Technical briefing paper

from the Wasting and Stunting Technical Interest Group

Updating evidence on the relationship between wasting and stunting

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Contents

Background

In 2014, ENN developed a technical briefing paper under the guidance of the Wasting and Stunting Technical Interest Group (WaSt TIG),[1] exploring the relationship between child wasting and stunting (1). For decades, wasting and stunting have been approached as separate conditions, largely disconnected in advocacy, programming, policy and financing (2). This has had negative implications for how children receive nutrition interventions and services across the world: support is often provided by different stakeholders who offer different interventions, often in different contexts, to different age groups and with different timeframes. The 2014 technical briefing paper reviewed available literature to understand what was known about the relationship between these two manifestations of undernutrition and to explore implications for the design and implementation of policies, programmes and research. The paper concluded that wasting and stunting often coexist in the same populations and often in the same children, though the extent to which this happened was seldom being reported. Seasonality was found to have a marked impact on both wasting and stunting prevalence, and wasting and stunting were found to share many common risk factors, including *in utero*. Both wasting and stunting were found to be associated with increased risk of mortality and evidence for wasting adversely affecting linear growth (stunting) was discussed. The paper found some encouraging operational research to suggest that both wasting and stunting may be reduced with similar preventative food-based approaches. The paper concluded that, while the evidence provided compelling reasons to bring wasting and stunting policy, programming and resourcing more closely together, critical research questions to better understand the process of wasting and stunting in individuals and of optimal programming strategies for preventing and treating both outcomes at a population level remained.

Since 2014, considerable work has been undertaken and published by the WaSt TIG (and others working in this field) to fill these research gaps. The resulting research papers and reports have answered many of the research questions outlined in the 2014 paper. Seven years later, a systematic review of evidence generated since the briefing paper was undertaken, authored by Susan Thurstans and colleagues from the WaSt TIG, and published in 2021 in the journal *Maternal and Child Nutrition*.[2] This technical brief outlines some of the key findings within the systematic review. It provides an opportunity to update the original briefing paper by bringing all the evidence together and considering the programmatic and policy implications in a new technical brief.

Systematic review method

The review of literature included studies published since the 2014 review,[3] from low- and middle-income settings (LMICs), exploring both change in weight/wasting and change in height/stunting, as well as the relationship between the two in children aged 0-59 months. The included studies focused on prevalence, physiological mechanisms and outcomes related to growth and mortality. Both peer-reviewed papers and grey literature were considered for inclusion. Studies exploring wasting and stunting separately were not included. Medline, Embase and Global Health databases through Ovid were searched. The database search identified 2,486 studies and reports and an additional 13 studies were identified for inclusion by WaSt TIG members. After removing duplicates and screening for relevance, a total of 44 studies and reports were included in the final review.

[1] The WaSt TIG is a group of global experts on child growth, nutrition and epidemiology. More information on WaSt TIG can be found on ENN's website: www.ennonline.net/ourwork/researchandreviews/wast/wasttigmembers

[2] Thurstans, S., Sessions, N., Dolan, C., Sadler, K., Cichon, B., Isanaka, S., Roberfroid, D., Stobaugh, H., Webb, P., & Khara, T. (2021). The relationship between wasting and stunting in young children: A systematic review. Maternal & Child Nutrition, e13246. https://doi.org/10.1111/mcn.13246 Available at: https://onlinelibrary.wiley.com/doi/10.1111/mcn.13246

[3] Documents from 2012 to the present were, however, included to account for any articles that might have been missed by the technical brief.

What have we learnt?

In the last seven years, new evidence has enhanced our understanding of wasting and stunting, particularly in terms of how children become wasted and/or stunted and how children experience these conditions over time. New evidence has further highlighted the relationship between wasting and stunting, including the common determinants and interconnected physiological processes leading to a child becoming wasted and/or stunted. We have built a greater understanding of how children who are treated for wasting recover and how wasting treatment impacts their linear growth. We also know more about the mortality-related consequences of being concurrently wasted and stunted,[4] how commonly concurrence occurs, which children are particularly at risk and how to best identify them. Based on this growing evidence base, some potential shifts and adaptations to policy and programme approaches may be needed. These findings are discussed below.

