programs to improve the quality of childcare practices and create healthier environments. While no systematic reviews are published on the link between maternal status and child nutritional status, multiple studies have found that women with higher levels of decision-making power and control over nutrition-related household decisions are less likely to have stunted children.³⁷⁻³⁹

Although interventions such as improvements in cooking stoves and fuels^{23,40-42} and housing ventilation could significantly improve child growth outcomes, further high-quality evidence is required on the linkages between household air pollution exposure and growth outcomes to increase confidence regarding the causal effect.

Maternal risk factors

The systematic reviews of maternal risk factors underscore the importance of intervening early, during the first 1,000-day window, to prevent child stunting. The findings of Christian et al.²⁵ highlight the value of interventions aimed at promoting healthy pregnancies and reducing fetal growth restriction and preterm birth. Birth spacing and age at first pregnancy are important predictors of healthy pregnancies, with implications for linear growth. The relationship between birth spacing and small for gestational age (SGA), a precursor for stunting, has also been explored.^{43,44} One meta-analysis reported that birth intervals of less than 18 months yielded significantly increased odds of SGA (pooled aOR: 1.51; 95 percent CI 1.31, 1.75),⁴³ suggesting an association between longer birth intervals



PATH/Gabe Bienczycki

and a reduced stunting risk. Further research with more comprehensive control of confounding factors is needed to better understand the relationship between birth spacing and stunting. Although systematic reviews have not yet evaluated the relationship between adolescent pregnancy and stunting, one meta-analysis⁴⁵ found that nulliparous women younger than 18 carried the highest risk of adverse neonatal outcomes, such as SGA, compared to other groups.

Maternal stature plays an important role in child growth outcomes. Although we identified only one systematic review examining the association between maternal stature and stunting in Ethiopia,²² other evidence exists on this relationship. One pooled analysis of 109 Demographic and Health Surveys from 54 countries found that a 1 centimeter increase in maternal height was associated with a significantly decreased risk of stunting (RR 0.97; 95 percent CI 0.97, 0.97).⁹

Finally, systematic reviews by both Surkan et al.²⁶ and Farías-Antúnez et al.²⁷ found that children of mothers suffering from depression face an increased risk of stunting. Interventions to treat maternal depression would benefit both mothers and their children, particularly during the first year of life.²⁷

Together, these reviews demonstrate the deep interconnections between maternal and child health, particularly given the intergenerational nature of growth faltering. Multisectoral interventions supporting the health and well-being of both mothers and children before, during, and after pregnancy can have resonating impacts that are mutually reinforcing across the life cycle. Accordingly,

global efforts to reduce stunting should focus not only on the child and more upstream, community-level and environmental risk factors, but also on evidence-based interventions to promote maternal and reproductive health.

Infection-related risk factors

Published evidence to date on infection-related risk factors shows a relationship between child growth and subclinical infections that contribute to nutrient malabsorption and impaired gut function. These infections stem from exposure to environmental contamination and poor hygiene practices. Exposure to animal waste also negatively affects child growth, yet many families in LMICs cohabit with livestock. Guidance on best practices for livestock management and housing must be incorporated into nutrition programs to prevent contamination of the air, water, and soil within the family's living space.

Environmental enteric dysfunction (EED), or chronic inflammation and dysfunction of the small bowel, is also linked to reduced child growth. Evidence now supports the relationships between intestinal inflammation and stunting, and systematic inflammation and stunting.²⁸ Historically, identifying EED within vulnerable populations has been difficult due to the absence of robust, established biomarkers. Almost half of the studies included in Harper et al.²⁸ used diarrhea as the case definition for EED, yet many EED cases are asymptomatic.⁴⁶ As this field continues to evolve, researchers are developing a standardized set of biomarkers to clarify the relationships between EED domains and stunting.



