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Exposure to cows is not associated with diarrhoea or impaired child growth in rural Odisha, India: a cohort study

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SUMMARY

Exposure to animal livestock has been linked to zoonotic transmission, especially of gastrointestinal pathogens. Exposure to animals may contribute to chronic asymptomatic intestinal infection, environmental enteropathy and child under-nutrition in low-income settings. We conducted a cohort study to explore the effect of exposure to cows on growth and endemic diarrhoea in children aged <5 years in a rural, low-income setting in the Indian state of Odisha. The study enrolled 1992 households with 2739 children. Height measurements were available for 824 children. Exposure to cows was measured as (1) the presence of a cowshed within or outside the compound, (2) the number of cows owned by a household, and (3) the number of cowsheds located within 50 m of a household. In a sub-study of 518 households, fly traps were used to count the number of synanthropic flies that may act as vectors for gastrointestinal pathogens. We found no evidence that environmental exposure to cows contributes to growth deficiency in children in rural India, neither directly by affecting growth, nor indirectly by increasing the risk of diarrhoea. We found no strong evidence that the presence of a cowshed increased the number synanthropic flies in households.

Key words: Gastrointestinal infections, zoonoses.

INTRODUCTION

The global burden of under-nutrition and stunting continues to be high. In India, despite economic growth and marked reductions in child mortality, under-nutrition and stunting remain common [1]. About 250 million Indians are classified as food-insecure [2]. The reasons for the astonishingly high rates of stunting in India, if international growth standards are used, remain largely unexplained. The comparability of growth data from South Asia with the WHO standard continues to be debated [3].

While many indicators of poverty are strong predictors of stunting and under-nutrition, the pathways by which poverty and inadequate intake of macro- and micronutrients cause under-nutrition may be less obvious than previously assumed [4]. Many widely used nutrition interventions only have a small impact on child growth, and do not make up for the growth retardation earlier in life [4, 5].

Apart from inadequate food intake, it has been suggested that children from poor families are exposed to frequent infections early in life, especially enteric infections, which are thought to impair growth and mental development. A vicious cycle has been proposed by

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which infection causes under-nutrition and under-nutrition in turn causes increased susceptibility to infection [6, 7]. However, it has also been shown that catch-up growth after an episode of diarrhoea is fast in most children, and almost fully compensates for the growth retardation and weight loss during an episode [4].

Children growing up in unhygienic conditions harbour a large number of pathogens [8] even if asymptomatic, and frequently display signs and laboratory markers of environmental enteropathy (EE) [4, 9]. EE is thought to result from exposure to environmentally occurring pathogens in settings characterized by poor hygiene, causing chronic inflammatory changes in the intestines, malabsorption and increased permeability of harmful intestinal products into the bloodstream. The implications of EE have recently gained wider interest in public health research, particularly in respect of providing a cleaner environment for children in poor settings [4, 9, 10].

Apart from poor access to water, sanitation and hygiene, exposure to animal livestock may increase the risk of EE and subsequently under-nutrition in low-income settings. Animal livestock may also contribute to the burden of diarrhoea and the observed vicious cycle between infection and malnutrition. Animal livestock may further increase the risk of gastrointestinal infections by attracting flies that carry pathogens to food or directly to humans. The recent GEMS study suggested a high burden of diarrhoeal disease due to *Cryptosporidium* which is also common in cattle [11]. In India cows play an important economic, nutritional, cultural and religious role, and are a ubiquitous feature of the Indian urban and rural landscape. If exposure to cows contributes to the high prevalence of stunting and diarrhoea in India, then the implications for public health and agricultural policy would be enormous. In this study, we explored whether exposure to cows is associated with an increased risk of diarrhoea, exposure to synanthropic flies (as vectors of gastrointestinal pathogens) and impaired child growth.

METHODS

Study design and setting

The study was conducted in the context of a cluster randomized trial to evaluate the effect of sanitation on child health between September 2010 and October 2013 in Puri, a coastal district of the state of Odisha (formerly, Orissa), India. Odisha has a population of 43 million people and is home to 12 million cows [12]. Trial design, setting and characteristics of the study population have already been described [13]. The trial included 100 rural villages spread across seven of the 11 blocks (an administrative sub-district) of Puri District. The intervention (latrine construction) was rolled out during 2011 in half of the villages. Households were eligible to participate in the study if they had a child aged <4 years or a pregnant woman living there. Households with a new baby born during the surveillance phase were also enrolled. The intervention had no impact on diarrhoea or stunting [14]. The present cohort study included children from all enrolled households regardless of intervention allocation.

