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Issue: Integrating Nutrition and Early Childhood Development Interventions

Water, sanitation, and hygiene (WASH), environmental enteropathy, nutrition, and early child development: making the links

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There is scarce research and programmatic evidence on the effect of poor water, sanitation, and hygiene (WASH) conditions of the physical environment on early child cognitive, sensorimotor, and socioemotional development. Furthermore, many common WASH interventions are not specifically designed to protect babies in the first 3 years of life, when gut health and linear growth are established. We review evidence linking WASH, anemia, and child growth, and highlight pathways through which WASH may affect early child development, primarily through inflammation, stunting, and anemia. Environmental enteropathy, a prevalent subclinical condition of the gut, may be a key mediating pathway linking poor hygiene to developmental deficits. Current early child development research and programs lack evidence-based interventions to provide a clean play and infant feeding environment in addition to established priorities of nutrition, stimulation, and child protection. Solutions to this problem will require appropriate behavior change and technologies that are adapted to the social and physical context and conducive to infant play and socialization. We propose the concept of baby WASH as an additional component of early childhood development programs.

Keywords: water; sanitation; hygiene; child development; nutrition; environmental enteropathy; stunting; anemia

Introduction

Child development refers to the ordered emergence of interdependent skills of sensorimotor, cognitivelanguage, and social-emotional functioning. 1 It is a complex phenomenon that is dependent on biological factors (such as nutrition), genetic factors, and the psychosocial and physical environment in which children are raised. Biological and psychosocial risk factors associated with poverty lead to inequalities in early child development (ECD), which undermine educational attainment, adult productivity, and contribute to intergenerational poverty.² Exposure to these multiple, co-occurring risks begins in early life and leads to widening disparities and developmental trajectories that cumulatively become more established.^{3,4} While recent decades have led to substantial gains in child survival, growth faltering

and developmental impairment remain pervasive in low- and middle-income countries.⁵ The multiple causes of poor growth and development will require an integrated approach to address the underlying risks.

Many risk factors for developmental deficits have been elucidated, and the potential role of hygiene must be considered in this context. The *Lancet* Child Development Series^{2,4} identified inadequate cognitive stimulation, stunting, iodine deficiency, and iron-deficiency anemia as key risks that prevent children from achieving their developmental potential. Other risk factors include intrauterine growth restriction (IUGR), malaria, lead exposure, human immunodeficiency virus (HIV) infection, institutionalization, and exposure to societal violence. There is emerging evidence of risks from prenatal maternal malnutrition, maternal stress,

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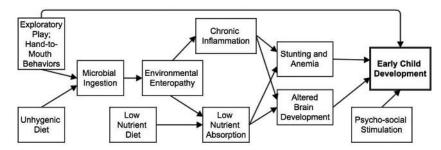


Figure 1. Summary of the relationships potentially linking poor hygiene in early childhood to child development.

and families affected by HIV. Evidence of adverse effects of environmental toxins on child development has been well documented^{6,7} with greatest attention toward lead, mercury, and polychlorinated biphenyls (PCBs), and more limited data on other heavy metals, solvents, and pesticides. Notably, in the two Child Development Series previously published in *The Lancet* there is no reference to any potential effect of hygienic and sanitary conditions of the physical environment on child development.^{2,4,8–10}

In this paper, we develop the argument that poor hygiene, resulting in microbial ingestion, is a risk factor for poor ECD. We review evidence on the links between clean water, sanitation, and hygiene (WASH), and stunting and anemia, which are known risk factors for child developmental deficits, and highlight how current WASH interventions fail to adequately protect children in the first 3 years of life. We advocate for a more holistic view of WASH oriented to babies in the first years of life and for the development of interventions targeted to this age group. The relationships that we focus on in this paper are summarized in Figure 1.

Undernutrition and poor child development

The relationship of malnutrition to child development is especially salient to our discussion of WASH because malnutrition may be the major mediator on the causal pathway between unhygienic environments and child development. Adequate nutrition during pregnancy and the first 2 years of life is necessary for normal brain development, which lays the foundation for future cognitive and social ability, school success, and productivity. An estimated 200 million children under the age of 5 years in low- and middle-income countries are at risk of not

achieving full developmental potential partly due to undernutrition. ¹⁰ Undernutrition affects brain development directly, and also affects physical growth, motor development, and physical activity, which may, in turn, influence brain development through both caregiver behavior and child interaction with the environment. ^{11,12} We focus on stunting and anemia in this review because of their plausible potential links to unhygienic environments, as postulated below.

