Stunting Persists despite Optimal Feeding: Are Toilets Part of the Solution?

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Children in developing countries have an average length-for-age that is already below the World Health Organization standard at birth, then show a further decline in linear growth over the first 24 months of life, with little or no recovery thereafter. Globally, 165 million children under 5 years are stunted, with associated sequelae of increased morbidity and mortality, lower school performance, reduced adult productivity and an elevated risk of nutrition-related chronic diseases [1].

Although it seems plausible that stunting develops because children have inadequate diets, complementary feeding interventions have only a modest impact on growth. Recurrent infections, particularly diarrhea, have been implicated in the etiology of stunting; however, whilst some studies report a significant impact of diarrhea on height, others indicate that catch-up growth occurs between episodes, leading to little or no long-term deficit in height attributable to diarrhea.

Children living in conditions of poor sanitation and hygiene are frequently exposed to pathogenic microbes through feco-oral transmission. Acute diarrhea likely represents only the tip of the 'enteric disease iceberg', with a substantial underlying burden of chronic, subclinical enteropathy (fig. 1). Several decades ago, it was noted that a small intestinal enteropathy, termed tropical enteropathy, is almost universal among people living in developing countries. This intestinal pathology, characterized by villous atrophy and a mucosal inflammatory infiltrate, much like celiac disease, was assumed to be an incidental finding until later studies reported an inverse association between increased intestinal permeability and length growth in infancy [2].

A series of recent studies has shed more light on this condition, which has been renamed environmental enteric dysfunction (EED), reflecting the broad disturbance in intestinal structure and function that likely arises from an apparent environmental insult. Gut damage appears
to arise early in infancy (from around 3 to 6 months of age), and markers of intestinal inflammation, intestinal permeability and systemic immune activation are inversely associated with linear growth [3, 4]. It is hypothesized that impaired gut barrier function enables translocation of intestinal microbes and microbial products, which stimulate pattern recognition receptors on innate immune cells in mesenteric lymph nodes and the liver, leading to chronic systemic inflammation (fig. 2), which may impair growth. Although the etiology of EED remains unclear, it is likely that recurrent exposure to fecal microbes alters the composition of the microbiota, leading to intestinal inflammation. EED is therefore viewed as a systemic condition arising from a primary subclinical gut lesion, which appears to be closely linked to environmental living standards.

Reducing feco-oral microbial transmission by improving water, sanitation and hygiene (WASH) may theoretically prevent or ameliorate EED and improve linear growth. There is evidence from a meta-analysis of water treatment and hand washing trials that WASH interventions have a small but measurable impact on linear growth; however, no trials to date have investigated the impact of improved sanitation on growth [5]. There is increasing interest in combining interventions during the first 1,000 days (from conception to 2 years of age) to address stunting. Two large cluster-randomized trials in Africa and Asia, SHINE and WASH Benefits, are currently evaluating the independent and combined effects of WASH

Fig. 1. The enteric disease iceberg. Diarrhea may be conceptualized as being the tip of the 'enteric disease iceberg', with a much greater burden of chronic, subclinical enteropathy (termed environmental enteric dysfunction) underlying clinically overt episodes of acute diarrhea. This figure is schematic and is not meant to depict relative proportions of diarrhea and enteropathy.
and infant feeding interventions on stunting in the first 2 years of life. These trials aim to provide an evidence base to guide policymakers how best to invest in public health interventions to ameliorate stunting to meet the ambitious World Health Assembly target of 40% reduction in stunting prevalence by 2025. Given the complex interplay of factors leading to stunting, such multisectoral interventions are likely required to substantially improve the growth and developmental potential of infants in developing countries.

**References**