Exposure variables

A baseline survey was conducted between September and October 2010 to collect information on household demographics, cow ownership, house structure, type of fuel used for cooking, and water and sanitation access. Between December 2012 and March 2013 a survey of all households in the study area regardless of the presence of a child aged <5 years was performed to assess compliance with the intervention, and to record the GPS location of every household. In this survey we further assessed the household size and presence and location of a cowshed in each household. Location of a cowshed belonging to a household were recorded as within the compound, or outside the compound. Outside cowsheds were usually in the immediate vicinity of the compound, rarely more than 20 m away. As a measure of human population density, we calculated the number of residents within 50 m of each household enrolled in the study. As a measure of cow population density we counted the number of cowsheds around each household. This measure included cowsheds recorded as being inside and outside a compound, even though the exact location of cowsheds outside a compound was not geo-referenced. Therefore, some of the cowsheds counted here as being within 50 m of a household may in fact lie somewhat outside the 50 m radius.

Outcome variables

Child growth

A baseline measure of recumbent length/height was taken in January 2012 of all children aged <2 years. The same children and those born during the study were measured again in October 2013. For this

analysis we only included measurements from the second survey. We measured recumbent length of children aged <2 years using Seca 417 boards (Seca, USA) with 1-mm increments. Height of children aged ⩾2 years was measured using a Seca 213 stadiometer. Back-checks on weight and height measurements were conducted in about 5% of the households selected at random. Height measures were transformed into height-for-age z-scores (HAZ) using the international growth standard from WHO Anthro software (WHO, Geneva).

Diarrhoea

The analysis of the association between exposure to cows and diarrhoea included all children aged <5 years enrolled in the trial. Between June 2011 and October 2013, households with children aged <5 years were followed up at 3-month intervals, resulting in a maximum of 10 rounds of observation per household. Children were excluded from the analysis once they were aged ⩾5 years. Seven-day period prevalence of diarrhoea was recorded based on reports from the primary caregiver [15, 16]. Following qualitative research and extensive piloting, three local terms for loose stools were identified and used for the questionnaire. Reported presence of any of these three conditions ⩾3 times (according to the WHO definition [17]) on at least 1 day during the past 7 days was defined as diarrhoea.

Synanthropic flies

We measured the number of synanthropic flies (*Musca domestica* and *M. sorbens*) by installing 24-h fly traps in a random subsample of 572 households (nine households per village) from a random sample of 64/100 study villages (32 control, 32 intervention villages). Following extensive piloting of different trapping methods, blue sticky cards (Agrisense BCS Ltd, UK), with both sticky surfaces exposed and each measuring 20 × 24·5 cm, were placed at a 45° angle on the floor inside the kitchen, or cooking area, at a minimum of 0·5 m from an open source of flame. Sampling was conducted over three consecutive days in each selected household. Fly counts were averaged at household level over the 3 days of observation. Of the households, 518/572 (90·6%) could be linked to the data on cowshed ownership and were used in the analysis.

Statistical analyses

The association between socioeconomic variables and cow ownership (Table 1) was calculated using linear regression analysis. Linear regression was also used to estimate the effect of exposure variables on HAZ (Table 2). Since multiple children were enrolled in some households, we specified household as a random effect in the model. The models displayed normality of residuals and approximate homoscedasticity.

The association between exposure variables and prevalence of diarrhoea was estimated using log-binomial models (binomial distribution, log-link). Household-level clustering was accounted for by generalized estimating equations (GEE) with robust standard errors.

Due to right skew, fly counts were $\log_{10}$-transformed which resulted in a near normal distribution with some zero-inflation. A count of 1 was added to all counts to remove zero values prior to log transformation, and subtracted after calculating the geometric mean (Williams mean) [18]. The association between presence of a cowshed in the household and $\log_{10}$ counts of synanthropic flies caught was estimated using the *t* test.