Iron is a structural component of hemoglobin and is also essential to brain development through its roles in myelination and neurotransmission. ¹³ Iron-deficiency anemia in Costa Rican infants was associated with alterations in affect and activity behaviors that are linked to functional isolation. ¹⁴ Anemic infants were easily tired, hesitant, less attentive, less playful, and less exploratory of their environment. Longitudinal studies have demonstrated deficits in cognition and school achievement from 4 to 19 years of age in children who were anemic in their first 2 years of life. ^{15,16}

Linear growth failure is also associated with poor cognitive and motor development. In pooled analyses from five birth cohorts in low- and middle-income countries, stunting at 24 months was associated with a 0.9 year reduction in schooling, delay in school enrollment, and 16% increased risk of failing at least one grade in school.¹⁷ In a large-scale nutrition supplementation trial in Guatemala, provision of a high-energy protein supplement during the first 3 years of life significantly increased height gain in early life, intellectual performance at 11–26 years of age, and intelligence scores and wages in men at 26–42 years of age.¹⁸

Although stunting and anemia are both clearly linked to malnutrition, dietary interventions alone have not normalized growth or hemoglobin levels in children from low-income contexts. A recent review of 38 efficacy studies utilizing nutrient-dense foods and supplements with or without nutrition education¹⁹ showed an approximate 0.7 Z-score gain in height for age (HAZ) at best; this is only one-third of the average deficit in Asian and African children (-2.0 Z).²⁰ Similarly, about half of the burden of pediatric anemia is not resolved through iron interventions.²¹

WASH and malnutrition

Poor conditions of WASH are associated with 6.6% of the global burden of disease and disability, and 2.4 million deaths annually due to diarrhea, subsequent malnutrition, and their consequences. 22 Most of this disease burden falls on children in lowincome countries. Some authors have claimed that poor WASH accounts for as much as 50% of maternal and childhood underweight, primarily through the well-described synergy between diarrheal diseases and undernutrition, whereby one increases vulnerability to the other.²³ On the other hand, the Lancet Maternal and Child Nutrition Series estimated that hygiene and sanitation interventions implemented with 99% coverage would reduce diarrhea incidence by 30%, which would, in turn, reduce the prevalence of stunting by only 2.4% at 36 months of age.²⁴ Thus, although the role of water and sanitation in regard to diarrhea has widely been studied, the relative contribution of diarrhea to stunting remains controversial.

An association of improved water supply and sanitation with better growth outcomes in children has been reported from cross-sectional, case-control, and prospective cohort studies. Using nationally representative cross-sectional samples from eight countries, Esrey²⁵ estimated that an improvement in sanitation was associated with 0.06-0.65 increments in HAZ. Improved water sources were associated with smaller benefits to height that were only apparent when sanitation was also improved. In a longitudinal cohort design, Peruvian children at 2 years of age with the worst conditions for water source, water storage, and sanitation were 1.0 cm shorter than children with the best conditions.²⁶ Similarly, Bangladeshi children younger than 4 years of age living in households with good water quality, improved toilets, and hand washing facilities had a HAZ 0.54 (95% CI: 0.06-1.01) greater than children that were not living in those conditions.²⁷

Other studies have also reported improved growth outcomes in children from households with either improved water supply, sanitation, or both in different countries.^{28–30} In a large prospective cohort study in Sudan, the risk of stunting was lowest in children who came from households with water and sanitation (multivariate RR = 0.79, 95% CI: 0.69–0.90).³¹ Among children who were stunted at baseline, those who came from households with water and sanitation had a 17% greater chance of reversing stunting than their peers from households without either facility. The effects of water quality and sanitation on child growth are complex and they may involve interaction between the two factors. A synergistic effect of water and sanitation on growth was reported among young children in Lesotho in a prospective study³⁰ and in the Esrey study described above,²⁵ but a similar synergy was not found in Sudan.³¹ These inconsistent findings might be explained by differences in hygienic conditions of the physical environment of the child and personal hygiene practices.

The randomized trial evidence for hand washing as a specific component of WASH is growing, with a 2008 Cochrane review of four studies from low- or middle-income countries showing an overall 32% decrease (IRR 0.68, 95% CI: 0.52-0.90) in diarrhea from hand washing interventions.³² Of particular salience to our topic is a recent trial of hand washing in Karachi, Pakistan, that found a large protective effect on diarrhea,³³ no effect on stunting,³⁴ and yet significant and meaningful effects on child motor and cognitive development that were consistent across all domains assessed.34 The authors concluded that there are multiple pathways through infections and inflammation, nutrient intake and metabolism, and caregiver interactions that could link hand washing to child development.

In a study of functional consequences of mild to moderate malnutrition in a rural area of central Mexico, WASH indicators were strongly and significantly associated with growth (height, weight, and height for weight) in children aged 6 and 30 months, when socioeconomic status, household size, and dietary intake were controlled in the statistical analysis. Two recent studies further support the role of WASH in child stunting; in a recent cross-sectional study, poor household hygiene was associated with lower HAZ independent of infant feeding practices, recent morbidity, household food

security, and socioeconomic status in 2- to 5-yearold children in Ethiopia. ³⁶ Children from the dirtiest households had 0.32 lower adjusted mean HAZ than children from the cleanest households. The evidence of this association at an age when stunting is more complete was thought to be due to cumulative negative effects of caregiver hygiene behaviors and poor domestic hygiene.

Experimental evidence is needed to examine the causality of these observations. However, a recent nonrandomized experimental design demonstrated that in a food-insecure region in Ethiopia, children aged 6-36 months from a WASH intervention area gained 0.33 Z score more in mean HAZ over 5 years (P=0.02) than children from three comparison villages who did not receive any additional intervention; all areas received the governmental Productive Safety Net Program.³⁷ The WASH intervention was comprehensive, including protected water supply, sanitation education, soap use, hand-washing practices, sanitary facility construction, cleanliness of the house, construction of separate housing for animals, and keeping water clean. In the same study, areas allocated to receive nutrition education or health education without WASH did not show effects on child growth.