A note on terminology

The terms **'wasted'** and **'stunted'** were introduced in the early 1970s by John Waterlow to differentiate, among underweight children, those who had a low weight in relation to their height (wasted) and those who had a low height in relation to their age (stunted). The terms **'wasting'** and **'stunting'** refer to a process rather than to a category. Stunting refers to a slowing or halting of linear growth compared to what is expected for the child's age (**linear growth faltering**). Wasting refers to a slowing or loss of body weight or ponderal growth compared to what is expected for the child's height (**ponderal growth faltering**) (1). The use of the term wasting to mean wasted and the use of the term stunting to mean stunted is quite widespread.

While being wasted has been described as an 'acute' condition and being stunted has been described as a 'chronic' condition, these terms may be misleading. Wasting, if untreated, can last several months and can occur many times through a child's young life (3, 4, 5). Similarly, stunting (having suboptimal linear growth) can manifest quickly in response to an acute event such as food shortage or disease (6). **Severe acute malnutrition** and **moderate acute malnutrition** are terms often used interchangeably with severe and moderate wasting to indicate severity of undernutrition. **Nutritional oedema (*kwashiorkor*)** is not included in this review, though it is a severe form of acute malnutrition and a separate criterion for treatment.

The terms wasted and stunted are used to classify children by comparing them to the WHO 2006 child growth standard population of healthy children growing in optimal conditions with adequate nutrition (7). A wasted child is defined as a child whose weight has fallen significantly below the weight of a healthy child of the same height and sex in the child growth standard (measuring WFH).[5] A stunted child is defined as a child whose height has fallen significantly below the height of a healthy child of the same age and sex in the child growth standard (measuring HFA)[6] (1). Mid-Upper Arm Circumference (MUAC) cut-offs are also used to identify wasting based on the evidence that low MUAC identifies children at high risk of death (8).

Underweight is a term used to describe children with a low weight in relation to their age (weight-for-age [WFA]) when compared to the median of the WHO 2006 child standard population. A child may be underweight because they are wasted, stunted or a combination of the two. Tracking WFA in infants and young children is a common tool of growth monitoring and promotion services to monitor growth patterns as it is able to detect changes in a child's growth at a relatively early stage (1).

[4] Defined as: Weight-for-Height (WFH) Z score (WHZ) < -2 with Height-for-Age (HFA) Z score (HAZ) < -2.
[5] Defined as: WFH Z score (WHZ) < -2.
[6] Defined as: HFA Z score (HAZ) < -2.

Understanding the onset, incidence and recovery of both wasting and stunting

Previously, much of the evidence for the associations between wasting and stunting, particularly at a population level, were based on available cross-sectional data. Cross-sectional data, although offering important insights, are problematic because they depict a child's anthropometric status at one point in time, not what they have gone through in terms of changes in weight and height to get to that point (i.e. the process of wasting and/or stunting). It also misrepresents the true burden of acute conditions in a population because it measures prevalence (the number of cases at one point in time) instead of incidence (the number of new cases that develop over time). In recent years, more longitudinal studies have become available, allowing insights into the dynamics of wasting and stunting in individuals throughout infancy and childhood and enabling a better understanding of incidence, duration, recovery and persistence of both manifestations of undernutrition.

Three linked studies have been particularly helpful in this regard, using pooled analyses of 18 longitudinal cohorts from 10 LMICs. The first study focuses on wasting (9), the second on stunting (10) (although the associations between the two forms of undernutrition are discussed throughout) and the third on the causes of wasting and stunting (11). The authors explored the differences between prevalence and incidence figures and found that 6% of children were wasted after birth (prevalence), but 33% had experienced at least one episode of wasting (incidence) by the age of 24 months. This means that the burden of wasting is likely far higher (as suggested above) than traditional cross-sectional studies have assumed (potentially five times higher) (9). Further corroborated analyses of secondary prevalence and incidence data from 352 sites in 20 countries suggest that the overall burden could have been underestimated by 4.6 times using prevalence data alone (5).

