Published by Oxford University Press on behalf of the International Epidemiological Association
International Journal of Epidemiology 2008;**37**:831–840
doi:10.1093/ije/dyn101

Impact of a city-wide sanitation intervention in a large urban centre on social, environmental and behavioural determinants of childhood diarrhoea: analysis of two cohort studies

Bernd Genser,[1]* Agostino Strina,[1] Lenaldo A dos Santos,[1] Carlos A Teles,[1] Matildes S Prado,[1] Sandy Cairncross[2] and Mauricio L Barreto[1]

Accepted 8 May 2008

Background Poor socioeconomic status (SES) increases diarrhoea risk, mostly mediated by lack of sanitation, poor infrastructure and living conditions. The effectiveness of a city-wide sanitation intervention on diarrhoea in a large urban centre in Northeast Brazil has recently been demonstrated. This article aims to explore how this intervention altered the magnitude of relative and attributable risks of diarrhoea determinants and the pathways by which those factors affect diarrhoea risk.

Methods We investigated determinants of prevalence of diarrhoea in two cohort studies conducted before and after the intervention. Each study enrolled pre-school children followed up for 8 months. For both cohorts, we calculated relative, attributable and mediated risks of diarrhoea determinants by a hierarchical effect decomposition strategy.

Results The intervention reduced diarrhoea and also changed attributable and relative risks of diarrhoea determinants by altering the pathways of mediation. Before the intervention SES was a major distal diarrhoea determinant (attributable risk: 24%) with 90% of risk mediated by other factors, mostly by lack of sanitation and poor infrastructure (53%). After the intervention, only 13% of risk was attributed to SES, with only 42% mediated by other factors (18% by lack of sanitation and poor infrastructure).

Conclusion The intervention reduced diarrhoea risk by reducing direct exposure to unfavourable sanitation conditions. At the same time it altered the effect and mediation pathways of most distal diarrhoea determinants, especially SES. This finding corroborates the importance of public sanitation measures in reducing the impact of poverty on diarrhoea. It also underlines the value of studying the impact of public health interventions to improve our understanding of health determinants.

Keywords Diarrhoea, risk factors, sanitation interventions, developing countries, socioeconomic status, poverty, Brazil

---

[1] Instituto de Saúde Coletiva, Federal University of Bahia, Salvador, Brazil.

[2] London School of Hygiene & Tropical Medicine, London, UK.

* Corresponding author. Instituto de Saúde Coletiva, Federal University of Bahia, Rua Basilio da Gama, s/n-Campus Universitario Canela, Salvador, Bahia 40.110-040, Brazil. E-mail: bernd.genser@bgstats.com

Introduction

Diarrhoeal diseases are a major child health problem in developing countries, especially in the growing urban areas.[1] Epidemiological studies conducted in various developing countries have shown that diarrhoea has a complex epidemiology. Diarrhoea risk is affected by numerous determinants on different causal levels, but the major role of poor socioeconomic status (SES) is well established.[2–6]

It is widely accepted that diarrhoea risk is affected by a hierarchy of risk factors, of which the distal factors are mediated through causal pathways involving the more proximal ones,[7] and hierarchical modelling techniques are mainly used to control for confounding and the risk factors identified one by one.[8] Rarely have the pathways themselves been the subject of study or any changes in them resulting from an intervention. Poverty is still one of the major distal determinants of diarrhoea, and is closely related to lack of sanitation, poor neighbourhood infrastructure and poor living conditions.[4,9] In urban settings, this is very clear because huge inequalities between rich and poor are expressed in marked social, environmental and health differences.[10] Children in the poorest stratum suffer from a high burden of various infectious diseases as a consequence of their unfavourable environment.[10]

The eradication of poverty in developing countries is not very likely during the next decades, but public interventions such as improving sanitation, water supply and neighbourhood infrastructure, food supply etc. seem to be immediate effective ways to improve child health. Several studies have shown the important role of inadequate water supply and sanitation in the occurrence of diarrhoeal diseases[11–14] and have shown that improving water supply and sanitation prevents them.[15–18]. However, very few studies have evaluated the impact of these interventions in urban settings, or of sanitation alone. Recently, an evaluative epidemiological study in Salvador, a large urban centre in Northeast Brazil, has shown the impact of a city-wide sanitation programme in controlling diarrhoeal diseases.[19] The evaluation was conducted using a robust observational methodology with precise measurement of the outcome in two cohorts before and after the intervention and with measurement of potential confounders at individual and contextual levels. The study showed a reduction in diarrhoea prevalence by 21% (95% CI 18–25%), from 9.2 (9.0–9.5) days per child-year before the intervention to 7.3 (7.0–7.5) days per child-year afterwards. By means of a novel hierarchical analysis strategy, the study also showed how different components of the intervention (e.g. the increased sewerage coverage, the improved water supply, etc) contributed to the decrease in diarrhoea risk.

There is some recognition that well-conducted health impact assessments, such as the study done in Salvador, have the potential to enhance recognition of societal determinants of health.[20] Large-scale sanitation programmes are complex interventions that affect the transmission of diarrhoeal diseases directly by reducing exposure to proximal risk factors, and also indirectly acting on distal ones. It is likely that such interventions act not only by changing the attributable risk of distal diarrhoea determinants (e.g. poor SES or poor living conditions), but also by modifying the mediating pathways by which these determinants act on the outcome. In this article, we further investigate the impact of the same city-wide sanitation programme on determinants of child diarrhoea.