Because of the large potential for confounding due to the inherent association between stunting, diarrhoea and poverty, all models were adjusted for a range of socioeconomic variables: house structure (dichotomized as concrete/pucca *vs.* mud and semi-pucca), education of the head of the household and carer (dichotomized as completed primary school *vs.* not completed), latrine ownership at baseline, water source in the compound, landownership (any *vs.* none), and membership of a scheduled caste/scheduled tribe (SC/ST), a classification used by the Indian Government to identify socially and economically disadvantaged communities. An asset index including ownership of phone, watch, TV, chair, mattress, bed, table, fan and/or bike was constructed using principal component analysis (Kaiser–Meyer–Olkin index 91%). The first component, explaining 46% of the variation of the items was used in the analysis. This percentage compares favourably with many other asset indices [19]. Latrines constructed during the intervention phase were disregarded in this analysis as they were rarely used and not shown to affect health outcomes [14]. All analyses were conducted in Stata v. 12 (Stata Corporation, USA).

Ethics

The study was conducted in the context of a trial which was approved by the Ethics Committee of the London School of Hygiene and Tropical Medicine, and in India by Xavier Institute of Management,

Table 1. *Socio-demographic characteristics of the study households and cow ownership*

	N	Cows per HH mean (S.D.)	Difference*	95% CI
Total	1992	1·4 (2·1)	–	–
Population density (residents of all ages within 50 m radius)				
0–100	653	1·3 (1·9)	(ref.)	
101–200	640	1·5 (2·6)	0·1	−0·1 to 0·4
>200	544	1·3 (1·6)	−0·1	−0·3 to 0·2
Household size				
1–4	487	0·9 (1·3)	(ref.)	
5–8	993	1·3 (1·6)	0·4	0·2 to 0·6
>9	341	2·3 (3·5)	1·3	1·1 to 1·6
Scheduled caste/tribe				
No	1588	1·5 (2·2)	(ref.)	
Yes	404	1·0 (1·7)	−0·5	−0·7 to −0·2
Head of HH completed primary school				
No	984	1·6 (2·5)	(ref.)	
Yes	1008	1·2 (1·7)	−0·3	−0·5 to −0·2
Mother/carer of child completed primary school				
No	601	1·1 (2·3)	(ref.)	
Yes	1391	1·5 (1·6)	0·4	0·2 to 0·6
House structure				
Pucca (concrete/cement)	791	1·7 (2·6)	(ref.)	
Semi-pucca	406	1·3 (1·9)	−0·3	−0·6 to −0·1
Mud	795	1·2 (1·6)	−0·5	−0·7 to −0·3
Land ownership				
Irrigated	1162	1·7 (2·0)	(ref.)	
Not irrigated	330	1·4 (3·1)	−0·3	−0·5 to 0·0
None	500	0·7 (1·2)	−1·0	−1·3 to −0·8
Dung as main fuel for cooking				
No	1571	1·3 (2·2)	(ref.)	
Yes	421	1·7 (1·8)	0·4	0·1 to 0·6
Water source in compound				
No	1422	1·3 (2·2)	(ref.)	
Yes	570	1·6 (2·0)	0·3	0·1 to 0·5
Owns latrine				
No	1786	1·4 (2·2)	(ref.)	
Yes	206	1·6 (1·7)	0·2	−0·1 to 0·5

HH, Household; CI, confidence interval.
* Linear regression analysis.

Bhubaneswar (XIMB), and Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar. The trial was registered with ClinicalTrials.gov (registration no. NCT01214785). Written informed consent was obtained from the male and/or female head of household prior to baseline data collection. No additional data collection was done specifically for the purposes of this study.