The longitudinal analyses of 18 cohorts further found that incidence of wasting peaked between birth and three months of life (9), (10), challenging the common understanding based on cross-sectional data that wasting peaks between the age of six and twenty-three months. These analyses concluded that "preventing and addressing early growth faltering/failure before six months of age is important and should be a high priority for global health programs" (9). The increased focus on wasting in infants under the age of six months (13) and evolution in our understanding of nutritional risk over the last decade also add weight to the call to rethink wasting treatment programming, which still largely focuses on children aged 6-59 months (14, 15) (12) Previous analysis has also found that a high proportion (30% and 20% respectively) of childhood wasting and stunting has it's origins in the foetal period (during pregnancy) (13) via the effect being born small has on malnutrition at birth and on infant's subsequent growth and mortality risk (12)(14). Findings of our systematic review further highlight the potential role of maternal drivers of wasting and stunting in children with characteristics such as maternal weight and high education achievement being found to be predictors of good nutrition at two years of age in terms of both weight and height (11, 16). Taken together these results suggest that targeting health and nutrition interventions toward women of reproductive age, pregnant women and mothers of at risk infants could be promising paths forward to prevent growth faltering amongst their children (11).

Longitudinal data have also shed light on recovery from being wasted, suggesting that there may be vulnerabilities associated with being born wasted and that vulnerabilities remain after a period of wasting and leave a child more likely to become wasted again. Ultimately, the majority of children in the cohort analysis recovered from being wasted (91.5% of moderate wasting episodes; 82.5% of severe wasting episodes) (9). However, on average, children born wasted did not catch up to the WFH of children who were not born wasted. Furthermore, children born wasted who recovered from being wasted became wasted again more frequently after the age of six months compared to children who were not born wasted. The longitudinal cohort data also found that there were a significant proportion of children who experienced regular periods of persistent wasting (being wasted for at least 50% of the time) over the first two years of life, the implications of which are discussed further below (9). These findings highlight how children move in and out of different nutritional states but remain vulnerable to the accumulation of nutrition deficits, while pointing to the importance of reaching children before they become wasted.

In the case of stunting (10), high incidence rates of stunting from birth to six months were also observed. Data showed that, although some children went on to recover from being stunted, they later continued to experience linear growth faltering, and more than 20% were stunted again in later measurements. Children who experienced linear growth failure[7] early were at a much higher risk of persistent growth failure and were between 2.0 to 4.8 times more likely to die by the age of 24 months than non-stunted children (11). This finding points to the critical importance of reaching children before they become stunted.

[7] Growth failure is a term used to describe a child's growth that is below the expected growth velocity compared to other healthy children of the same age.

There is now more evidence for the relationship between wasting and stunting

More evidence amassed for associations between wasting and stunting

Wasting and stunting appear to be associated to a degree beyond coincidence. One large meta-analysis of cross-sectional studies from 51 countries showed that, in 37 countries, a positive and significant association between wasting and stunting was seen: i.e., the proportion of children concurrently wasted and stunted was greater than that expected by chance given the prevalence of wasting and stunting in the populations. This finding suggests that, in the presence of one condition, the likelihood of developing the other condition increases (15), though the direction of the association could not be established in this analysis (15).

Additional studies have further elaborated on this. A study from Senegal found that the proportion of wasted children increased with the degree of stunting and that the proportion of stunting increased with the degree of wasting (16). A study from Malawi found a strong association between poor linear growth and relapse to severe and moderate wasting after treatment for wasting (17).