Methods

Salvador, capital of Bahia state in Northeastern Brazil (population 2.5 million) has health problems typical of a large urban centre. The improvement in water supply in the 1980s and 1990s (by 1997 over 90% of the city's households had access to piped water) must have had some effect on childhood diarrhoea, but this was never measured. In 1997, the prevalence of child diarrhoea measured in a longitudinal study was 9.2 days per child-year. A large sanitation programme, initiated in 1997, known as *Bahia Azul* or '*Blue Bay*', was implemented with the objective of increasing the proportion of the population with an adequate sewer connection from 26% to 80%; details of the intervention are given elsewhere.[19] To assess the epidemiological impact of the intervention an evaluation study was conducted composed of two longitudinal studies.[19] Each consisted of a cohort of children aged 0–36 months at baseline, recruited from households selected from the same 24 sentinel areas chosen to represent the parts of the city without access to sewerage. The sampling design of the study has been described in detail elsewhere.[19,21,22] The first study, carried out before the intervention, enrolled 832 children beginning in September 1997 and followed them over a period of up to 15 months (until November 1998). The second study was conducted after the intervention, beginning in October 2003 and enrolled 992 children with a follow-up of up to 8 months (until May 2004). To make the two cohorts comparable, the analysis of the first cohort was restricted to the first 8 months of follow-up.

Diarrhoea data were collected by twice-weekly home visits carried out by 15 fieldworkers. During each visit, the field worker questioned the mother or child's caretaker about the number and consistency of bowel movements, and the occurrence of additional symptoms (such as fever, vomiting and blood in stool) over the preceding 3–4 days. A day with diarrhoea was defined by the occurrence of three or more liquid or loose stools starting when the child woke in the morning.[23] As outcome variable, we chose prevalence of diarrhoea, defined as the number of diarrhoea days divided by the total number of days under observation.[24,25] Days with missing diarrhoea information were excluded from the person's time at risk. Stool samples were collected only once during the follow-up and examined for the presence of intestinal parasites (Ascaris lumbricoides, Trichiuris trichiura, Giardia lamblia). The mother or child's caretaker was visited at home and given a numbered, labelled container, which was collected the next morning, placed on ice and taken immediately for analysis the same day. Each stool sample was examined using the Kato-Katz method for the presence and number of helminth eggs.

In addition, at the beginning of both cohort studies, individual and household questionnaires were applied by fieldworkers to assess potential child and household confounding variables. These included SES, living and sanitation conditions of the households and child-related variables (birth weight and breast feeding). Anthropometric measurements of nutritional status were done at baseline, and height-for-age *Z*-scores were calculated by use of the EPINUT programme (version 6.0).[26] The fieldworkers were also trained to check a list of 23 forms of hygienic or unhygienic behaviour by the child or the child's caretaker if they were observed during two visits every week. On the basis of this information, a composite hygiene behaviour score was calculated for each child. Details of the hygiene behaviour observations have been reported elsewhere.[27] In addition, for each sentinel area contextual variables were assessed, on the basis of environmental surveys that were done in 1997 (for cohort 1) and 2004 (for cohort 2). These surveys used similar methodologies and their unit of sampling was the 100 m stretch of road running 50 m to either side of each sampled house.[10]

Statistical analysis

Statistical analysis was conducted according to a predefined conceptual model (shown in Figure 1) that reflects our hypotheses about how diarrhoea determinants are grouped in blocks, the inter-relationships existing between these blocks and the pathways by which these variables act on diarrhoea prevalence. The conceptual framework selected is similar to a model that has been used previously in the same context to analyse determinants of diarrhoea incidence.[4] Following the framework we implemented a hierarchical effect decomposition strategy (HED) to quantify the effect of risk factors on different levels and to disentangle direct and mediated effects of these factors. Details of the HED are described elsewhere.[28,29]

First, descriptive statistics were calculated to examine the distribution of covariates before and after the intervention. Then, bivariate analyses were carried out to identify potential diarrhoea determinants by calculating prevalence ratios (PR) (exposed vs non-exposed children) and 95% CI. Robust CIs were calculated by using a resampling technique to adjust for intra-cluster correlation due to children recruited from the same sentinel areas.[30] Second, we fitted a sequence of mixed effects Poisson regression

