Impact of contaminated household environment on stunting in children aged 12–59 months in Burkina Faso

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ABSTRACT

Background Stunting affects 165 million children worldwide, with repercussions on their survival and development. A contaminated environment is likely to contribute to stunting: frequent faecal-oral transmission possibly causes environmental enteropathy, a chronic inflammatory disorder that may contribute to faltering growth in children. This study's objective was to assess the effect of contaminated environment on stunting in Burkina Faso, where stunting prevalence is persistently high.

Methods Panel study of children aged 1–5 years in Kaya. Household socioeconomic characteristics, food needs and sanitary conditions were measured once, and child growth every year (2011–2014). Using multiple correspondence analysis and 12 questions and observations on water, sanitation, hygiene behaviours, yard cleanliness and animal proximity, we constructed a 'contaminated environment' index as a proxy of faecal-oral transmission exposure. Analysis was performed using a generalised structural equation model (SEM), adjusting for repeat observations and hierarchical data.

Results Stunting (<2 SD height-for-age) prevalence was 29% among 3121 children (median (IQR) age 36 (25–48) months). Environment contamination was widespread, particularly in rural and peri-urban areas, and was associated with stunting (prevalence ratio 1.30; p=0.008), controlling for sex, age, survey year, setting, mother's education, father's occupation, household food security and wealth. This association was significant for children of all ages (1–5 years) and settings. Lower contamination and higher food security had effects of comparable magnitude.

Conclusions Environment contamination can be at least as influential as nutritional components in the pathway to stunting. There is a rationale for including interventions to reduce environment contamination in stunting prevention programmes.

INTRODUCTION

Stunting affects about 165 million children worldwide and one of three children under five in sub-Saharan Africa (SSA).[1] Stunting is a measure of children's vulnerability: stunted children have impaired immunity, are more susceptible and vulnerable to infections,[2] and are at higher risk of developing metabolic diseases.[3] They also risk delayed physical and cognitive development, as well as lower productivity as adults.[4 5]

Intrauterine growth retardation and non-adequate caloric and nutrient uptake and intake are the two commonly cited causes of faltering child growth.[6] The pathogenesis of stunting is complex and not yet well understood, but increasing evidence[7–11] suggests that environmental enteropathy disorder (EED) might play a significant role in children with faltering growth. Environmental enteropathy, a subclinical condition of the small intestine, alters gut permeability and reduces intestinal absorption of nutrients.[11–13] Its causes have not yet been completely identified, but continuous exposure to enteric pathogens via faecal-oral transmission seems key to its pathogenesis.[14–16]

Children living in developing countries are heavily exposed to contact and ingestion of faecal pathogens.[17 18] Transmission occurs mainly through the so-called Five F's: food, fluids, flies, fingers and field/floors.[19] Microbiological studies have shown the presence of enteric pathogens in household soil, fomites, stored water, utensils and hands of caregivers in rural Tanzania, Zimbabwe and Bangladesh.[9 17 18] Geophagy and hand-to-mouth contacts are also frequent among children aged 6–24 months, which is also the most critical window for stunting development.[20] Geophagy was reported in up to 28% of children aged 6–30 months in a recent study in Bangladesh,[9] and frequent hourly contact with soil, hands or objects, such as utensils or toys, has been observed in young children in Zimbabwe.[21] Stunting has been found to be associated with water and/or sanitation access[8 22–24] and caregivers' handwashing.[10 25] Proximity of animals[26] and reported geophagy[9] have also been identified as markers of EED and linked to higher risk of stunting.

With some exceptions,[22 24] most studies published until now on faecal-oral transmission and stunting are cross-sectional. The causal forces at work are still poorly understood. We also know very little concerning the overall effect on child growth of a whole spectrum of factors hindering hygiene and potentially causing exposure to faecal pathogens.

