

Beyond wasted and stunted—a major shift to fight child undernutrition

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Child undernutrition refers broadly to the condition in which food intake is inadequate to meet a child's needs for physiological function, growth, and the capacity to respond to illness. Since the 1970s, nutritionists have categorised undernutrition in two major ways, either as wasted (i.e., low weight for height, or small mid-upper arm circumference) or stunted (i.e., low height for age). This approach, although useful for identifying populations at risk of undernutrition, creates several problems: the focus is on children who have already become undernourished, and this approach draws an artificial distinction between two idealised types of undernourished children that are widely interpreted as indicative of either acute or chronic undernutrition. This distinction in turn has led to the separation of programmatic approaches to prevent and treat child undernutrition. In the past 3 years, research has shown that individual children are at risk of both conditions, might be born with both, pass from one state to the other over time, and accumulate risks to their health and life through their combined effects. The current emphasis on identifying children who are already wasted or stunted detracts attention from the larger number of children undergoing the process of becoming undernourished. We call for a major shift in thinking regarding how we assess child undernutrition, and how prevention and treatment programmes can best address the diverse causes and dynamic biological processes that underlie undernutrition.

The problem framed

Child undernutrition refers broadly to the condition in which food intake does not meet the needs of physiological function, growth, and the capacity to respond to illness. Despite substantial progress in preventing and treating child undernutrition in the past three decades, approximately 5 million annual deaths in children younger than 5 years occur worldwide,¹ of which nutrition-associated factors still account for almost half of these deaths.² In the longer term, child undernutrition is associated with adverse effects on health and human capital.³

The overarching strategy to address these issues is to bring together nutrition-specific interventions, targeting both mothers and their offspring in populations at risk, with nutrition-sensitive approaches that address broader underlying factors such as women's empowerment, food systems, socioeconomic factors, and disease prevention.⁴ These efforts are central to Sustainable Development Goal 2.2, which aims to end all forms of malnutrition by 2030, and to reach internationally agreed targets on stunting and wasting in children under 5 years by 2025.⁵ Our focus in this Viewpoint is on how the efforts to meet this goal are shaped by the way that agencies conceptualise undernutrition at the level of the individual child.

Since the 1970s, nutritionists have categorised under-nutrition in two major ways. Children are defined as wasted and in need of treatment if they have a low weight-for-height or small mid-upper arm circumference, which are taken to indicate acute undernutrition. Children are defined as stunted if they have a low height-for-age, which is taken to indicate chronic undernutrition. Children classified as underweight have a low weight-for-age, which can be due to wasting or stunting, or both, so the index of undernutrition is composite. These markers are widely used to assess child undernutrition at the population level and a high prevalence of children who are wasted or stunted is considered a public health problem.⁶ At the level of programmatic design and interventions, however, the two categories of under-nutrition are approached very differently.

Wasted children have a high risk of dying, which can often be rapidly reduced by nutritional therapy.⁷ Making therapy available is thus considered crucial to prevent deaths associated with child wasting. Conversely, children categorised as stunted have had poor growth in height over long periods, including foetal development. This growth faltering is not amenable to rapid nutritional correction and is therefore considered to require prevention rather than treatment.⁶ We argue that these views have become entrenched, leading to the separation of these outcomes in terms of policy, guidance, programme interventions, and financing: at the individual level, acute and chronic undernutrition are now viewed as separate conditions, and are routinely reported as distinct outcomes among policy makers.⁸

What is poorly recognised is that the anthropometrical indices used to categorise individual children as wasted and stunted are only superficial proxies for the physiological and functional consequences of the underlying processes of undernutrition. In this sense, anthropometry is analogous to a sign, reflecting a range of possible causative factors acting on individuals and societies. We argue that reliance on anthropometry leads to a spurious dichotomisation and categorisation of child undernutrition, and this problem is propagated to the population level when describing the prevalence of children who are wasted or stunted. Not only does this approach give a profoundly misleading representation of the complex causality of undernutrition, it also needlessly narrows the approaches to its prevention and treatment.

Here, we review research highlighting the fundamental common ground between weight faltering and linear growth faltering. This evidence underpins our call for a shift in how we prevent these processes from leading to detrimental effects for children, their families, and communities.

New evidence

Inadequate weight gain, weight loss, and linear growth faltering are all the result of multiple processes in which the body responds to diverse causes acting on both individuals and societies.⁹ The causes that act on individuals include poor growth in utero, dietary inadequacy, infectious diseases, and intestinal inflammation or dysfunction,^{10,11} although the relative importance of these causes varies by individual and setting.¹² The causes that act on societies include poverty, disempowerment, food insecurity, poor health services, and poor facilities for water, sanitation, and hygiene (WASH). The rate at which children become wasted and stunted often differs, in part because weight can be lost whereas height cannot. However, both conditions might already be present at birth and persist concurrently over time, hence the notion that the two categories reflect acute versus chronic undernutrition separately is incorrect.

As the processes of becoming wasted and stunted have common causes, the two forms are expected to often occur in the same child, a situation termed concurrence. Yet this issue has only attracted attention among the international nutrition community since 2017. The first study¹³ formally to address this issue analysed data on children between 6 months and 59 months in 84 countries and reported a pooled prevalence of concurrence of 3·0%, ranging from 0% to 8·0% in individual countries. This pooled data analysis was followed by an analysis estimating that 15·9 million children worldwide are affected by concurrence.⁸ Our reanalysis^{14,15} of data on survival shows that mortality risk for children concurrently wasted and stunted is equal to that of the most severe form of being wasted. Several different factors might underlie this elevated mortality risk.