PATH/Evelyn Hockstein

No systematic reviews were identified on the many other infection-related risk factors, such as diarrhea, helminth infection, or malaria. However, evidence does exist on these relationships. One multicountry analysis concluded that a child's odds of stunting by 24 months of age significantly increased with every five episodes of diarrhea (aOR=1.13; 95 percent CI 1.07, 1.19). Frequent infections reduce the likelihood of catch-up growth. Researchers have also found a direct link between increased risk of stunting and diarrheal infection due to *Cryptosporidium*, *Campylobacter*, and enteroaggregative *Escherichia coli*.⁴⁸⁻⁵¹ Further, certain risk factors, such as parasitic infection, can be difficult and unethical to study in the long-term, making it impossible to quantify associated height deficits. Regardless, deworming interventions are recommended by the World Health Organization and provide many benefits to child health, even though systematic reviews have not consistently documented their impact on linear growth.⁵²⁻⁵⁵ Finally, recent studies have identified malaria as a significant risk factor for stunting, but the only (non-systematic) review conducted to date could not conclusively list malaria as a determinant of stunting or quantify the impact of malarial interventions on stunting.⁵⁶

In sum, while evidence-based approaches to prevent and treat infections are critical to ensuring optimal child growth and development, further research is needed to better understand the complex relationships between subclinical infections and linear growth. The body of literature in this area is rapidly evolving. Improved

understanding of interventions to prevent and treat EED and the bacterial and parasitic infections that contribute to stunting is expected to have substantial impact in the coming decades.

CONCLUSION

Stunting remains a difficult indicator to improve within the limited time frame of typical impact assessments. We still have a lot to learn about what works (and what does not) to move the needle on this complex indicator of growth and development. Knowing which risk factors to target for greatest impact, and which interventions most effectively target those risk factors, will further our progress. Successful interventions will need to target multiple risk factors. At this time, however, it is not always clear which dimensions of each risk factor are best targeted due to the complexity of implementing and evaluating multi-sectoral interventions.

Although many of the reviews included in this technical brief focus on maternal and household risk factors for stunting, child stunting is reflective of broader, social and community-level challenges, many of which reside outside of the control of individual caregivers.⁷ It is clear that sustainable solutions for childhood stunting will not arise from single-sector approaches. Nutrition-specific interventions should be supported and reinforced by nutrition-sensitive programs that target the many underlying risk factors for stunting.

We emphasize the urgent need for multisectoral, integrated approaches that target not only the child during the first 1,000 days of life, but also women and mothers, families, communities, and the environments we all share.

References

1. UNICEF, World Health Organization (WHO), World Bank Group. *Levels and Trends in Child Malnutrition: UNICEF / WHO / World Bank Group: Joint Child Malnutrition Estimates: Key Findings of the 2017 Edition*. New York, Geneva, Washington, DC; UNICEF, WHO, World Bank Group; 2017. Available at <https://data.unicef.org/wp-content/uploads/2017/05/JME-2017-brochure.pdf>.
2. Victora CG, de Onis M, Hallal PC, Blössner M, Shrimpton R. Worldwide timing of growth faltering: revisiting implications for interventions. *Pediatrics*. 2010;125(3). Available at <http://pediatrics.aappublications.org/content/125/3/e473.long>.
3. United Nations Sustainable Development Knowledge Platform website. Sustainable Development Goal 2 page. Available at <https://sustainabledevelopment.un.org/sdg2>. Accessed February 9, 2018.
4. World Health Organization (WHO). *WHA Global Nutrition Targets 2025: Stunting Policy Brief*. Geneva: WHO; 2014. Available at http://www.who.int/nutrition/topics/globaltargets_stunting_policybrief.pdf.
5. World Health Organization (WHO). *WHO Child Growth Standards: Length/Height-for-Age, Weight-for-Age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-Age: Methods and Development*. Geneva: WHO; 2006. Available at http://www.who.int/childgrowth/standards/Technical_report.pdf?ua=1.
6. de Onis M, Branca F. Childhood stunting: a global perspective. *Maternal & Child Nutrition*. 2016;12(Suppl 1):12-26. Available at <https://doi.org/10.1111/mcn.12231>.
7. Perumal N, Bassani DG, Roth DE. Use and misuse of stunting as a measure of child health. *The Journal of Nutrition*. 2018;148(3):311-315. Available at <https://academic.oup.com/jn/article/148/3/311/4930811>.