Data from programmes that treat severely wasted children have also shown a potential association between wasting and stunting, as high proportions of children admitted for wasting treatment were classified as stunted. In Nigeria, out of 472 children in a community management of acute malnutrition programme, 82.8% of children were stunted compared to a national stunting prevalence estimate of 32% (18). In Niger, out of 1,542 children who were treated for and recovered from severe wasting, 79.0% were stunted and 49.0% were severely stunted (19), in comparison to a national stunting prevalence of 39.9% in the same year (20). In Uganda, out of 788 children with uncomplicated severe wasting, 48.7% of children experienced concurrent wasting and stunting (21). These findings further suggest that the relationship between wasting and stunting is much stronger than could simply be explained by chance.

Interconnected physiological processes that occur during wasting and stunting

This association between wasting and stunting may be related to interconnected physiological processes. Wasting and stunting occur when nutrient intakes do not meet the body's requirements for growth and immune response to illness, or to compensate for nutrient losses (22). As a result, the body draws on nutritional reserves, mainly fat and muscle, to maintain essential metabolic processes (23). Infection reduces appetite and impacts nutrient absorption and metabolism, which also leads to increased risk of wasting and stunting. This process can be exacerbated by pre-existing gut inflammation (environmental enteric

dysfunction)[8] and a disturbed gut microbiome (24, 25). On the other hand, wasted and stunted children are extremely susceptible to infectious diseases due to reduced immunity. Children who are concurrently wasted and stunted have been found to be at an increased risk of infectious disease compared to those experiencing only one anthropometric deficit (26-28).

The loss of fat is associated with both wasting and stunting, although more inconsistently with stunting (22, 29-31). Fat is important in ensuring the functioning of the immune system, which demands increased energy when stimulated by infection. This suggests that the loss of fat may act as an additional mechanism linking wasting and stunting with increased susceptibility to infection and consequent mortality (22). Muscle mass loss also occurs in both wasting and stunting, particularly when there is also infection. Reduced muscle mass increases the risk of death during infections (22). Muscle mass in relation to bodyweight tends to increase with age, and thus infants are particularly vulnerable to the effects of undernutrition and associated mortality (22).

Leptin – a hormone produced primarily by fat cells that is responsible for the regulation of energy, hunger and metabolism, as well as for stimulating immune function – may also play a role in the relationship between wasting, stunting and mortality. Low leptin levels are highly correlated with low body fat stores and are associated with increased risk of mortality (31). Leptin may also have an effect on bone growth, which may explain the reduced linear growth observed in wasted children (22). We do not as yet have a full understanding of all the interconnected physiological processes and more research is needed in this regard.

Common causes and determinants

Wasting and stunting have several shared risk factors. One extensive literature review found no risk factors for wasting that were not also associated with risk of stunting (32). The range of shared risk factors is reflected in the UNICEF Conceptual Framework on Determinants of Maternal and Child Nutrition, which outlines the enabling, underlying and immediate determinants of undernutrition (described in the UNICEF 2021 Nutrition Strategy).[9]

Known immediate causal factors for both wasting and stunting include dietary inadequacy, infectious diseases, malabsorption due to (for example) diarrhoea, poor quality and quantity of complementary feeding and intrauterine growth restrictions (26), (33), (11, 34). Underlying determinants include maternal factors such as poor maternal nutrition (11), high parity (repeated pregnancies) (11), low maternal education levels (11, 21], impaired maternal mental health (35) and maternal stature (11), all of which are likely to play important roles, as well as low birthweight and/or length (11) and poor care and feeding practices (26), (33, 36). Enabling determinants such as poor economic conditions (11), conflict (32) and seasonal factors (as outlined below) (16) have also been found to contribute to both conditions. Furthermore, determinants can influence (interact with) each other in complex ways and that are difficult to disentangle.