RESULTS

The demographic characteristics of the study households in relation to the number of cows owned are shown in Table 1. Population density was not associated with the number of cows owned. Smaller households had fewer cows. Higher education level of the mother/carer was associated with more cows owned, education of the head of the household with fewer cows owned. Households using dung as the main fuel source had more cows. Other than that, indicators of higher socioeconomic status (not SC/ST, land ownership, house structure, water source in compound, latrine ownership) were largely associated with higher cow numbers owned. Forty-four percent of the study households did not own cows (Fig. 1*a*). The number of cowsheds located within a 50 m radius of a house

Table 2. *Association between socioeconomic indicators, exposure to cows and height-for-age z score*

	N	HAZ mean (S.D.)	Difference*	95% CI	Adjusted difference*	95% CI
Total	824	−1·41 (1·19)	–	–	–	–
By socioeconomic factors						
Population density (residents of all ages within 50 m radius)						
0–100	249	−1·55 (1·20)	(ref.)	–	(ref.)	–
101–200	280	−1·28 (1·16)	0·25	0·03 to 0·46	0·14	−0·06 to 0·34
>200	241	−1·43 (1·22)	0·11	−0·12 to 0·33	0·02	−0·20 to 0·24
Change in HAZ per additional 100 residents within 50 m	770	−1·41 (1·19)	0·01	−0·07 to 0·08	−0·01	−0·09 to 0·07
Change in HAZ per additional household member	770	−1·41 (1·19)	0·02	−0·01 to 0·04	0·0	−0·02 to 0·03
Scheduled caste/tribe						
No	654	−1·19 (1·16)	(ref.)		(ref.)	
Yes	170	−1·89 (1·20)	−0·58	−0·79 to −0·37	−0·33	−0·55 to −0·11
Head of HH completed primary school						
No	444	−1·58 (1·16)	(ref.)			
Yes	380	−1·22 (1·20)	0·33	0·15 to 0·50	0·14	−0·03 to 0·31
Mother/carer of child completed primary school						
No	217	−1·87 (1·17)	(ref.)			
Yes	607	−1·25 (1·16)	0·62	0·43 to 0·81	0·30	0·09 to 0·50
House structure						
Pucca (concrete/ cement)	364	−1·18 (1·16)	(ref.)			
Semi-pucca	155	−1·37 (1·08)	−0·20	−0·43 to 0·03	−0·05	−0·28 to 0·18
Mud	305	−1·72 (1·23)	−0·54	−0·73 to −0·35	−0·19	−0·40 to 0·02
Land ownership						
Irrigated	486	−1·26 (1·17)	(ref.)		(ref.)	
Not irrigated	150	−1·45 (1·25)	−0·15	−0·4 to 0·1	−0·04	−0·26 to 0·18
None	188	−1·77 (1·14)	−0·50	−0·7 to −0·3	−0·12	−0·34 to 0·10
Water source in compound						
No	591	−1·53 (1·17)	(ref.)		(ref.)	
Yes	233	−1·10 (1·19)	0·41	0·23 to 0·60	0·13	−0·06 to 0·33
Owns latrine						
No	742	−1·47 (1·18)	(ref.)		(ref.)	
Yes	82	−0·92 (1·22)	0·52	0·25 to 0·82	0·07†	−0·24 to 0·37
Exposure to cows						
Cowshed ownership						
None	212	−1·52 (1·24)	(ref.)		(ref.)	
Outside compound	155	−1·32 (1·22)	0·17	−0·09 to 0·43	0·00	−0·25 to 0·25
In compound	398	−1·39 (1·16)	0·10	−0·11 to 0·31	0·03	−0·17 to 0·23
Number of cows owned						
0	322	−1·49 (1·12)	(ref.)		(ref.)	
1–2	336	−1·40 (1·26)	0·11	−0·16 to 0·32	−0·02	−0·21 to 0·17
⩾3	166	−1·29 (1·18)	0·19	−0·06 to 0·37	−0·03	−0·26 to 0·21
Number of cowsheds within 50 m of house						
<10	212	−1·53 (1·22)	(ref.)		(ref.)	
10–19	223	−1·43 (1·21)	0·08	−0·16 to 0·32	−0·05	−0·28 to 0·20
⩾20	335	−1·33 (1·17)	0·16	−0·06 to 0·37	−0·01	−0·23 to 0·20
Dung used as main fuel for cooking						
No	656	−1·48 (1·18)	(ref.)		(ref.)	
yes	168	−1·16 (1·20)	0·31	0·10 to 0·52	0·20	0·00 to 0·41

HAZ, Height-for-age *z* score; HH, household; CI, confidence interval.