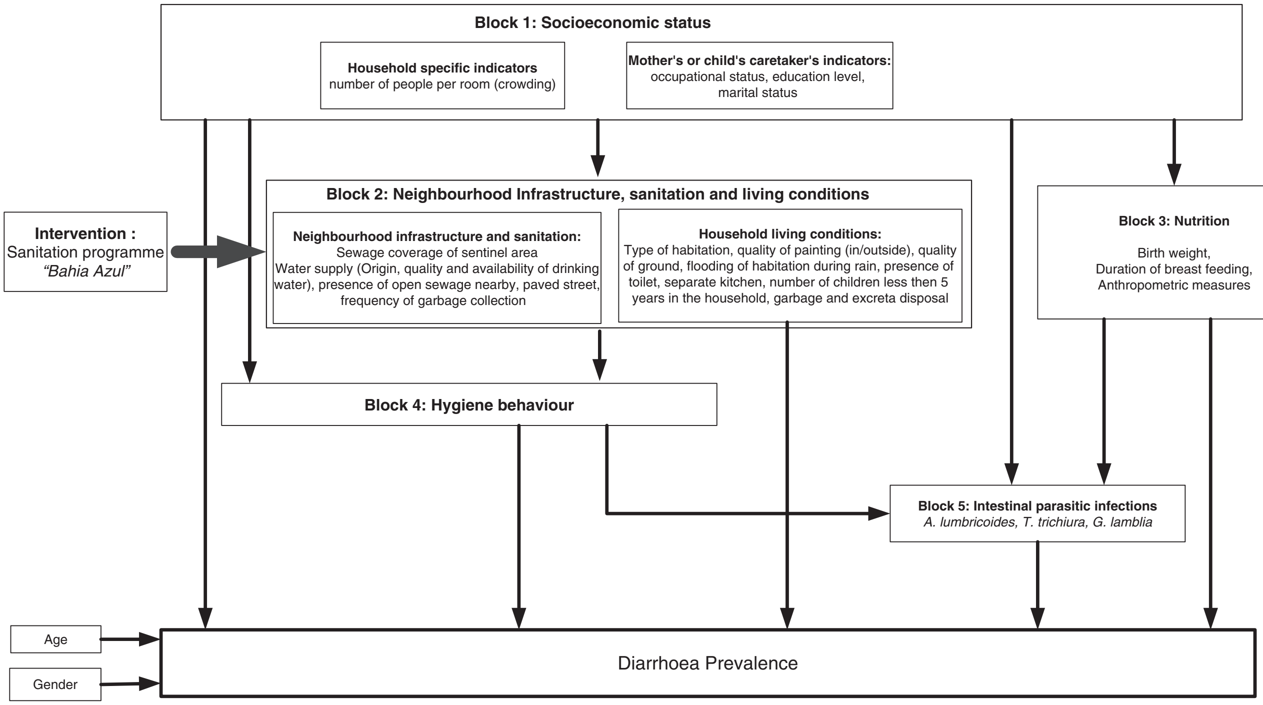

Figure 1 Conceptual framework visualizing the inter-relationships between potential risk factors, the sanitation intervention and diarrhoea prevalence

models each including different blocks of variables to obtain multivariate PR. All models were adjusted for confounding by age and gender; adjustment for intracluster correlation was done by including a gamma-distributed random effect. For multivariate analysis, missing data for explanatory variables were filled by imputation of the mean for quantitative variables and the mode for categorical ones, assuming that there was no association between diarrhoea risk and the probability of observing it on the day (assuming 'missing at random'). Variable selection in each block was done using a backward selection procedure using a *P*-value of 0.1.

Briefly, we started with model A (including only block 1) that sought to estimate the overall effect of SES. Followed by model B additionally including variables from block 2 (neighbourhood infrastructure, sanitation and living conditions) and model C additionally including block 3 variables (nutrition) and so on. By comparing the effect estimate (e.g. for SES, the PR of the poorest group vs other groups) before and after adjusting for the next block in the hierarchy of the framework (e.g. block 2) we get an estimate how much of the effect of the block has been mediated by the block on the pathway. Some authors have called this proportion of excess risk explained the 'mediation proportion'.[28]

In addition, we calculated multivariate adjusted estimates of attributable risks from the relative risks for both exposed subjects and the whole study population. Finally, we extended the idea of 'excess risk explained' to attributable risks and quantified the fraction of attributable risk mediated by the pathway by comparing the 'overall' estimates of attributable risk of each block with the estimates that were adjusted for blocks on the pathway. All statistical analyses were carried out using the statistical software package STATA (version 9.2, STATA Corporation, College Station, TX, USA).

Ethics

Ethical approval for the study was given by the ethics review board of the Instituto de Saúde Coletiva, Federal University of Bahia. Written informed consent to participate in the study was obtained from the guardians of all studied children.

Results

The study population was selected from the population of the city, as described elsewhere.[19] For this study, we selected children that were followed up for at least 90 days and for whom all study variables of the conceptual framework in Figure 1 (except for data about intestinal parasites) were available. The first cohort, before the intervention, consisted of 832 children (mean age 18.1 months, SD: 9.8, median follow-up: 239 days). The second cohort, after the intervention, consisted of 992 children (mean age 17.1 months, SD: 9.7, median follow-up: 201 days). The prevalence of diarrhoea decreased from 9.2 diarrhoea days per year (95% CI: 8.9–9.6) before the intervention to 7.3 diarrhoea days per year (95% CI: 6.9–7.5) after the intervention, a reduction of 21%.

Table 1 summarizes the results of the HED approach showing all factors that showed multivariate associations with diarrhoea prevalence ($P < 0.05$). For each factor, estimates of PR and 95% CI are shown. According to the principle of HED,[7] the PR shown refer to the 'overall effect', i.e. the effect adjusted for all potential confounding variables from the same or upper levels of the conceptual framework (Figure 1) but not adjusted for mediating variables on the pathway to the outcome. For instance, the overall effect of block 2 (sanitation, neighbourhood infrastructure and living conditions) is adjusted for age, gender and SES (located one causal level above) and block 3 (located on the same causal level) but not for the effect of blocks 4 and 5 (that are potential mediating variables). In addition, the proportion of multivariate attributable risks is shown for both exposed individuals (AR) (i.e. quantifying the diarrhoea risk in exposed subjects that is due to exposure) and the total study population (ART) (i.e. quantifying how much risk in the total population can be attributed to exposure). In addition, we show the cumulative proportion of ART for each block of variables. For SES we observed a decrease in ART from 24% in cohort 1 to 13% in cohort 2. This decrease could not be explained either by changes in the distribution of this variable or by changes in the magnitude of association from cohort 1 to cohort 2. We observe a higher PR for the poorest (stratum 4) but a decrease in stratum 3.