Seasonal patterns in wasting and stunting are related

It is well known that, at a population level, wasting and stunting rates vary and in many contexts peak seasonally, alongside seasonal variations in disease, household/ income generation activity levels and food availability. Recent studies in the Gambia have highlighted that individual infants who are wasted in the first wet season (the so-called 'hunger season') of their life are more likely to be wasted in their second wet season, even if they had recovered in the intervening dry season (the so-called 'harvest season') (16). It was also found that infants born at the start of the wet season did not catch up in weight during the first three months of life to the same extent as their peers born in the dry season. These infants experienced more wasting during childhood and had an increased risk of becoming stunted by the age of two (16). Multiple cohort analyses have also produced similar findings, with average WFH scores varying greatly depending on the month in which the child was born (almost a full standard deviation variation). Birth month also influenced the effect of season on WFH trajectories, which persisted through a child's second year of life (9).The findings reflect those found in the 2014 review which also highlighted evidence at population level of seasonal peaks in stunting following wasting peaks. These findings suggest that seasonal patterns in wasting and stunting may be related and that, in some contexts, children born in certain months of the year may be at heightened risk of becoming wasted in particular. No studies exploring the effect these seasonal patterns in wasting may have on subsequent stunting were identified during our review.

In individual children wasting can predict subsequent stunting and vice versa

Wasting episodes appear to contribute to subsequent stunting. In fact, some evidence suggests that stunting is in part a biological response to previous episodes of being wasted (16). Longitudinal data from the Gambia showed that being wasted increases the odds of becoming stunted within the next three months by a factor of 3.2: i.e., a child is three times more likely to be stunted three months later,

[8] Environmental enteric dysfunction is damage to the gut following repeated infection and diarrhoeal illness. This damage means the gut cannot absorb nutrients well, and thus contributes to poor nutrition. This happens even without ongoing diarrhoea, so may be 'invisible'.

[9] https://www.unicef.org/media/92031/file/UNICEFNutritionStrategy 2020-2030.pdf

based on being currently wasted, compared to a non-wasted child (13). Multi-country longitudinal data show that persistent wasting from birth to the age of six months is strongly associated with stunting incidence in older ages (11). These studies demonstrate that there may be a time lag whereby wasting is followed by stunting in later months. Reasons for this could be that the body's response to weight faltering is to slow or halt linear growth until weight is gained and infection is treated (19), (37). An analysis from Niger exploring linear growth during wasting treatment suggests that HFA deteriorates during the period of treatment, where rapid weight gain is typical (19). Where linear growth was seen during treatment, the children had less severe wasting and stunting (19, 38) and fewer infections at baseline (38). This suggests that untreated infection may also impede linear growth during wasting treatment.

Similar findings from Senegal suggest that linear growth may increase during wasting treatment, with improving health status (39). Age factors may also play a role, with data from the Gambia suggesting that wasting is more detrimental to linear growth the later it occurs and recovery of HFA is more likely if wasting occurs early (16). For example, a longitudinal study of infants and children 0-24 months in LMIC countries (40) found no long-term effect of one period of wasting in the first 6 months of life on HFA at 18–24 months if no further wasting was experienced after that time, suggesting that one episode of wasting in this age group is not enough to slow/halt linear growth. These findings highlight that, although wasting is commonly viewed as transient and treatable, its effects are longer-term and could result in linear growth slowing. Despite the link between periods of wasting and subsequent stunting, it is important to note that stunting is far more common than the prevalence of earlier wasting can fully explain. Stunting can also coexist with a high overweight prevalence in some populations (41). This suggests multiple and diverse causal pathways for stunting.

Although less evidence exists, some research has also pointed to a direct relationship whereby stunting leads to wasting, although to a lesser extent than wasting leading to stunting. Two longitudinal studies have demonstrated a strong and significant effect of stunting on the risk of subsequent wasting (16, 42). In the Gambia, it was found that a child was 1.5 times more likely to be wasted three months later if the child was, at the time, classified as being stunted, regardless of the child's wasting status at that point in time (16). The degree of stunting affected the level of risk, with more severe stunting more likely to result in wasting (16). The physiological mechanisms for this relationship are less clear, but the findings emphasize that the relationship between wasting and stunting is complex and may be bidirectional(16).