WASH and environmental enteropathy

How does WASH exert these effects on child growth and development? Two longstanding observations have been newly integrated in a hypothesis that poor sanitation and hygiene cause stunting not only through diarrhea, but also through the subclinical condition, environmental enteropathy (EE).³⁸ The first observation is that the introduction of antibiotics to chicken feed led to a trophic response in poorly growing chicks reared in unhygienic and unsanitary environments. Antibiotic-treated chicks reared in dirty environments attained normal rates of growth and skeletal muscle accretion.^{39,40} However, chicks raised in hygienic and sanitary conditions had high rates of growth and skeletal muscle accretion, compared to their dirty counterparts. Adding antibiotics to feeds did not have an additional effect on growth. Subsequently, more research on poultry showed that chronic immune stimulation with consequent mediation of catabolic and antitrophic metabolic processes was responsible for growth impairments in chicks raised in unhygienic environments. 41,42

Solomons *et al.*⁴³ suggested that a similar phenomenon of impaired growth occurs in children growing in poor hygiene and sanitation conditions. Even when children are not apparently infected, the microbial-laden environment may provide a low-level chronic immune stimulation with catabolic consequences that result in poor growth.

The second observation concerns enteropathy and growth failure in African children. In The Gambia, growth faltering was not associated with dietary inadequacy or clinical diarrhea but with the ratio of urinary lactulose to mannitol—an indicator of subclinical intestinal permeability. This accounted for 39% of ponderal and 43% of linear growth failure.44 A subsequent study by the same investigators demonstrated that translocation of immunogenic macromolecules across a permeable gut leads to stimulation of the systemic immune processes and subsequent growth impairment.⁴⁵ Intestinal permeability of the Gambian infants was abnormal and associated with growth impairment (r = -0.41, P < 0.001). Elevated plasma concentrations of endotoxin and immunoglobulin (Ig) G-endotoxin core antibody were also associated with growth impairment and measures of mucosal enteropathy. This research was seminal in showing mechanisms strongly linking chronic asymptomatic mucosal enteropathy to growth failure.

Humphrey³⁸ has recently hypothesized that exposure to larger quantities of fecal bacteria due to poor sanitation and hygiene is the cause of this enteropathy, now termed as EE. She also hypothesized that the primary causal pathway from poor sanitation and hygiene to stunting is EE and not diarrhea. EE is an energy-intensive subclinical condition, characterized by villous atrophy, crypt hyperplasia, increased permeability, inflammatory cell infiltrate, and modest malabsorption.⁴⁶ These processes impair absorptive and barrier functions of the small intestine mucosa lining, causing growth to falter.

Chronic immune stimulation arising from EE may also be an underlying cause of anemia. Anemia of inflammation is the second most prevalent form of anemia, after iron-deficiency anemia. ⁴⁷ Inflammation disturbs iron homeostasis by reducing iron absorption and by diversion of iron from the circulation into storage sites of the reticuloendothelial system, subsequently limiting availability of iron for erythroid progenitor cells and iron-restricted erythropoiesis. ⁴⁷ Inflammation also reduces plasma

retinol, which is essential to erythropoiesis. These processes cause anemia by inhibiting iron uptake by erythroblasts, and may also affect iron utilization by other target tissues critical for child development, including muscle and brain.

Implications for children's play and feeding environment in low-income households

Contamination of the domestic environment with animal and human feces in poor households is ubiquitous. Human and animal feet carry feces deposited in the open, bringing diverse microbes and pathogens into the domestic environment and the immediate vicinity of infants and young children.⁴⁸ Infants and young children are frequently exposed to poultry feces in poor-resource settings. In Peruvian households, toddlers' hand contact with poultry feces occurred 2.9 (SD 3.0) times, on average, and an average of 3.9 (SD 4.6) feces-to-mouth episodes was observed per household in 12 hours. 49 Campylobacter jejuni, a pathogenic bacterium important in causing dysenteric diarrhea, was isolated in chicken feces up to 48 h after deposition in these Peruvian slums. 49 Similarly, fecal contamination of infant and young children's play areas was reported in 66% of households in Bangladesh (Zeitlin et al., as cited in Ref. 49). About half of the mothers reported seeing a child touch or eat animal feces in the previous two weeks.

In a recent in-depth observation study of 23 households in rural Zimbabwe, three infants actively ingested 11.3 \pm 9.2 handfuls of soil (mean \pm SD) and two ingested chicken feces 2 ± 1.4 times in 6 hours.⁵⁰ Infant play and feeding areas were frequently contaminated with fecal bacteria in these households. The majority of households' kitchen floors (82%) and soil samples (64-82%) were contaminated with Escherichia coli, the most definitive indicator of fecal contamination. Exploratory ingestion of soil and chicken feces was identified as the predominant fecal-oral transmission pathway of bacteria in infants and toddlers (3-18 months old), due to the very high bacterial load of these substances.³⁶ In an earlier study in rural Zimbabwe, a nosocomial pathogenic bacteria, Clostridium difficile, was isolated in 37% of soil samples, 17% of chicken feces, and 6% of water samples collected from 146 households.⁵¹ More than half of these isolates in soil and chicken feces were toxigenic strains. Using specific molecular techniques, human Bacteriodales, pathogenic *E. coli*, enterovirus, and rotavirus genes were detected in soil samples from rural households that used pit latrines in Tanzania.⁵² Soil samples collected from inside the house and food preparation areas had a higher concentration of fecal indicator bacteria and general *Bacteriodales* compared to soil collected near or inside the latrine. There were no significant differences between the level of fecal contamination of the household environment between households using pit latrines with a concrete slab (improved sanitation) and those using one without a concrete slab.⁵²

