FOOD for Thought

To WASH or Not to WASH

Klaus Kraemer
Managing Director, *Sight and Life* Foundation

"Everything should be made as simple as possible, but not simpler"
Albert Einstein

At the end of April, I attended the American Society for Nutrition's scientific sessions at Experimental Biology 2017 in Chicago. Christine Stewart (University of California, Davis) and Rebecca Stoltzfus (Cornell University, Ithaca) organized an excellent session, presenting the long-awaited results of two large-scale, cluster-randomized trials in rural Bangladesh and Kenya, integrating WASH (Water, Sanitation, Hygiene) and nutrition interventions. The WASH Benefits Study[1] aimed to systematically investigate whether water quality, sanitation, handwashing, and improved infant and young child feeding practices (IYCF) can, independently or in combination, improve linear growth in the first two years of life. (IYCF covers breastfeeding and improved feeding practices, including the provision of lipid-based nutritional food supplement [LNS].) The rationale is that repeated bouts of infectious diseases, such as diarrhea, environmental enteric dysfunction (EED), and macro- and micronutrient deficiencies are thought to be leading causes of child stunting. The Sanitation Hygiene Infant Nutrition Efficacy (SHINE)[2] trial in Zimbabwe, with a comparable design but a stronger focus on mitigating exposure to chicken feces, was also on the agenda, but the results of this trial will only be available later this year.

The WASH Benefits study team invested considerable time and effort in reducing child exposure to enteric pathogens, viz. through the provision of plastic potties for children, improved pit latrines, water chlorination, and handwashing stations with soapy water near the latrines and the kitchen. Moreover, a behavior change communication (BCC) component was central to the study, emphasizing IYCF, hand washing after toilet use and before food preparation, the use of latrines for defecation, and the safe disposal of feces from the compound. Nonetheless, alone or in combination, the WASH interventions did not prevent linear growth faltering at 12 and 24 months. However, the LNS which was added to the child's complementary food did have a small, but consistently significant, positive impact on linear growth.

What happened?

Without any doubt, the reduction of child stunting is a thorny issue. I still believe that WASH interventions are important. WASH interventions appear to be sufficient to prevent infectious diseases such as diarrhea (interestingly, this was the case in Bangladesh, but not in Kenya), but EED prevention may require measures which are more comprehensive; fecal-oral transmission of pathogens appears to be difficult to control. In Bangladesh, the WASH Benefits Study reported frequent hand-mouth contact by children, indicating a potential source of bacteria, viruses, and parasites. Unfortunately, the results are pending on EED biomarkers and soil-transmitted helminth infections from the WASH Benefits Study and the SHINE trial. They will provide important information on future directions.

"We must better understand the underlying causes of stunting, including the biology of linear growth"

Evidently, in order to effectively reduce stunting, we must better understand the underlying causes of stunting, including the biology of linear growth. Danaei and co-workers[3] identified 18 key risk factors of stunting from an analysis in 137 developing countries, grouped in five clusters (maternal nutrition and infection, teenage pregnancy and short birth spacing, fetal growth restriction and preterm birth, child nutrition and infection, and the environment) with fetal growth restriction, preterm birth and environmental factors (WASH, use of biomass fuel) being the most prominent.

Great progress

Some countries have made tremendous progress in terms of reducing stunting, while others have not. Hossain and colleagues[4]

WASH interventions may not be enough on their own to prevent environmental enteric dysfunction (EED).

evaluated the average annual rate of reduction (AARR) for stunting in 19 low- and middle-income countries, which varied from 0.6% to 8.4%. Successful programs in countries (AARR ≥ 3%) were characterized by a combination of political commitment, multisectoral collaboration, community engagement, a community-based service delivery platform, and good program coverage and compliance. At a recent nutrition event during the World Bank-IMF's spring meeting, Rwanda set the goal of reducing the stunting rate from 38% to zero in the next three or four years.[5] Although Rwanda has effective policies in place and has made good progress as far as stunting reduction is concerned, with the current trajectory, additional efforts will still be required to meet the 2025 World Health Assembly goal of 24%.[6] Given the tremendous effect that reducing stunting has on the development of human capital, I hope that this ambitious goal is indeed achievable.

> "Stunting should be a development indicator rather than a nutrition target of SDG2"

Context matters

We must better understand the context children are living in and design interventions accordingly. We could also decide simply to wait until stunting goes away, while poverty declines, since a GDP (gross domestic product) increase of 10% is associated with a drop of 6% in the stunting rate.[7] I know this is not a good idea, but infrastructure programs – when funding is available for them as a consequence of increased GDP – may be as important as cumbersome BCC interventions. For example, in a large-scale program run by the Mexican government to improve housing, dirt floors were replaced with concrete.[8] The replacement reduced parasitic infection and diarrhea, and also reduced anemia and improved child cognitive development, but no effect on anthropometric indicators was observed. Simple-solution concrete floors are much easier to keep clean. Recognizing the many underlying factors of stunting, I am adamant that stunting should be a development indicator rather than a nutrition target of SDG2 (see *Sight and Life* 30 (1), 2016).