Stunting may influence wasting treatment response

As noted above, there is a high prevalence of stunting among severely wasted children admitted to wasting treatment programmes. While there are inconsistencies in results, there is some evidence that this influences treatment response. Data from Uganda found lower recovery rates in stunted children compared with non-stunted children during wasting treatment (58.0% vs. 65.4%), higher rates of non-response (18.7% vs. 9.8%) and a longer length of stay (63 days vs. 56 days), but also greater weight gain (2.2 g/kg/day vs. 1.7 g/kg/day) (21). Similarly, in Malawi, children who experienced poor linear growth were more likely to relapse to moderate or severe wasting than those whose linear growth rate was maintained or increased (17). In contrast, however, a study from Niger found that levels of stunting had no impact on weight gain during or after wasting treatment (19). One study exploring longer-term outcomes after wasting treatment showed that some recovery in height of previously wasted children was possible, but that these children still demonstrated more severe stunting than sibling and age-and-sex-matched community controls (43). While further research is required, this finding highlights the need to consider stunting status within wasting treatment programmes in that stunted children appear to have a higher risk of poor outcomes within such treatment programmes.

Consequences of wasting and stunting when they occur together

Increased mortality risk for concurrent wasting and stunting

Children with concurrent wasting and stunting are at high risk of mortality and the level of excess mortality is not explained by the severity of wasting and stunting alone. This finding suggests a multiplicative effect on mortality of having both deficits (11, 15, 16, 42, 44-47). An analysis of mortality cohort data from 10 countries found that children who are wasted, stunted and underweight have a 12-fold elevated risk of mortality compared to children with no nutritional deficits (45). A later analysis of nutrition surveys in 51 countries found that all children who were wasted and stunted were also underweight and, therefore, that the same elevated mortality estimate is applicable to them (15). A recent longitudinal analysis of eight datasets showed that all measures of early growth failure were significantly associated with a higher risk of death by the age of 24 months. Those most strongly associated with death were severe underweight before the age of six months, concurrent wasting and stunting and persistent wasting under the age of six months (11). Being concurrently wasted and stunted appears to amplify the risk of death to levels comparable to that seen in children with the most severe form of wasting.

The burden of concurrent wasting and stunting is relatively large

Analysis of 84 countries found the pooled prevalence of concurrence was 3.0%, ranging from 0.0% to 8.0% in different countries (48). Building on this, the Global Nutrition Report 2018 carried out a global level analysis which indicated that 3.62% of children under five years globally are both stunted and wasted, equating to 15.95 million children worldwide (49). Other published studies (12 in total) have found similar levels of concurrence, with prevalence ranging from 12% in Niger (50) to 1.4% in Ghana (33). The findings indicate that fragile and conflict-affected states (FCAS) appear to be disproportionately affected, with higher rates of concurrent wasting and stunting than stable contexts (the large-scale study found a pooled prevalence of 3.6% in FCAS compared with 2.24% in stable contexts). This finding emphasises the increased vulnerability of children growing up in FCAS countries (48, 51).

While rates of concurrence vary greatly depending on context, the findings indicate that concurrent wasting and stunting affects a significant number of children and, given the mortality risks associated with this condition, these children require special attention. Furthermore, prevalence estimates are likely to be an underestimation of the true burden of concurrence. Better assessments of the actual burden of wasting is needed, as children can move in and out of periods of this condition throughout the year.

Younger children and males appear to be more at risk of concurrent wasting and stunting

Wasting, stunting and concurrent wasting and stunting appear to be more prevalent in boys than in girls (15, 21, 48, 52). A recently published systematic review and meta-analysis (53) found that, in studies that examined wasting, boys had higher odds of being wasted than girls (pooled OR 1.26). Similarly, boys had a higher odds of being stunted and also underweight than girls (pooled OR 1.29 for stunting; pooled OR 1.14 for underweight). This finding has also been noted in wasting treatment programme data, particularly in relation to concurrent wasting and stunting (18, 19, 21). Data from one treatment programme in Uganda found that, despite higher overall admissions for females, there were more males with concurrent wasting and stunting within the admitted group (21).