Thus, fecal contamination of children's play and feeding environments is a constant and cumulative health risk during the critical window of a child's growth and development. By combining behavioral ingestion data and microbiological data from Zimbabwe, it was estimated that ingestion of homestead soil amounts to *E. coli* intake similar to or greater than that from untreated drinking water, and that *E. coli* intake from ingestion of chicken feces is 4000 times greater than that from either untreated drinking water or soil.⁵⁰

Several recent studies have also shown that infant and young children's foods are frequently contaminated with fecal indicator bacteria, especially when those foods have been stored and fed at later times. Infant or young children's foods were heavily contaminated in studies in peri-urban Mali, Bangladesh, and Zanzibar, Tanzania. ^{53–56} Surprisingly, infant foods were not found to contain *E. coli* in rural Zimbabwe, although about half of household drinking water samples were contaminated. ⁵⁰

Proposed causal chain from poor hygiene to ECD

We hypothesize that poor cognitive, sensorimotor, and socioemotional development are mediated in part through anemia of inflammation and stunting resulting from poor gut health and chronic immune stimulation, in addition to other wellestablished causes of developmental deficits. The intermediate biological mechanisms underlying the concomitant environmental risks and diet deficiencies on the one end, and a child's developmental outcomes on the other, interact and overlap in a complex manner. For instance, clinical or subclinical inflammation prevents iron absorption in contexts with limited micronutrient-rich foods. Anemia induced by inflammation may also contribute to poor

mother-child interaction behavior, which is critical to cognitive and socioemotional development in children. Even though some of the diet-induced deficits in brain development may be ameliorated by positive child and caregiving experiences (e.g., psychosocial stimulation), the constant state of immune stimulation, even in the absence of overt clinical disease, could have cumulative effects with adverse developmental outcomes in contexts of poverty.

These nutritional and environment risks⁵⁷ retard growth and affect the mother-child behavior patterns that are critical to better development. Normal development requires a balance between time spent with caregivers and time spent freely exploring the environment.14 As described earlier, anemic infants are more likely to be hesitant of exploring their environments and to cling to their caregivers. Poor physical growth may also influence development through caregiver behavior and the child's interaction with the environment.⁵⁸ Caregivers may treat children who are small for their age as younger than they actually are, resulting in less appropriate stimulation and therefore altered brain development. In addition, children with such a constant state of immune stimulation may also be irritable or withdrawn, eliciting negative treatment from caregivers. Lower activity due to frequent illness or reduced aerobic capacity could limit a child's exploratory behavior and initiation of interactions with the caregiver. These mechanisms may contribute to delayed motor and cognitive development as seen in children with protein-energy malnutrition⁵⁹ and iron-deficiency anemia.13

The programmatic gap between WASH and ECD

Integrated ECD interventions, such as the Essential Package developed by CARE, Save the Children, and other key stakeholders, 60 and Care for Development developed by the United Nations Children's Fund (UNICEF) and World Health Organization (WHO), 61 acknowledge the importance of preventing frequent child illnesses and promoting clean water and positive hygiene and sanitation practices. These programs do not, however, include more specific, holistic, age-targeted approaches to preventing the microbial burden encountered by young children in their play and feeding environments.

WASH interventions have focused on improved sanitation, point-of-use water treatment, and maternal hand washing. None of these interventions address the important vectors of soil, poultry feces, and infant foods highlighted in this review. Smallscale pilot interventions for food hygiene in young child feeding have been implemented successfully in Bangladesh and Mali,53,54 using critical control points as the framework for training mothers. Fecal indicator bacteria in children's foods decreased substantially after training mothers. For example, in peri-urban Mali, mothers were trained on handwashing with soap, and safe food preparation, heating, cooling, and storage.⁵⁴ Both of these studies were small (30 mothers) and relatively short term (3 months), and the authors called for larger trials to test feasibility and efficacy at scale.

We know even less about how to interrupt the ingestion of contaminated soils and animal feces. Hand washing interventions usually focus on hand washing by mothers and other caregivers, but most commonly the hands that enter an infant's mouth are his or her own.⁵⁰ Many of these key events for baby hand washing occur frequently and at unpredictable times, and are difficult to keep track of. Infants and young children in resource-poor contexts frequently crawl on contaminated soil and surfaces, as they explore dirty objects from the ground. In addition to a lack of knowledge about the environmental risks that babies are exposed to, most caregivers have time constraints and carry out multiple tasks concurrently, limiting their capacity to attend to such random hand washing events.

Interventions to separate infants and toddlers from free-range poultry have so far not been successful. Corralling poultry was intermittent in a shanty town in Peru even with extensive orientation and technical support, and proved to be ineffective in separating children from contact with chicken feces.⁵⁷ Commonly perceived barriers to corralling chickens at all times include the commitment and high cost of feeding the birds, building and maintaining corrals, and purchasing vaccines. Many growers in this setting attributed human characteristics to birds: they want to run, play, eat food they like, scratch (for chickens), and swim (for ducks). They also held the common belief that freerange chickens have better tasting eggs and meat. In short, existing WASH interventions are not designed to interrupt the primary vectors of fecal-oral transmission for children within the first 2 years of life, the critical window for stunting, anemia, and poor child development.