8. Black RE, Victora CG, Walker SP, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. *The Lancet*. 2013;382(9890):427–451. Available at [http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736\(13\)60937-X.pdf](http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(13)60937-X.pdf).
9. Özaltin E, Hill K, Subramanian SV. Association of maternal stature with offspring mortality, underweight, and stunting in low- to middle-income countries. *Journal of the American Medical Association*. 2010;303(15):1507–1516. Available at <https://jamanetwork.com/journals/jama/fullarticle/185712>.
10. Victora CG, Adair L, Fall C, et al. Maternal and child undernutrition: consequences for adult health and human capital. *The Lancet*. 2008;371(9609):340–357. Available at [http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736\(07\)61692-4.pdf](http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(07)61692-4.pdf).
11. Prendergast AJ, Humphrey JH. The stunting syndrome in developing countries. *Paediatrics and International Child Health*. 2014;34(4):250–265. Available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4232245/>.
12. Stewart CP, Ianotti L, Dewey KG, Michaelsen KF, Onyango AW. Contextualizing complementary feeding in a broader framework for stunting prevention. *Maternal & Child Nutrition*. 2013;9(Suppl 2): 27–45. Available at <https://doi.org/10.1111/mcn.12088>.
13. Martorell R, Zongrone A. Intergenerational influences on child growth and undernutrition. *Paediatric and Perinatal Epidemiology*. 2012;26(Suppl 1):302–314. Available at <https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1365-3016.2012.01298.x>.
14. Danaei G, Andrews KG, Sudfeld CR, et al. Risk factors for childhood stunting in 137 developing countries: a comparative risk assessment analysis at global, regional, and country levels. *PLoS Medicine*. 2016;13(11):e1002614. Available at <http://journals.plos.org/plosmedicine/article?id=10.1371/journal.pmed.1002614>.
15. World Health Organization (WHO). Stunted Growth and Development: Context, Causes, and Consequences. Geneva: WHO; 2017. Available at http://www.who.int/nutrition/childhood_stunting_framework_leaflet_en.pdf?ua=1.
16. Bhutta ZA, Das JK, Rizvi A, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *The Lancet*. 2013;382(9890):452–477. Available at [http://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(13\)60996-4/abstract](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(13)60996-4/abstract).
17. Ruel MT, Alderman H, the Maternal and Child Nutrition Study Group. Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? *The Lancet*. 2013;382(9891):536–551. Available at [http://www.thelancet.com/journals/lancet/article/PIIS0140-6736\(13\)60843-0/abstract](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(13)60843-0/abstract).
18. Uman LS. Systematic reviews and meta-analyses. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*. 2011;20(1):57–59. Available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3024725/>.
19. Larsen DA, Grisham T, Slawsky E, Narine L. An individual-level meta-analysis assessing the impact of community-level sanitation access on child stunting, anemia, and diarrhea: evidence from DHS and MICS surveys. *PLoS Neglected Tropical Diseases*. 2017;11(6):e0005591. Available at <https://doi.org/10.1371/journal.pntd.0005591>.