As expected, after the construction of the new sewer system, attributable risk due to block 2a (neighbourhood infrastructure, sanitation and living conditions) changed substantially. The increase in ART from 24% to 47% was mostly related to the construction of the new 'Bahia Azul' project sewers that, even adjusted for SES, explained 28% of the diarrhoea risk. A decrease in ART was observed for water supply, on the one hand because there were more houses with piped water (coverage increased from 74% to 96%), and on the other hand by reducing the strength of association (e.g. PR for inadequate water supply diminished from 1.75 to 1.32). For open sewage nearby, despite the decrease in the frequency of exposure (47% to 23%) the PR increased from 1.09 to 1.47, resulting in a substantial increase of AR from 4% to 10%.

In contrast, for variables of block 2b (household and living conditions) we did not observe any substantial change in PR. Except for type of floor (PR decreased from 1.30 to 1.03) the distribution and strength of association with these risk factors was similar before and after the intervention. Attributable and relative risks for

Table 1 Estimates of PR and attributable risks of factors associated with diarrhoea prevalence before and after the sanitation intervention

	Cohort 1 (before, $n=832$ children)					**Cohort 2 (after, $n=992$ children)**				
	Children *n* (%)	PR	95% CI	AR[a]	ART[b]	Children *n* (%)	PR	95% CI	AR[a]	ART[b]
Block 1: Socioeconomic status										
Socioeconomic index					24[c]					13[c]
1 (richest)	119 (14)	1.00				164 (17)	1.00			
2	207 (25)	1.04	0.91–1.19	4		279 (28)	1.11	0.97–1.26	10	
3	312 (38)	**1.33**[f]	**1.18–1.50**[f]	25		335 (34)	**1.19**[f]	**1.05–1.35**[f]	16	
4 (poorest)	194 (23)	**1.49**[f]	**1.31–1.69**[f]	33		214 (22)	**1.62**[f]	**1.39–1.88**[f]	38	
Block 2a: Sanitation and neighbourhood infrastructure					24[d]					47[d]
Connections to sewer[e]										
>75% of houses in area	183 (22)	1.00			0	268 (27)	1.00			28
>50%, ⩽75% of houses in area	251 (30)	0.94	0.84–1.04	0		441 (45)	**1.59**[f]	**1.43–1.76**[f]	33	
>25%, ⩽50% of houses in area	314 (37)	**0.74**[f]	**0.66–0.82**[f]	0		199 (20)	**1.21**[f]	**1.07–1.38**[f]	16	
⩽25% of houses in area	84 (10)	1.12	0.98–1.28	11		84 (8)	**1.87**[f]	**1.61–2.18**[f]	47	
Open sewage nearby					4					10
Absent	442 (53)	1.00				766 (77)	1.00			
Present	390 (47)	**1.09**[f]	**1.01–1.08**[f]	8		226 (23)	**1.47**[f]	**1.34–1.60**[f]	32	
Water supply					11[c]					0[c]
Piped water inside the household	613 (74)	1.00				865 (96)	1.00			
Access to piped water close to the household	102 (12)	**1.19**[f]	**1.06–1.34**[f]	16		98 (11)	0.74	0.64–0.87	0	
No access to piped water	117 (14)	**1.75**[f]	**1.59–1.93**[f]	43		29 (3)	**1.32**[f]	**1.07–1.57**[f]	24	
House served by paved street					9					9
Yes	256 (31)	1.00				369 (41)	1.00			
No	576 (69)	**1.14**[f]	**1.04–1.24**[f]	12		623 (59)	**1.14**[f]	**1.04–1.25**[f]	13	
Block 2b: Household living conditions					10[d]					11[d]
Type of housing					2					6
House/apartment	765 (92)	1.00				912 (92)	1.00			
Shack	67 (8)	**1.23**[f]	**1.05–1.43**[f]	20		80 (8)	**1.29**[f]	**1.09–1.53**[f]	23	
Floor conditions					4					0
Ceramic/cement	721 (87)	1.00				955 (96)	1.00			
Unpaved floor	111 (13)	**1.30**[f]	**1.15–1.47**[f]	25		37 (4)	1.03	0.84–1.27	3	
Flooding of house during rain					4					5
No	689 (68)	1.00				867 (87)	1.00			
Yes	143 (17)	**1.24**[f]	**1.13–1.36**[f]	19		125 (13)	**1.49**[f]	**1.34–1.65**[f]	35	
Block 3: Nutrition					18[d]					17[d]
Birth weight					7					5
⩾2500 g	713 (86)	1.00				861 (87)	1.00			
<2500 g	119 (14)	**1.51**[f]	**1.37–1.67**[f]	34		131 (13)	**1.37**[f]	**1.24–1.53**[f]	27	
Anthropometry (height for age)					9					5
⩾−1 (normal)	657 (79)	1.00				821 (83)	1.00			
<−1 (subnutrition)	175 (21)	**1.46**[f]	**1.34–1.59**[f]	32		171 (17)	**1.28**[f]	**1.17–1.40**[f]	22	

(continued)