First, increasing evidence exists of temporal links between the two manifestations of undernutrition. Findings from a longitudinal study¹⁶ of children in the Gambia support earlier research that showed that being wasted increases the risk of subsequently becoming stunted.¹⁷ These associations indicate that the body responds to weight faltering by slowing linear growth, and so children affected become more susceptible to subsequent periods of weight faltering than those who do not experience height faltering.¹⁶ Evidence also indicates that seasonal stresses, perhaps related to food and nutrient availability or infections, might underlie the temporal connections between weight faltering and linear growth faltering. For example, several studies have shown that new cases of children categorised as stunted occur seasonally, following peaks in those categorised as wasted.^{16,18} Therefore, weight faltering seems to represent a more malleable, short-term response to nutritional stress than linear growth faltering. These dynamic associations have implications for prevention and treatment strategies.

Second, both weight faltering and linear growth faltering are associated with detrimental effects on body composition. As with the loss of muscle mass during severe weight loss, stunted children show persistent deficits in lean mass, including small organ size¹⁹ and lower muscle mass, which raises mortality risk during infections.²⁰

Furthermore, children who are wasted or stunted have lower fat mass,^{19,21,22} which might harm systemic immune function through the mediating effects of the hormone leptin. Because leptin also stimulates bone growth, this effect might explain why children who are wasted have slow linear growth.²⁰ These organ and tissue deficits might therefore represent a common mechanism linking weight and height faltering with increased risk of dying. In the short term, weight and height deficits respond differently to current nutritional treatment strategies. Intensive nutritional treatment of children who are wasted, most of whom are also stunted, leads to rapid weight gain, but has been widely shown to have little immediate effect on linear growth.²³ However, over a longer period of several years, some recovery in the height of previously wasted children has been shown,²⁴ although the magnitude of this recovery might reflect a degree of survivor bias.

These different responses led to concern that the intensive nutritional treatment for children who are wasted might promote excessive fat accretion in stunted children, placing them at long-term risk of overweight or obesity and non-communicable disease. However, excessive fat accretion does not seem to accompany standard treatment regimens with highly fortified foods. A study²¹ of supplementary food given to young, undernourished children found that an average of 93% of weight gained over 3 months comprised lean tissue, and further a analysis²² showed that short children showed no tendency to gain more fat than children who were not short. These findings are consistent with a similar study²⁵ of therapeutic intervention. Collectively, these studies suggest that nutritional therapy for children who are wasted need not be altered if the child is also stunted.

Other interventions have also been tested, for example targeting environmental conditions such as WASH. In severely undernourished children in Chad, adding a household WASH intervention to an outpatient therapeutic feeding programme reduced the time required for recovery and increased the recovery rate.²⁶ However, community-based WASH interventions have had minimal success in preventing linear growth faltering, and improved infant and young child feeding practices appear to be more effective targets for intervention than WASH.^{27–29}

What needs to change?

On the basis of the evidence described, we believe that now is the time for a major change in how childhood undernutrition is understood and managed.

Approaching weight and linear growth faltering as separate problems does not recognise the fact that individual children are at risk of both conditions, might be born with both, pass from one state to the other over time, and accumulate risks to their health and life through their combined effects. Moreover, the emphasis on identifying children once they are defined as having one or other form of undernutrition detracts crucial attention from the much larger number of children who are in the process of becoming undernourished, and in whom harmful effects of the causes of wasting and stunting are already present.³⁰

We call for change in five areas: (1) focus more research and practice on the concurrent and dynamic biological processes and pathways that underlie the entire spectrum of child weight and linear growth faltering; (2) develop innovative and early markers to predict, identify, and monitor children at short-term and long-term risk of weight and linear growth faltering; (3) research maternal factors from adolescence through pregnancy that effect in utero and postnatal child weight and linear growth faltering; (4) evaluate preventive interventions, universal and seasonal, for children at risk of weight or linear growth faltering, including in countries affected by protracted crisis; and (5) review and adjust therapeutic interventions to ensure that the children at highest mortality risk, including as a result of dual weight and height deficits, are included.

We believe that a major shift is needed to drive substantial improvements in the efforts to successfully prevent and treat child undernutrition. To achieve these aims, we call for additional research to gain a better understanding of the synergy and temporal relationships between weight and linear growth faltering, and how this interaction affects the risk of morbidity and mortality. We also call for greater recognition of the need to target the distal factors that drive undernutrition, which are essential to reduce its manifestation. Given that child undernutrition is often established by the time a child is born, our approach might facilitate greater coordination between interventions targeting adolescent girls and mothers, and those aiming to prevent child undernutrition, than what is currently the case

in programme intervention design. This change will have profound implications for policy and programme organisation that are relevant to both the short-term consequences of undernutrition (such as reducing the risks of infection and child mortality) and long-term outcomes (such as promoting human capital and reducing the risk of non-communicable disease). In turn, this change will further the development of comprehensive nutrition programmes to pursue Sustainable Development Goal 2.2, to end malnutrition in all its forms by 2030.⁵

Contributors

This Viewpoint was conceived by the Emergency Nutrition Network Coordinated Wasting-Stunting Technical Interest Group and developed by all authors. JCKW wrote the first draft, and all other authors contributed to revising it.

Declaration of interests

We declare no competing interests.

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