Over the last year, I have been involved in a group which has been studying the serum metabolome of stunted and non-stunted children in Malawi.[9–12] Stunting was associated with low levels of all essential amino acids, choline, carnitine, arachidonic acid, and docosahexaenoic acid. These nutrients are important for protein synthesis, growth (including long-bone growth), energy formation, and brain development. A common feature of all these nutrients is that they are abundantly present in animal-source foods, such as eggs, milk, fish, and meat. These results therefore corroborate the long-known benefits of nutrient-dense animal-source food for complementary feeding.[13]

Could EED, which causes epithelial atrophy, malabsorption, and inflammation, be key to the management of stunting? Our metabolomics work has demonstrated that confirmed EED is associated with major metabolic alterations in the Malawian cohort.[14] In addition to bacteria, viruses, and parasites, aflatoxin – one of the most potent and stable toxins on earth from the *Aspergillus* species – is thought to contribute to EED. Billions of people are exposed to aflatoxin daily, and the problem seems to be growing rather than diminishing. We also know little about what role an abnormal gut microbiota might play in EED. Beneficial gut barrier *Bifidobacteria* have been associated with improved weight gain in formula-fed infants.[15] It is therefore worrying that, according to the work of Michael Zimmermann's group from ETH Zurich, iron supplementation even in low doses reduces *Bifidobacteria* and *Lactobacilli*, and increases pathogenic bacteria such as *Escherichia* and *Shigella* in the microbiome of Kenyan children.[16] The good news, however, is that Daniela Paganini, who is from the same group, reported at Experimental Biology that the addition of prebiotic galacto-oligosaccharide can mitigate the negative iron effects on the microbiome, and that it even improves iron absorption.

> "(Micro)nutrient-dense animal-source food should be available for complementary feeding"

There is no simple solution to the issue of stunting. In addition to poverty alleviation, leading to a cleaner household environment, (micro)nutrient-dense animal-source food should be available for complementary feeding. It is also conceivable that some nutrients at higher levels or special additives such as pre- and probiotics, choline or essential fatty acids would provide an advantage for children living under environmentally compromised conditions. Albert Einstein once observed that "Everything should be made as simple as possible, but not simpler." Much more work remains to be done, both in the lab and in the field.

With warm regards,

Klaus Kraemer
Managing Director, *Sight and Life* Foundation

References

01. Arnold BF, Null C, Luby SP et al. Cluster-randomised controlled trials of individual and combined water, sanitation, hygiene and nutritional interventions in rural Bangladesh and Kenya: the WASH Benefits study design and rationale. BMJ Open 2013;3:e003476. DOI:10.1136/bmjopen-2013-003476.
02. The Sanitation Hygiene Infant Nutrition Efficacy (SHINE) Trial Team. The Sanitation Hygiene Infant Nutrition Efficacy (SHINE) Trial: Rationale, Design, and Methods. Clin Infect Dis 2015; 61(7):S685-S702. DOI: https://doi.org/10.1093/cid/civ844.
03. Danaei G, Andrews KG, Sudfeld CR et al. Risk Factors for Childhood Stunting in 137 Developing Countries: A Comparative Risk Assessment Analysis at Global, Regional, and Country Levels. PLoS Med 2016;13(11): e1002164. doi:10.1371/journal.pmed.1002164.
04. Hossain M, Choudhury N, Adib Binte Abdullah K et al. Evidence-based approaches to childhood stunting in low and middle income countries: a systematic review. Arch Dis Child 2017: pii: archdischild-2016-311050. doi: 10.1136/archdischild-2016-311050.
05. www.compact2025.org/2017/04/25/investing-innutrition-to-unlock-human-capital-and-economic-prosperity/" www.compact2025.org/2017/04/25/investing-innutrition-to-unlock-human-capital-and-economic-prosperity/ (accessed May 19, 2017).
06. https://ec.europa.eu/europeaid/sites/devco/files/nutritiongraphs-rwanda-2016_en.pdf.
07. Ruel MT, Alderman H, the Maternal and Child Nutrition Study Group. Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? Lancet 2013:382, doi: http://dx.doi.org/10.1016/S0140-6736(13)60843-0.
08. Cattaneo MD, Galiani S, Gertler PJ et al. Housing, health, and happiness. AEJ: Economic Policy 2009;1:75–105; www.aeaweb.org/articles.php?doi=10.1257/pol.1.1.75 (accessed May 19, 2017).
09. Semba RD, Shardell M, Sakr Ashour FA et al. Child stunting is associated with low circulating essential amino acids. EBioMedicine 2016;6:24–52.
10. Semba RD, Shardell M, Trehan I et al. Metabolic alterations in children with environmental enteric dysfunction. Sci Rep 2016;6:28009.
11. Semba RD, Zhang P, Gonzalez-Freire M et al. The association of serum choline with linear growth failure in young children from rural Malawi. Am J Clin Nutr 2016;104(1):191–7.
12. Semba RD, Trehan I, Li X et al. Environmental enteric dysfunction is associated with carnitine deficiency and altered fatty acid oxidation. EBioMedicine 2017;17:57–66.
13. Dror DK, Allen LH. The importance of milk and other animal-source foods for children in low-income countries. Food Nutr Bull 2011;32(3):227–43.
14. Semba RD, Shardell M, Trehan I et al. Metabolic alterations in children with environmental enteric dysfunction. Sci Rep. 2016;6:28009. doi: 10.1038/srep28009.
15. Gough EK, Prendergast AJ, Kuda E et al for the Sanitation Hygiene Infant Nutrition Efficacy (SHINE) Trial Team. Assessing the Intestinal Microbiota in the SHINE Trial. Clin Infect Dis 2015;61(7):S738–S744. doi: 10.1093/cid/civ850.
16. Jaeggi T, Kortman GA, Moretti D et al. Iron fortification adversely affects the gut microbiome, increases pathogen abundance and induces intestinal inflammation in Kenyan infants. Gut 2015;64(5):731