20. Akombi BJ, Agho KE, Hall JJ, Wali N, Renzaho AMN, Merom D. Stunting, wasting and underweight in sub-Saharan Africa: a systematic review. *International Journal of Environmental Research and Public Health*. 2017;14(8):863. Available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5580567/>.
21. Wamani H, Åström AN, Peterson S, Tumwine JK, Tylleskär T. Boys are more stunted than girls in Sub-Saharan Africa: a meta-analysis of 16 demographic and health surveys. *BMC Pediatrics*. 2007;7(17):1–10. Available at <https://bmcpediatr.biomedcentral.com/articles/10.1186/1471-2431-7-17>.
22. Abdulahi A, Shab-Bidar S, Rezaei S, Djafarian K. Nutritional status of under five children in Ethiopia: a systematic review and meta-analysis. *Ethiopian Journal of Health Sciences*. 2017;27(2):175–188. Available at <http://dx.doi.org/10.4314/ejhs.v27i2.10>.
23. Bruce NG, Dherani MK, Das JK, et al. Control of household air pollution for child survival: estimates for intervention impacts. *BMC Public Health*. 2013;13(Suppl 3): S8. Available at <https://bmcpublichealth.biomedcentral.com/articles/10.1186/1471-2458-13-S3-S8>.
24. Dewey KG, Cohen RJ. Does birth spacing affect maternal or child nutritional status? A systematic literature review. *Maternal and Child Nutrition*. 2007;3(3):151–173. Available at <https://doi.org/10.1111/j.1740-8709.2007.00092.x>.
25. Christian P, Lee SU, Donahue Angel M, et al. Risk of childhood undernutrition related to small-for-gestational age and preterm birth in low- and middle-income countries. *International Journal of Epidemiology*. 2013;42(5):1340–1355. Available at <https://www.ncbi.nlm.nih.gov/pubmed/23920141>.
26. Surkan PJ, Kennedy CE, Hurley KM, Black MM. Maternal depression and early childhood growth in developing countries: systematic review and meta-analysis. *Bulletin of the World Health Organization*. 2011;89:608–615E. Available at <http://www.who.int/bulletin/volumes/89/8/11-088187/en/>.
27. Fariás-Antúnez S, Xavier MO, Santos IS. Effect of maternal postpartum depression on offspring's growth. *Journal of Affective Disorders*. 2018;228:143–152. Available at <https://doi.org/10.1016/j.jad.2017.12.013>.
28. Harper KM, Mutasa M, Prendergast AJ, Humphrey J, Manges AR. Environmental enteric dysfunction pathways and child stunting: a systematic review. *PLoS Neglected Tropical Diseases*. 2018;12(1):1–23. Available at <https://doi.org/10.1371/journal.pntd.0006205>.
29. Penakalapati G, Swarthout J, Delahoy MJ, et al. Exposure to animal feces and human health: a systematic review and proposed research priorities. *Environmental Science & Technology*. 2017;51(20):11537–11552. Available at <https://pubs.acs.org/doi/abs/10.1021/acs.est.7b02811>.
30. Luby SP, Rahman M, Arnold BF, et al. Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Bangladesh: a cluster randomised controlled trial. *The Lancet Global Health*. 2018;6(3):e302–e315. Available at [https://doi.org/10.1016/S2214-109X\(17\)30490-4](https://doi.org/10.1016/S2214-109X(17)30490-4).
31. Null C, Stewart CP, Pickering AJ, et al. Effects of water quality, sanitation, handwashing, and nutritional interventions on diarrhoea and child growth in rural Kenya: a cluster-randomised controlled trial. *Lancet Global Health*. 2018;6(3):e316–e329. Available at [https://doi.org/10.1016/S2214-109X\(18\)30005-6](https://doi.org/10.1016/S2214-109X(18)30005-6).
32. Honorati M, Gentilini U, Yetmtsov RG. *The State of Social Safety Nets 2015*. Washington, D.C.: World Bank Group; 2015. Available at <http://documents.worldbank.org/curated/en/415491467994645020/The-state-of-social-safety-nets-2015>.