This longitudinal study is intended to improve understanding of the role of faecal-oral transmission on stunting. Its objective is to isolate and estimate the magnitude of the effects of a contaminated environment favouring faecal-oral transmission in a cohort of children aged 1–5 years in Burkina Faso, where stunting remains extremely high and only modest improvements have been observed over the past 20 years.[27]

Structural equation model (SEM) analysis was used in order to take into account the complex systems of confounders and mediating factors involved in the emergence of stunting. Models were based on the following hypotheses derived from the

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literature and experts' suggestions (figure 1): (1) stunting is influenced by two endogenous proximal determinants—contaminated environment and ability to fulfil nutrition needs—which are themselves influenced by structural household determinants and child characteristics; (2) a contaminated environment is more likely to be observed in poor households in non-urban settings lacking public water and sanitation infrastructure; (3) the ability of a household to fulfil its food needs is constrained by the household's economic condition, the head of the family's employment and residential setting.

METHODOLOGY

Study design, sampling and data collection

The data come from a household panel study set up in 2011 as part of the Kaya Health and Demographic Surveillance System (HDSS).[28 29] The panel includes 2004 randomly selected households in urban, peri-urban and rural areas. In this context, 'peri-urban' refers to new, infrastructure-poor settlements built in the periphery of Kaya. Households were surveyed once a year between 2011 and 2014. In 2014, a survey on environmental conditions was conducted among the 1435 households of the panel that had children under 5 years. It combined direct observations and a questionnaire administered by the interviewer (details on the items collected and observed are provided in online supplementary table S1). Analysis was limited to the children living in the 1401 households with complete data for the environmental survey who were 12–59 months of age between 2011 and 2014 (n=3121). Of these children, 2881 (92%) had complete data for each variable used in the SEM models.

Variables

Data collected yearly for each child were: recent episodes of illness and health-seeking behaviours; growth (weight, height, mid-upper arm circumference); and health (body temperature, rapid diagnostic test for malaria, haemoglobin). Stunting was defined as a height-for-age z-score less than two SDs, using the WHO's growth standards.[30] Data on household socioeconomic characteristics, food needs and environmental contamination were collected once. To account for contaminated environment favourable to faecal-oral transmission, an index derived by multiple correspondence analysis (MCA) was constructed. This 'contaminated environment index' (CEI) was based on 12 items related to: access to water and sanitation (3 items), handwashing (3 items), presence of animals in the courtyard (3 items) and yard cleanliness (3 items) (see online supplementary tables 1 and 2).

An index reflecting the household's ability to fulfil its food needs was constructed based on responses to four questions on food security. Structural determinants and potential confounders considered were child sex and age, survey year, residential setting (urban/peri-urban/rural), mother's education, head of household's occupation, distance to the closest health centre, and socioeconomic status (SES). SES was measured using an index also constructed by MCA and including 20 variables pertaining to family possessions, housing conditions and energy sources used at home. A specific index was constructed for each of the urban, rural and peri-urban settings. All indexes were ultimately recoded based on quartiles of distribution.

Analysis

To assess the complex system of relationships, a generalised SEM was used, adjusting for repeated observations and hierarchical data (observations nested in children, themselves nested in households). The final model is presented in figure 1 (other models are presented in online supplementary table 3 and figures). In all, the system of equations includes an ultimate dependent variable (stunting), two proximal intermediary variables (contamination, food security) and three distal intermediary variables (education, occupation, wealth) that are subject to the influence of context. The link functions correspond to the nature of the dependent variables; Robust Poisson Regression was used for stunting.[31] All analyses were conducted with Stata V.14 (Stata Corp). Predicted values, prevalence ratios and marginal effects (prevalence differences and ratios) were derived using Stata postestimation commands.

Ethical approval was obtained from the Burkina Faso Health Research Ethics Committee and the Ethical Committee of the Centre de Recherche du CHUM in Montreal. All participants' guardians provided written consent to the annual interview questionnaire and to their children's laboratory tests and clinical assessment. Data were used in conformity with the Kaya HDSS policy (authorisation 1KH002-2015).

