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EDITORIAL

Stunting: There Is More Than Meets the Eye

More than just a defensive stunt, the title aptly implies more than it appears at first say. By definition, stunted growth is a reduced growth rate in human development, the etiology of which can be traced to fetal development brought on by the malnourished mother, onto the early childhood years. Once established, children may never regain the lost opportunity for height as a result of stunting. Clearly, stunting results from chronic undernutrition, which retards linear growth. In measurement, a child whose height-for-age is less than -2SD of a reference population is considered stunted, because the chances of the child’s height being normal are less. In many developing countries, stunted growth commonly affects a large percentage of children manifesting malnutrition in general. As such, stunting is a problem that has not been solved.

In defense of height, one could always articulate – height is not might. Or, is it? The angle with which to take on the challenge of answering such a provocative statement can prove difficult to address, primarily because of the multi-faceted implications of stunting and in comparison to social, physical and mental markers. There will always be the next door stunted human being who excels academically, or plays sports to the maximum and what have you. Yet, have you ever wondered what else could that individual have achieved if he or she has not been stunted? Additionally, what practical biomarkers for diagnosis would characterise functional changes brought about by stunting; or vice versa, by improved height-for-age? Irrespective of its use as a risk predictor, there is equal interest in the social and health benefits of being tall, and social and health implications of being stunted, and whether or not well-developed height will improve performance, or reduce risks for chronic disease conditions.

Health experts now recognise the long-term effects of early undernutrition. Evidence abounds on how early malnutrition increases the risk of numerous chronic diseases later in life, such as diabetes, hypertension, renal disease, and cardiovascular disease. Although the role of nutrition in the development of disease condition is relatively well established, the potential contribution of stunting to chronic deleterious effects has remained controversial. While faltering growth easily manifests itself in height, stunting may also be accompanied by vital organs not being fully developed, thus leading to premature death later in life.

Socially and economically, childhood undernutrition may lead to inability to perform simple physical tasks, diminishes adult intellectual ability and work capacity, causing economic hardships for individuals, their families and societies. The cycle goes around, thus producing malnourished women, again with tendencies to deliver premature or small babies who are most likely to suffer from suboptimal growth and development. Poverty, ill-health and stunting are passed on from generation to generation, presenting a thin line between genetics and environment as determinants to stunting. That height in humans represents a complex genetic make up while stunting is an environmentally inclined manifestation is universally accepted. Thus, the propensity for linear growth by virtue of genes becomes a case of “lost in translation” in stunting. Indeed, linear growth is inheritable but highly sensitive to environmental conditions.

While our understanding of the underpinnings of stunting has been evolving, much has yet to be done, particularly in the light of interventions. At a time when the concept of underweight is a convenient rationale to nutrition interventions, the problem of stunting plays second to importance, if not urgency. Could it be so because of the irreversibility of the
condition of stunting? Or, of the seemingly blurred, if not difficult to prove, causal relationships between genes and environment and their resultant height? Over time, thinking on how to reduce malnutrition has shifted from just the problem of underweight, to undernutrition, linear growth included, and even overnutrition. Whereas previous efforts focused almost exclusively on identifying and rehabilitating malnourished children, current efforts gear towards interventions of combined nutritional and disease prevention and treatment.

If indeed stunting becomes a rationale for intervention, the question that may be asked next is, at what stage – considering the foregoing reiterations on what may have led to stunting. Factors that have limited progress in this area include the lack of agreement on how to identify the appropriate target population. Complicated decisions that have to be made are accumulating evidence that people who showed reduced growth rate during infancy and childhood, but who subsequently showed catch-up growth, have higher susceptibility to developing abdominal obesity rather than lean mass or of skeletal growth. Perhaps one major goal in preventing stunting is to discover a pathway to unfaltering linear growth and yet avoiding a positive energy balance and its accompanying metabolic syndrome. Related to this is the importance of aiming for optimal diets, which includes paying attention to dietary quality in both macro and micronutrients.

On the other hand, would the above not make it easier to decide for early or preventive interventions thus avoiding the issue of catch-up growth? After all, an examination on the possible reasons that may have led to a child’s faltering growth trajectories become sufficient grounds to justify any well-meaning and thought of interventions. Thus, the value of the life cycle approach when it comes to supporting potential growth of individuals and population groups if stunting is to be abated. In other words, stunting being of a chronic nature, shall we then step backwards, or forward, i.e. on the pre-pregnant stage of a mother, as a cycle would go, to be able to intervene?

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