Compounding these challenges is the need for young children to experience the world in a sensorimotor way through their bodies and interactions with the environment. In recent decades, research has strengthened knowledge about the connection between play and childhood development, especially during early and middle childhood. Jean Piaget⁶² and Lev Vygotsky⁶³ were among the first to link play with cognitive development. Stimulating play affords young children the opportunity to master skills in memory, information processing, and other cognitive abilities necessary for learning.⁶⁴ Studies by Bodrova and Leong show a direct link between play in young children and memory, school adjustment, oral language development, and improved social skills.⁶⁵ Thus, quality play in early childhood is a prelude to positive functioning later on in development.66

Interaction with the environment in play is a critical area for increased attention in environmental design research and innovation in support of early childhood development. Infants experience their environment through sensory perception and exploration, which, in turn, contributes to the learning and development of important cognitive concepts.⁶⁶ Visual perceptual information contributes to the development of infants' cognitive understanding of motion, depth, and event sequencing.⁶⁷ Cognitive and motor development takes place through manual activities and tactile exploration within the environment. The environment provides rich sources of information about shape, texture, consistency, object properties, and the development of object representations and auditory signals from environmental sounds contribute to word learning and joint attention.⁶³ The unique contribution of sensory stimuli from each of these sensory modalities maximizes perceptual learning and cognitive development and occurs specifically through infants' play and exploration of their microsystem environment. Children learn about their world through exploration and consolidate that information in their play.66 Thus, interaction with the environment through play supports the total development—social, cognitive, affective, emotional, and physical—of all children.66 Yet, for many young children in poor households, the environment is a substantial microbiological threat to health

Designing a protective space: considerations for ECD

To address babies' environmental hygiene, we propose a clean and protective play space, designed to protect, stimulate, and promote learning for babies in the context of their culture and family structure. The play space should allow active and socially relevant play, which is required for healthy brain growth. New baby products, such as a play space, should be provided with education to promote acceptability and appropriate adoption. Care should be taken to educate mothers and other caregivers about the need to maintain healthy social interaction in the context of such an intervention, and the effects on child physical, social, and cognitive development will need to be evaluated.

Child play happens in a cultural context, and while there are universal developmental schedules, play unfolds around culturally influenced behaviors. 66 Therefore, play spaces must be responsive to the dominant adult concepts of childrearing and social interaction.⁶⁹ Culturally variable dimensions include the participation of specific play partners, the extent of child initiations of social pretend play with caregivers, the various functions of social pretend play in interaction, and specific themes. 66,70 We introduced American playpens in a small pilot study in Zimbabwe but elicited some negative reactions from the community for a number of reasons, primarily related to cultural concepts of play. For instance, mothers commented that their "child will not have room to experiment," and "people need to understand why the child is being kept in a playpen," in reference to the general cultural practice of not using any child containment. In the focus group, one mother was adamant about not using a playpen, expressing concern about her child hurting himself. She said that the American playpens were "too small... my baby needs all the yard to explore."

From the hygiene perspective, a play space is only helpful if it can be kept clean. Both floor and walls must be considered in this respect, and the walls need to be designed to keep chickens out. Household members, especially those who directly care for the child, must perceive the potential benefits to outweigh the burdens of any new technology. For a

mother (or other caregiver) with many demands on her time, the convenience of a play space (or inconvenience of keeping it clean) is likely to be a major factor influencing use.

The safety of protective play space design is also imperative. International standards for full-size baby cribs can provide a basis for safety. Safe designs will follow applicable safety specifications, performance requirements, structural integrity, use of nontoxic materials, and design requirements to prevent choking, pinching, shearing, entrapment, cuts, or entanglement on elements such as corner postextensions.⁷¹

Research, program, and policy implications

The research needed to act on the link between WASH and ECD will be multidisciplinary and inquiring of local realities and conceptions. Expertise in hygiene, design, nutrition, food science, and child development needs to be brought together with cultural concepts of play and childcare. Ethnographic methods employed in each of these fields of research are powerful tools for understanding the problem and its solutions.

Holistic inquiries into WASH are not new. In studies conducted 25 years ago, 35 investigators made the intriguing discovery that the rural Mexican women who were participating in the longitudinal growth study already had a holistic concept of WASH practices, which they termed as organizada. According to key informant interviews, women who had a clean house, whose young children were neat, clean in appearance, and wore clean clothes, and whose older children went to school and did their homework were organizada. Whether they covered their dishes to keep off flies, had clean work surfaces in the kitchen area, washed their children's hands and face before and after they ate, had clean finger nails—all of these were characteristic of women who were organizada. It is unlikely that rural Mexican women in the 1980s were unique in articulating a cultural construct we refer to as WASH-which also includes elements, such as homework, that do not fit our idea of WASH. It is probable that there are many other communities and cultures in which emic interpretations and formulations of WASH are to be found. Identifying these and examining the areas of overlap and nonoverlap with the concepts of researchers and intervention designers is a first step

toward sharpening our own conceptual frameworks and improving the effectiveness of interventions.