Table 1 Continued

	Cohort 1 (before, $n = 832$ children)					**Cohort 2 (after, $n = 992$ children)**				
	Children n (%)	PR	95% CI	AR[a]	ART[b]	Children n (%)	PR	95% CI	AR[a]	ART[b]
Exclusive breast feeding					2					7
<6 months	686 (82)	1.00				841 (85)	1.00			
⩾6 months	146 (18)	**1.13**[f]	**1.01–1.25**[f]	12		151 (15)	**1.53**[f]	**1.39–1.69**[f]	35	
Block 4: Hygiene behaviour										
Hygiene behaviour score					30[c]					15[c]
Good	175 (21)	1.00				266 (27)	1.00			
Intermediate	504 (61)	**1.44**[f]	**1.30–1.60**[f]	22		421 (42)	**1.15**[f]	**1.04–1.26**[f]	13	
Bad	153 (18)	**1.92**[f]	**1.71–2.16**[f]	48		305 (31)	**1.37**[f]	**1.24–1.52**[f]	27	
Block 5: Intestinal parasitic infections					3[d]					1[d]
A. lumbricoides	679 (82)	1.00								
	153 (18)	0.85	0.77–0.94	0	0	108 (12)	1.07	0.94–1.22	7	1
T. trichiura	719 (86)	1.00								
	113 (14)	**1.33**	**1.19–1.48**[f]	23	3	41 (5)	1.00	0.79–1.26	0	0
G. lamblia	742 (89)	1.00								
	90 (11)	0.95	0.85–1.07	0	0	44 (5)	0.86	0.71–1.04	0	0

[a] Adjusted proportion of attributable risk of diarrhoea due to exposure in exposed individuals, estimates were calculated from the predicted rates for each stratum resulting from the multivariate estimates of relative risk.

[b] Adjusted proportion of attributable risk of diarrhoea due to exposure in the entire study population, the estimate is based on the rate in unexposed subjects adjusted for covariates.

[c] For variables with more than two categories the first category was considered as the stratum of unexposed individuals.

[d] Sum of proportion of attributable risk of all variables of the block.

[e] The coverage shown for the first cohort refers to connections to any existing sewer system before the intervention, the coverage shown for the second cohort refers to the new ‘Bahia Azul’ project sewers.

[f] P values refer to the Wald test statistic (testing whether the regression coefficient of the Poisson regression model was significantly different from zero).

nutritional variables also did not change substantially. The reduction of attributable risk due to unhygienic behaviour was more striking (30% to 15%). Intestinal parasites did not increase diarrhoea risk except for *T. trichiura*, whose PR was 1.33 before the intervention, but diminished after the intervention (PR = 1.00).

Table 2 summarizes the ART with respect to the total study population showing how each block of variables contributed to the diarrhoeal prevalence. We observed a reduction of attributable risk of poor SES (−11%) without any substantial change in the distribution of this risk factor in the study population. As expected, the sewerage intervention substantially changed the population attributable risk of block 2 (increase of 24%), even adjusted for SES, lack of sanitation and neighbourhood infrastructure explained an additional 23% of the diarrhoea risk in cohort 2. Improving sanitation also reduced the attributable risk due to negative hygiene behaviour (−15%), mostly by reducing the magnitude of association (Table 1) rather than by improving the behaviour of the population. Nutritional status also had an effect on diarrhoea risk, but attributable risks were quite similar in both cohorts.

In addition, by our HED approach we were able to investigate whether the intervention also changed the pathways by which the risk factors acted on the diarrhoea occurrence. By fitting a sequence of regression models each adjusting for different blocks of variables we quantified the proportion of effect mediated by other variables on the pathway. Effect measures were derived from the multivariate estimates of attributable risk that refer to the overall effect of each block (shown in Table 1) and estimates step-by-step adjusted for other blocks of variables on the pathway (not shown). The results of this effect decomposition analysis are shown in Figure 2. We found that in both cohorts a large fraction of the effect of SES was mediated by other factors. The proportion of attributable risk thus mediated decreased substantially after the intervention. Before the intervention, 90% of the effect of SES was mediated; 53% by block 2, 24% by block 3, 11% by block 4 and 2% by block 5. In contrast, after the intervention only 42% of effect of SES was mediated by the factors of our conceptual framework; 18% by block 2, 9% by block 3 and 15% by block 4. Sanitation infrastructure and nutritional status proved to be more directly

Table 2 Change in population attributable risk of the study population due to exposure to diarrhoea determinants before and after the intervention

	Population attributable risk (%)		
	Cohort 1	Cohort 2	Change
Block 1: Socioeconomic status	24	13	−11
Block 2: Neighbourhood infrastructure, sanitation and living conditions	34	58	+24
Block 2a: Neighbourhood infrastructure and sanitary conditions	24	47	+23
Block 2b: Household living conditions	10	11	+1
Block 3: Nutrition	18	17	−1
Block 4: Hygiene behaviour	30	15	−15
Block 5: Intestinal parasitic infections	3	1	−2