In terms of age, it appears that concurrent wasting and stunting peaks between the age of 12 months and the age of 30 months (11, 18, 21, 42, 44, 48), with younger

children being the most affected (21). In Senegal, it was found that males were more likely to be concurrently wasted and stunted below the age of 30 months, but less likely to be wasted above 30 months at the same level of stunting (42). Age and sex therefore appear to be fundamental factors affecting the occurrence of concurrent wasting and stunting.

Measuring WFA and MUAC may help identify children at risk of concurrent wasting and stunting

Given that concurrent wasting and stunting leads to a heightened risk of mortality, there is an urgent need to explore how we might identify these children, or indeed whether they are already being captured within our existing approaches. Several studies point towards the utility of WFA Z scores to do just that. Data from Senegal found that the combined use of WFA Z score (WAZ) < -2.8 Z score and severely low MUAC < 115mm identified near-term deaths associated with concurrent wasting and stunting and with severe wasting, as defined by WFH Z score < -3 (WHZ). Studies from Niger found that MUAC was the best predictor of mortality in children aged six months to 59 months, followed by WAZ (54). A recent paper analysed 12 similar mortality cohorts that included children experiencing wasting and concurrent wasting and stunting, and found that WAZ < -3 or MUAC < 115mm detected all, or nearly all, deaths associated with severe wasting (including WHZ < -3) or stunting (55).

These findings suggest that WAZ identifies children at high risk of mortality, which MUAC and WHZ alone might not. Therefore, the use of MUAC with the addition of WAZ might effectively identify those children at most risk and in need of treatment. However outstanding questions as to the intensity of treatment required for this group remain. WAZ is widely used in many countries across several community-based health and nutrition interventions, including growth monitoring and promotion as well as maternal child health and integrated management of childhood illness programming at primary healthcare facilities. It must be noted, however, that challenges with accurately determining a child's age in some contexts hinders uptake of the use of WAZ. These findings suggest that wasting treatment services may achieve a higher impact if both WAZ and MUAC admission criteria are considered. Given the wide existing use of WAZ in growth monitoring, it could also provide a tool for linking preventative and treatment approaches for undernutrition in children.

Implications for policies and programmes

The evidence amassed thus far offers important considerations for programming. Such considerations have been highlighted in this technical brief and include the following.

- Consider long-term overlapping vulnerabilities: wasted children are more at risk of becoming stunted and are more at risk of having subsequent wasting episodes than

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non-wasted children, while stunted children are more at risk of becoming wasted. Children who have been identified with one or the other anthropometric deficit should be considered in prevention programmes that include targeting for both outcomes.

- Develop common prevention strategies to tackle all forms of undernutrition: targeting for prevention programmes should prioritise multiple risk factors for undernutrition rather than anthropometric cut-offs or geographical targeting (as is typical in stunting prevention programmes). Such targeting should consider seasonality of undernutrition and childhood diseases (for example acute respiratory infection, diarrhoea and malaria) and population subgroups defined by sex, socioeconomic status, maternal risk factors, care and feeding practices and birth characteristics.
- Consider children with concurrent wasting and stunting within wasting treatment programming: given their elevated mortality risk, mechanisms to identify and target these children with appropriate treatment should be prioritised and scaled up within programmes. Although questions remain on the intensity of treatment these children require.
- Ensure younger children (those under the age of 24 months) are prioritised for treatment interventions given their increased risk of concurrent wasting and stunting and vulnerability to recurrent wasting.
- Adopt strategies to better identify severe wasting and concurrent wasting and stunting in this age group (under 24 months) and maximise coverage of treatment. For example, the Management of At-Risk Mothers and Infants Care Pathway[10] can be used as a strategy for scaling up interventions for infants aged 0-6 months.
- Understand sex-specific data: while sex-specific data are routinely analysed and reported in nutrition surveys, they should be used in nutrition programming to better identify and understand what differences exist. Analysis should assess if any sex difference in programme admissions is reflective of sex differences in undernutrition burden.
- Consider joint contextual and causal analyses for both wasting and stunting (given the commonalities in risk factors) as a basis for joined up and effective prevention programming. Seasonality should be given special consideration in certain contexts.

