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Perspective: What Does Stunting Really Mean? A Critical Review of the Evidence

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Summary

- The authors of this article challenge the current view that improving linear growth will subsequently lead to improvements in the correlates.
- The authors suggest that current evidence and understanding of mechanisms does not support this **causal** thinking—with the exceptions of poor birth outcomes and difficult birth.
- Instead the authors state that linear growth retardation is **associated** with (but does not cause) delayed child development, reduced earnings in adulthood, and chronic disease.
- Question for donors, program planners, and researchers: **Does a focus on linear growth retardation and stunting help to improve the well-being of children? Or is it an imperfect indicator?**



Introduction

- The focus on linear growth retardation and stunting has facilitated communication with policy makers, enabled successful advocacy for nutrition, and mobilized policy makers and donors to pay attention to undernutrition and its consequences.
- But what are stunting's actual consequences?
- The authors argue that along with the strong emphasis on linear growth retardation and stunting, there has been some confusion and misunderstanding about its meaning among researchers, donors, and agencies active in nutrition.



Linear growth retardation and stunting: what's the difference?

- **Linear growth retardation** (or linear growth faltering) is defined as a failure to reach one's linear growth potential. Linear growth retardation implies that (groups of) children are too short for their age, but does not imply that they are stunted. The number of children suffering from linear growth retardation is much higher than the number of children that are stunted.
- **Stunting** is defined as having a height-for-age z score (HAZ) $< -2SD$. HAZ is calculated by subtracting an age- and sex-appropriate median value from a standard population and dividing by the SD of the standard population. The 2006 WHO growth standards are the recommended standard. In a healthy population, $\sim 2.5\%$ of all children have a HAZ $< -2SD$. A higher percentage $< -2SD$ is indicative of a deficient growth environment. Children who are stunted are a subset of those with linear growth retardation.



Objectives of the Article

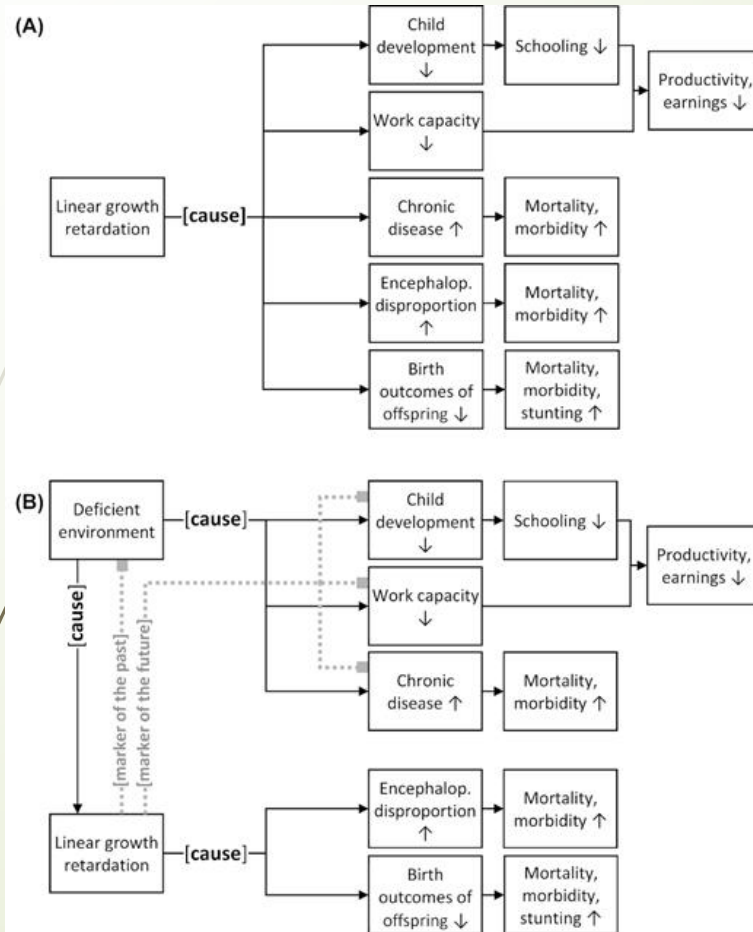
Goal: To show that many outcomes commonly presented as consequences of linear growth retardation and stunting are not causally linked.

1. Illustrate how the nutrition community has emphasized the consequences of linear growth retardation and stunting and how this “**causal**” view has been widely adopted.
2. Critically review the scientific evidence linking linear growth retardation and stunting to other outcomes.
3. Recommend a fundamentally different evidence-based way of making use of linear growth retardation and stunting as measures of global development.