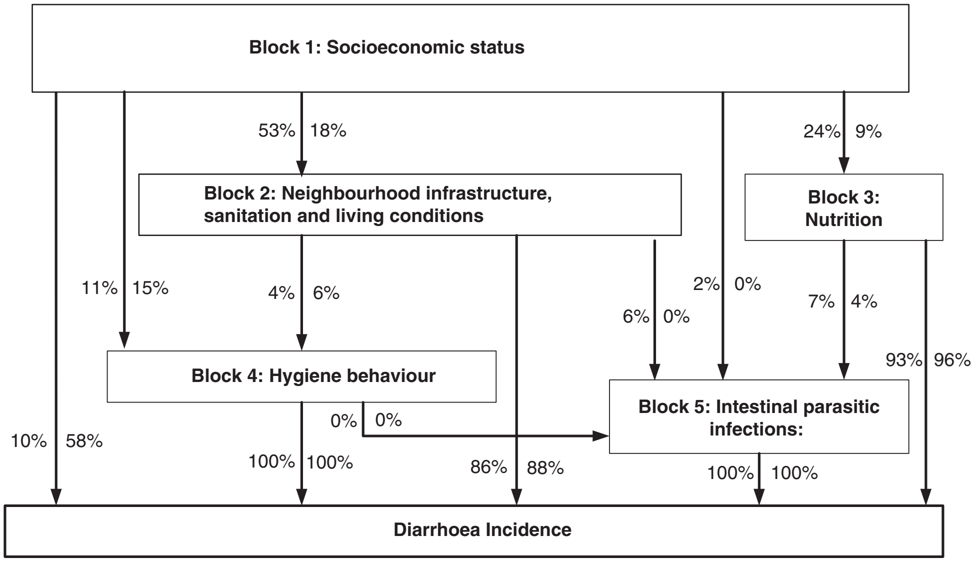

Figure 2 Proportion of population attributable risk explained by mediating factors (mediating proportion) before and after the intervention, displayed in the left and right side of each arrow, respectively. The numbers close to the arrows (left: before the intervention, right: after the intervention) show the proportion of attributable risk (of the block the arrow comes from) mediated by the block the arrow goes to. The estimates of the mediated proportion (MP) have been derived from the adjusted (ART_{adj}) and unadjusted estimates (ART_{unadj}) of multivariate attributable risks according to the formula: $MP = (ART_{unadj} - ART_{adj})/ART_{unadj} \times 100$

associated with diarrhoea risk; only 14% of block 2 were mediated by other variables before and 12% after the intervention, while mediation of block 3 was 7% before and 4% after the intervention.

Discussion

The aim of this study was to investigate the impact of a city-wide sanitation intervention in a large urban centre in Northeast Brazil on determinants of child diarrhoea. We identified diarrhoea determinants in two cohort studies conducted before and after the intervention and quantified the magnitude of relative, attributable and mediated risk of these factors by a HED strategy based on a predefined conceptual framework.

As a main result of the present analysis we found that the city-wide sanitation intervention besides generating a substantial decrease in child diarrhoea prevalence, shown elsewhere,[9] changed the magnitude of attributable and relative risks of many proximal and distal factors and also altered the pathways by which SES acted on the diarrhoea occurrence.

As expected, our study showed that the intervention substantially altered diarrhoea occurrence directly by reducing exposure to proximate factors that are directly related to diarrhoea (e.g. lack of adequate sanitation, neighbourhood infrastructure or household conditions). For instance, by increasing the coverage of sewer connections the intervention reduced exposure to open puddles and streams of sewage. However, the multivariate relative risk for ‘open sewage’ increased. This finding suggests that after the intervention, this manifestation of inadequate sanitation was less closely associated with poverty. Before the intervention, it was mainly the poor who lacked sewerage; after the intervention, other factors, such as local topography making sewer construction difficult, are more likely to have come into play, weakening the association between poverty and poor sanitation.

Improving sanitation also reduced the relative risk of inadequate floor conditions in the household and the effect of unhygienic behaviour on diarrhoea. A cement floor is easier to clean, and cleaning the floor assumes greater importance in the prevention of diarrhoea if people's shoes are likely to be contaminated with sewage. So does hygienic behaviour such as washing hands and cleaning objects that fall on the floor. Thus, both changes would be expected if, due to the increasing sewerage coverage, there was a decrease in the burden of pathogens carried into the domestic environment—for instance on shoes and on hands. As a consequence, conditions that before the intervention were strongly associated with diarrhoea occurrence (e.g. inadequate floor conditions or unhygienic behaviours) became less risky and 'caused' less diarrhoea, even without changes in their frequencies.

This new finding has several implications. Much of the global interest devoted to diarrhoea control is focused on behavioural changes;[27,31] however, while unhygienic behaviour was the most important single risk factor in the first cohort (Table 1), and without major changes on its frequency, its role decreased in consequence of the environmental improvements which occurred.

The most rewarding finding derived from our analysis was that the intervention substantially altered the attributable risk of poor SES without changing its relative risk or the prevalence of poverty in the city. Before the intervention, poverty was a major determinant of diarrhoea (attributable risk: 24%). Most of the effect of SES was mediated (mediating proportion 90%) indicating that poor SES was closely related to other more proximate diarrhoea determinants such as lack of sanitation, inadequate environmental infrastructure and poor living conditions. This finding of mediation of SES is not very surprising and completely in line with the results of a previous study in the same city that investigated risk factors for diarrhoea incidence.[4]

After adjustment for SES, access to the existing sewer system did not explain any diarrhoea risk. This finding can be explained by the fact that before the intervention poor SES was closely related to lack of sanitation, and also that the pre-existing sewerage was ineffective due to poor construction and maintenance.[10] After the intervention, the situation had changed: SES had a less important role (attributable risk: 13%) but diarrhoea occurrence was strongly related to the coverage of the new sewage system (attributable risk: 28%). These results suggest that the progress of the intervention (e.g. the coverage of the new sewer system) and its effect on diarrhoea occurrence seems to have happened independently of SES. While economic constraints delayed connection of some of the poorest households to the sewer system, the intervention was a city-wide public measure planned to benefit all the households in the areas covered.[32] Moreover, the evaluation study[11] found that the protective effect was associated with the overall coverage of the neighbourhood, rather than with access to the system by the household of the individual child. The evaluation study also showed that the intervention was most effective in very poor areas, which had previously had the highest diarrhoea risk.[19] Thus, the intervention decreased the association between poverty and unfavourable environmental conditions *directly* by reducing the population's exposure to environmental risk factors. The intervention also altered the attributable risk of SES *indirectly* by changing the magnitude of relative risk of variables on the pathways by which poor SES was mediated, e.g. floor conditions, anthropometry and hygiene behaviour.