RESULTS

Households in urban areas showed a higher level of education for the mother, higher food security and lower environment contamination (table 1). Overall, access to improved water sources and sanitation was poor; the presence of animals, their excreta or garbage in yards was widespread; only 64% of caregivers reported washing their hands with soap more than twice a day; only 48% had observable soap available and 10% or less had a dedicated place for handwashing. Food security was also

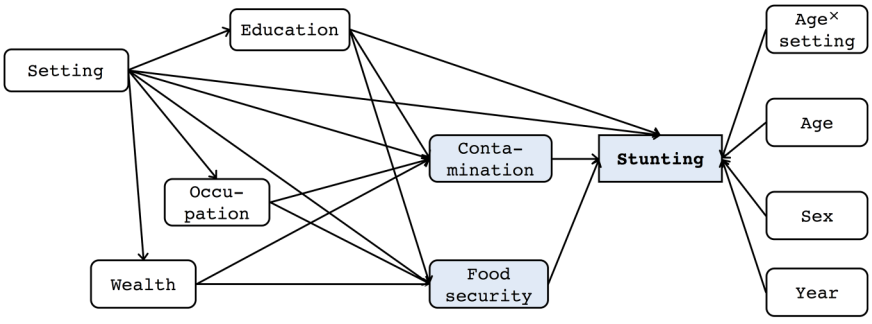

Figure 1 Final generalised structural equation model. Age: child's age in 4 categories; Age×setting: interaction of age with residential setting; Occupation: father's occupation is in agriculture; Education: mother's education; Contamination: contaminated environment index; Food security: score of household food security; Setting: residential setting (urban, peri-urban, rural); Sex: child's sex; Stunting: binary outcome; Wealth: socioeconomic status score; Year: year of survey. Hierarchical model: observations are clustered by child (level 1), and children are clustered by household (level 2).

Table 1 Sample description, by setting

	Urban	Peri-urban	Rural
Households, n (%)	580 (41)	203 (15)	618 (44)
Household characteristics at entry:			
Mother's education: none, % (95% CI)	83 (81 to 85)	94 (92 to 96)	96 (95 to 97)
Father's occupation: agriculture, % (95% CI)	53 (51 to 55)	84 (81 to 87)	95 (94 to 96)
Number of children 12–59 months, median (IQR)	4 (3–5)	4 (3–6)	5 (3–7)
Members/room, median (IQR)	2.0 (1.5–2.5)	2.0 (1.7–2.6)	2.0 (1.7–2.5)
Household food security: %(95% CI)*			
1. Three meals/day for all family members	61 (59 to 63)	62 (59 to 65)	45 (43 to 47)
2. Food supply all year long	57 (55 to 59)	58 (55 to 61)	36 (34 to 38)
3. Food variety and quality all year long	28 (26 to 29)	18 (15 to 21)	10 (8 to 11)
4. Means to buy food and cooking condiments	43 (41 to 45)	25 (22 to 28)	12 (11 to 13)
Food security score†, median (IQR)	6 (4–7)	5 (4–7)	4 (3–6)
Sources of contamination‡: % (95% CI)			
1. Source of water:			
Tap on premises	33 (31 to 35)	2 (1 to 4)	0 (0 to 1)
Public tap	53 (51 to 55)	44 (41 to 48)	14 (13 to 16)
Public pump or unimproved	14 (13 to 16)	53 (50 to 57)	85 (84 to 87)
2. Time to fetch water, minutes; median (IQR)	10 (3–30)	25 (15–30)	30 (20–40)
3. Sanitation:			
Improved and not shared	63 (61 to 65)	24 (21 to 27)	12 (11 to 13)
Improved and shared	26 (24 to 28)	21 (18 to 24)	13 (12 to 14)
Unimproved	11 (10 to 12)	55 (51 to 59)	74 (72 to 76)
4. Observed area dedicated to handwashing	10 (9 to 11)	2 (1 to 3)	5 (4 to 6)
5. Caregiver handwashing/day with soap			
One time or less	15 (13 to 16)	20 (18 to 23)	32 (30 to 34)
Two times	8 (7 to 9)	11 (8 to 13)	12 (11 to 13)
More than 2 times	77 (75 to 79)	69 (66 to 72)	56 (54 to 58)
6. Observed soap for handwashing	62 (60 to 64)	43 (40 to 46)	37 (35 to 39)
7. Reported animal access to the yard	52 (50 to 54)	71 (68 to 74)	86 (84 to 87)
8. Observed animals in the yard	68 (66 to 70)	76 (73 to 79)	86 (84 to 87)
9. Observed animal excreta in the yard	63 (61 to 65)	76 (73 to 79)	90 (88 to 91)
10. Observed garbage in the yard	69 (67 to 71)	79 (76 to 82)	85 (83 to 86)
11. Observed stagnating water in the yard	14 (12 to 15)	22 (19 to 25)	31 (29 to 33)
12. Observed open containers in the yard	59 (57 to 61)	61 (58 to 65)	71 (69 to 73)
Contaminated environment index§, median (IQR)	0.5 (−0.2–1.3)	−0.5 (−0.8–0.5)	−0.7 (−1.0–−0.3)