* Linear regression with random effect to adjust for multiple observations within households.

† House structure omitted from model due to collinearity.

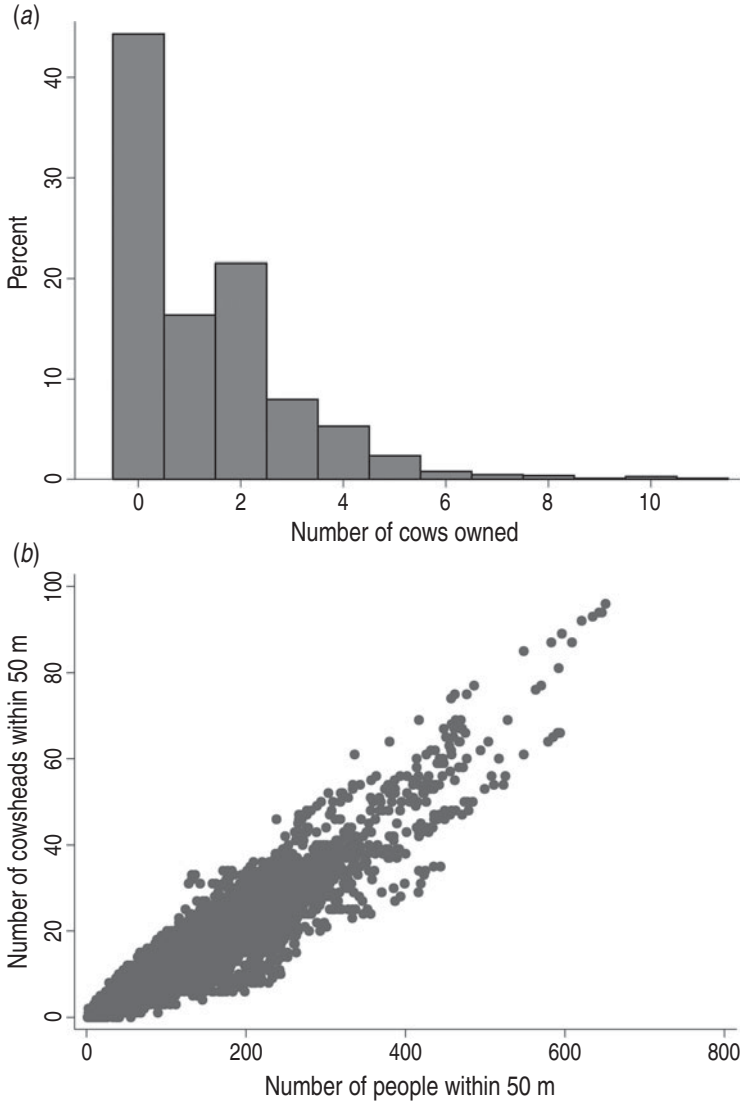

Fig. 1. (*a*) Number of cows owned per household, (*b*) association between population density (number of residents within 50 m of a house) and number of cowsheds within 50 m ($r = 0{\cdot}93$).

was highly correlated with population density ($r = 0{\cdot}93$, Fig. 1*b*).

The mean HAZ was −1·41 (S.D. = 1·19, Table 1). Boys ($n = 433$) were 0·19 *z* scores taller than girls ($n = 391$) [HAZ −1·32 *vs.* −1·52, 95% confidence interval (CI) of the difference 0·03–0·36]. The effect of exposure to cows and child growth (HAZ) is shown in Table 2. After adjusting for socioeconomic indicators, SC/ST status and carer not having completed primary school were strongly associated with lower HAZ scores. By contrast, after adjusting for socioeconomic indicators, there was no evidence that the number of cows owned, a cowshed within the compound, the number of cowsheds within 50 m, or cow dung used as main fuel for cooking were associated with lower HAZ scores.

There was a steady decline in HAZ with age (Fig. 2). On average, children lost about 0·25 *z* scores per year in the first 4 years of life (95% CI −0·32 to −0·18). Children in households surrounded by fewer than 10, 10–19 and >20 cowsheds within 50 m lost on average 0·21, 0·19 and 0·26 *z* scores per year, respectively (test for interaction between age and number of cows: $P = 0{\cdot}59$). There was also no evidence for an interaction affecting HAZ between age and in-compound presence of a cowshed ($P = 0{\cdot}69$) and between age and the number of cows owned ($P = 0{\cdot}99$). Thus, there was no evidence that exposure to cows led to a more rapid decrease in HAZ with age.