This study, using a comparison of the epidemiological situation before and after a city-wide sanitation programme, shows how improving sanitation affects the contribution of other determinants of diarrhoea risk that did not change significantly during the period of the intervention (e.g. SES). In addition, by implementing a HED strategy we were able to estimate the effects of risk factors operating at different levels and also to disentangle direct and intermediate effect components involved in diarrhoea occurrence in a complex urban setting. It is a relevant contribution on the search for interventions targeted to reduce socioeconomic inequalities in health.[33]

A limitation of our study was that the comparison was conducted using different children (though from the same sentinel areas), so that bias due to sampling error and confounding due to unobserved child specific variables could not be completely excluded. Moreover, since it was a longitudinal study over several years several unmeasured factors might have changed during the time of the study. However, it is not likely that these changes would account for the findings of our study with respect to the main study question, i.e. whether a complex sanitation intervention was able to change relative, attributable risks and pathways of important diarrhoea determinants. Our major finding was that the intervention changed the attributable risk and the pathways of SES without changing the prevalence of poverty. Before the intervention, the effect of SES was mostly mediated by poor living conditions and lack of access to sanitation. Since the intervention improved sanitation and environment infrastructure of the study households independent of their SES, poverty was less related to poor sanitation after the intervention. It is much more likely that the intervention, rather than changes in other unobserved variables, accounts for this major finding. A further limitation of our study was that the HED approach is only valid when there is no confounding at the intermediate levels of the framework, an assumption that we have discussed in detail elsewhere.[4,28]

In conclusion, our study shows that a sanitation intervention in a large urban centre can have a substantial impact on the epidemiology of child diarrhoea. Improving sanitation decreases the attributable risk of diarrhoea determinants directly by reducing exposure to these factors, or indirectly by changing the strength of associations with them. An indirect effect of the sanitation intervention is to alter the pathways by which socioeconomic factors act on the outcomes. In particular, the intervention reduced the risk attributable to poor SES, a major health determinant in developing countries, without changing the prevalence or distribution of poverty. This finding underlines the importance of public sanitation measures as part of a group of compensatory policies in reducing the impact of poverty and social inequalities on poor child health. Moreover, the interesting findings of the hierarchical analysis approach used in this study underline the importance of studying the impact of public health interventions in order to understand inter-relationships and complex pathways of health determinants.

Acknowledgements

The authors thank the fieldwork team and the families that participated in the study. This study was supported by PRONEX—Centres of Excellence Programme; CNPq, Brazil (661086/1998-4); the Department of Infrastructure, State Government of Bahia; Fundação de Amparo à Pesquisa do Estado da Bahia (FAPESB), Brazil (1360/2006); the Wellcome Trust, UK (072405/Z/03/Z and 082569/Z/07/Z to BG).

Conflict of interest: None declared.

References

[1] Kosek M, Bern C, Guerrant RL. The global burden of diarrhoeal disease, as estimated from studies published between 1992 and 2000. *Bull World Health Organ* 2003;**81:**197–204.

[2] Molbak K. The epidemiology of diarrhoeal diseases in early childhood. A review of community studies in Guinea-Bissau. *Dan Med Bull* 2000;**47:**340–58.

[3] Punyaratabandhu P, Sangchai R, Vathanophas K, Athipanyakom S, Varavithya W. Risk factors for childhood diarrhea in an urban community, Bangkok, Thailand. *J Med Assoc Thai* 1993;**76:**535–41.

[4] Genser B, Strina A, Teles CA, Prado MS, Barreto ML. Risk factors for childhood diarrhea incidence: dynamic analysis of a longitudinal study. *Epidemiology* 2006;**17:**658–67.

[5] Chowdhury MK, Gupta VM, Bairagi R, Bhattacharya BN. Does malnutrition predispose to diarrhoea during childhood? Evidence from a longitudinal study in Matlab, Bangladesh. *Eur J Clin Nutr* 1990;**44:**515–25.

[6] Yeager BA, Lanata CF, Lazo F, Verastegui H, Black RE. Transmission factors and socioeconomic status as determinants of diarrhoeal incidence in Lima, Peru. *J Diarrhoeal Dis Res* 1991;**9:**186–93.

[7] Victora CG, Huttly SR, Fuchs SC, Olinto MT. The role of conceptual frameworks in epidemiological analysis: a hierarchical approach. *Int J Epidemiol* 1997;**26:**224–27.

[8] Fuchs SC, Victora CG. Risk and prognostic factors for diarrheal disease in Brazilian infants: a special case-control design application. *Cad Saude Publica* 2002;**18:** 773–82.

[9] Assis AM, Barreto ML, Santos NS, de Oliveira LP, Dos Santos SM, Pinheiro SM. [Inequality, poverty, and childhood health and nutritional conditions in Northeast Brazil]. *Cad Saude Publica* 2007;**23:**2337–50.