*Surveyed in 2013.
†Summary score ranging from 0 to 8.
‡Surveyed in 2014.
§Factorial index ranging from −1.4 to +2.8.
Improved sanitation, use of latrines with slabs; Unimproved sanitation, open defaecation, use of latrine without slab; Unimproved water source, unprotected well, other sources.

suboptimal, the worst situation being in rural areas. For example, more than 40% of households in urban and peri-urban areas and more than 60% in rural areas experienced food shortages during the year (table 1).

A total of 3121 children aged 12–59 months were followed between 2011 and 2014, with a median (IQR) of 3 (2–4) measurements per child. Age (median, IQR: 36, 25–48 months) and sex (51% male) were similar in all settings. The overall prevalence of stunting was 29% and was significantly higher among boys (32% vs 26%, χ^2 $p<0.001$, table 2). The gap between sexes diminished with age and was no longer significant in the fifth year of life. Among children aged 12–23 months, one in three was stunted, with no difference across settings. The situation improved with age in urban areas and, to a lesser extent, in rural areas, but persisted in peri-urban settings (figure 2). Finally, despite the short time period for capturing secular trends, a constant and significant decrease in stunting prevalence was noticeable between 2011 and 2014 across all age categories (table 2).

Table 2 presents the observed prevalence of stunting and predicted prevalence from multilevel Poisson models, adjusted for repeated measures and clustering in families. As hypothesised, stunting prevalence was strongly associated with higher environmental contamination. Children in the most contaminated environments (4th CEI quartile) had a 40% higher risk of stunting than those in the least contaminated environments, with a mean 8 percentage points difference in prevalence between the two groups ($p<0.001$). Other factors associated with a higher risk of stunting were low household food security, male sex, younger age, rural and peri-urban settings, father's occupation in agriculture, uneducated mother, and earlier year of survey (all $p<0.05$).

Risk of contaminated environment was significantly higher in rural and peri-urban settings (43% and 27% of households with highest contamination vs 5% in urban areas); in households with the lowest food security score (highest contamination for 37% vs 11%); with no education (27% vs 13%) and with

Table 2 Observed prevalence of stunting and prevalence ratio and prevalence difference across groups (n=2881)