Adjusting for socioeconomic factors, there was some evidence that high human population density

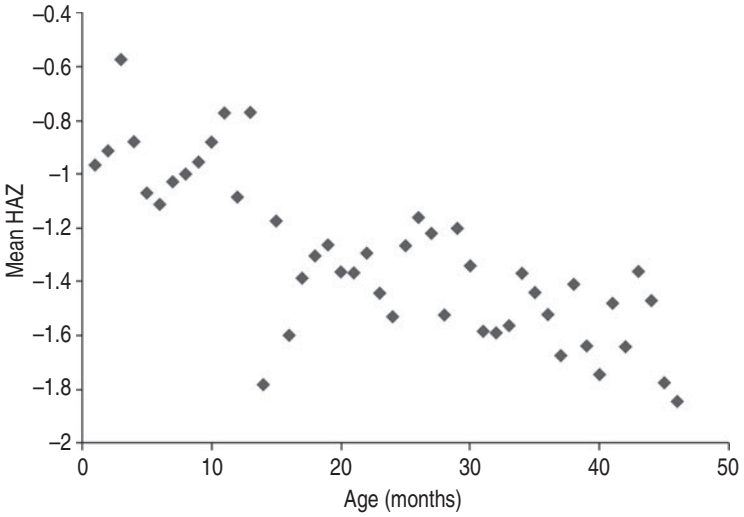

Fig. 2. Association between age and height-for-age *z* score (HAZ). Age was rounded to full months. Individual HAZ values were averaged within month.

was associated with a higher prevalence of diarrhoea. A lower prevalence of diarrhoea was reported for children with SC/ST background. A water source in the compound was protective against diarrhoea.

In crude and adjusted analysis, there was no evidence that cow ownership, cowshed density within 50 m of a household or use of cow dung for cooking increased the prevalence of diarrhoea (Table 3). There was no evidence for an interaction between exposure to cows and season: neither during the rainy nor the dry seasons were cow ownership, cowshed density within 50 m of a household or use of cow dung for cooking associated with an increased prevalence of diarrhoea (data not shown).

The Williams mean of fly counts was 14·7 in households without a cowshed ($n = 143$), 18·9 in households with a cowshed outside the compound ($n = 93$) and 19·5 in households with a cowshed in the compound ($n = 282$). There was suggestive evidence that fly counts were higher in households with a cowshed within the compound compared to households without a cowshed (+0·12 $\log_{10}$, 95% CI −0·02 to 0·25). The difference between households with a cowshed outside the compound compared to households without a cowshed was inconclusive due to a wide confidence interval (+0·10 $\log_{10}$, 95% CI −0·08 to 0·28).

DISCUSSION

We found no evidence that environmental exposure to cows contributes to growth deficiency in children in rural India, neither directly by affecting growth, nor indirectly by increasing the risk of diarrhoea.

Environmental exposure to cows has been shown to increase the risk of infection and disease outbreaks of many gastrointestinal pathogens such as rotavirus [20], *E. coli* (including O157) [21, 22], *Schistosoma japonicum* [23, 24], *Cryptosporidium* spp., and *Giardia intestinalis*. For example, a study of asymptomatic infection with *G. intestinalis* and *Cryptosporidium* conducted in Ethiopia revealed exposure to cattle as a risk factor for both [25], A further study from Ethiopia found evidence for zoonotic transmission of *Cryptosporidium* to HIV-positive individuals [26]. One study from Egypt found that animal contact increased the risk of *Cryptosporidium* infection [27]. However, further analysis in the same study suggested different transmission dynamics of human and cattle and little spatial overlap [27]. Using sequence typing, the same study indicated a predominant anthropogenic cycle of infection of *G. intestinalis* in children with diarrhoea, despite the high prevalence of *G. intestinalis* in ruminants in the study area [28].