Finally, and more broadly, the research described in this brief has highlighted problems in underestimating the actual burden of wasting, which has knock-on effects for our ability to understand both the magnitude of the condition and its implications on how it interacts with other manifestations of undernutrition, particularly stunting. There is an urgent need to better measure the incidence of wasting and stunting rather than to rely on prevalence data. A better understanding of incidence of wasting and stunting and their concurrence might more effectively inform policies and target programme resources.

At a policy level, this brief recommends that governments explore policy cohesion between wasting and stunting policies as outlined in the latest UNICEF Nutrition Strategy (2020-2030).[11] The Global Action Plan for child wasting and the Scaling Up Nutrition Strategy 3.0 have created opportunities to explore joined programming for wasting and stunting and to develop mechanisms by which these can be achieved. Donors should continue to examine their financing arrangements so that wasting and stunting can be simultaneously prevented in both humanitarian and development contexts, and to allow wasting treatment to be scaled to prevent near-term mortality and enable improved nutrition and growth outcomes.

There is more we still need to know about the relationship between wasting and stunting

While research in understanding the relationship between wasting and stunting has advanced tremendously in recent years, operational research is needed to guide optimal treatment approaches, particularly for children experiencing concurrent wasting and stunting. Areas of further operational research include the following:

- Understanding physiological mechanisms linking wasting and stunting, in particular a better understanding of when best to intervene to interrupt the accumulation of vulnerabilities;
- Understanding factors that lead to children's early vulnerability to develop wasting and/or stunting (even before birth) to inform optimal prevention packages;
- Understanding and evidencing the range of optimal care packages (including intensity and duration of treatment) for high-risk children, including those with concurrent wasting and stunting;
- Exploring whether wasting treatment programmes can be adapted to prevent and improve linear growth, to support children who are vulnerable to or experiencing stunting;
- Exploring the effect of timing and intensity of interventions that can best have an impact on preventing seasonal peaks in wasting and stunting; and
- Exploring the time windows by which we define concurrent wasting and stunting – considering time lags between children experiencing wasting and stunting events (as opposed to measuring concurrence at one point in time) and what the implications of this are on mortality risk.

[10] www.ennonline.net/ourwork/research/mami
[11] https://www.unicef.org/media/92031/file/UNICEFNutritionStrategy2030.pdf

Concluding remarks

A significant amount of evidence has been generated since the 2014 ENN technical briefing paper that further supports the existence of a strong relationship between wasting and stunting, with important implications for policy and practice. We now know that wasting and stunting incidence rates are high from birth to the age of six months, with many children born with nutritional deficits. It is also now apparent that children can move in and out of different nutrition states, but remain vulnerable to the accumulation of these nutrition deficits. We know that wasting and stunting are inextricably linked with common causes and risk factors for both conditions, even before birth, as well as with shared physiological processes. We know that wasting and stunting appear to be associated beyond what can be ascribed to coincidence; that wasting episodes can contribute to subsequent stunting; and that stunted children are at risk of subsequent wasting episodes. We know that concurrent wasting and stunting is not uncommon, with national estimates in the population aged six months to 59 months reaching 9% and an estimated global burden of 16 million cases. We know that these children face a 12-fold mortality risk compared to their healthy peers. We know that using a combination of WAZ and MUAC anthropometric measures can help identify these children. We also know that younger males seem more vulnerable to wasting and concurrent wasting and stunting, and that seasonality plays a role in vulnerability.

While research is needed to further enrich our understanding of the relationship between wasting and stunting, particularly operational research to explore ways of tackling both wasting and stunting in prevention and treatment interventions, there is sufficient evidence to start shifting the divide between wasting and stunting in research, policy, financing and programming. To do this, policy and practice need to address undernutrition through a lifecycle approach, from preventative interventions targeting adolescents and women of reproductive age, to infants and young children and the treatment of undernutrition in the young child.

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