Characteristics	Stunting‡ %	Prevalence ratio§ (95% CI)	Prevalence difference§ % (95% CI)
Contaminated environment index			
1st quartile	23.0	–	–
2nd quartile	30.7	1.3 (1.1 to 1.6)*	6.5 (2.4 to 10.6)
3rd quartile	30.1	1.3 (1.1 to 1.6)*	5.6 (1.6 to 9.6)
4th quartile (highest contamination)	32.8	1.4 (1.2 to 1.7)*	8.0 (3.9 to 12.1)
Household food security score			
1st quartile	33.3	–	–
4th quartile (highest food security)	24.5	0.7 (0.6 to 0.9)*	−7.0 (−11.4 to −2.6)
Mother's education			
None	30.3	–	–
Primary or above	19.4	0.6 (0.5 to 0.8)*	−9.6 (−13.8 to −5.5)
Sex			
Male	32.2	–	–
Female	26.4	0.8 (0.7 to 0.9)*	−5.3 (−8.0 to −2.8)
Age (in months)			
12–23	31.9	–	–
24–35	32.0	1.0 (0.9 to 1.1)	−0.4 (−3.9 to 3.2)
36–47	31.2	1.0 (0.8 to 1.1)	−1.0 (−4.5 to 2.5)
48–59	22.5	0.7 (0.6 to 0.8)*	−8.0 (−11.4 to −4.8)
Setting			
Urban	25.8	–	–
Peri-urban	35.4	1.4 (1.1 to 1.6)*	7.9 (3.0 to 12.9)
Rural	30.4	1.2 (1.1 to 1.4)*	3.5 (0.4 to 6.7)
Father's occupation			
Agriculture	30.5	1.2 (1.1 to 1.4)*	4.3 (0.9 to 7.7)
Other	25.2	–	–
Wealth score			
1st quartile	31.3	–	–
4th quartile (richest)	26.8	0.8 (0.7 to 1.0)	−4.1 (−8.3 to 0.2)
Year of survey			
2011	33.8	–	–
2012	30.3	0.9 (0.8 to 1.0)	−2.9 (−6.6 to 0.7)
2013	29.7	0.9 (0.8 to 1.0)	−3.3 (−6.9 to 0.2)
2014	24.8	0.7 (0.6 to 0.9)*	−7.3 (−10.8 to −3.8)

‡All p for χ^2<0.05; *p<0.05;
§First category as reference for prevalence ratio and prevalence difference; prevalence from multilevel Poisson model adjusted for repeated measures and clustering.

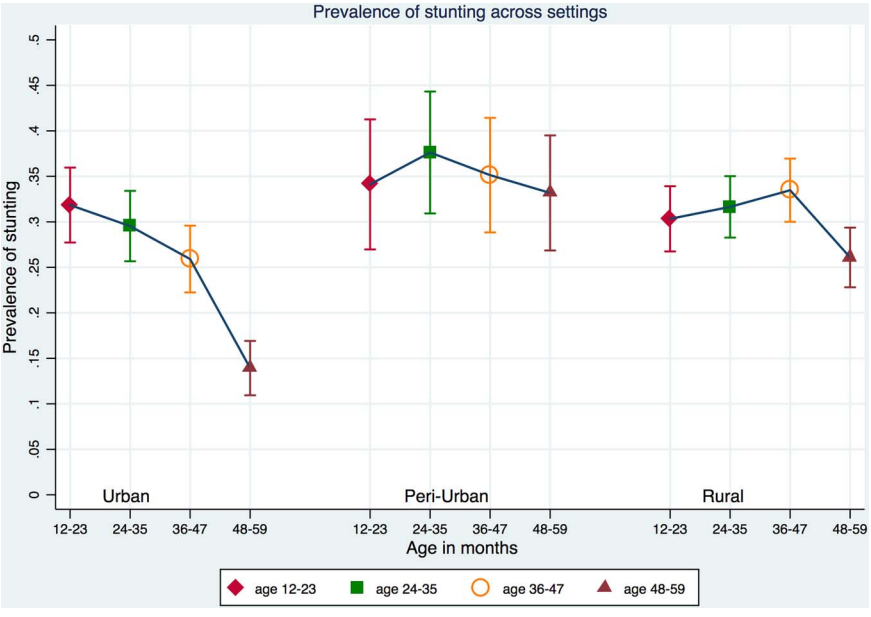

Figure 2 Observed prevalence of stunting across settings by age.

agriculture as the main income (32% vs 5%; all p<0.05) (see online supplementary table S4).