We propose the concept of baby WASH as an additional focal component of early childhood development programs, motivated by the dual concerns of EE and infectious diseases. The essential goal of baby WASH is to interrupt the key fecal-oral vectors of babies' hands and hand-to-mouth activity, paying attention to animal feces as well as human feces. This emphasis on the baby does not replace more general household-level interventions, which may reduce overall contamination of the household environment, but rather targets WASH interventions to the individual of most concern to ECD and nutrition: the very young child. Research by our group and others leads us to conclude that baby WASH interventions require baby hand washing at key times and creation of a hygienic and protective play environment, in addition to hygienic infant feeding and household hand washing and sanitation interventions. ECD programs and interventions should include appropriate behavior change and technologies that are adapted to the social and physical context, and conducive to infant play and socialization. Care should be taken to preserve infants' exploration of their environment when designing such technologies.

The following set of research questions is key to understanding how to integrate WASH most effectively into nutrition and ECD programs:

- Is microbial ingestion a primary cause of EE in infants and young children?
- If so, does microbial ingestion from animal feces matter equally as much as human feces?
- Is the concept of a protective play space culturally, socially, and economically feasible for rural low-income households?
- Would such a play space decrease microbial ingestion?
- If so, how large is the protective effect of a play space compared to other baby WASH interventions such as infant food hygiene?
- How can behavior-change education be developed to support the hygienic protection of infants and young children, with or without new technologies such as play spaces?
- Does effective baby WASH programming contribute independently or synergistically to

child development, along with nutrition and stimulation?

On the basis of both empirical and theoretical evidence, we have argued that the specific components of WASH interventions need to be expanded in support of ECD. Child health, nutrition, growth, and development are interlinked, and are influenced by the hygiene of the immediate environment in which the baby begins to explore the world. In addition to expanding the scope of interventions, it is also important to broaden the conceptual structure of WASH as an aspect of child nutrition and development interventions, and not simply as the sum of toilets, caregiver hand washing, and water purification. WASH should be defined holistically as broadly encompassing the hygiene-related aspects of the physical and behavioral environment in which children are being raised. At the household level, where it directly affects children's growth and development, effective WASH management is a composite of multiple factors. The interactions of these various factors produce the WASH conditions and processes of concern for research and intervention planning and implementation.

Conflicts of interest

The authors declare no conflicts of interest.

References

- 1. UNICEF. 2006. Programming Experiences in Early Child Development. New York: UNICEF.
- Engle, P.L., M.M. Black, J.R. Behrman, et al. 2007. Strategies to avoid the loss of developmental potential in more than 200 million children in the developing world. Lancet 369: 229–242.
- 3. Evans, G.W. & L.A. Marcynyszyn. 2004. Environmental justice, cumulative environmental risk, and health among lowand middle-income children in upstate New York. *Am J Public Health* **94**: 1942–1944.
- Walker, S.P., T.D. Wachs, J.M. Gardner, et al. 2007. Child development: risk factors for adverse outcomes in developing countries. Lancet 369: 145–157.
- UNICEF. 2012. Committing to Child Survival: A Promise Renewed. New York: UNICEF.
- Evans, G.W. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423–451.
- Lanphear, B.P., C.V. Vorhees & D.C. Bellinger. 2005. Protecting children from environmental toxins. PLoS Med 2: e61.
- Engle, P.L., L.C. Fernald, H. Alderman, et al. 2011. Strategies for reducing inequalities and improving developmental outcomes for young children in low-income and middleincome countries. Lancet 378: 1339–1353.