Cow products and excreta have long been used in traditional Indian medicine [29]. Cow dung is regarded as an important antiseptic. In our study area, it is used by households with mud floors for cleaning purposes and to improve the appearance of mud surfaces. Applying cow dung to the umbilical stump post-delivery is not an uncommon practice in parts of Africa and South Asia that, however, is associated with neonatal tetanus [30].

While it seems clear that exposure to cows can lead to symptomatic human infection and occasional

Table 3. *Association between socioeconomic indicators, exposure to cows and diarrhoea in children aged <5 years*

	N	Diarrhoea, 7-day period prevalence	RR*	95% CI	Adjusted RR*	95% CI
Total	2739	0·10	–	–	–	–
By socioeconomic factors						
Population density (residents of all ages within 50 m radius)						
0–100	943	0·09	(ref.)	–	(ref.)	–
101–200	976	0·10	1·0	0·9–1·2	1·0	0·9–1·1
>200	820	0·11	1·2	1·1–1·4	1·1	1·0–1·3
RR per additional 100 residents within 50 m	2739	–	1·1	1·1–1·2	1·1	1·0–1·1
RR per additional household member	2739	–	1·00	0·99–1·01	1·00	0·99–1·02
Scheduled caste/tribe						
No	2194	0·10	(ref.)		(ref.)	
Yes	545	0·08	0·8	0·7–0·9	0·7	0·6–0·9
Head of HH completed primary school						
No	1409	0·11	(ref.)			
Yes	1330	0·10	0·9	0·8–1·0	0·9	0·9–1·1
Mother/carer of child completed primary school						
No	779	0·11	(ref.)			
Yes	1960	0·10	0·9	0·8–1·0	0·9	0·8–1·1
House structure						
Pucca (concrete/ cement)	1117	0·10	(ref.)			
Semi-pucca	559	0·09	0·9	0·8–1·1	0·9	0·7–1·0
Mud	1063	0·11	1·1	1·0–1·2	1·0	0·9–1·1
Land ownership						
Irrigated	676	0·10	(ref.)		(ref.)	
Not irrigated	482	0·10	1·0	0·9–1·1	1·0	0·8–1·1
None	1581	0·10	1·0	0·9–1·2	1·0	0·8–1·1
Water source in compound						
No	1960	0·11	(ref.)		(ref.)	
yes	779	0·08	0·8	0·7–0·9	0·8	0·7–0·9
Owns latrine						
No	2447	0·10	(ref.)		(ref.)	
Yes	292	0·08	0·7	0·6–0·9	0·8†	0·7–1·1
Season						
Dry season (October– May)	–	0·08	(ref.)		–	–
Wet season (June– September)	–	0·14	1·9	1·7–2·0	–	–
Exposure to cows						
Cowshed ownership						
None	792	0·10	(ref.)		(ref.)	
Outside compound	521	0·10	1·0	0·8–1·1	1·0	0·9–1·2
In compound	1382	0·10	1·0	0·9–1·1	1·0	0·9–1·1
Number of cows owned						
0	1138	0·11	(ref.)		(ref.)	
1–2	1048	0·10	0·9	0·8–1·1	1·0	0·8–1·1
⩾3	553	0·10	0·9	0·8–1·1	1·0	0·8–1·1
Number of cowsheds within 50 m of house						
<10	802	0·10	(ref.)		(ref.)	
10–19	791	0·09	1·0	0·9–1·2	1·0	0·8–1·1
⩾20	1146	0·11	1·2	1·0–1·3	1·1	0·9–1·2
Dung used as main fuel for cooking						
No	2163	0·10	(ref.)		(ref.)	
Yes	576	0·09	0·9	0·9–1·0	0·9	0·8–1·1

RR, Risk ratio; CI, confidence interval; HH, household.

* Log-binomial regression with generalized estimating equations to adjust for multiple observations within households.

† House structure omitted from model due to collinearity.

outbreaks of gastrointestinal disease, the contribution of cow exposure to the overall burden of endemic (as opposed to epidemic) diarrhoea is less clear, given that diarrhoea is caused by a large number of pathogens most of which are easily transmitted among humans without requiring an animal reservoir. Our study suggests that the importance of direct exposure to cows for disease transmission may be small relative to other sources of infection. These results mirror those from a similar large-scale analysis from Vietnam that found no association between household or neighbourhood exposure to different types of livestock (e.g. poultry, cows, buffalos, pigs) and hospital admission for diarrhoea in children aged <5 years [31].