Taking into consideration endogeneity and confounding factors in SEM, children from households with the highest contamination had a relative risk of stunting of 1.30 (p=0.008) when compared to children from households with the lowest contamination (table 3 and online supplementary table S5). The prevalence ratio (PR) of stunting was comparable for children in the quartile with lowest contamination (PR 0.74) and in the best quartile for food security (PR 0.80, p=0.04). Residential setting and father's occupation were predictors of both risk of higher household contamination and lower food security. Interaction terms between age and setting were significant. In urban areas, the relative prevalence of stunting was 0.43 (95% CI 0.34 to 0.54) for children aged 48–59 months compared to those aged 12–23 months, while in rural and peri-urban areas the risk was high at all ages, with a relative risk of stunting in children aged 48–59 months being higher in rural and peri-urban areas versus urban ones (table 3).

Figure 3 presents predicted prevalence of stunting for different levels of contamination by age. For younger children (12–23 months old), the prevalence gap between least (1st quartile) and most contaminated (4th quartile) was comparable across settings: 7.8 percentage points (95% CI 1.8 to 13.9) in urban settings; 8.0 (2.0 to 13.9) in peri-urban ones; and 6.6 (1.9 to 11.3) in rural ones. Just as the prevalence of stunting decreased with age in urban areas, the gap between the least and most exposed children decreased substantially with age in urban settings, resulting in a difference of 3.3 percentage points (0.7 to 6.0) in the age bracket 48–59 months. For children in peri-urban and rural areas, the risk difference remained constant between 6 and 7 percentage points at all ages, with a difference of 7.4% (1.9% to 12.9%) and 5.7% (1.6% to 9.7%), respectively, at age 48–59 months (figure 2).

Sensitivity analyses were used to test the model's robustness. Results for the contamination–stunting relationships were comparable when changing: (1) the dependent variable: continuous (standardised height-for-age), ordinal variable (severe stunting, moderate or none) or binary stunting outcome; (2) the link functions of the generalised linear models: logistic, negative binomial or Poisson with robust estimators;[31] (3) the specification of error terms: covariant or not; and (4) the confounders included. The interaction between contamination and setting was explored, but was not significant.

Table 3 Adjusted prevalence ratio of stunting for child and household variables (n=2881)

Characteristic	Adjusted prevalence ratio (95% CI)†
Contaminated environment index (1st quartile as reference)	
2nd–3rd quartile	1.23 (1.04 to 1.45)*
4th quartile (highest contamination)	1.30 (1.07 to 1.58)*
Household food security score (1st quartile as reference)	
2nd and 3rd quartiles	0.92 (0.80 to 1.07)
4th quartile (highest food security)	0.82 (0.67 to 0.99)*
Mother's education (none as reference)	
Primary or more	0.69 (0.52 to 0.90)*
Sex (male as reference)	
Female	0.81 (0.73 to 0.90)*
Setting by age in months (urban as reference in each age)	
Peri-urban 12–24	1.07 (0.85 to 1.36)
Rural 12–24	0.92 (0.77 to 1.10)
Peri-urban 24–35	1.24 (1.00 to 1.54)
Rural 24–35	1.04 (0.87 to 1.23)
Peri-urban 36–47	1.35 (1.07 to 1.69)*
Rural 36–47	1.23 (1.03 to 1.46)*
Peri-urban 48–59	2.34 (1.77 to 3.09)*
Rural 48–59	1.85 (1.45 to 2.38)*
Year (2011 as reference)	
2012	0.89 (0.82 to 0.97)*
2013	0.88 (0.80 to 0.97)*
2014	0.74 (0.67 to 0.82)*

*p<0.05.
†Adjusted prevalence ratio as estimated by SEM Poisson model. Model presented in figure 1.
SEM, structural equation model.

DISCUSSION

The prevalence of stunting in the study population was high, consistent with the country's report in the annual SMART surveys.[27] Despite a gradual decline between 2011 and 2014 (from 34% to 25%, p<0.001), the persistent high prevalence of stunting confirms that it is still a major public health issue.