What the Nutritional Science Community Is Telling the World about Linear Growth Retardation and Stunting

- Linear growth retardation and stunting are associated with undesirable short-, medium-, and long-term outcomes in 5 domains:
 - 1) Delayed child development leading to lower school achievement and reduced earnings
 - 2) Reduced physical strength and work capacity, leading to reduced earnings
 - 3) Physiologic changes, contributing to adult noncommunicable diseases and increased mortality
 - 4) Increased risk of cephalopelvic disproportion, leading to dystocia, mortality, and morbidity
 - 5) Undesirable birth outcomes in the next generation, i.e., low birth weight or small-for-gestational-age (SGA) infants more likely to die or not grow adequately.




Framework showing importance of linear growth retardation and proposed framework distinguishing between child linear growth




Catch-up growth

- The causal thinking has also triggered research on catch-up growth.
- Catch-up growth refers to accelerated growth that reduces a child's accumulated height deficit. Some studies reported an **association** between catch-up growth and child development, concluding that promoting growth during infancy and early childhood might contribute to better child development.
- The authors argue that this is misleading because much of the catch-up growth work has assumed that linear growth retardation and stunting negatively affect child (cognitive) development, and recovery from linear growth retardation or stunting is presented as if it will lead to improved cognitive outcomes.
- The authors aim to show that there is no evidence that linear growth and cognitive development are **causally** linked. They state that these studies merely confirm that better linear growth is **associated** with better cognitive development.




What Is the Evidence about Outcomes of Linear Growth Retardation and Stunting?



Linear growth retardation and developmental delays

- Linear growth retardation is **associated** with reduced cognition and motor development in middle- and low-income countries.
- Linear growth retardation and poor development are **associated** through a set of shared determinants (suboptimal nutrition, inadequate care, and repeated infections). However, linear growth retardation is not part of the mechanistic path leading to delayed cognitive, motor, or socioemotional development.
- Ultimately, the authors conclude that there is no evidence that linear growth retardation (or stunting) **causes** delays in child development, and based on our current understanding of mechanisms, it is not likely that they are **causally** related.



Linear growth retardation and earnings

- Earnings have been associated with height.
 - Taller individuals have more schooling and better skills, which could explain the association, but the height-earnings association remains after controlling for cognitive and socioemotional capacity.
- Reasons to question the **causality** of this association.
 - No evidence for a credible biological (or other) mechanism that would explain the effect of stature on earnings at the population level.
 - The height-earnings association in developed economies indicates that relative height (rather than height in absolute terms) is of importance. Therefore, the association will not disappear when linear growth retardation is eliminated since that would not remove the distribution of heights at the population level.
- The authors conclude that a **causal** link between linear growth retardation (or stunting) and lower earnings is not supported by current evidence.



Linear growth retardation and chronic diseases

- Environmental influences during early development, such as poor nutrition, increase chronic disease risk later in life.
- Much early work on the developmental origins of disease focused on birth weight and infant size as measures of exposure, which may have contributed to the belief that linear growth retardation and stunting are a cause of adult chronic disease risk.
- Based on current knowledge, however, linear growth retardation and stunting are not part of the mechanistic path. Recent evidence from carefully conducted epidemiologic studies **does not show an association between linear growth retardation (or stunting) and a number of chronic disease risk factors.**
- The authors conclude that the evidence does not support a causal link between linear growth retardation (or stunting) and chronic disease.




Linear growth retardation and cephalopelvic disproportion

- ❑ Linear growth retardation at childhood reduces adult height.
- ❑ Shorter stature in women at adulthood, in turn, is associated with a higher risk of dystocia or difficult labor.
- ❑ Mechanical dystocia, or cephalopelvic disproportion, is a major cause of maternal and neonatal mortality and morbidity.
- ❑ The association between maternal height and difficult labor is mediated by the size of the pelvic inlet; shorter women have a smaller pelvic inlet and are thus more likely to suffer from a mismatch between the size of fetal head and the dimensions of the birth canal. Since both stature and pelvic size are linked to skeletal size, the authors state that the association between linear growth retardation at childhood and obstructed labor at adulthood is *causal*.




Linear growth retardation and birth outcomes

- A short mother is more likely to have SGA children.
- This association is considered **causal** and due (in part) to maternal physical constraints associated with short stature.
- SGA children are at increased risk of neonatal and infant mortality and morbidity during the neonatal period and beyond. Being SGA is responsible for up to 20% of stunting in children between the ages of 1 and 5 y.
- Maternal short stature is associated with an estimated 6 million SGA births in low- and middle-income countries annually. Eliminating SGA births that are due to maternal short stature would reduce neonatal deaths by an estimated 3.6% (or 97,200 deaths globally).
- The authors conclude that linear growth retardation at childhood is **causally** linked to an increased risk of giving birth to SGA children. Eliminating maternal short stature would have a modest effect on neonatal mortality.