The concept of EE suggests that most environmentally transmitted gastrointestinal pathogens lead to under-nutrition in children by causing a chronic inflammatory state in the intestines, and only to a lesser extent by causing diarrhoea [4, 9]. While EE has been found to be associated with both unhygienic conditions and stunting, we found no evidence that exposure to cows contributes to this assumed causal pathway. In our study, socioeconomic, caste and educational factors were most strongly associated with under-nutrition. However, education of the head of the household and carer, water access, house structure, assets, land ownership and caste combined only explained 12% (R^2) of the variation in HAZ in our study population. EE from unhygienic conditions, but perhaps not from exposure to cows, may explain a share of the variability in HAZ unaccounted for by the factors measured in our study.

Households with a cowshed in the compound had slightly higher fly counts than households without a cowshed. Further analysis of the fly counts will be published in a separate paper. Preliminary analysis revealed no clear trends towards presence of cows increasing the number of flies in a household (data not shown). This finding suggests that attraction of synanthropic flies to the human environment may largely be due to inadequacies in waste management or lack of barriers to keep flies away from spaces used for cooking rather than presence of cows or cow dung.

Our analysis has two major limitations. First, unmeasured and imprecisely measured confounders could explain the lack of association between exposure to cows and the study outcomes. Under-nutrition and diarrhoea are likely to be strongly associated with poverty, while in our study area cow ownership was more common in wealthier households. In multivariate regression analysis, these two associations may have cancelled each other out, resulting in no effect. Multivariate adjustment can only partially address confounding as, conceivably, many true confounders were not measured in our study while those that were could not be measured with perfect accuracy [32].

The second major limitation of our study lies in the absence of a true control group. Similar to public health risks such as passive smoking or air pollution, few participants in our study population may have had no exposure to cows at all in their daily life. While the magnitude of cow exposure across different measures varied considerably within the study area (Fig. 1*b*), the analysis would have benefitted from including fully unexposed households. Cowsheds and cows are proxy markers of exposure to cow dung as the most obvious potential source of infection from cattle. Families without cows may collect cow dung elsewhere for use in the household. We did not collect data on the presence of cow dung in and around each household which may have provided a more accurate estimate of exposure to cow dung as opposed to cow ownership or the presence of a cowshed. In the Vietnamese study that similarly found no effect of cow exposure on child health, exposure to livestock animals was very common. Unlike in our study, the kinds of animals differed greatly within the study area, i.e. there were many households with little or no cows in their neighbourhood. The absence of a protective effect in this group of households in Vietnam suggests that the lack of effect found in both studies may not alone be due households in the low exposure categories still being sufficiently exposed to experience a health risk.

Other limitations include imprecision in constructing the variable for the number of cowsheds located within 50 m of a house as the exact location of cowsheds outside the compound was not recorded (see Methods section). Further, the baseline measure assessing socioeconomic status and the number of cows owned was conducted 2 years before the collection of the geospatial data that included variables on the size of all households in the study area and presence of a cowshed. Child growth was recorded nearly 3 years after the baseline survey. Some of the study households may have undergone changes in socioeconomic status and number of cows owned during that time. Diarrhoea measurements were on self- or carer-reported symptoms. No attempts were made to identify severe episodes (e.g. those leading to hospital admission).

To conclude, the children in this study were at a high risk of stunting and diarrhoea. Exposure to

cows did not appear to contribute to this disease burden. Our findings are compatible with the notion that most transmission of gastrointestinal disease occurs among humans. If EE critically contributes to stunting in rural India, then exposure to human excreta rather than cow dung may be the primary cause of chronic gut inflammation. However, corroborating this hypothesis may require further research including in-depth analysis of transmission pathways of gastrointestinal pathogens in low-income settings. Molecular methods including microbial source tracking able to distinguish between animal and human faecal exposure is being applied in this trial [14] and in other studies [33], and may contribute to our understanding of the causes of diarrhoea and stunting.

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DECLARATION OF INTEREST

None.

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