After controlling for endogeneity and other known determinants of stunting, children in the most contaminated environments appeared to have a 30% higher probability of being stunted than those living in the least contaminated ones. The magnitude of this risk was comparable to living in households with lower food security (PR 1.20). These findings indicate a strong relationship between exposure to faecal-oral transmission and faltering growth.[8–10 22 25] They are also consistent with the suspected linkage between stunting[11] and the triad of environment contamination, faecal-oral transmission and environmental enteropathy.[15 16] The magnitude of the effect of contaminated environment on stunting is comparable to those found in studies in various settings in Bangladesh;[9 10] India;[25] and peri-urban Peru.[22]

Intervention studies are needed to test the effectiveness of complex multiple interventions in improving linear growth, especially the combined impact of nutrition-specific and nutrition-sensitive interventions, such as WASH. Several ongoing multi-country trials are expected to help fill this gap.[32–34] Interventions aimed at improving water and sanitation access as well as the hygiene and cleanliness of houses and yards, and reducing human contact with animal excreta, may be effective in reducing EED and stunting.[13 21] In addition, we need to identify the most effective targets and delivery methods for the environmental components of these complex stunting prevention programmes.

Living in a rural or peri-urban setting was a robust predictor of household contamination, incapacity to fulfil food needs and faltering growth. An 'urban advantage' favouring children living in cities has been repeatedly reported for low income and middle income countries,[35] including Burkina Faso,[36 37] but what was noticeable in this study was the magnitude of the 'periurban disadvantage'. In this study context, peri-urban areas host a heterogeneous population of migrants and local poor in the periphery of a small town with limited public infrastructure. Even though they are not very densely populated areas, they concentrate vulnerabilities and unsanitary conditions in a way that is somewhat comparable to what is seen in urban slums. According to Fink *et al*,[37] children in peri-urban slums of small towns have worse health outcomes than children living in urban areas or even slums of bigger cities. People living in the same context share an array of environmental, social and services-related determinants of health, beyond the ones we could

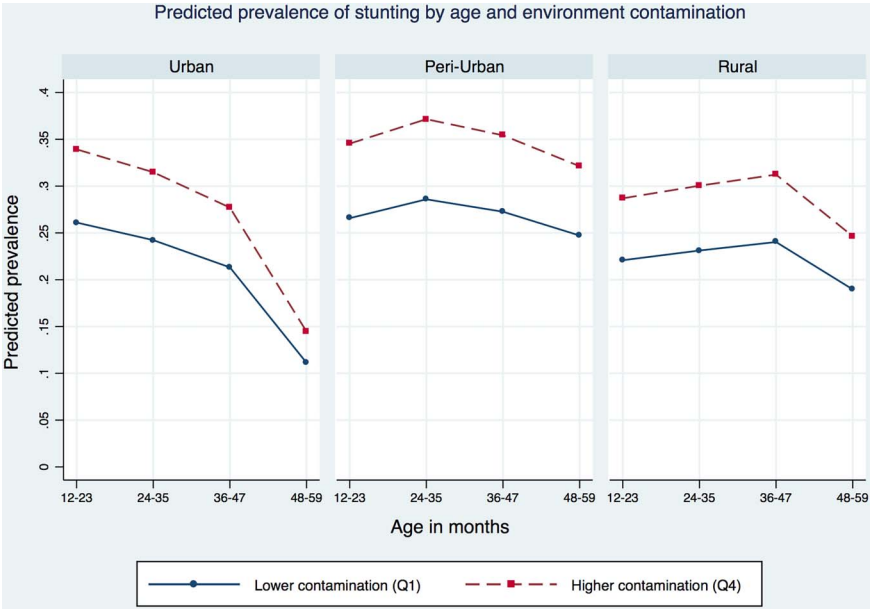

Figure 3 Predicted prevalence of stunting for high and low environment contamination, by setting and age (SEM model). SEM, structural equation model.

control for.[38] Most likely, context-level contamination also plays an important role in child health. In a study in rural Ecuador, neighbourhood sanitation was a stronger predictor of child growth than house sanitation coverage.[39] Neighbourhood-level data on sanitation, water access and hygiene would help identify the specific effect of neighbourhood contamination on child stunting, within the broader effect of residential setting.