[10] Milroy CA, Broja PC, Barros FR, Barreto ML. Evaluating sanitary quality and classifying urban sectors according to environmental conditions. *Environ Urban* 2001;**13:**235–56.

[11] Baltazar JC, Solon FS. Disposal of faeces of children under two years old and diarrhoea incidence: a case-control study. *Int J Epidemiol* 1989;**18 (4 Suppl 2):**S16–19.

[12] Rahman M, Rahaman MM, Wojtyniak B, Aziz KM. Impact of environmental sanitation and crowding on infant mortality in rural Bangladesh. *Lancet* 1985;**2:**28–31.

[13] Victora CG, Smith PG, Vaughan JP *et al*. Water supply, sanitation and housing in relation to the risk of infant mortality from diarrhoea. *Int J Epidemiol* 1988;**17:**651–54.

[14] Checkley W, Gilman RH, Black RE *et al*. Effect of water and sanitation on childhood health in a poor Peruvian peri-urban community. *Lancet* 2004;**363:**112–18.

[15] Clasen T, Schmidt WP, Rabie T, Roberts I, Cairncross S. Interventions to improve water quality for preventing diarrhoea: systematic review and meta-analysis. *Br Med J* 2007;**334:**782.

[16] Clasen T, Cairncross S, Haller L, Bartram J, Walker D. Cost-effectiveness of water quality interventions for preventing diarrhoeal disease in developing countries. *J Water Health* 2007;**5:**599–608.

[17] Pruss A, Kay D, Fewtrell L, Bartram J. Estimating the burden of disease from water, sanitation, and hygiene at a global level. *Environ Health Perspect* 2002;**110:**537–42.

[18] Moraes LR, Cancio JA, Cairncross S, Huttly S. Impact of drainage and sewerage on diarrhoea in poor urban areas in Salvador, Brazil. *Trans R Soc Trop Med Hyg* 2003;**97:**153–58.

[19] Barreto ML, Genser B, Strina A *et al*. Effect of city-wide sanitation programme on reduction in rate of childhood diarrhoea in northeast Brazil: assessment by two cohort studies. *Lancet* 2007;**370:**1622–28.

[20] Krieger N, Northridge M, Gruskin S *et al*. Assessing health impact assessment: multidisciplinary and international perspectives. *J Epidemiol Community Health* 2003; **57:**659–62.

[21] Barreto ML, Strina A, Prado M *et al*. Saneamento básico e saúde: fundamentos científicos para avaliação do impacto epidemiólogico do programa de saneamento ambiental da Bahia de Todos os Santos (Bahia Azul). In: Heller l ML, Monteiro TC *et al*. (eds). *Saneamento e Saúde nos Países em Desenvolvimento*. Rio de Janeiro: CC&P Editores, 1997. pp. 7–35.

[22] Teixeira MG, Barreto ML, Costa MC, Strina A, Martins D, Jr., Prado M. Sentinel areas: a monitoring strategy in public health. *Cad Saude Publica* 2002;**18:**1189–95.

[23] Baqui AH, Black RE, Yunus M, Hoque AR, Chowdhury HR, Sack RB. Methodological issues in diarrhoeal diseases epidemiology: definition of diarrhoeal episodes. *Int J Epidemiol* 1991;**20:**1057–63.

[24] Morris SS, Cousens SN, Kirkwood BR, Arthur P, Ross DA. Is prevalence of diarrhea a better predictor of subsequent mortality and weight gain than diarrhea incidence? *Am J Epidemiol* 1996;**144:**582–88.

[25] Schmidt WP, Luby SP, Genser B, Barreto ML, Clasen T. Estimating the longitudinal prevalence of diarrhea and other episodic diseases: continuous versus intermittent surveillance. *Epidemiology* 2007;**18:**537–43.

[26] Prado MS, Cairncross S, Strina A, Barreto ML, Oliveira-Assis AM, Rego S. Asymptomatic giardiasis and growth in young children; a longitudinal study in Salvador, Brazil. *Parasitology* 2005;**131**(Pt 1):51–56.

[27] Strina A, Cairncross S, Barreto ML, Larrea C, Prado MS. Childhood diarrhea and observed hygiene behavior in Salvador, Brazil. *Am J Epidemiol* 2003;**157:**1032–38.

[28] Ditlevsen S, Christensen U, Lynch J, Damsgaard MT, Keiding N. The mediation proportion: a structural equation approach for estimating the proportion of exposure effect on outcome explained by an intermediate variable. *Epidemiology* 2005;**16:**114–20.

[29] Ditlevsen S, Keiding N, Christensen U, Damsgaard MT, Lynch J. Mediation proportion. *Epidemiology* 2005;**16:**592.

[30] Fawzi WW, Herrera MG, Willett WC *et al*. Dietary vitamin A intake and the risk of mortality among children. *Am J Clin Nutr* 1994;**59:**401–08.

[31] Curtis V, Cairncross S. Effect of washing hands with soap on diarrhoea risk in the community: a systematic review. *Lancet Infect Dis* 2003;**3:**275–81.

[32] TCEB. Evaluation of the sanitation programme of the ''Todos os Santos''-Bay. Tribunal de Contas do Estado da Bahia, Salvador, Brazil, 2005.

[33] Mackenbach J, Bakker M, Sihto M, Diderichsen F. Strategies to reduce socioeconomic inequalities in health. In: Mackenbach J, Bakker M (eds). *Reducing Inequalities in Health: A European Perspective*. London: Routledge, 2002. pp. 25–49.