Distinguishing between linear growth retardation and stunting as a marker compared with as an outcome

- Based on the evidence, the authors identify two distinct uses for linear growth retardation and stunting:
 - The association between linear growth retardation and other outcomes makes it a useful *marker*.
 - Reflective of past exposure to an inadequate environment.
 - Predictive of the future: Since linear growth and poor cognition share some of the same determinants, improvements in these determinants can be expected to improve both growth and cognition.
 - In areas of high stunting prevalence, two messages are implied: 1) children are growing up in a deficient environment; 2) as a consequence of living in this environment, they are unlikely to realize their full potential in the future.



Distinguishing between linear growth retardation and stunting as a marker compared with as an outcome


- ❓ The causal links with difficult birth and poor birth outcomes make linear growth retardation and stunting outcomes of *intrinsic value*.
- ❓ A reduction in linear growth retardation or stunting is expected to directly improve these outcomes as part of the mechanistic path and not merely indicative of other outcomes.



Just Semantics? Some practical implications




Improving linear growth is often not necessary

- For many nutrition outcomes, nutrition interventions will have positive, meaningful and observable effects before linear growth improves.
 - Equating lack of impact on linear growth to program failure discounts the importance of other outcomes and interventions to improve them.
 - Several nutrition interventions are effective at improving child well-being but have no effect on linear growth.
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Improving linear growth is not sufficient

- Children who experience adequate growth, may still lack adequate stimulation at home or attend poor-quality pre- and primary schools.
 - Addressing the associated outcomes with linear growth retardation directly may be more efficient than focusing on improvements in linear growth.
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Eliminating fatalism

- The observations that 1) it is during the first two years of life that most rapid growth failure occurs, and 2) interventions beyond this age have little impact on improved linear growth have led to a view that interventions beyond the 1,000 day period are unlikely to have meaningful effects.
- Linear growth retardation occurs beyond the first two years and biological windows of opportunity for its improvement do not always coincide with windows for other outcomes.
- Nutrition, health and development efforts need to extend beyond the first two years. Research is needed to determine the potential for improving nutritional status beyond the 1,000 day period, to test different interventions on undernutrition and to identify optimal timing for improving these outcomes.



Getting other sectors on board for nutrition-sensitive interventions

- Nutrition-sensitive interventions both address the underlying causes of undernutrition (e.g., poverty) and incorporate specific nutrition goals and actions.
- Nutrition-sensitive programming contribute to improvements in access or consumption of high-quality diets or reduce poverty / food insecurity, but should not expected to directly improve child growth.



The proposed way forward

- **The need for specificity:** Donors, program planners and researchers should use specific terminology and be explicit about reasons for focusing on linear growth.
- **Population assessment:** The severity of linear growth retardation and stunting in groups of children can be used to compare countries or regions within a country. It can also be used to monitor progress of children of the same age distribution over time.
- **Counting cases:** Limitations exist when using stunting (HAZ <-2SD) to count the number of affected children. Counting the number of stunted children underestimates the number of children affected by inadequate environment due to a shift in the entire HAZ distribution.
- **Programs, interventions, impact evaluation:** Recommend the use of primary outcomes to eliminate the risk of basing a programs' success on its ability to improve linear growth. Assessing linear growth as a secondary outcome may be useful to evaluate if a program was able to improve the full set of conditions necessary for linear growth.



Discussion Question:

- What do you think about the author's position that there is not enough evidence to support the claim that improvements in linear growth will decrease negative outcomes in childhood and later in life?



Discussion Question:

- The authors state that there is no evidence that linear growth and cognitive development are causally linked.
- What do you think about this? Is it enough that linear growth is associated with better cognitive development? Should the nutrition community continue to use this indicator even if the positive outcomes are only associated (and not causally linked) with stunting?



Discussion Question:

- The authors argue that children who grow adequately, but who lack adequate stimulation at home or attend poor-quality preschool and primary education, are unlikely to fully develop. Should we be more focused on behavior change, early childhood development, and caregiving practices at the household level for improvements in cognitive development, chronic disease and earning potential? Do we think stunting is no longer a useful indicator for these concerns?

Discussion Question:

- How is the authors' perspective supported by emerging models like the Nurturing Care Framework?





Discussion Question:

- The authors argue that programs that focus on nutrition-sensitive agriculture programs, and nutrition-sensitive social protection programs should not be expected to directly improve child growth, and therefore should be taken out as a primary outcome indicator. Even for nutrition-specific programs, they state that linear growth retardation and stunting should not be a primary outcome for the purposes of evaluating programs and interventions. Do you agree? If we don't move stunting, does that mean that program failed?
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