In this study, the effect of residential setting on child growth was also differential by age, and differences between urban and non-urban settings were more evident for older children (4–5 years old). In a multicountry study, Kyu *et al*[40] also found that older children living in slums had a higher risk of stunting than children of the same age living in urban areas. According to Kyu, this difference could be the result of the former group's prolonged exposure to an unfavourable environment. Longitudinal studies could help distinguish between effects on stunting attributable to long-term exposure to a slum environment and those reflecting the progression of stunting from its onset at a younger age.

Other child and household characteristics strongly associated with stunting were male sex and low level of education of the mother. A higher risk of stunting for males has been found in other SSA studies.[41 42] There is no agreement yet as to whether it is due to biological factors or inequalities in provision of food and healthcare. Mother's education, a known determinant of child growth,[6] was significantly associated with contamination and stunting even in this population with very limited formal education (5% had primary education, 3% secondary or more).

Strengths and limitations

The 4-year window of observation was chosen to capture changes in children's condition over time and apprehend some of the dynamics of child stunting, something which cannot be easily achieved through cross-sectional studies. Another strength of the study is its measurement of environmental contamination, using an index that includes broad potential sources of faecal-oral transmission. Although further improvements could certainly increase the index's content validity, it already has the advantage of including influential elements beyond access to water and sanitation, the only two factors considered in commonly used scores. The index appears to be reasonably unidimensional, explaining 70% of the variance (see additional table in online appendix 1). The association between the index and stunting was also extremely stable during the modelling process, further confirming the index's validity.

One limitation of the study is that data on household contamination, SES and food security were collected only once (socioeconomic status in 2011, food security in 2013 and household contamination in 2014). Although it is reasonable to assume that these structural characteristics were relatively stable in this context, some minor changes might have occurred over time within some households. Second, the final models were based on a subsample consisting of the 92% non-missing observations. Secondary comparisons were made to search for possible differences between the final and initial samples. No evidence of a possible selection bias was observed. Finally, although SEM analyses were based on what we believe to be a fairly robust theoretical model and controlled for key confounding factors, the scope of the analysis remains limited, and the study can only provide preliminary evidence regarding the magnitude of the effects of contaminated environments on stunting. In addition, residual confounding from other socioeconomic variables could not be excluded. There is still a long way to go in untangling the complex and intertwined determinants of stunting in developing countries.

CONCLUSION

Progress towards reducing the global burden of stunting has been limited and ultimately disappointing in SSA. More effective, feasible and diversified approaches are needed to win the battle against faltering growth. This study suggests that in the pathway to stunting, the exposure of young children to a contaminated environment is extremely influential, possibly as much as food security. This result, together with previous evidence on links between faecal-oral transmission, EED and stunting, supports the argument that stunting prevention programmes should include interventions to reduce environmental contamination and faecal-oral transmission. Such interventions, targeting toddlers and young children, could extend beyond water and sanitation to consider a broader spectrum of possible sources of environmental contamination.

What is already known on this subject

Stunting can be related to exposure to a contaminated environment favouring faecal-oral transmission, possibly via the subclinical inflammatory disorder known as environmental enteropathy. Young children living in low income and middle income countries are continuously exposed to faecal pathogens through poor environmental conditions and frequent hand-to-mouth contact.

What this study adds

Environment contamination was measured using an index including household access to water and sanitation; caregivers' hygiene; yard cleanliness; and proximity of animals. Contamination was associated with stunting for all ages (1–5 years). Both contamination and stunting were higher in non-urban areas. Interventions to prevent stunting might consider targeting various different sources of contamination and focusing on the most deprived settings.

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Impact of contaminated household environment on stunting in children aged 12–59 months in Burkina Faso

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Notes