- Walker, S.P., T.D. Wachs, S. Grantham-McGregor, et al. 2011. Inequality in early childhood: risk and protective factors for early child development. *Lancet* 378: 1325–1338.
- Grantham-McGregor, S., Y.B. Cheung, S. Cueto, et al. 2007. Developmental potential in the first 5 years for children in developing countries. *Lancet* 369: 60–70.
- Pollitt, E., K.S. Gorman, P.L. Engle, et al. 1995. Nutrition in early life and the fulfillment of intellectual potential. J Nutr 125: 11115–1118S.
- Olney, D.K., E. Pollitt, P.K. Kariger, et al. 2007. Young Zanzibari children with iron deficiency, iron deficiency anemia, stunting, or malaria have lower motor activity scores and spend less time in locomotion. J Nutr 137: 2756–2762.
- Lozoff, B. 1998. Explanatory mechanisms for poorer development in iron-deficient anemic infants. In *Nutrition*, *Health and Child Development*. 162–178. Washington, DC: Pan American Health Organization.
- Lozoff, B., N.K. Klein, E.C. Nelson, et al. 1998. Behavior of infants with iron-deficiency anemia. Child Dev 69: 24–36.
- Lozoff, B., E. Jimenez & J.B. Smith. 2006. Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. Arch Pediatr Adolesc Med 160: 1108–1113.
- Lozoff, B., J. Beard, J. Connor, et al. 2006. Long-lasting neural and behavioral effects of iron deficiency in infancy. Nutr Rev 64: S34–43; discussion S72–91.
- Martorell, R., P. Melgar, J.A. Maluccio, et al. 2010. The nutrition intervention improved adult human capital and economic productivity. J Nutr 140: 411–414.
- Ramirez-Zea, M., P. Melgar & J. A. Rivera. 2010. INCAP Oriente longitudinal study: 40 years of history and legacy. J Nutr 140: 397–401.
- Dewey, K.G. & S. Adu-Afarwuah. 2008. Systematic review of the efficacy and effectiveness of complementary feeding interventions in developing countries. *Matern Child Nutr* 4(Suppl 1): 24–85.
- Victora, C.G., M. de Onis, P.C. Hallal, et al. 2010. Worldwide timing of growth faltering: revisiting implications for interventions. *Pediatrics* 125: e473–480.
- Stoltzfus, R.J., L. Mullany & R.E. Black. 2002. Iron deficiency and global burden of disease. In Comparative Quantification of Health Risks: The Global and Regional Burden of Disease due to 25 Selected Major Risk Factors. Cambridge: Harvard University Press.
- Pruss-ustun, A.B.R., F. Gore & J. Bartram. 2008. Safer Water, Better Health: Costs, Benefits and Sustainability of Interventions to Protect and Promote Health. Geneva: World Health Organization.
- World Bank. 2008. Environmental Health and Child Survival: Epidemiology, Economics, Experience. Washington, DC: World Bank.
- Bhutta, Z.A., T. Ahmed, R.E. Black, et al. 2008. What works? Interventions for maternal and child undernutrition and survival. Lancet 371: 417–440.
- 25. Esrey, S.A. 1996. Water, waste, and well-being: a multicountry study. *Am J Epidemiol* **143**: 608–623.
- Checkley, W., R.H. Gilman, R.E. Black, et al. 2004. Effect of water and sanitation on childhood health in a poor Peruvian peri-urban community. *Lancet* 363: 112–118.

- Lin, A., B.F. Arnold, S. Afreen, et al. 2013. Household environmental conditions are associated with enteropathy and impaired growth in rural Bangladesh. Am J Trop Med Hyg 89: 130–137.
- Daniels, D.L., S.N. Cousens, L.N. Makoae & R.G. Feachem. 1991. A study of the association between improved sanitation facilities and children's height in Lesotho. Eur J Clin Nutr 45: 23–32
- Magnani, R.J., N.B. Mock, W.E. Bertrand & D. C. Clay. 1993. Breast-feeding, water and sanitation, and childhood malnutrition in the Philippines. *J Biosoc Sci* 25: 195–211.
- Esrey, S.A., J.P. Habicht & G. Casella. 1992. The complementary effect of latrines and increased water usage on the growth of infants in rural Lesotho. *Am J Epidemiol* 135: 659-666.
- Merchant, A.T., C. Jones, A. Kiure, et al. 2003. Water and sanitation associated with improved child growth. Eur J Clin Nutr 57: 1562–1568.
- Ejemot-Nwadiaro RIE, J.E., M.M. Meremikwu & J.A. Critchley. 2008. Hand washing for preventing diarrhoea. *Cochrane Database Syst Rev* 23: CD004265.
- Luby, S.P., M. Agboatwalla, J. Painter, et al. 2006. Combining drinking water treatment and hand washing for diarrhoea prevention, a cluster randomised controlled trial. Trop Med Int Health 11: 479–489.
- Bowen, A., M. Agboatwalla, S. Luby, et al. 2012. Association between intensive handwashing promotion and child development in Karachi, Pakistan: a cluster randomized controlled trial. Arch Pediatr Adolesc Med 166: 1037–1044.
- Allen, L.H., A. Chavez & G.H. Pelto. 1987. The Mexico Project. Collaborative Research Support Program on Food Intake and Human Function. Final Report. 398 Storrs, CT: University of Connecticut.
- 36. Ngure, F. 2012. Environmental Hygiene, Food Safety and Growth in Less than Five Year Old Children in Zimbabwe and Ethiopia. 133 Ithaca: Cornell University.
- Fenn, B., A.T. Bulti, T. Nduna, et al. 2012. An evaluation of an operations research project to reduce childhood stunting in a food-insecure area in Ethiopia. Public Health Nutr 15: 1746–1754.
- Humphrey, J.H. 2009. Child undernutrition, tropical enteropathy, toilets, and handwashing. *Lancet* 374: 1032–1035.
- Libby, D.A. & P.J. Schaible. 1955. Observations on growth responses to antibiotics and arsonic acids in poultry feeds. *Science* 121: 733–734.
- Coates, M.E., R. Fuller, G.F. Harrison, et al. 1963. A comparison of the growth of chicks in the Gustafsson germ-free apparatus and in a conventional environment, with and without dietary supplements of penicillin. Br J Nutr 17: 141–150.
- 41. Klasing, K.C. & B.J. Johnstone. 1991. Monokines in growth and development. *Poult Sci* **70**: 1781–1789.
- Klasing KCj, B.J., B.N. Benson. 1991. Implications of an immune response on growth and nutrient requirements of chicks. In *Recent Advances in Animal Nutrition*. W.C. Haresign & D.J.A. Cole, Eds.: 135–146. Oxford: Butterworth-Heinemann.
- Solomons, N.W., M. Mazariegos, K.H. Brown & K. Klasing.
 1993. The underprivileged, developing country child: envi-