33. de Groot R, Palermo T, Handa S, Ragno LP, Peterman A. Cash transfers and child nutrition: pathways and impacts. *Development Policy Review*. 2017;35(5):621–643. Available at <https://doi.org/10.1111/dpr.12255>.
34. Manley J, Gitter S, Slavchevska V. How effective are cash transfers at improving nutritional status? *World Development*. 2013;48:133–155. Available at <https://doi.org/10.1016/j.worlddev.2013.03.010>.
35. Manley J, Slavchevska V. Are cash transfers the answer for children in sub-Saharan Africa? A literature review. Working Paper No. 2016-12. Towson: Towson University Department of Economics; 2016. Available at <http://webapps.towson.edu/cbe/economics/workingpapers/2016-12.pdf>.
36. Roelen K, Devereux S, Abdulai AG, et al. *How to Make 'Cash Plus' Work: Linking Cash Transfers to Services and Sectors*. 2017. Innocenti Working Paper 2017-10. Florence: UNICEF Office of Research; 2017. Available at <https://www.unicef-irc.org/publications/pdf/IDS%20WP%20CORRECTED%20Sept%202017.pdf>.
37. Taukobong HF, Kincaid MM, Levy JK, et al. Does addressing gender inequalities and empowering women and girls improve health and development programme outcomes? *Health Policy and Planning*. 2016;31(10):1492–1514. Available at <https://doi.org/10.1093/heapol/czw074>.
38. Shroff M, Griffiths P, Adair L, Suchindran C, Bentley M. Maternal autonomy is inversely related to child stunting in Andhra Pradesh, India. *Maternal and Child Nutrition*. 2009;5(1):1–16. Available at <https://doi.org/10.1111/j.1740-8709.2008.00161.x>.
39. Smith LC, Ramakrishnan U, Ndiaye A, Haddad L, Martorell R. *The Importance of Women's Status for Child Nutrition in Developing Countries*. Research Report 131. Washington, D.C.: International Food Policy Research Institute; 2003. Available at <https://ageconsearch.umn.edu/bitstream/16526/1/r030131.pdf>.
40. Mishra V, Retherford RD. Does biofuel smoke contribute to anaemia and stunting in early childhood? *International Journal of Epidemiology*. 2007;36(1):117–129. Available at <https://doi.org/10.1093/ije/dyl234>.
41. Kyu HH, Georgiades K, Boyle MH. Maternal smoking, biofuel smoke exposure and child height-for-age in seven developing countries. *International Journal of Epidemiology*. 2009;38(5):1342–1350. Available at <https://doi.org/10.1093/ije/dyp253>.
42. Tielsch JM, Katz J, Thulasiraj RD, et al. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India. *International Journal of Epidemiology*. 2009;38(5):1351–1363. Available at <https://doi.org/10.1093/ije/dyp286>.
43. Kozuki N, Lee AC, Silveira MF, et al. The associations of birth intervals with small-for-gestational-age, preterm, and neonatal and infant mortality: a meta-analysis. *BMC Public Health*. 2013;13(Suppl 3):S3. Available at <https://doi.org/10.1186/1471-2458-13-S3-S3>.
44. Conde-Agudelo A, Rosas-Bermúdez A, Kafury-Goeta AC. Birth spacing and risk of adverse perinatal outcomes: a meta-analysis. *JAMA*. 2006;295(15):1809–1823. Available at <https://jamanetwork.com/journals/jama/article-abstract/202711?redirect=true>.
45. Kozuki N, Lee ACC, Silveira MF, et al. The associations of parity and maternal age with small-for-gestational age, preterm, and neonatal and infant mortality: a meta-analysis. *BMC Public Health*. 2013;13(Suppl 3): S2. Available at <https://doi.org/10.1186/1471-2458-13-S3-S2>.