- ronmental contamination and growth failure revisited. *Nutr Rev* **51**: 327–332.
- Lunn, P.G., C.A. Northrop-Clewes & R.M. Downes. 1991.
 Intestinal permeability, mucosal injury, and growth faltering in Gambian infants. *Lancet* 338: 907–910.
- Campbell, D.I., M. Elia & P.G. Lunn. 2003. Growth faltering in rural Gambian infants is associated with impaired small intestinal barrier function, leading to endotoxemia and systemic inflammation. *J Nutr* 133: 1332–1338.
- Haghighi, P. & P.L. Wolf. 1997. Tropical sprue and subclinical enteropathy: a vision for the nineties. *Crit Rev Clin Lab Sci* 34: 313–341.
- Weiss, G. & L.T. Goodnough. 2005. Anemia of chronic disease. N Engl J Med 352: 1011–1023.
- Curtis, V., S. Cairncross & R. Yonli. 2000. Domestic hygiene and diarrhoea – pinpointing the problem. *Trop Med Int* Health 5:22–32
- Marquis, G.S., G. Ventura, R.H. Gilman, et al. 1990. Fecal contamination of shanty town toddlers in households with non-corralled poultry, Lima, Peru. Am J Public Health 80: 146–149.
- Ngure, F.M., J.H. Humphrey, M.N. Mbuya, et al. 2013. Formative research on hygiene behaviors and geophagy among infants and young children and implications of exposure to fecal bacteria. Am J Trop Med Hyg 89: 709–716.
- Simango, C. 2006. Prevalence of Clostridium difficile in the environment in a rural community in Zimbabwe. *Trans R* Soc Trop Med Hyg 100: 1146–1150.
- 52. Pickering, A.J., T.R. Julian, S.J. Marks, *et al.* 2012. Fecal contamination and diarrheal pathogens on surfaces and in soils among Tanzanian households with and without improved sanitation. *Environ Sci Technol* **46**: 5736–5743.
- Islam, M.S., Z.H. Mahmud, P.S. Gope, et al. 2013. Hygiene intervention reduces contamination of weaning food in Bangladesh. Trop Med Int Health 18: 250–258.
- Toure, O., S. Coulibaly, A. Arby, et al. 2013. Piloting an intervention to improve microbiological food safety in Peri-Urban Mali. Int J Hyg Environ Health 216: 138–145.
- Islam, M.A., T. Ahmed, A.S. Faruque, et al. 2012. Microbiological quality of complementary foods and its association with diarrhoeal morbidity and nutritional status of Bangladeshi children. Eur J Clin Nutr 66: 1242–1246.
- Kung'u, J.K., K.J. Boor, S.M. Ame, et al. 2009. Bacterial populations in complementary foods and drinking-water in households with children aged 10–15 months in Zanzibar, Tanzania. J Health Popul Nutr 27: 41–52.
- Harvey, S.A., P.J. Winch, E. Leontsini, et al. 2003. Domestic poultry-raising practices in a Peruvian shantytown: implications for control of *Campylobacter jejuni*-associated diarrhea. Acta Trop 86: 41–54.
- Levitsky, D.A. & R.H. Barnes. 1972. Nutritional and environmental interactions in the behavioral development of the rat: long-term effects. *Science* 176: 68–71.
- Pollitt, E., K.S. Gorman, P.L. Engle, et al. 1993. Early supplementary feeding and cognition: effects over two decades. Monographs of the Society for Research in Child Development. Serial No. 235, 58, No. 7.
- Inter-Agency Taskforce on HIV and ECD. 2012. The Essential Package: Holistically Addressing the Needs of Young

- Vulnerable Children and Their Caregivers Affected by HIV and AIDS. Consultative Group on Early Childhood Care and Development.
- 61. UNICEF WHO. 2012. Care for Child Development.
- 62. Piaget, J. 1962. Play, Dreams, and Imitation in Childhood. New York: Norton.
- Vygotsky, L. 1978. Interaction between learning and development. In Mind in Society: The Development of Higher Psychological Processes. M.J. Cole, V. John-Steiner, S. Scribner & E. Souberman, Eds.: 79–91. Boston: Harvard University Press
- 64. Piek, J.P., L. Dawson, L.M. Smith & N. Gasson. 2008. The role of early fine and gross motor development on later motor and cognitive ability. *Hum Mov Sci* 27: 668–681.
- Bodrova, E. & D.J. Leong. 2003. The importance of being playful. *Educ Leadership* 60: 50–53.

- Johnson, J.E., J. F. Christie & F. Wardle. 2005. Play, Development, and Early Education. Boston: Pearson Education, Inc.
- 67. Lerner, R. 1986. Concepts and Theories of Human Development. New York: Random House.
- 68. Brown, S.V.C. 2009. Play. How It Shapes the Brain, Opens the Imagination, and Invigorates the Soul. New York: Penguin.
- Haight, W.L., X.L. Wang, H.H. Fung, et al. 1999. Universal, developmental, and variable aspects of young children's play: a cross-cultural comparison of pretending at home. Child Dev 70: 1477–1488.
- 70. Gray, P. 2007. The special value of children's age-mixed play. *Am J Play* **3**: 500–522.
- 71. Anonymous. 2012. Standard consumer safety specification for non-full-size baby cribs/play yards. In *ASTM Standard F406*. West Conshohocken, PA: ASTM International.