46. Guerrant RL, Leite AM, Pinkerton R, et al. Biomarkers of environmental enteropathy, inflammation, stunting, and impaired growth in children in northeast Brasil. *PLoS One*. 2016;11(9):e0158772. Available at <https://doi.org/10.1371/journal.pone.0158772>.
47. Checkley W, Buckley G, Gilman RH, et al. Multi-country analysis of the effects of diarrhoea on childhood stunting. *International Journal of Epidemiology*. 2008;37(4):816–830. Available at <https://doi.org/10.1093/ije/dyn099>.
48. Amour C, Gratz J, Mduma E, et al. Epidemiology and impact of *Campylobacter* infection in children in 8 low-resource settings: results from the MAL-ED study. *Clinical Infectious Diseases*. 2016;63(9):1171–1179. Available at <https://doi.org/10.1093/cid/ciw542>.
49. Checkley W, Epstein LD, Gilman RH, Black RE, Cabrera L, Sterling CR. Effects of *Cryptosporidium parvum* infection in Peruvian children: growth faltering and subsequent catch-up growth. *American Journal of Epidemiology*. 1998;148(5):497–506. Available at <https://www.ncbi.nlm.nih.gov/pubmed/9737562>.
50. Kirkpatrick BD, Noel F, Rouzier PD, et al. Childhood cryptosporidiosis is associated with a persistent systemic inflammatory response. *Clinical Infectious Diseases*. 2006;43(5):604–608. Available at <https://doi.org/10.1086/506565>.
51. Rogawski ET, Guerrant RL, Havt A, et al. Epidemiology of enteroaggregative *Escherichia coli* infections and associated outcomes in the MAL-ED birth cohort. *PLoS Neglected Tropical Diseases*. 2017;11(7):e0005798. Available at <https://doi.org/10.1371/journal.pntd.0005798>.
52. Welch VA, Ghogomu E, Hossain A, et al. Mass deworming to improve developmental health and wellbeing of children in low-income and middle-income countries: a systematic review and network meta-analysis. *The Lancet Global Health*. 2017;5(1):e40–e50. Available at [https://doi.org/10.1016/S2214-109X\(16\)30242-X](https://doi.org/10.1016/S2214-109X(16)30242-X).
53. Taylor-Robinson DC, Maayan N, Soares-Weiser K, Donegan S, Garner P. Deworming drugs for soil-transmitted intestinal worms in children: effects on nutritional indicators, haemoglobin, and school performance. *Cochrane Database Systematic Review*. 2015;23(7):CD000371. Available at <https://www.ncbi.nlm.nih.gov/pubmed/26202783>.
54. Hall A, Hewitt G, Tuffrey V, de Silva N. A review and meta-analysis of the impact of intestinal worms on child growth and nutrition. *Maternal & Child Nutrition*. 2008;4(Suppl 1):118–236. Available at <https://doi.org/10.1111/j.1740-8709.2007.00127.x>.
55. World Health Organization (WHO). *Deworming in Children*. Geneva: WHO; 2018. Available at <http://www.who.int/elena/titles/deworming/en/>.
56. Jackson BD, Black RE. A literature review of the effect of malaria on stunting. *Journal of Nutrition*. 2017;147(11):2163S–2168S. Available at <https://doi.org/10.3945/jn.116.242289>.
57. Ruel MT, Quisumbing AR, Balagamwala M. Nutrition-sensitive agriculture: what have we learned so far? *Global Food Security*. In press. Available at <https://doi.org/10.1016/j.gfs.2018.01.002>



This work was funded by an educational grant from the Sight and Life Foundation. Sight and Life is a humanitarian nutrition think tank working to innovate in nutrition towards eradicating all forms of malnutrition in children and women of childbearing age and so improve the lives of the world's most vulnerable populations.



www.path.org

PATH is the leader in global health innovation. An international nonprofit organization, we save lives and improve health, especially among women and children. We accelerate innovation across five platforms—vaccines, drugs, diagnostics, devices, and system and service innovations—that harness our entrepreneurial insight, scientific and public health expertise, and passion for health equity. By mobilizing partners around the world, we take innovation to scale, working alongside countries primarily in Africa and Asia to tackle their greatest health needs. Together, we deliver measurable results that disrupt the cycle of poor health. Learn more at www.path.org.

STREET ADDRESS
2201 Westlake Avenue
Suite 200
Seattle, WA 98121 USA

MAILING ADDRESS
PO Box 900922
Seattle, WA 98109 USA